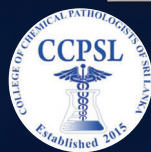


Under the Auspices of



COLLEGE OF CHEMICAL PATHOLOGISTS OF SRI LANKA

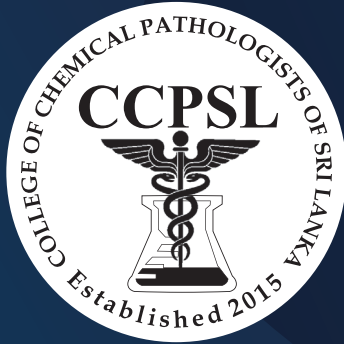
11th ANNUAL ACADEMIC SESSIONS 2026

*Quality, Innovation and Interdisciplinary Collaboration
in the Era of personalised Medicine*



10th and 11th July 2026

📍 Shangri La Hotel, Colombo



COLLEGE OF CHEMICAL PATHOLOGISTS OF SRI LANKA

11th Annual Academic Sessions 2026

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MESSAGES

MESSAGE FROM THE PRESIDENT



Dr Ganga Withana Pathirana
MBBS, Dip Path, MD(Chemical Pathology)
Consultant Chemical Pathologist
Teaching Hospital Ratnapura
Sri Lanka

It gives me great pleasure to welcome you to the 11th Annual Academic Sessions of the College of Chemical Pathologists of Sri Lanka, to be held on 10th and 11th July 2026 at Shangri-La Hotel, Colombo.

The theme for this year, “Quality, Innovation and Interdisciplinary Collaboration in the Era of Personalized Medicine” was chosen to reflect the direction in which our specialty is moving. Chemical Pathology has always been closely linked to patient care, but its role is becoming even more important as clinical practice becomes more precise, data-driven and individualized. The quality of our laboratory work, the way we adopt new technology, and the strength of our collaboration with clinical teams will all determine how effectively we serve our patients.

The Annual Academic Sessions this year, held under the auspices of the International Federation of Clinical Chemistry and Laboratory Medicine (IFCC) and the Asia-Pacific Federation for Clinical Biochemistry and Laboratory Medicine (APFCB), brings together a wide range of areas relevant to daily practice and the future development of laboratory medicine. The sessions will cover important clinical and laboratory topics, including cardiovascular and metabolic disease, neurological biochemistry, women’s health, chronic kidney disease, oncology, diabetes, toxicology, paediatric disorders, point-of-care testing and laboratory management. There will also be a strong focus on quality systems, accreditation, traceability, assay performance, ethics, automation and quality indicators.

The Annual Academic Sessions will include a dedicated programme for medical laboratory scientists, a valued component of our sessions over the years. The participation of local and international resource persons, clinicians, laboratory professionals, trainees, researchers and industry partners will add greatly to the value of these sessions.

A two-day industrial exhibition will be held concurrently with the academic sessions, providing an opportunity to view current technologies and interact with industry representatives who support the development of laboratory medicine.

On behalf of the Council of the College of Chemical Pathologists of Sri Lanka, I warmly invite you to join us for the 11th Annual Academic Sessions. I hope this meeting will be a useful and enjoyable opportunity to learn, share experiences, renew professional friendships and strengthen our collective commitment to improving laboratory medicine in Sri Lanka.

I look forward to welcoming you to Shangri-La Hotel, Colombo, in July 2026.

Dr Ganga Withana Pathirana
President
College of Chemical Pathologists of Sri Lanka

MESSAGE FROM THE CHIEF GUEST



Hon Dr Nalinda Jayatissa
Minister of Health and Mass Media
Sri Lanka

It gives me great pleasure to send this message on the occasion of the 11th Annual Academic Sessions of the College of Chemical Pathologists of Sri Lanka, held under the theme “Quality, Innovation, and Interdisciplinary Collaboration in the Era of Personalised Medicine.”

Laboratory medicine is an essential component of a strong health system. Accurate, timely, and clinically meaningful laboratory results support correct diagnosis, guide treatment decisions, monitor disease progression, and improve patient outcomes. In this context, chemical pathology plays a vital role in strengthening healthcare delivery across Sri Lanka.

The theme selected for this year’s sessions is highly relevant. Quality ensures confidence in every laboratory report. Innovation enables health services to advance through new technologies, improved workflows, digital systems, and advanced diagnostic methods. Interdisciplinary collaboration brings professionals together to provide more comprehensive, patient-centred care. These principles are essential as we work towards a health system that is modern, equitable, efficient, and responsive to the needs of our people.

The Ministry of Health recognises the importance of strengthening laboratory services through sustainable procurement, proper maintenance of equipment, continuous training of human resources, laboratory information systems, quality assurance, and support for national reference services. Investment in laboratory medicine is an investment in patient safety, clinical excellence, and public health preparedness.

I am pleased to note that the College continues to make significant contributions to professional development, academic leadership, guideline development, quality improvement, and capacity building in laboratory medicine. The inclusion of a dedicated Medical Laboratory Science Programme is also commendable, as medical laboratory scientists and technologists are indispensable members of the diagnostic team.

I extend my sincere congratulations to the President, Council, Joint Secretaries, Organising Committee, Scientific Committee, resource persons, and all participants of the 11th Annual Academic Sessions. I am confident that this event will provide a valuable platform for knowledge sharing, professional collaboration, and future planning.

I wish the College of Chemical Pathologists of Sri Lanka and the 11th Annual Academic Sessions every success.

Hon Dr Nalinda Jayatissa
Minister of Health and Mass Media

MESSAGE FROM THE GUEST OF HONOUR



Professor Jayantha Balawardane
MBBS, MD, FRCP [Edin], FRCP [Lond] FSLCO, FSLCPM
Professor in Oncology
Sir John Kotelawala Defence University
Chancellor, Uva Wellassa University

It is both a privilege and a pleasure to extend my greetings on the occasion of the 11th Annual Academic Sessions of the College of Chemical Pathologists of Sri Lanka.

I warmly congratulate the President, Council Members, and the Organizing Committee for their dedication and hard work in organizing this important academic gathering. The College has consistently demonstrated its commitment to advancing the field of chemical pathology and strengthening laboratory medicine in Sri Lanka through education, research, and professional excellence.

This year's theme, "Quality, Innovation, and Interdisciplinary Collaboration in the Era of Personalized Medicine," is both timely and highly relevant. As healthcare continues to evolve, personalized medicine is transforming the way diseases are diagnosed, monitored, and treated. In this rapidly changing landscape, maintaining the highest standards of quality, embracing innovation, and fostering collaboration across disciplines are essential to delivering optimal patient care.

Chemical pathologists play a vital role in this transformation. Through the integration of advanced diagnostic technologies, data-driven decision-making, and close collaboration with clinicians and other healthcare professionals, they contribute significantly to the realization of precision healthcare. Academic forums such as these provide an invaluable platform for sharing knowledge, exploring emerging developments, and strengthening partnerships that will shape the future of medicine.

I am confident that the scientific deliberations and professional interactions during these sessions will be both enriching and inspiring. May this event foster new ideas, meaningful collaborations, and innovative approaches that will further strengthen the practice of chemical pathology in Sri Lanka and beyond.

I wish the 11th Annual Academic Sessions of the College of Chemical Pathologists of Sri Lanka every success and extend my best wishes to all participants for a productive and rewarding conference.

With warm regards,

Professor Jayantha Balawardane
Professor in Oncology
Sir John Kotelawala Defence University
Chancellor, Uva Wellassa University

MESSAGE FROM THE DIRECTOR GENERAL OF HEALTH SERVICES



Dr Asela Gunawardena
Director General of Health Services
Ministry of Health and Mass Media
Sri Lanka

It is with great pleasure that I extend my warmest greetings and best wishes to the College of Chemical Pathologists of Sri Lanka on the occasion of its 11th Annual Academic Sessions, to be held on the 10th and 11th of July 2026 at Shangri-La Hotel, Colombo.

This year's theme, "Quality, Innovation, and Interdisciplinary Collaboration in the Era of Personalised Medicine" is timely and highly relevant to the evolving needs of modern healthcare. Chemical Pathology plays a vital role in patient care, from early diagnosis and disease monitoring to guiding treatment decisions, ensuring patient safety, and supporting evidence-based clinical practice. In an era where medicine is rapidly moving towards precision and personalised care, the contribution of laboratory medicine has become more important than ever.

The academic programme, with its wide range of symposia, plenary lectures, medical laboratory science sessions, and industrial exhibition, provides an excellent platform for consultants, trainees, medical laboratory scientists, technologists, clinicians, researchers, and industry partners to share knowledge, exchange experiences, and strengthen professional collaboration. The participation of both local and international experts will undoubtedly enrich the scientific value of this event.

I commend the College of Chemical Pathologists of Sri Lanka for its continued commitment to improving laboratory quality, promoting innovation, strengthening interdisciplinary partnerships, and supporting continuous professional development. These efforts contribute significantly to improving healthcare delivery and ensuring that patients across the country receive reliable, timely, and high-quality diagnostic services.

I hope that the proceedings of the 11th Annual Academic Sessions will inspire all participants to apply new knowledge, embrace innovation, and uphold the highest standards of laboratory practice. I wish the President, Council, Organising Committee, resource persons, and all participants every success in this important academic event.

Dr Asela Gunawardena
Director General of Health Services
Ministry of Health

MESSAGE FROM THE HEALTH SECRETARY



Dr Anil Jasinghe
Secretary
Ministry of Health and Mass Media
Sri Lanka

It is with great pleasure that I extend my warm greetings and best wishes to the College of Chemical Pathologists of Sri Lanka on the occasion of its 11th Annual Academic Sessions and Scientific Exhibition 2026, held under the timely and meaningful theme, “Quality, Innovation, and Interdisciplinary Collaboration in the Era of Personalised Medicine.”

I am pleased to recognize the valuable contribution made by Chemical Pathologists, medical laboratory technologists, postgraduate trainees, medical officers, scientists, and all laboratory professionals towards strengthening diagnostic services and improving patient care in Sri Lanka.

Chemical Pathology plays a vital role in modern healthcare by supporting accurate diagnosis, disease monitoring, treatment decisions, preventive medicine, and public health services. In the current era of personalised medicine, the integration of quality laboratory services, innovative technologies, and interdisciplinary collaboration is essential to deliver more precise, effective, and patient-centred care. The chosen theme reflects the College’s vision to advance laboratory medicine in response to the evolving needs of the healthcare system. It also highlights the importance of close collaboration between clinicians, laboratory specialists, researchers, technologists, and policymakers in improving health outcomes for the people of Sri Lanka.

The 11th Annual Academic Sessions and Scientific Exhibition provide an excellent platform for knowledge sharing, academic discourse, research, innovation, and professional development. I commend the College of Chemical Pathologists of Sri Lanka for its continued commitment to maintaining high standards in Chemical Pathology, supporting postgraduate training, promoting quality improvement, and contributing to the development of laboratory services in the country.

I sincerely congratulate the President, Council Members, Organising Committee, resource persons, and all participants for their dedication and hard work in organizing this important event.

I wish the 11th Annual Academic Sessions and Scientific Exhibition 2026 every success.

Dr Anil Jasinghe
Secretary
Ministry of Health and Mass Media

MESSAGE FROM THE DEPUTY DIRECTOR GENERAL LABORATORY SERVICES



Dr H M A Thilakaratne
Deputy Director General (Laboratory Services)
Ministry of Health and Mass Media
Sri Lanka

It is with great pleasure that I convey this message to the abstract book of the 11th Annual Academic Sessions and Scientific Exhibition 2026 of the College of Chemical Pathologists of Sri Lanka, held under the timely theme, “Quality, Innovation and Interdisciplinary Collaboration in the Era of Personalized Medicine.”

Laboratory services are an essential pillar of the healthcare system. They contribute significantly to accurate diagnosis, disease monitoring, treatment decisions, screening programmes, and public health activities. In this context, Chemical Pathology plays a vital role by providing reliable biochemical investigations, expert interpretation, and valuable support for clinical decision-making.

The theme of this year’s academic sessions is especially relevant at a time when laboratory medicine is rapidly advancing. Ensuring quality at every stage of the testing process, introducing appropriate innovations, and strengthening collaboration among laboratory professionals, clinicians, administrators, and policymakers are essential to delivering safe, efficient, and patient-centred services.

I greatly appreciate the contribution made by the College of Chemical Pathologists of Sri Lanka in promoting academic development, research, quality improvement, and professional standards in laboratory medicine. The inclusion of scientific abstracts in this publication reflects the commitment of professionals to continuous learning, service improvement, and evidence-based practice.

I extend my sincere congratulations to the President, Council, Organizing Committee, and all members of the College for organizing this important academic event. I wish the 11th Annual Academic Sessions and Scientific Exhibition 2026 every success.

Dr H M A Thilakaratne
Deputy Director General (Laboratory Services)
Ministry of Health and Mass Media

MESSAGE FROM THE DIRECTOR, LABORATORY SERVICES



Dr Suranga Dolamulla
MBBS (Sri Lanka), MSc, MD (Medical Administration),
PhD (University of York)
Consultant Medical Administrator
Director, Laboratory Services
Ministry of Health and Mass Media
Sri Lanka

It is with great pleasure that I extend my warm greetings and best wishes to the College of Chemical Pathologists of Sri Lanka on the occasion of its 11th Annual Academic Sessions 2026.

The theme of this year's sessions, "Quality, Innovation, and Interdisciplinary Collaboration in the Era of Personalised Medicine," is both timely and highly relevant to the evolving landscape of healthcare. As laboratory medicine continues to play a pivotal role in clinical decision-making, chemical pathology remains at the forefront of advancing precision diagnostics, evidence-based treatment, and patient-centred care.

The College of Chemical Pathologists of Sri Lanka has consistently demonstrated leadership in promoting excellence in laboratory services, professional development, research, and quality assurance. Through its academic and professional initiatives, the College has made a significant contribution to strengthening laboratory medicine and improving healthcare outcomes in Sri Lanka.

As the Director of Laboratory Services, I am particularly encouraged by the increasing emphasis on innovation, digital transformation, quality management systems, and multidisciplinary collaboration within laboratory services. These developments are essential for enhancing the efficiency, reliability, and accessibility of diagnostic services across the country. The ongoing modernisation of laboratory information systems and the integration of laboratory data into national health information platforms further highlight the critical role of laboratory professionals in the healthcare system.

I commend the organisers for bringing together specialists, researchers, clinicians, scientists, and healthcare administrators to share knowledge, exchange experiences, and explore emerging developments in the field. Such academic forums provide invaluable opportunities to foster collaboration and strengthen professional networks that ultimately benefit patient care.

I wish the President, Council Members, and Organising Committee of the College of Chemical Pathologists of Sri Lanka every success in conducting this important scientific event. I am confident that the sessions will be intellectually stimulating and will contribute significantly to the advancement of laboratory medicine in Sri Lanka.

Dr Suranga Dolamulla
Director, Laboratory Services
Ministry of Health and Mass Media

MESSAGE FROM THE IFCC PRESIDENT



Professor Dr Tomris Ozben

EuSpLM, PhD, DSc

President, International Federation of Clinical Chemistry and Laboratory Medicine

Specialist in Medical Biochemistry

Past-President, European Federation of Clinical Chemistry and Laboratory Medicine

Past President, Balkan Clinical Laboratory Federation

Professor of Medical Biochemistry, Akdeniz University Faculty of Medicine, Antalya, Türkiye

University of Modena and Reggio Emilia, Clinical and Experimental Medicine PhD School, Modena, Italy

On behalf of the International Federation of Clinical Chemistry and Laboratory Medicine, it is my great pleasure to extend warm greetings and sincere congratulations to the College of Chemical Pathologists of Sri Lanka on the occasion of the 11th Annual Academic Sessions and Scientific Exhibition CCPSL 2026, to be held on 10th and 11th July 2026 at Shangri-La, Colombo.

This important annual scientific gathering provides an excellent platform for education, scientific exchange, professional networking, and collaboration among chemical pathologists, laboratory medicine specialists, clinicians, researchers, laboratory scientists, and partners from the diagnostic industry. By bringing together professionals from different disciplines, the College continues to strengthen the role of laboratory medicine in patient care, public health, and modern healthcare delivery.

The theme of this year's meeting, "Quality, Innovation, and Interdisciplinary Collaboration in the Era of Personalized Medicine" is both timely and highly relevant. Advances in molecular diagnostics, biomarkers, automation, artificial intelligence, digital health, and data-driven decision-making are rapidly transforming the field of laboratory medicine. At the same time, quality remains the foundation of reliable laboratory practice, ensuring that test results are accurate, clinically meaningful, and beneficial to patient outcomes.

The IFCC strongly supports initiatives that promote scientific excellence, quality improvement, innovation, education, and international collaboration. Meetings such as CCPSL 2026 play an important role in sharing knowledge, encouraging research, inspiring young professionals, and strengthening laboratory medicine services at national, regional, and international levels.

It is also my pleasure to invite colleagues to participate in the XXVII IFCC WorldLab Congress, to be held in New Delhi from 25th to 29th October 2026. WorldLab 2026 will bring together laboratory professionals, clinicians, researchers, healthcare decision-makers, and industry partners from across the world, providing an outstanding platform for scientific exchange, collaboration, and global engagement. The Congress will also host the IFCC Young Scientist Forum, with opportunities for early-career professionals to participate, network, and engage with leading experts in the field.

I warmly congratulate the Council of the College of Chemical Pathologists of Sri Lanka, the organizing committee, speakers, authors, participants, and all those contributing to this important event. On behalf of the IFCC, I extend my best wishes for a successful, productive, and memorable meeting, and look forward to the continued contributions of the College to the advancement of laboratory medicine and healthcare excellence.

Professor Dr Tomris Ozben
IFCC President

MESSAGE FROM THE ABFCB PRESIDENT



Professor Tony Badrick
President
Asia Pacific Federation for Clinical Biochemistry
and Laboratory Medicine (APFCB)

On behalf of the APFCB community, I would like to lend my support to the CCPSL as they provide a world-class conference to the region. The Sri Lankan College have consistently created highly regarded scientific meetings in a beautiful country. These meetings have focused on contemporary issues in laboratory medicine presented by experts.

The theme for this meeting “is Quality, Innovation, and Interdisciplinary Collaboration in the Era of Personalised Medicine” which should interest every clinical professional in the region. Precision medicine should provide more targeted treatments with fewer complications, but that will require laboratories to provide the evidence needed to identify a specific illness and monitor response and recurrence. These requirements require accurate, precise measurements of biomarkers (not all new), an understanding of disease and treatment response, and the ability to deliver all of this cost-effectively.

I wish to congratulate the CCPSL on its ongoing educational activities in Sri Lanka, despite the significant geopolitical uncertainty worldwide.

Professor Tony Badrick
APFCB President

MESSAGE FROM THE JOINT SECRETARIES



► **Dr Thathsarani Vithana Pathirana**
MBBS, Dip Chem Path, MD (Chemical Pathology)
Consultant Chemical Pathologist
Teaching Hospital
Badulla

Dr S I Majitha
MBBS, Dip Path, MD (Chemical Pathology)
Consultant Chemical Pathologist
Lady Ridgeway Hospital for Children
Colombo



It is with great pleasure that we extend our warm greetings to all distinguished guests, speakers, chairpersons, delegates, postgraduate trainees, medical laboratory technologists, scientists, sponsors, and well-wishers on the occasion of the 11th Annual Academic Sessions and Industrial Exhibition of the College of Chemical Pathologists of Sri Lanka.

This important academic event provides a valuable platform for scientific exchange, professional development, and collaboration in Chemical Pathology and Laboratory Medicine. As diagnostic services continue to advance, the role of Chemical Pathologists remains essential in ensuring quality, accuracy, and clinical relevance in patient care.

We sincerely appreciate the guidance of the President and Council, the dedication of the Organizing and Scientific Committees, and the valuable contributions of all resource persons, participants, and industry partners who have supported this event.

We hope that this abstract book will serve as a meaningful record of the scientific work presented and inspire continued learning, research, and excellence in Chemical Pathology.

We warmly welcome you and wish you a successful and memorable academic experience.

Dr Thathsarani Vithana Pathirana
Dr S I Majitha
Joint Secretaries
College of Chemical Pathologists of Sri Lanka



COUNCIL

COLLEGE OF CHEMICAL PATHOLOGISTS OF SRI LANKA, THE COUNCIL 2026



Sitting :
(Left to right)

Dr Thathsarani Vithana Pathirana, Dr Imala Jayasinghe, Dr Rajitha Samarasinghe, Dr Thamara Herath, Dr Ganga Withana Pathirana,
Dr Thushara Hewageegana, Dr Shyamalee Gunarathna, Dr Mihika Fernando, Dr S I Majitha.

Standing-1st row :
(Left to right)

Dr B K T P Dayanath, Dr Neranjana Vithanage, Dr Deepani Siriwardhana, Dr Ushani Jayawardane, Dr Maduri Vidanapathirana,
Dr Nadeen Senanayake, Dr Manjula Dissanayake, Dr Gaya Katulanda, Dr Chandrika Meegama, Dr Dilinika Perera,
Dr Kisali Hirimuthugoda, Dr Dulani Jayawardana.

Standing-2nd row :
(Left to right)

Dr Thushari Vithanage, Dr Samadhi Madanayaka, Dr Rasangika Gunasekara, Dr Gawri Abeynayake, Dr Nangai Kularatnam, Dr Vithegi
Kesavan, Dr Udara Senarathne, Dr H W Dilanthi, Dr Fasilaas Kiyamudeen, Dr Eshani Senanayake.

COLLEGE OF CHEMICAL PATHOLOGISTS OF SRI LANKA

COUNCIL - 2026

President	Dr Ganga Withana Pathirana
President-Elect	Dr Thamara Herath
Immediate Past - President	Dr Thushara Hewageegana
Joint Secretaries	Dr S I Majitha Dr Thathsarani Vithana Pathirana
Treasurer	Dr Shyamalee Gunarathna
Co-Editors	Dr Imala Jayasinghe Dr Mihika Fernando
Council Members	Dr Chandrika Meegama Dr Deepani Siriwardhana Dr Gaya Katulanda Dr B K T P Dayanath Dr Kisali Hirimuthugoda Dr Manjula Dissanayake Dr Dulani Jayawardana Dr Vithegi Kesavan Dr Dilinika Perera Dr Neranjana Vithanage Dr Ushani Jayawardane Dr Nangai Kularatnam Dr H W Dilanthi Dr Gawri Abeynayake Dr Thushari Vithanage Dr Nadeen Senanayake Dr Maduri Vidanapathirana Dr Eshani Senanayake Dr Fasilaas Kiyamudeen Dr Samadhie Madanayaka Dr Udara Senarathne Dr Rasangika Gunasekara
Honorary Advisor	Dr Rajitha Samarasinghe



PROGRAMMES



ACADEMIC PROGRAMME

11th Annual Academic Sessions 2026 College of Chemical Pathologists of Sri Lanka

DAY 1 | 10th July 2026

TIME	SESSION / TOPIC	RESOURCE PERSON
Symposium 1 From Risk to Block - Lipids and Troponin in Cardiac Disease.		
8.30 am - 9.00 am	Troponins: Light Guiding Through Cardiac Night	Dr Gawri Abeynayake 
9.00 am - 9.30 am	What is New in Lipid Management? Treat Early, Reach Targets, and Protect the Heart	Dr M R Mubarak 
9.30 am - 10.00 am	Beyond LDL and HDL — Where Advanced Lipids Tell the Tale	Dr Gayani Weerasinghe 
10.00 am - 10.30 am	Plenary 1 Decoding the CSF ; Biochemical Clues in Neurological Diseases	Dr Kishara Gooneratne 
10.30 am - 11.00 am Morning Tea		
11.00 am - 11.30 am	Plenary 2 From Cradle to Crown - Biochemical Evidence when Woman's Life Turns Around	Dr Thushara Hewageegana 
Symposium 2 From Stone to Skeleton — Unravelling CKD and Its Metabolic Sequelae		
11.30 am - 12.00 pm	Kidneys in Strain - A Silent Island Pain	Dr Udana Ratnapala 
12.00 pm - 12.30 pm	Renal Stones: Prevention,Biochemical Investigations and Management	Dr Farhan Ahmed 
12.30 pm - 1.00 pm	Investigations in Mineral and Bone Disorder of Chronic Kidney Disease (CKD-MBD)	Dr Samuel Vasikaran 
1.00 pm - 2.00 pm Lunch		
Symposium 3 From Marker to Malignancy — Laboratory Insights in Oncology		
2.00 pm - 2.30 pm	When Proteins Divide and Diagnoses Arise: The Power of Serum Protein Electrophoresis	Dr Rajitha Samarasinghe 
2.30 pm - 3.00 pm	Markers that Speak — Detecting Cancer We Seek	A/Prof Robert Hawkins 
3.00 - 3.30 pm Evening Tea		

ACADEMIC PROGRAMME

11th Annual Academic Sessions 2026 College of Chemical Pathologists of Sri Lanka

DAY 2 | 11th July 2026

TIME	SESSION / TOPIC	RESOURCE PERSON
Symposium 4 From Samples to Systems — The Art of Laboratory Management		
9.00 am - 9.30 am	Acceptance Testing in Chemical Pathology: Verifying Performance Before Patient Care	Dr Gayani Weerasinghe 
9.30 am - 10.00 am	Predict the Risk, Perfect the Test	A/Prof Robert Hawkins 
10.00 am - 10.30 am	From Error to Excellence: Six Sigma and Measurement of Uncertainty in Laboratory Medicine	Dr Sakunthala Jayasinghe 
10.30 am - 11.00 am	Morning Tea	
11.00 am - 11.30 am	Plenary 3 Rapid and Bright; Guiding Care Right - POCT	Dr Saswati Das 
Symposium 5 From Glucose to Control — Biochemical Insights into Diabetes Mellitus		
11.30 am - 12.00 pm	Aetiological diagnosis of Diabetes;Methods and Usefulness	Prof Prasad Katulanda 
12.00 pm - 12.30 pm	Sugar in Motion - Continuous Monitoring for Better Notion.	Dr Leslie Lai 
12.30 pm - 1.00 pm	Plenary 4 From Vein to Vial - Drugs of Abuse and the Role of Chemical Pathology in Post-Mortem Trials	Snr Prof Muditha Vidanapathirana 
1.00 pm - 2.00 pm	Lunch	
Symposium 6 From Code to Cortex — Understanding Hormonal and Genetic Disorders in Children		
2.00 pm - 2.30 pm	Congenital Adrenal Hyperplasia: From Enzyme Block to Hormonal Balance	Dr Navoda Atapattu 
2.30 pm - 3.00 pm	From Pathway to Therapy - Solving the Riddle of Porphyria	Dr Thathsarani Vithana Pathirana 
3.00 pm - 3.30 pm	Tiny Genes, Major Scenes: The Metabolic Story in Between	Dr Vindya Subasinghe 
3.30 pm - 4.00 pm	Oral Presentations, Prize Giving and Closing Ceremony	
4.00 pm - 4.30 pm	Evening Tea	

MEDICAL LABORATORY SCIENCE PROGRAMME

11th Annual Academic Sessions 2026 College of Chemical Pathologists of Sri Lanka

DAY 1 | 10th July 2026

TIME	SESSION / TOPIC	RESOURCE PERSON
8.30 am - 9.00 am	Welcome Speech	President, CCPSL, Dr Ganga Withana Pathirana 
9.00 am - 9.30 am	The Unbroken Chain of Trust: Metrological Traceability in Laboratory Medicine	Dr Kisali Hirimuthugoda 
9.30 am - 10.00 am	From Hidden Sugar to Guided Care: The Laboratory Journey in Diabetes Diagnosis and Care	Dr Leslie Lai 
10.00 am - 10.30 am	Morning Tea	
10.30 am - 11.00 am	Indicators of Excellence: Measuring Quality in Laboratory Performance	Dr Neranjana Vithanage 
11.00 am - 11.30 am	Pathways to Parenthood: Investigating the Hidden Causes of Subfertility	Dr Dilinika Perera 
11.30 am - 12.00 pm	When Pathways Lose Their Way: Unveiling Inborn Errors of Metabolism	Dr Eresha Jasinge 
12.00 pm - 12.30 pm	When Electrolytes Fall Out of Tune: Understanding Common Abnormalities	Dr Nangai Kularatnam 
12.30 pm - 1.30 pm	Lunch	
1.30 pm - 2.00 pm	Lipid Measurements in the Management of Cardiovascular Diseases	Dr Chandrika Meegama 
2.00 pm - 2.30 pm	From Control to Confidence: Quality in Chemical Pathology	Dr Sakunthala Jayasinghe 
2.30 pm - 3.00 pm	Where Science Meets Conscience: Ethics in the Laboratory	Dr Saswati Das 
3.00 pm - 3.30 pm	Evening Tea	

MEDICAL LABORATORY SCIENCE PROGRAMME

11th Annual Academic Sessions 2026 College of Chemical Pathologists of Sri Lanka

DAY 2 | 11th July 2026

TIME	SESSION / TOPIC	RESOURCE PERSON
8.30 am - 9.00 am	Hormonal Harmony: Understanding Thyroid Function	Dr Deepani Siriwardhana 
9.00 am - 9.30 am	The Art of Accurate Speed: Laboratory Automation	Dr Gaya Katulanda 
9.30 am - 10.00 am	Listening to the Language of Renal Metabolic Function	Dr Farhan Ahmed 
10.00 am - 10.30 am	Morning Tea	
10.30 am - 11.00 am	Bridging Complaints and Clinical Clues: How to Investigate Complaints from Clinicians	Dr Dulani Jayawardana 
11.00 am - 11.30 am	Silent Streams, Diagnostic Dreams: Analysis of Body Fluids	Dr Manjula Dissanayake 
11.30 am - 12.00 pm	Echoes of Error: Assay Interference in the Clinical Laboratory	Dr Thamara Herath 
12.00 pm - 12.30 pm	Between Dose and Destiny: Therapeutic Drug Monitoring	Dr Thushara Hewageegana 
12.30 pm - 1.30 pm	Lunch	
1.30 pm - 2.00 pm	Tracing Cancer Through Hidden Clues: Tumour markers	Dr Fasilaas Kiyamudeen 
2.00 pm - 3.00 pm	Quiz	Dr Thushari Vithanage Dr Nadeen Senanayake Dr Maduri Vidanapathirana Dr Janeesha Liyanagunawardana Dr Dinithi Denipitiya 
3.00 pm - 3.30 pm	From Standards to Strength: The Way Forward to Accreditation	Dr H W Dilanthi 
3.30 pm - 4.00 pm	The Silent Giver, the Speaking Liver: Interpreting the Liver Function Test	Dr Ushani Jayawardane 
4.00 pm - 4.30 pm	Evening Tea	

INAUGURATION CEREMONY PROGRAMME

6.30 pm	–	Invitees take their seats
6.45 pm	–	National Anthem
6.50 pm	–	Lighting of the traditional oil lamp
6.55 pm	–	Welcome Address by Dr Thathsarani Vithana Pathirana, Joint Secretary, CCPSL
7.00 pm	–	Induction of the new President by the Immediate Past President, Dr Thushara Hewageegana
7.05 pm	–	Address by the President, CCPSL, Dr Ganga Withana Pathirana
7.10 pm	–	Address by the Chief Guest, Hon Dr Nalinda Jayatissa, Minister of Health and Mass Media, Sri Lanka
7.25 pm	–	Awarding of the medal to the Past President
7.35 pm	–	Address by the Guest of Honour, Prof Jayantha Balawardane, Chancellor, Uva Wellassa University of Sri Lanka; Professor in Oncology, University Hospital, Kotelawala Defence University
7.55 pm	–	Presentation of CCPSL Felicitation Awards
8.00 pm	–	Conferment of CCPSL Fellowships
8.10 pm	–	Awards
8.15 pm	–	CCPSL Oration 2026 by Dr Samuel Vasikaran, Consultant Chemical Pathologist, PathWest Laboratory Medicine, Western Australia
8.45 pm	–	Vote of Thanks by Dr S I Majitha, Joint Secretary, CCPSL
8.50 pm	–	Cultural Show
9.20 pm	–	Ceremonial procession leaves the hall
9.25 pm	–	Reception



HONOURS



FELLOWSHIP AWARDS



CITATION FOR THE CONFERMENT OF FELLOWSHIP



Associate Professor Peter Stewart
MBBS, BSc(Med), FRACP, FRCPA, MBA
 Chemical Pathologist
 4Cyte Pathology, Australia
 Associate Professor of Biochemistry and Medicine
 University of Sydney
 Australia

Associate Professor Peter Stewart is a distinguished Chemical Pathologist and Metabolic Physician from Australia, whose career has been marked by outstanding contributions to clinical biochemistry, metabolic medicine, laboratory quality, accreditation, specialist training, and professional leadership. He is currently a Chemical Pathologist at 4Cyte Pathology, Sydney, Australia. He previously served as Clinical Director at Royal Prince Alfred and Liverpool Hospitals in Sydney. He also holds an academic appointment as Associate Professor of Biochemistry and Medicine at the University of Sydney and has obtained a Master of Business Administration from Macquarie University, Sydney.

Associate Professor Stewart is a Past President of the Royal College of Pathologists of Australasia (RCPA). He served as Chairman of the RCPA Quality Assurance Programs for eleven years and was a ministerial appointee to the National Pathology Accreditation Advisory Council for fifteen years. He also chaired the Pathology Clinical Committee of the Australian Medicare Benefits Schedule Review. His contribution to laboratory accreditation and quality standards has been substantial. He has served as a lead Technical Assessor in the Australian accreditation system for more than twenty years and is currently Chairman of the Pathology Testing Accreditation Advisory Committee of the National Association of Testing Authorities (NATA), Australia.

His major clinical and research interests include the laboratory and molecular diagnosis of porphyria, new treatments for porphyria, and the adoption of new technologies into laboratory practice.

Of particular significance to the College of Chemical Pathologists of Sri Lanka is Associate Professor Stewart's contribution to the training and mentorship of Sri Lankan overseas trainees. Through his guidance, trainees have gained valuable exposure to advanced laboratory medicine, including metabolic medicine, molecular diagnostics, quality systems, and modern diagnostic technologies. This contribution has supported capacity building and the development of Chemical Pathology services in Sri Lanka.

Associate Professor Stewart has also been a generous and supportive mentor, providing professional guidance and encouragement to Sri Lankan trainees and contributing meaningfully to their development as specialists.

In recognition of his distinguished service, professional leadership, and significant contributions to Chemical Pathology, metabolic medicine, laboratory quality, accreditation, specialist training, and the advancement of laboratory medicine in Sri Lanka and the wider region, the College of Chemical Pathologists of Sri Lanka is honoured to confer upon Associate Professor Peter Stewart the Fellowship of the College.

CITATION FOR THE CONFERMENT OF FELLOWSHIP



Late Dr Srilal Dharmadasa Atukorala
MBBS, MD (Microbiology), Dip. Bact. (Manchester), FRCPath
Consultant Microbiologist
Former Head, Department of Pathology
National Hospital of Sri Lanka

Dr Srilal Dharmadasa Atukorala was a distinguished clinical microbiologist, educator, examiner and professional leader whose contribution to pathology, microbiology and laboratory medicine in Sri Lanka has been both pioneering and enduring.

Born in 1945, he was educated at Royal College, Colombo, and later graduated MBBS in 1972. Following internship at the General Hospital, Colombo, and Castle Street Hospital, he served in the anti-venereal disease campaign before proceeding to the United Kingdom on a World Health Organization fellowship, obtaining the Diploma in Microbiology and Membership of the Royal College of Pathologists. On returning to Sri Lanka, Dr Atukorala was appointed Clinical Bacteriologist at the National Hospital of Sri Lanka, where he served with distinction until his retirement in 2005. From 1992, he also served as Head of the Department of Pathology. Under his leadership, the Department of Microbiology developed as a major centre for diagnostic service, postgraduate training and laboratory standards.

Dr Atukorala's expertise was recognised internationally through his longstanding association with the World Health Organization. He served as a WHO Visiting Scientist at Columbia Presbyterian Medical Centre, New York, and at hospitals affiliated with Harvard Medical School in Boston. He was also invited on several occasions as a WHO Consultant and Temporary Adviser, supporting infection-control and rational antibiotic-use initiatives in Bhutan, Bangladesh, Maldives and Nepal. He contributed as a co-author to WHO guidelines on infection control for South-East Asia and authored Ministry of Health publications on infection control and rational antibiotic use.

A respected academic and postgraduate trainer, he published more than twenty papers, delivered many orations and guest lectures, and served the Postgraduate Institute of Medicine, University of Colombo, as Coordinator and member of the Board of Study in Pathology and as a member of the Board of Study in Microbiology. He also served as an examiner for the Diploma in Pathology and the MD in Microbiology.

As a founder member and council member of the College of Pathologists of Sri Lanka, Dr Atukorala contributed significantly to the College's academic mission and professional development. This Fellowship is conferred in recognition of his outstanding service to microbiology, pathology, postgraduate education, infection control and the advancement of laboratory medicine in Sri Lanka and the region.

CCPSL ORATION 2026

BON(E) VOYAGE: A JOURNEY IN BONE RESEARCH AND CLINICAL PRACTICE

**Dr Samuel Vasikaran****MBBS, MSc, MAACB, MD, FRCPA, FFSc(RCPA)**

Consultant Chemical Pathologist

PathWest Laboratory Medicine

Western Australia

The fully grown adult skeleton is a dynamic organ that is continuously remodelled in response to mechanical stress and metabolic factors. The key cells that drive remodelling—osteoblasts, osteoclasts, and osteocytes—are regulated by a network of signalling molecules and hormones. Genetic factors are central to the development of bone tissue and structure in utero and during childhood growth. Bone tissue is composed of a type I collagen matrix, which confers strength, and calcium hydroxyapatite mineral, which provides rigidity to the skeleton.

Monogenic diseases with skeletal impacts include osteogenesis imperfecta (OI), osteopetrosis or dense bone disease, and various mineralisation defects resulting in rickets or osteomalacia. These include hypophosphatasia, due to alkaline phosphatase deficiency; disorders of vitamin D metabolism or its receptor; and X-linked hypophosphataemia (XLH), caused by overproduction of fibroblast growth factor 23 (FGF23). The laboratory plays a key role in diagnosing genetic diseases and derangements in bone metabolism. In addition to X-rays for diagnosing skeletal abnormalities, dual-energy X-ray absorptiometry (DXA) is used to diagnose osteoporosis, while the trabecular bone score (TBS) assesses trabecular microarchitecture using DXA images. High-resolution peripheral quantitative CT (HR-pQCT) provides detailed assessment of changes in bone microarchitecture.

Major advances in the treatment of bone disorders include enzyme replacement therapy with asfotase alfa for hypophosphatasia, monoclonal antibody therapy against FGF23 with burosumab for X-linked hypophosphataemia, and progress in experimental gene therapy approaches to correct or silence pathogenic mutations in OI. Gene editing to repair mutations also offers the promise of a permanent cure. Treatment of osteoporosis, the commonest bone disorder in adults, has seen major progress with menopausal hormone therapy, osteoclast inhibitors that block bone resorption, anabolic agents such as PTH analogues that stimulate osteoblasts, and the dual-action sclerostin antibody romosozumab. Combined and sequential treatment strategies offer important advantages and long-term solutions for preventing fractures and their associated morbidity and mortality.



FACULTIES

INTERNATIONAL FACULTY



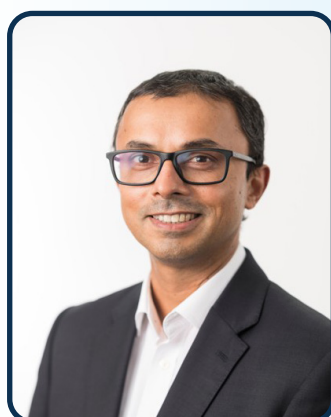
INTERNATIONAL FACULTY



Dr Gayani Weerasinghe

MBBS, Dip Chem Path, MD (Chem Path), FRCPath (UK), EuSpLM
Consultant Chemical Pathologist and Co-Clinical Director for Pathology
Buckinghamshire Healthcare NHS Trust
Honorary Consultant Chemical Pathologist
Oxford University Hospitals NHS Trust
United Kingdom

Dr Gayani Weerasinghe is a Consultant Chemical Pathologist with specialist interests in lipid disorders and metabolic bone disease. She has extensive experience in clinical and laboratory Chemical Pathology, including quality improvement, accreditation clinical governance, and laboratory leadership. She is involved in lipid optimisation and familial hypercholesterolaemia services in Buckinghamshire, supporting cardiovascular disease prevention and early detection of inherited lipid disorders. She also contributes to undergraduate medical education at the University of Oxford and serves as an examiner in Clinical Biochemistry for the Royal College of Pathologists, UK.



Dr Farhan Ahmed

**MBBS, FRCPath(UK), FRCP (London), MRCP (UK), SCE(M-
RCP Diabetes & Endocrinology (UK), MSc Clinical Chemis-
try (UK), MSc Public Health (Sydney)**

Consultant in Chemical Pathology and Metabolic Medicine
Milton Keynes University Hospital, United Kingdom
Honorary Consultant, Oxford University Hospitals NHS Trust, United Kingdom
Honorary Senior Clinical Lecturer in Clinical Biochemistry, Nuffield Division of Clin-
ical Laboratory Sciences, University of Oxford
Honorary Senior Clinical Lecturer, University of Buckingham
United Kingdom

Dr Farhan Ahmed is a Consultant in Chemical Pathology and Metabolic Medicine, Clinical Lead, and Clinical Director at Milton Keynes University Hospital. He also serves as an Honorary Consultant at Oxford University Hospitals and as a Senior Clinical Lecturer at the University of Oxford and the University of Buckingham Medical School. He is an examiner for the Royal College of Pathologists, UK, and has contributed nationally as a lipid expert in Familial Chylomicronaemia Syndrome, including support for NICE technology appraisals. He is also a Principal Investigator in clinical trials evaluating novel lipid-lowering therapies. With over 20 years of NHS experience, Dr Ahmed has published in peer-reviewed journals and has been invited as a keynote speaker at international conferences.

INTERNATIONAL FACULTY



Dr Samuel Vasikaran

MBBS, MSc, MAACB, MD, FRCPA, FFSc(RCPA)

Consultant Chemical Pathologist

PathWest Laboratory Medicine

Western Australia

Sam Vasikaran is a Consultant Chemical Pathologist at PathWest Laboratory Medicine, Western Australia, where he has served for over three decades. His major research interest is in bone metabolism, and he has published more than 150 peer-reviewed papers and book chapters. He is currently Chair of the IFCC Committee on Bone Metabolism and Vice President of the Asia Pacific Federation of Clinical Biochemistry and Laboratory Medicine. He was also formerly Clinical Professor in Pathology at the University of Western Australia.



Dr Leslie Lai

MBBS, MD

Consultant Chemical Pathologist

Laboratory Director

Premier Integrated Labs, Gleneagles Hospital Kuala Lumpur
Malaysia

Dr Leslie Lai is a Consultant Chemical Pathologist and Laboratory Director of Premier Integrated Labs, Gleneagles Hospital Kuala Lumpur Branch, Malaysia. He has held senior academic positions in the United Kingdom and Malaysia, including Professor of Clinical Biochemistry and Metabolic Medicine at Universiti Putra Malaysia and Dean of the Postgraduate Medical School at the International Medical University, Kuala Lumpur. He served as President of the Asia-Pacific Federation for Clinical Biochemistry and Laboratory Medicine from 2010 to 2016 and Chair of the IFCC Education and Management Division from 2015 to 2020. He currently chairs the IFCC Nominations Committee and has published extensively in international peer-reviewed journals.

INTERNATIONAL FACULTY



Associate Professor Robert Hawkins
MB ChB, FRCPA, MHealSc, MD
Senior Consultant Chemical Pathologist
Tan Tock Seng Hospital
Singapore

Associate Professor Robert Hawkins is a Senior Consultant Chemical Pathologist at Tan Tock Seng Hospital in Singapore. He is a medical graduate of the University of Auckland, New Zealand, and a Fellow of the Royal College of Pathologists of Australasia. He also holds a research MD and a master's degree in Information Science.

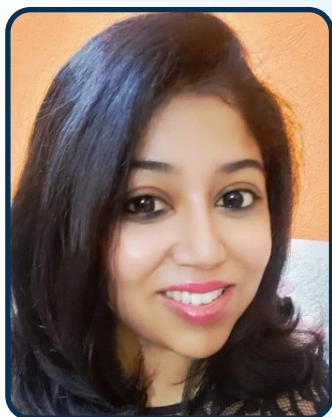
His academic contributions include more than 80 scientific articles and reviews published in peer-reviewed journals.



Dr Sakunthala Jayasinghe
MBBS, Dip Path, MD(Chemical Pathology), FRCPath(UK)
Consultant Chemical Pathologist
LabPLUS, Auckland City Hospital
Auckland
New Zealand

Dr Sakunthala Jayasinghe is a Consultant Chemical Pathologist at LabPLUS, Auckland City Hospital, Auckland, New Zealand. She has more than 15 years of experience in Chemical Pathology, having trained and worked in Sri Lanka, the United Kingdom, and New Zealand. Her main areas of interest include laboratory automation, quality control, and laboratory accreditation. She has also worked as an assessor for medical laboratory accreditation in Sri Lanka and New Zealand. In addition, she has clinical experience in lipidology and is actively involved in teaching clinical biochemistry to medical students and other healthcare professionals.

INTERNATIONAL FACULTY



Dr Saswati Das

MBBS, MD, PGDHHM, IFCAP, FAACC, FADLM, FCAP

Senior Medical Specialist in Biochemistry

New Delhi

India

Dr Saswati Das is a Senior Medical Specialist in Biochemistry, New Delhi, where she oversees chemical pathology diagnostics, molecular medicine, quality management, and laboratory services. She completed her MBBS and MD from the University of Delhi and subsequently trained in the NHS, United Kingdom, as an international fellow. She has more than 80 peer - reviewed indexed publications and serves on the editorial boards of several scientific journals. Her research interests include cardiobiochemistry, endocrinology, epigenetics, point-of-care testing, quality assurance, risk assessment, and laboratory test utilisation.

LOCAL FACULTY



LOCAL FACULTY



Dr Gawri Abeynayake

MBBS, MD (Chemical Pathology), FRCPath (UK)

Consultant Chemical Pathologist at Teaching Hospital Kalutara
An Honorary Consultant Chemical Pathologist at Sir John Kotalawela
Defence University Hospital



Dr M R Mubarak

MBBS, MD, FACC, FESC, FAPSC, FCCP, FRCP (Lond)

Consultant Cardiologist
Lanka Hospitals
Colombo



Dr Kishara Gooneratne

MBBS(Col), MD(Col), MRCP(UK)

Consultant Neurologist
Senior Lecturer in Medicine
Faculty of Medicine
University of Kelaniya



Dr Thushara Hewageegana

MBBS, Dip Path, MD (Chemical Pathology)

Consultant Chemical Pathologist
Teaching Hospital
Anuradhapura



Dr Udana Ratnapala

MBBS, MD, FRCP (UK), FRCP (London), MRCP (Nephrology), FISN, SISN, DipUKMED (LSTM), PG Certificate in Nephropathology (ISN-ANIO) PG Certificate in Transplantation (Liverpool)

Consultant Nephrologist
District General Hospital
Chilaw

LOCAL FACULTY



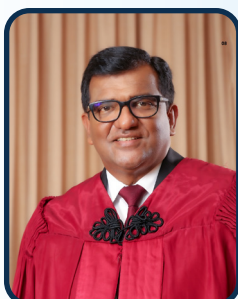
Dr Rajitha Samarasinghe

MBBS, Dip Path, MD (Chemical Pathology)

Consultant Chemical Pathologist

National Cancer Institute

Maharagama



Vidya Jyothi Professor Prasad Katulanda

MBBS, MD, FRCP (Lon), FCCP, FACE, DPhil (oxon)

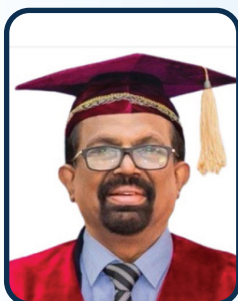
Professor and Head, Department of Clinical Medicine

University of Colombo

Consultant Endocrinologist and Diabetologist,

National Hospital of Sri Lanka

Colombo



Senior Professor Muditha Vidanapathirana

MBBS (Col), Pg.DLM (Col), MD (Col), MA in criminology (SJP)

FFFLM- Fellow of Faculty of Forensic and Legal Medicine (RCP-UK)

Dean, Faculty of Medicine, Uva Wellassa University, Badulla, Sri Lanka

Chair Professor of Forensic Medicine, Department of Forensic Medicine,

Faculty of Medical Sciences, University of Sri Jayewardenepura, Nugegoda



Dr Navoda Atapattu

MBBS, MD, DCH, MRCPCH, FRCPCH

Consultant Paediatric Endocrinologist

Lady Ridgeway Hospital for Children

Colombo



Dr Thathsarani Vithana Pathirana

MBBS, Dip Chem Path, MD (Chemical Pathology)

Consultant Chemical Pathologist

Teaching Hospital

Badulla

LOCAL FACULTY



Dr Vindya Subasinghe
MBBS, DCH, MD (Paediatrics), MRCPCH (UK)
Consultant Clinical Geneticist
Lady Ridgeway Hospital for Children
Colombo



Dr Kisali Hirimuthugoda
MBBS, Dip Path, MD (Chemical Pathology)
Consultant Chemical Pathologist
Colombo South Teaching Hospital
Kalubowila



Dr Neranjana Vithanage
MBBS, Dip Path, MD (Chemical Pathology)
Consultant Chemical Pathologist
Sri Jayewardenepura General Hospital
Nugegoda



Dr Dilinika Perera
MBBS, Dip Path, MD (Chemical Pathology), Associate Member RC-Path (UK)
Consultant Chemical Pathologist
Teaching Hospital
Kurunegala



Dr Eresha Jasinge
MBBS, Dip Path, MD (Chemical Pathology)
Consultant Chemical Pathologist
Chemical Pathology Laboratory
Ninewells Hospital (Pvt) Ltd
Colombo 05

LOCAL FACULTY



Dr G A M Kularatnam

MBBS, Dip Path, MD (Chemical Pathology)

Genomic Laboratory
National Cancer Institute
Maharagama



Dr Chandrika Meegama

MBBS, Dip Path, MD (Chemical Pathology), FAACC (USA)

Consultant Chemical Pathologist
Asiri Central Laboratory, Colombo 05
MCARD & Laboratory, Kalubowila



Dr Deepani Siriwardhana

MBBS, Dip Path, MD (Chemical Pathology)

Specialist in Chemical Pathology
Senior Lecturer
Department of Biochemistry and Clinical Chemistry Faculty of Medicine University of Moratuwa



Dr Gaya Katulanda

MBBS, Dip Path, MD (Chemical Pathology), Dip RCPATH (UK)

Consultant Chemical Pathologist
National Hospital of Sri Lanka
Colombo



Dr Dulani Jayawardana

MBBS, Dip Path, MD (Chemical Pathology)

Consultant Chemical Pathologist
National Hospital
Kandy

LOCAL FACULTY



Dr Manjula Dissanayake

MBBS, Dip Path, MD (Chemical Pathology)

Consultant Chemical Pathologist

Teaching Hospital

Karapitiya



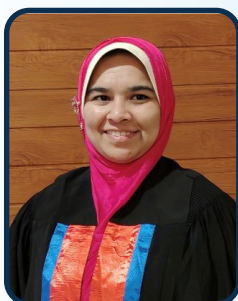
Dr Thamara Herath

MBBS, Dip Path, MD (Chemical Pathology)

Consultant Chemical Pathologist

Medical Research Institute

Colombo



Dr Fasilaas Kiyamudeen

MBBS, MD (Chemical Pathology), Dip RCPATH(UK)

Consultant Chemical Pathologist

Sirimavo Bandaranaike Specialised Children's Hospital

Peradeniya



Dr Thushari Vithanage

MBBS, Dip Chem Path, MD (Chemical Pathology)

Consultant Chemical Pathologist

District General Hospital

Matara



Dr D N Senanayake

MBBS, Dip Chem Path, MD (Chemical Pathology)

Consultant Chemical Pathologist

District General Hospital

Kegalle

LOCAL FACULTY



Dr Maduri Vidanapathirana

MBBS, Dip Chem Path, MD (Chemical Pathology), FRCPath(UK)

Consultant Chemical Pathologist

Senior Lecturer

Department of Pathology

Faculty of Medical Sciences

University of Sri Jayewardenepura



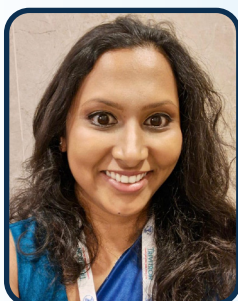
Dr Janeesha Liyanagunawardana

MBBS, MD (Chemical Pathology)

Associate Specialist Chemical Pathologist

District General Hospital

Mullaitivu



Dr Dinithi Denipitiya

MBBS, MD (Chemical Pathology)

Associate Specialist Chemical Pathologist

Lady Ridgeway Hospital for Children

Borella



Dr Dilanthi Warawita

MBBS, Dip Chem Path, MD (Chemical Pathology)

Consultant Chemical Pathologist

Senior Lecturer

Department of Biochemistry and Molecular Biology

Faculty of Medicine

University of Colombo



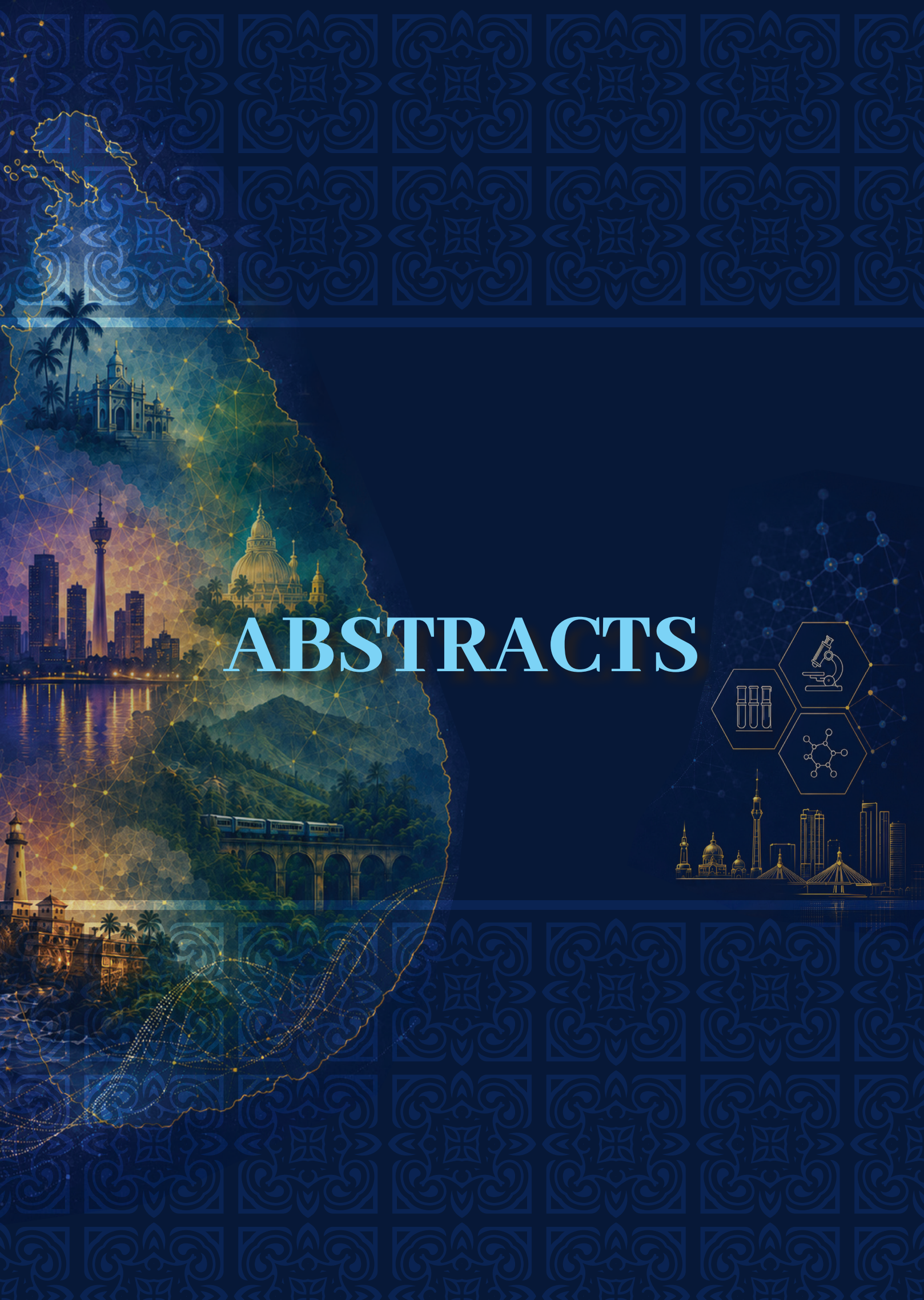
Dr Ushani Jayawardena

MBBS, Dip Chem Path, MD (Chemical Pathology)

Consultant Chemical Pathologist

Colombo North Teaching Hospital

Ragama



ABSTRACTS



SPEAKER ABSTRACTS



DAY 01**TROPONIN – LIGHT GUIDING THROUGH
CARDIAC NIGHT****Dr Gawri Abeynayake**

Cardiac troponin is the cornerstone biomarker for the diagnosis of acute myocardial infarction (AMI). The transition from conventional to high-sensitivity cardiac troponin assays (hs-cTn) has revolutionized emergency cardiovascular care by enabling earlier detection of myocardial injury and faster rule-out of acute coronary syndrome.

While this evolution has maximized sensitivity and negative predictive value, it has also challenged clinicians by identifying a broader spectrum of differential diagnoses, with a modest reduction in specificity. In line with updated American Heart Association and European Society of Cardiology guidelines, modern diagnostic algorithms use rapid serial sampling, such as 0/1-hour or 0/2-hour protocols, to significantly reduce the “troponin-blind” interval and improve risk stratification for AMI.

Furthermore, the advent of high-sensitivity point-of-care testing promises to expedite clinical decision-making by reducing delays associated with central laboratory turnaround times.

This presentation will review conventional versus high-sensitivity cardiac troponin assays, current evidence and guideline recommendations, challenges related to accelerated diagnostic algorithms, and the emerging utility and clinical impact of point-of-care troponin testing.

**WHAT IS NEW IN DYSLIPIDAEMIA MANAGEMENT TREAT EARLY,
REACH TARGET AND PROTECT THE HEART****Dr MR Mubarak**

Cardiovascular disease (CVD) remains the leading cause of death globally, causing approximately 20 million deaths annually. Most premature cardiovascular deaths can be prevented through effective control of major risk factors, including hypertension, diabetes, dyslipidaemia, smoking, obesity, unhealthy diet, and physical inactivity.

Dyslipidaemia is a fundamental driver of the atherosclerotic disease process. The primary objective of dyslipidaemia management is to reduce cardiovascular morbidity and mortality. Contemporary lipid management is undergoing a paradigm shift from merely treating elevated cholesterol levels to reducing lifelong exposure to atherogenic lipoproteins. Growing evidence indicates that cumulative lifetime exposure to atherogenic lipoproteins is a major determinant of cardiovascular risk, highlighting the importance of early intervention.

Low-density lipoprotein cholesterol (LDL-C) is recognised as the principal atherogenic lipoprotein and remains the primary target of therapy. Current guidelines recommend LDL-C targets according to an individual's cardiovascular risk profile, with increasingly intensive lipid lowering recommended for those at highest risk.

Non-high-density lipoprotein cholesterol (non-HDL-C) reflects the burden of all atherogenic lipoproteins and provides important information regarding residual cardiovascular risk, particularly that associated with triglyceride-rich lipoproteins. Recent guidelines recognise non-HDL-C as a co-primary treatment target.

This is especially relevant in South Asians, who commonly exhibit an atherogenic dyslipidaemia phenotype characterised by elevated triglycerides and low high-density lipoprotein cholesterol (HDL-C) levels.

Recent lipid guidelines also emphasise the growing roles of apolipoprotein B (ApoB) and lipoprotein(a) [Lp(a)] in cardiovascular risk assessment and risk reduction strategies.

Effective lipid management begins with accurate interpretation of the lipid profile. Many laboratory reports continue to rely on population-derived reference intervals that may not reflect guideline-directed, evidence-based therapeutic targets. A well-designed lipid report should not merely identify abnormal values but should also support risk-based clinical decision-making, enabling clinicians to achieve recommended lipid targets and improve cardiovascular outcomes.

BEYOND LDL AND HDL — WHERE ADVANCED LIPIDS TELL THE TALE

Dr Gayani Weerasinghe

Traditional lipid measurements, such as LDL cholesterol (LDL-C) and high-density lipoprotein cholesterol (HDL-C), remain central to cardiovascular risk assessment. However, they do not fully capture lipid-related risk. Advances in lipidology support the use of extended lipid testing in selected patients to improve risk stratification and guide personalised management.

Key advanced lipid markers include lipoprotein(a) [Lp(a)], apolipoprotein B (ApoB), apolipoprotein A1 (ApoA1), non-HDL cholesterol, and emerging biomarkers. Inflammatory markers such as high-sensitivity C-reactive protein (hs-CRP), alongside emerging research markers including LDL particle number (LDL-P), small dense LDL, and genetic risk scores, may improve risk prediction beyond conventional lipid measures. Lp(a) is a genetically determined risk factor associated with premature coronary artery disease and aortic valve disease. ApoB reflects the total number of atherogenic lipoprotein particles and may outperform LDL-C in predicting risk, particularly in diabetes, obesity, and metabolic syndrome. Non-HDL cholesterol offers a simple measure of total atherogenic cholesterol, especially when triglycerides are elevated.

These markers are particularly useful in familial hypercholesterolaemia, premature cardiovascular disease, metabolic disorders, and in patients with residual risk despite optimal lipid-lowering therapy. They can help guide intensification of treatment, including statins, ezetimibe, PCSK9 inhibitors, and newer agents targeting Lp(a). Practical issues include assay standardisation, interpretation, and availability in routine practice. Current guidelines increasingly recognise their role within preventive cardiology and NHS cardiovascular disease prevention pathways.

Overall, extended lipid profiling enhances early identification of high-risk individuals, supports targeted therapy, and improves long-term cardiovascular outcomes.

DECODING THE CSF: BIOCHEMICAL CLUES IN NEUROLOGICAL DISEASES

Dr Kishara Gooneratne

Cerebrospinal fluid (CSF) examination remains a powerful diagnostic window into neurological disease, particularly when interpreted together with the clinical syndrome, timing of sampling, paired serum results, and neuroimaging. This lecture will provide a practical framework for “decoding” CSF biochemical abnormalities in common and high-stakes neurological presentations.

The talk will review the interpretation of CSF protein, glucose and CSF glucose ratio, lactate, chloride, albumin quotient, IgG index, and oligoclonal bands, highlighting how these markers help differentiate bacterial, tuberculous, fungal, and viral meningoencephalitis; autoimmune and demyelinating disorders; malignant infiltration; subarachnoid haemorrhage; and selected metabolic or mitochondrial conditions.

Particular emphasis will be placed on biochemical patterns that should trigger urgent action, such as hypoglycorrhachia, markedly elevated lactate, albuminocytological dissociation, and inflammatory intrathecal immunoglobulin synthesis.

The session will also address pre-analytical and interpretive pitfalls, including traumatic taps, delayed glucose processing, absence of paired blood glucose, prior antibiotic exposure, and age-related variation in protein. Using clinical vignettes, the lecture will link laboratory patterns to bedside decision-making, demonstrating how CSF biochemistry can narrow the differential diagnosis, guide early treatment, and indicate when further microbiological, immunological, cytological, or genetic testing is required.

FROM CRADLE TO CROWN — BIOCHEMICAL EVIDENCE WHEN A WOMAN’S LIFE TURNS AROUND

Dr Thushara Hewageegana

Gynaecological disorders often present a diagnostic challenge for clinicians. Overlapping clinical features, atypical presentations, unreliable clinical histories, and the need for invasive procedures may create significant diagnostic dilemmas. Acute presentations and unstable clinical conditions further complicate management. Some gynaecological disorders occur in early pregnancy, while others are encountered in non-pregnant women, adding to the complexity.

Laboratory investigations play a vital role in the management of gynaecological disorders; however, their interpretation may be challenging. Correlation of clinical history, examination findings, laboratory results, and radiological findings has improved diagnostic sensitivity. International guidelines and their recent updates have helped bring greater consensus to patient management.

KIDNEY IN STRAIN – A SILENT ISLAND PAIN: CKD IN SRI LANKA

Dr Udana Ratnapala

Chronic kidney disease (CKD) has emerged as a major public health challenge in Sri Lanka, imposing a substantial burden on individuals, families, and the healthcare system. While diabetes and hypertension remain the leading causes of CKD worldwide, Sri Lanka faces the additional challenge of CKD of uncertain aetiology (CKDu), particularly affecting rural farming communities in the North Central, Uva, and North Western Provinces. The disease often progresses silently, with many patients presenting at advanced stages, when opportunities for prevention and early intervention have been missed.

Over the past decade, Sri Lanka has witnessed a growing demand for renal replacement therapy, including haemodialysis, peritoneal dialysis, and kidney transplantation. This increasing burden places significant pressure on limited healthcare resources and highlights the urgent need for strengthened prevention strategies, early detection programmes, and equitable access to renal care.

This lecture will provide an overview of the epidemiology, causes, and current trends of CKD in Sri Lanka, discuss the unique challenges posed by CKDu, and explore innovative approaches to prevention and integrated renal care. Drawing on national experiences and regional initiatives, the session will highlight opportunities to improve outcomes through public awareness, primary care engagement, and sustainable health policy interventions.

Ultimately, addressing CKD requires a coordinated national response to alleviate what remains a largely silent but devastating burden on the island nation.

RENAL STONES: PREVENTION, BIOCHEMICAL INVESTIGATIONS AND MANAGEMENT

Dr Farhan Ahmed

Renal stone disease is a common and recurrent disorder with a substantial clinical and economic impact worldwide. Despite advances in surgical management, recurrence prevention and metabolic evaluation remain underutilized in routine clinical practice. This lecture will present a practical, case-based approach to the prevention, biochemical investigation, and medical management of nephrolithiasis, integrating current evidence with real-world clinical decision-making.

Through representative clinical cases, the lecture will explore the spectrum of stone disease, including calcium oxalate, uric acid, and cystine stones. Each case will illustrate key diagnostic and therapeutic challenges, emphasizing how targeted biochemical investigations, including serum metabolic profiles, stone composition analysis, and 24-hour urine studies, can identify modifiable risk factors and guide personalized management. The lecture will highlight preventive strategies tailored to individual metabolic abnormalities, with a focus on hydration, dietary interventions, optimization of urinary chemistry, and lifestyle modification. Pharmacological management will be discussed in the context of specific patient scenarios to demonstrate the rationale for evidence-based treatment selection.

Special attention will be given to recurrent stone formers, systemic disorders associated with nephrolithiasis, and the importance of long-term follow-up in reducing recurrence and preserving renal function. Case discussions will provide attendees with practical insights into translating biochemical data into effective, patient-centred management plans.

By combining clinical cases with guideline-based recommendations, this lecture aims to enhance clinicians' confidence in adopting a precision-medicine approach to renal stone disease, ultimately improving prevention strategies, treatment outcomes, and quality of patient care.

LABORATORY INVESTIGATIONS IN MINERAL AND BONE DISORDER OF CHRONIC KIDNEY DISEASE (CKD-MBD)

Dr Samuel Vasikaran

Laboratory investigations are important for the assessment and appropriate management of patients with mineral and bone disorder of chronic kidney disease (CKD-MBD). Mineral disorders are assessed by measurement of serum calcium, phosphate, magnesium, parathyroid hormone (PTH), and 25-hydroxyvitamin D [25(OH)D], in addition to creatinine.

Ionised calcium at the patient's pH is the recommended marker of calcium homeostasis, rather than total calcium. PTH is the main hormone involved in the regulation of calcium and bone balance and is used for monitoring CKD-MBD. However, laboratory measurement of PTH is affected by inter-method differences due to the lack of standardisation of commercial immunoassays.

Vitamin D, a prohormone, is important for bone health, and its deficiency can cause osteomalacia or contribute to the development of osteoporosis, both of which are components of renal osteodystrophy. Vitamin D metabolism is impaired in advanced CKD, amplifying the effects of deficiency on bone health. Lack of consensus on optimal serum 25(OH)D concentrations for bone health is further complicated by measurement issues in commercial immunoassays. Liquid chromatography–tandem mass spectrometry (LC-MS/MS) assays may be more reliable.

Calculated maximal tubular reabsorption of phosphate normalised to glomerular filtration rate (TmP/GFR) is useful in the assessment of phosphate status. Fibroblast growth factor 23 (FGF23), while important in phosphate homeostasis and known to accumulate in CKD, is not useful for the clinical management of CKD-MBD.

Bone-specific alkaline phosphatase (BALP) and tartrate-resistant acid phosphatase isoform 5b (TRACP5b) have been designated as reference markers of bone formation and resorption, respectively, in CKD-associated osteoporosis. BALP and TRACP5b reflect histomorphometric indices of bone turnover with reasonable accuracy in patients with advanced CKD and may be useful in initial assessment, treatment selection, and monitoring of patients with CKD-associated osteoporosis.

WHEN PROTEINS DIVIDE AND DIAGNOSES ARISE: THE POWER OF PROTEIN ELECTROPHORESIS

Dr Rajitha Samarasinghe

Protein electrophoresis is a laboratory technique in which serum globulins are separated into distinct fractions, including alpha-1, alpha-2, beta-1, beta-2, and gamma globulins. It is commonly used in the diagnosis, monitoring, and follow-up of plasma cell disorders, with both serum and urine samples playing important roles at different stages of assessment and management.

Interpretation of protein electrophoresis patterns can sometimes be challenging, as appearances may vary depending on the underlying clinical condition, sample type, and analytical platform used. Conditions such as acute inflammation, chronic inflammation, light chain disease, and nephrotic syndrome may produce characteristic or overlapping electrophoretic patterns, which can influence diagnosis and subsequent patient management.

In addition, separation patterns may differ between gel and capillary electrophoresis platforms, further highlighting the importance of careful interpretation. This presentation will explore the diagnostic value of protein electrophoresis, common interpretative challenges, and the need to correlate electrophoretic findings with clinical and laboratory information.

MARKERS THAT SPEAK DETECTING CANCER WE SEEK

Associate Professor Robert Hawkins

Tumour markers are high-volume, high-risk tests that are frequently misunderstood by clinicians. Small numerical changes often drive major clinical decisions, yet methodological differences between platforms can cause direct patient harm. Their primary clinical utility lies in monitoring therapy response and surveillance for recurrence; their role in diagnosis and screening is limited, and single results are rarely meaningful in isolation.

The recently published CLSI C65 guideline provides a structured laboratory framework organised around the three-phase total testing process: pre-examination, examination, and post-examination. Key pre-analytical considerations vary by marker: PSA is affected by prostatic procedures, ejaculation, and several medications, including 5-alpha-reductase inhibitors and thiazide diuretics; CA125 may be transiently elevated during menstruation or after pelvic surgery; calcitonin requires immediate storage on ice due to instability; and cannabis use can interfere with hCG measurement.

A central analytical concern is non-interchangeability. Most major markers, including CEA, CA19-9, CA125, CA15-3, hCG, thyroglobulin, and calcitonin, lack international reference standards and show significant between-method bias. Serial monitoring is only clinically valid when the same platform is used consistently. Any method change requires parallel testing, bias documentation, and sustained clinician notification.

Biological variation data demonstrate that most tumour markers have an index of individuality well below 0.6, making population-based reference intervals poorly suited for individual patient monitoring. Decision limits, patient-specific baselines, and reference change values are more appropriate interpretive tools.

For reporting, C65 recommends cumulative and, ideally, graphical formats; inclusion of the analytical method; and the use of decision limits rather than reference intervals where applicable, but not both.

DAY 02

ACCEPTANCE TESTING IN CHEMICAL PATHOLOGY: VERIFYING PERFORMANCE BEFORE PATIENT CARE

Dr Gayani Weerasinghe

Acceptance testing is a critical component of laboratory quality management and plays a vital role in ensuring patient safety before new analytical systems are introduced into routine clinical service. In Chemical Pathology, verification of analyser performance prior to implementation is essential to confirm that instruments, assays, and associated systems meet predefined operational, analytical, and clinical requirements.

Key areas include verification of analytical performance characteristics such as precision, accuracy, linearity, carryover, reference intervals, and comparability with existing methods. Acceptance testing also includes risk assessment, documentation, quality indicators, and compliance with ISO 15189 accreditation requirements. In the context of laboratory automation, digital connectivity, and evolving service demands, acceptance testing remains a key component of current medical laboratory practice.

This presentation highlights the collaborative role of laboratory professionals, clinicians, biomedical scientists, and industry partners in ensuring a safe and effective transition from validation to routine patient testing. Real-world examples from laboratory practice will be used to demonstrate common challenges encountered during analyser implementation, including troubleshooting unexpected performance issues, managing workflow disruptions, and ensuring continuity of patient care.

Effective acceptance testing not only assures technical performance but also strengthens clinical confidence in laboratory results, ultimately supporting accurate diagnosis, treatment decisions, and improved patient outcomes.

PREDICT THE RISK, PERFECT THE TEST

Associate Professor Robert Hawkins

ISO 15189:2022 places risk management at the heart of medical laboratory quality systems. Unlike previous iterations, the 2022 standard embeds risk-based requirements across the entire testing cycle, from sample collection and transport through analytical processes to result reporting and clinical communication. Laboratory management holds overall responsibility for establishing a risk management policy, approving risk assessments, and ensuring regular review with documented outcomes.

Risk is commonly assessed in terms of likelihood and consequence, with the primary focus on potential harm to patients. Two complementary approaches are required: prospective risk management, applied to planned changes such as new test introduction, analyser installation, or laboratory information system upgrades; and reactive risk management, triggered by incidents, external quality assurance failures, or nonconforming work.

While specific risk scoring tools, such as risk matrices, are optional, laboratories must demonstrate that actions taken are proportional to the severity of potential harm. A structured risk management system should incorporate a risk register, defined assessment triggers, escalation rules, linkage to quality indicators, and integration with corrective and preventive action processes. The hierarchy of risk controls favours process redesign and built-in safeguards over reliance on training alone, which represents the weakest form of control.

Supporting standards, including ISO 22367:2020 for the application of risk management and ISO 35001:2019 for biorisk management, are available but not mandatory. Quality indicators across the pre-analytical, analytical, and post-analytical phases serve as objective evidence that risk controls are effective. Residual risks must be communicated transparently to clinical users.

Common audit findings include outdated risk registers, inconsistent risk scoring, absent residual risk statements, and failure to link risk management activities to measurable improvement. Auditors seek evidence of a functioning system, not documentation alone.

FROM ERROR TO EXCELLENCE: SIX SIGMA AND MEASUREMENT OF UNCERTAINTY IN LABORATORY MEDICINE

Dr Sakunthala Jayasinghe

Maintaining quality and minimizing errors in the clinical laboratory is essential for safe patient care. However, laboratories must achieve this amid increasing resource and budget constraints. Therefore, quality should be maintained while minimizing wastage and maximizing efficiency. Two methods that are increasingly gaining popularity are Six Sigma and measurement uncertainty.

Six Sigma is a method of process improvement that focuses on minimizing variability in process outputs. It can help answer one of the most commonly asked questions in laboratory quality control: how often should QC be run? The Six Sigma model allows laboratories to evaluate the effectiveness of their current QC processes. Its most common use is to support a risk-based approach to QC, where the optimum QC frequency and multi-rule procedure can be based on the sigma score of the test in question.

Measurement uncertainty relates to the doubt that exists in the result of any measurement. In a hospital or healthcare environment, the clinician must be confident that any change identified in a patient's test result is due to a change in the patient's status, rather than variation within the laboratory test system. This is especially critical at clinical decision levels. Two values are needed to quantify uncertainty: the width of the margin or interval, and the confidence level, which states how certain we are that the "true value" lies within that margin. In addition to being a requirement for accreditation, maintaining acceptable measurement uncertainty adds value to the laboratory.

Six Sigma and measurement uncertainty can be used to increase efficiency in QC practices. Both statistical calculations, however, may involve time-consuming and labour-intensive data analysis. The ideal solution would be for laboratories to utilize a programme with automatic QC data upload and automated statistical calculations.

RAPID AND BRIGHT: GUIDING CARE RIGHT POINT-OF-CARE TESTING

Dr Saswati Das

Point-of-care testing (POCT) is the analysis of patient specimens outside the clinical laboratory, near or at the site of patient care. It is essentially a laboratory test conducted outside the laboratory setting, usually by appropriately trained non-laboratory staff, although it also encompasses patient self-monitoring. POCT provides rapid results near the patient, allowing immediate clinical action.

The key driver for POCT is the concept that clinical decision-making may be delayed when samples are sent to the central clinical laboratory. Devices used for point-of-care testing come in a variety of forms, including basic dipsticks, handheld devices such as glucose meters, and sophisticated bench-top analysers. A healthcare professional may use a handheld device to perform a test at the patient's bedside. Alternatively, a blood sample may be collected from the patient and sent to a satellite laboratory, where it is processed and tested using instrumentation. Both are examples of point-of-care testing.

There are many challenges associated with POCT, mainly related to quality assurance. POCT is often performed by clinical staff rather than laboratory-trained personnel, which may lead to errors due to limited understanding of the importance of quality control and quality assurance practices. POCT is usually more expensive than testing performed in the central laboratory and requires significant laboratory support to ensure quality testing and meet accreditation requirements.

The justification for POCT depends on demonstrating that a more timely result, with a shorter turnaround time (TAT), can provide a clinically important advantage in decision-making compared with testing performed in the central laboratory. This talk will focus on the role of the laboratory in developing a hospital point-of-care testing policy, with emphasis on the quality assurance of POCT.

AETIOLOGICAL DIAGNOSIS OF DIABETES: METHODS AND CLINICAL UTILITY IN THE ERA OF PRECISION MEDICINE

Professor Prasad Katulanda

Diabetes mellitus is a heterogeneous group of metabolic disorders characterized by hyperglycaemia resulting from diverse underlying mechanisms. While the majority of patients are classified as having type 1 or type 2 diabetes, a significant proportion have specific aetiological subtypes, including monogenic diabetes, pancreatogenic diabetes, endocrinopathy-associated diabetes, drug-induced diabetes, and latent autoimmune diabetes in adults (LADA). Accurate identification of the underlying aetiology is increasingly important, as therapeutic approaches, prognosis, risk of complications, and implications for family screening differ substantially among these subtypes.

This presentation will review contemporary approaches to the aetiological diagnosis of diabetes, emphasizing the complementary roles of clinical assessment, biochemical investigations, immunological markers, and genetic testing. Clinical indicators such as age at onset, family history, obesity, metabolic syndrome, ketosis, and associated autoimmune or pancreatic disorders provide important diagnostic clues.

Biochemical methods, including C-peptide measurement, markers of insulin resistance, lipid profiles, and high-sensitivity C-reactive protein (hs-CRP), will be discussed. The role of pancreatic autoantibodies, including glutamic acid decarboxylase 65 (GAD65), IA-2, zinc transporter 8 (ZnT8), and islet cell antibodies, in identifying autoimmune diabetes will be highlighted. Advances in molecular diagnostics, particularly in the detection of monogenic forms of diabetes such as maturity-onset diabetes of the young (MODY), will also be reviewed.

The clinical utility of aetiological diagnosis will be illustrated through precision medicine approaches, including intensive insulin therapy for type 1 diabetes, cardiometabolic risk-reducing therapies for type 2 diabetes, sulfonylurea responsiveness in HNF1A/HNF4A-MODY, and treatment de-escalation in GCK-MODY. The presentation will emphasize how mechanism-based classification can improve patient outcomes and support personalized diabetes care.

SUGAR IN MOTION: CONTINUOUS MONITORING FOR BETTER NOTION

Dr Leslie Lai

Continuous glucose monitoring (CGM) provides real-time, dynamic information about blood glucose concentrations, unlike intermittent finger-prick capillary blood glucose measurements. CGM measures interstitial fluid glucose concentrations at frequent, regular intervals, allowing glycaemic patterns, glucose variability, and trends to be assessed.

CGM metrics include time in range (TIR), time in tight range (TITR), time above range (TAR), time below range (TBR), glycaemic variability, expressed as coefficient of variation (CV), and glucose management indicator (GMI). CGM allows glycaemic control to be assessed more accurately than HbA1c alone.

CGM can improve glycaemic control by increasing TIR, reducing hypoglycaemia, and lowering HbA1c levels. It is particularly beneficial for individuals with hypoglycaemic unawareness, type 1 diabetes, type 2 diabetes on insulin therapy, and women with pre-existing diabetes or gestational diabetes during pregnancy. A standardised visual presentation of CGM data, termed the ambulatory glucose profile (AGP), allows easy clinical interpretation and aids individualised therapy adjustments.

The accuracy of sensors, typically expressed as mean absolute relative difference (MARD), continues to improve and should ideally be less than 10%. CGM devices can be used alone or linked to insulin pumps and automated insulin delivery devices, including closed-loop artificial pancreas systems, to optimise insulin dosing decisions in relation to real-time glucose data.

Artificial intelligence and machine learning are being developed and implemented into CGM systems for predictive modelling, allowing early detection and intervention for hypoglycaemia and hyperglycaemia, as well as supporting personalised decision-making. More widespread use of CGM is limited by cost, accessibility, and the need for patient education to maximise its effective use.

FROM VEIN TO VIAL: DRUGS OF ABUSE AND BIOCHEMICAL TRUTHS BEYOND THE FINAL TRIAL

Senior Professor Muditha Vidanapathirana

Substance abuse has accompanied human civilization since antiquity, with evidence of alcohol and psychoactive drug use dating back several millennia. From ancient opium consumption in Asia to the ritualistic and medicinal use of cannabis in Sri Lanka, psychoactive substances have shaped cultures, warfare, medicine, and crime. In the modern era, synthetic drugs such as amphetamine-type stimulants (ATS), methamphetamine, MDMA, and novel psychoactive substances have transformed patterns of abuse and intensified medico-legal challenges worldwide.

This presentation explores the historical evolution, pharmacology, biochemical pathways, forensic interpretation, and medico-legal implications of drug and alcohol abuse. Particular emphasis is placed on ethanol metabolism, amphetamine-related toxicity, and the neurochemical effects of stimulants and sedatives on dopamine, norepinephrine, and serotonin pathways. Acute and chronic clinical manifestations, including psychosis, cardiovascular complications, hyperthermia, dependency, withdrawal syndromes, and sudden death, are discussed in relation to forensic investigations.

The lecture further examines the scientific principles and limitations of alcohol and drug testing, including clinical assessment, field sobriety testing, breath analysis, saliva testing, immunoassays, and confirmatory toxicological techniques such as gas chromatography–mass spectrometry. Special attention is given to postmortem toxicology, endogenous alcohol production, postmortem redistribution, and interpretation of toxicological findings during autopsy practice.

Sri Lankan trends in drug abuse, emerging synthetic substances, legal provisions, and the increasing social and public health burden associated with addiction are highlighted. The presentation integrates biochemical science with forensic practice to demonstrate how toxicological evidence bridges clinical medicine, law enforcement, and judicial processes. Ultimately, it emphasises that behind every toxicology report lies a complex biochemical narrative extending beyond death, from the vein to the vial.

CONGENITAL ADRENAL HYPERPLASIA BLOCK TO BALANCE

Dr Navoda Atapattu

Congenital adrenal hyperplasia (CAH) is an autosomal recessive disorder caused primarily by *CYP21A2* mutations. This leads to 21-hydroxylase deficiency, which impairs cortisol synthesis. Without cortisol, the negative feedback loop fails, triggering chronic adrenocorticotrophic hormone (ACTH) overproduction and severe hyperandrogenism. Clinical phenotypes vary from severe classic salt-wasting and simple virilising forms to mild, non-classic variants.

Newborn screening identifies CAH by measuring 17-hydroxyprogesterone (17-OHP) in dried blood spots. Modern protocols integrate second-tier liquid chromatography–tandem mass spectrometry (LC-MS/MS) profiling to reduce false positives. Treatment requires supraphysiological glucocorticoids to suppress hypothalamic-pituitary-adrenal axis hyperactivity, with mineralocorticoids added for salt-wasting disease. Long-term surveillance focuses on monitoring growth velocity, bone age, and blood pressure, as well as screening for testicular adrenal rest tumours (TARTs).

Traditionally, clinicians have monitored biochemical disease control using 17-OHP, androstenedione (A4), and plasma renin activity (PRA). The standard strategy aims for near-normal A4, mid-normal PRA, and 17-OHP levels roughly twice the upper limit of normal. However, 17-OHP tracking is highly unstable due to marked diurnal fluctuations, physiological variability, and weak correlation with actual tissue androgen exposure. This instability may lead to therapeutic mismanagement, resulting in inadvertent over- or under-treatment.

To overcome these challenges, the monitoring landscape is shifting towards stable, adrenal-specific 11-oxygenated androgens, particularly 11-ketotestosterone (11KT) and 11 β -hydroxyandrostenedione (11OHA4). Because these biomarkers correlate directly with androgen receptor activation, they offer a reliable and biologically relevant picture of disease control. This allows clinicians to suppress hyperandrogenism effectively while safely minimising the risks of glucocorticoid overtreatment.

FROM PATHWAY TO THERAPY: SOLVING THE RIDDLE OF PORPHYRIA

Dr Thathsarani Vithana Pathirana

Porphyria remains one of the more challenging and fascinating areas in clinical biochemistry, where disturbances in a single metabolic pathway can give rise to a wide spectrum of clinical presentations. Although rare, porphyrias are important to recognise, as delayed diagnosis may lead to significant morbidity and missed opportunities for appropriate treatment and family counselling. This lecture will provide a practical overview of porphyria, moving from the haem biosynthetic pathway to clinical presentation, laboratory diagnosis, and current therapeutic approaches. It will highlight how different pathway defects translate into distinct patterns of disease, and how careful clinical and biochemical assessment can guide the diagnostic process. Particular emphasis will be placed on the role of the laboratory in recognising porphyria, selecting appropriate investigations, ensuring correct sample handling, and interpreting results in the clinical context. The lecture will also touch on the value of molecular testing, family screening, and recent advances in targeted therapy. By linking biochemical mechanisms with patient care, this session aims to make porphyria more understandable, clinically relevant, and approachable for both clinicians and laboratory professionals.

TINY GENES, MAJOR SCENES: THE METABOLIC STORY IN BETWEEN

Dr Vindya Subasinghe

Inherited metabolic disorders represent a unique interface between genetics and chemical pathology, where subtle alterations in DNA can lead to profound biochemical disturbances and significant clinical consequences. This presentation explores the journey from gene to phenotype, illustrating how defects in metabolic pathways manifest as characteristic laboratory signatures that are often first recognised by chemical pathologists.

Through selected examples, including urea cycle disorders, organic acidaemias, and conditions identified through newborn screening, the session highlights the complementary roles of biochemical investigations and genomic technologies in diagnosis and management. The evolving relationship between metabolomics and genomics has transformed the diagnostic landscape, enabling earlier recognition, improved variant interpretation, and the implementation of precision therapies.

Chemical pathologists play a pivotal role not only in detecting metabolic abnormalities but also in providing functional evidence that supports clinical and molecular diagnoses. As healthcare increasingly adopts integrated multi-omics approaches, collaboration between clinical geneticists, chemical pathologists, and other specialists becomes essential.

This symposium emphasises that while genes may initiate the story, metabolites often reveal its most important chapters.

Abstracts of Oral Presentations Research and Audits



ABSTRACTS OF ORAL PRESENTATIONS

RESEARCH AND AUDITS

- OP-RP 01** Cardiovascular Risk Misclassification with Non-Fasting Lipid Testing: A Paired Study in a Sri Lankan Population
- OP-RP 02** Derivation of a Laboratory-Specific Corrected Calcium Equation and Comparison with Payne's Equation in Hospitalized Patients at the Teaching Hospital, Jaffna
- OP-RP 03** Evaluation of a High-Sensitivity Cardiac Troponin I (hs-cTnI) Assay and Determination of the 99th Percentile Cut-Off for a Healthy Population in Sri Lanka
- OP-AR 01** Sustainability Audit in Biochemistry and Molecular Biology Research Laboratories: Opportunities for Carbon Reduction in a Sri Lankan Academic Setting

ABSTRACTS OF ORAL PRESENTATIONS

RESEARCH AND AUDITS

OP–RP 01

Cardiovascular Risk Misclassification with Non-Fasting Lipid Testing: A Paired Study in a Sri Lankan Population

Fernando KM¹, Fernando JN², Welhenge CU², Fernando DP³, Madanayaka S⁴, Mettananda KCD⁵, De Silva ST^{6,7}, Dayanath BKTP⁸

¹Department of Biochemistry, Faculty of Medicine, University of Kelaniya, Sri Lanka

²University Medical Unit, National Hospital of Sri Lanka, Sri Lanka

³Faculty of Medicine, University of Colombo, Sri Lanka

⁴Department of Chemical Pathology, Colombo North Teaching Hospital, Sri Lanka

⁵Department of Pharmacology, Faculty of Medicine, University of Kelaniya, Sri Lanka

⁶University Medical Unit, Colombo North Teaching Hospital, Sri Lanka

⁷Department of Medicine, Faculty of Medicine, University of Kelaniya, Sri Lanka

⁸Dorevitch Pathology, Melbourne, Victoria, Australia

kavindyamjn14@gmail.com

Introduction

Current international guidelines endorse non-fasting lipid testing; however, their applicability to South Asian populations, characterized by higher cardiometabolic risk and distinct dietary patterns, remains uncertain. Misclassification of cardiovascular risk in this population may have significant clinical consequences.

Methods

A paired observational study was conducted among 257 adults with both fasting and non-fasting lipid profiles. Analytical agreement was assessed using correlation, Bland–Altman analysis, and concordance within predefined clinical limits. Clinical impact was evaluated using guideline-based thresholds for LDL-C, triglycerides, and non-HDL-C, with reclassification analysis and McNemar's test.

Results

Non-fasting samples showed significantly higher triglyceride concentrations and lower calculated LDL-C concentrations compared with fasting samples ($p < 0.001$). Although correlation was strong for total cholesterol ($r = 0.94$) and LDL-C ($r = 0.90$), clinical agreement was poor.

Concordance within acceptable limits was low for LDL-C (29.5%) and triglycerides (26.0%), but higher for non-HDL-C (57.4%) and HDL-C (87.6%).

Clinically significant discordance was observed. At the LDL-C threshold of 100 mg/dL, 17.1% of individuals were reclassified, predominantly downward (16.0%), indicating potential systematic underestimation of cardiovascular risk. In contrast, triglycerides showed substantial upward reclassification (20.2%) at the 150 mg/dL threshold ($p < 0.001$). Non-HDL-C demonstrated greater stability, with lower reclassification (12.5%).

ABSTRACTS OF ORAL PRESENTATIONS

RESEARCH AND AUDITS

Conclusion

Non-fasting lipid testing results in clinically meaningful misclassification, driven primarily by triglyceride-related variability in calculated LDL-C. This may lead to underestimation of cardiovascular risk and potential undertreatment. Non-HDL-C appears more robust and may be a preferable marker in the non-fasting state. These findings question the universal applicability of non-fasting lipid guidelines to South Asian populations and support a more cautious, context-specific approach.

Keywords

cholesterol, LDL, triglycerides, cardiovascular diseases

ABSTRACTS OF ORAL PRESENTATIONS

RESEARCH AND AUDITS

OP–RP 02

Derivation of a Laboratory-Specific Corrected Calcium Equation and Comparison with Payne's Equation in Hospitalized Patients at the Teaching Hospital, Jaffna

Dilshan HPT¹, Lakmin AGS¹, Senadhipathi SMSS¹, Tharshan R¹, Sivakumar H¹

hptharakadilshan98@gmail.com

Introduction

Serum calcium exists in bound and ionized forms, with ionized calcium being the clinically relevant fraction. Since direct measurement of ionized calcium is technically demanding, corrected calcium equations are commonly used as substitutes. The most widely used equation was described by Payne in 1973. However, its accuracy varies depending on albumin and calcium methods and analyzer type, necessitating a laboratory- and population-specific equation to enhance patient outcomes.

Methods

This retrospective study used laboratory records of medical ward inpatients at Teaching Hospital, Jaffna, Sri Lanka. Adults aged ≥ 18 years with concurrent serum calcium, albumin, ALP, ALT, potassium, and creatinine results were included. Patients attending Endocrinology, Haematology, Oncology, or Nephrology clinics, and those failing Payne's biochemical thresholds, were excluded. These thresholds were ALT > 63 U/L, ALP > 116 U/L, creatinine > 200 $\mu\text{mol/L}$, and potassium outside 3.5–5.1 mmol/L.

Total calcium and albumin were measured using the Arsenazo III and bromocresol green methods, respectively. Records were randomly divided into derivation ($n = 600$) and validation ($n = 303$) groups. The derivation group was used to develop the equation by linear regression, while the validation group was used to assess its validity and agreement with Payne's equation using Bland–Altman analysis. Ethical approval was obtained from the Ethics Review Committee, University of Jaffna.

Results

Mean total calcium and albumin were 2.21 mmol/L and 34.859 g/L, respectively. Linear regression produced the following equation:

Corrected calcium = Total calcium + $0.0227 \times (34.859 - \text{Albumin})$

The slope was 0.0227, intercept was 1.4188, and r was 0.699. The shrinkage value, calculated as R^2 derivation – R^2 validation, was 0.0068, indicating internal validity. The mean absolute deviation of the derived equation was lower than that of Payne's equation: 0.1337 versus 0.1909.

Bland–Altman analysis in 109 hypoalbuminaemic patients showed a mean difference of 0.087 mmol/L, with 95% limits of agreement of 0.068–0.105 mmol/L. Agreement in calcium status classification between the two equations was 75.22% in hypoalbuminaemia and 61.45% in normoalbuminaemia.

Conclusion

Calcium status classifications varied between the two equations, highlighting the importance of laboratory- and population-specific corrected calcium equations.

Keywords: albumin, albumin-adjusted calcium, calcium, corrected calcium equation

ABSTRACTS OF ORAL PRESENTATIONS

RESEARCH AND AUDITS

OP–RP 03

Evaluation of a High-Sensitivity Cardiac Troponin I (hs-cTnI) Assay and Determination of the 99th Percentile Cut-Off for a Healthy Population in Sri Lanka

Jayasinghe S¹, Tennakoon A¹, Kuruppu D², Wijesinghe C²

¹Department of Pathology, Faculty of Medicine, University of Peradeniya, Sri Lanka

²Transfusion Medicine Unit, Teaching Hospital, Peradeniya, Sri Lanka

hbvs.jayasinghe@gmail.com

Introduction

Measurement of cardiac troponin is vital for the diagnosis of acute myocardial infarction. Knowing the 99th percentile upper reference limit (URL) of high-sensitivity cardiac troponin I (hs-cTnI) in the local population supports its appropriate use in clinical practice. This study aimed to verify the analytical performance and identify the URL of hs-cTnI measured by a single assay in a cohort of the Sri Lankan population.

Methods

Performance characteristics, including precision, limit of blank (LoB), limit of detection (LoD), and linearity, were verified, and comparison with a method-established laboratory was performed. Leftover blood samples from blood donors were used to determine the 99th percentile URL, considering them as “healthy” subjects based on a self-administered questionnaire and basic physical examination.

High-sensitivity cardiac troponin I was measured on the Siemens Dimension EXL platform using dedicated reagents from Siemens Healthcare Diagnostics, Newark, USA. The 99th percentile was calculated according to CLSI C28-A3 guidelines.

Results

The analytical performance of the assay was as claimed by the manufacturer. The coefficient of variation was <10% at the 99th percentile URL, and >50% of the selected healthy population had hs-cTnI levels above the LoD, confirming that it is a high-sensitivity assay.

A total of 140 females and 140 males aged 18–60 years were included to establish the 99th percentile URL. The overall 99th percentile was 22.0 ng/L, with a 90% confidence interval of 17.6–25.8 ng/L. A significant difference was observed between males and females. The sex-specific cut-offs, calculated by the robust method, were 13.1 ng/L for females, with a 90% confidence interval of 9.3–17.4 ng/L, and 27.7 ng/L for males, with a 90% confidence interval of 21.4–33.4 ng/L.

The 99th percentile URL was lower for the local population than the levels quoted by the manufacturer.

Conclusion

The Siemens Dimension EXL hs-cTnI method demonstrated acceptable analytical performance and met the criteria required for a high-sensitivity assay. The difference between the locally derived and manufacturer-quoted levels highlights the need to establish the 99th percentile URL for the local population for each method and platform used.

Keywords

high-sensitivity troponin I, reference interval, verification, 99th percentile upper reference limit

ABSTRACTS OF ORAL PRESENTATIONS

RESEARCH AND AUDITS

OP-AR 01

Sustainability Audit in Biochemistry and Molecular Biology Research Laboratories: Opportunities for Carbon Reduction in a Sri Lankan Academic Setting

Jayaweera JATS¹, Thoradeniya T¹, Dilanthi HW¹

¹Department of Biochemistry and Molecular Biology, Faculty of Medicine, University of Colombo, Sri Lanka
fmpg2024mb003@stu.cmb.ac.lk

Introduction

In 2015, the combined climate footprint of healthcare facilities accounted for 4.4% of total global carbon emissions. Sri Lanka's Nationally Determined Contributions emphasize climate resilience in research facilities through energy efficiency. This audit assessed sustainability compliance in Sri Lankan research laboratories and estimated potential carbon emission reductions.

Methods

A cross-sectional audit was conducted in three postgraduate research laboratories at the Department of Biochemistry and Molecular Biology, Faculty of Medicine, University of Colombo, across four domains: electricity use, water consumption, waste management, and hazardous chemical handling. Data were collected through staff interviews, document review, and structured observation, with compliance assessed on a four-point scale adapted from certified accreditation systems. A single trained investigator conducted all audits. Carbon emissions were calculated based on equipment energy consumption using Sri Lanka's grid emission factor of 0.62 kg CO₂/kWh.

Results

The lowest compliance was observed in water conservation at 22.2%, primarily due to inefficient washing practices and lack of leak detection systems. Waste management showed the highest compliance at 53.3%, with reuse and segregation practices. Electricity use at 36.4% and hazardous chemical handling at 44.8% revealed gaps, including unoptimized freezer management, poor organization, and non-compliance with WHO storage recommendations.

Annual carbon emissions from selected equipment exceeded 3 tCO₂ per laboratory, surpassing green laboratory benchmarks of <1.5 tCO₂/laboratory/year given by certified accreditation systems such as My Green Lab. Implementing corrective actions could reduce emissions by approximately 27%, equivalent to 0.8 tCO₂ per laboratory annually. Extrapolation to registered laboratories nationally (n = 586) suggested a potential reduction of approximately 469 tCO₂ annually.

Conclusion

This pilot audit identified critical sustainability gaps within academic laboratory settings. An evidence-based Standard Operating Procedure was developed targeting key areas. Implementation of this SOP offers a practical, low-cost framework for reducing carbon emissions and contributing to Sri Lanka's Nationally Determined Contribution targets. Wider adoption, followed by periodic re-audit, is essential to achieve sustained and measurable impact.

Keywords

sustainability, Laboratory audit, research environment, waste management, water conservation

Abstracts Of Oral Presentations Case Reports And Case Series



ABSTRACTS OF ORAL PRESENTATIONS

CASE REPORTS AND CASE SERIES

- OP - CR 01** Biochemical Evaluation of Persistent Hypophosphataemia: Diagnostic Utility of Fibroblast Growth Factor-23 Measurement
- OP - CR 02** When Mood Stabilisation Disrupts Calcium Homeostasis: Lithium-Induced Hyperparathyroidism A Case Report
- OP - CR 03** Beyond the Textbook: When Calcium Tells a Different Story – A Diagnostic Twist in Gitelman Syndrome
- OP - CR 04** Biochemical Pattern Recognition of Cisplatin-Induced Renal Tubular Electrolyte Wasting Using Fractional Excretion Analysis in Pancreato-Biliary Adenocarcinoma: A Case Report
- OP - CR 05** The Renin Paradox in Cushing's Syndrome: Resolution Following Adrenal Tumour Resection

ABSTRACTS OF ORAL PRESENTATIONS

CASE REPORTS AND CASE SERIES

OP-CR 01

Biochemical Evaluation of Persistent Hypophosphataemia: Diagnostic Utility of Fibroblast Growth Factor-23 Measurement

Jayasuriya NUW¹, Siddiqa OMOM¹, Arooran T¹, Thusyanthi N¹, Mendis S², Katulanda GW¹

¹Department of Chemical Pathology, National Hospital – Sri Lanka, Colombo, Ministry of Health, Sri Lanka.

²Medical Unit, National Hospital – Sri Lanka, Colombo, Ministry of Health, Sri Lanka.

nuwa25101987@gmail.com

Introduction

Tumour-induced osteomalacia (TIO) is a rare paraneoplastic disorder characterised by fibroblast growth factor-23 (FGF23)-mediated renal phosphate wasting, leading to chronic hypophosphataemia and osteomalacia. Systematic biochemical evaluation is essential for diagnosis, particularly when imaging findings are inconclusive.

Case Presentation

A 35-year-old previously healthy man presented with a six-month history of progressive generalised weakness and bone pain. Initial investigations showed hypophosphataemia of 1.31 mg/dL, with markedly elevated 24-hour urinary phosphate excretion of 732.9 mg/24 h; reference <129.7 mg/24 h. Alkaline phosphatase was elevated at 321 IU/L. Calcium and magnesium levels were normal, with normal renal, hepatic, and thyroid function.

Parathyroid hormone was within the upper normal limit at 58 pg/mL. Serum 25-hydroxyvitamin D was adequate at 60.57 ng/mL, while 1,25-dihydroxyvitamin D was inappropriately normal for the severity of hypophosphataemia. DEXA scan demonstrated osteoporosis, with a lumbar spine Z-score of -2.8. PET imaging revealed multiple insufficiency fractures and FDG-avid skeletal lesions, including a C3 vertebral lesion and a metabolically active left thyroid nodule. Biopsies were non-diagnostic, and FGF23 was not assessed initially. On re-presentation, persistent hypophosphataemia of 1.4 mg/dL, elevated fractional excretion of phosphate of 21%, and reduced TmP/GFR of 1.31 mg/dL confirmed renal phosphate wasting. The intact serum FGF23 level, measured by immunoassay, was markedly elevated at 1119 pg/mL; reference range 23.2–95.4 pg/mL. Repeat PET imaging demonstrated persistent FDG-avid thyroid and femoral lesions without definitive tumour localisation.

Discussion and Conclusion

This case highlights the importance of biochemical algorithms in evaluating persistent hypophosphataemia. In suspected TIO, assessment of renal phosphate handling and FGF23 levels is crucial, even when imaging findings are inconclusive. Early integration of specialised biochemical testing may reduce diagnostic delay and guide targeted tumour localisation in suspected TIO.

Keywords

tumour-induced osteomalacia, FGF23, hypophosphataemia

ABSTRACTS OF ORAL PRESENTATIONS

CASE REPORTS AND CASE SERIES

OP-CR 02

When Mood Stabilisation Disrupts Calcium Homeostasis: Lithium-Induced Hyperparathyroidism – A Case Report

Samaratunga EPA¹, Antonypillai CN², Premadasa MGTW¹, Ekanayaka CK¹, Sampath WGP¹, Basnayaka AM¹, Jayawardana RDP¹

¹Department of Chemical Pathology, National Hospital Kandy

²Diabetes and Endocrine Unit, National Hospital Kandy

m31359@pgim.cmb.ac.lk

Background

Lithium is used in the management of bipolar disorder and may result in hyperparathyroidism as a long-term complication. Lithium alters the calcium-sensing receptor set point, resulting in inappropriate PTH secretion and hypercalcaemia, which may persist for years or become permanent even after cessation of treatment.

Case Presentation

A 68-year-old male, a known patient with bipolar affective disorder and dementia, presented with altered behaviour of one week's duration. He had recent-onset polyuria and constipation. He had been on lithium carbonate, risperidone, quetiapine, and benzhexol for five years.

On examination, he was mildly dehydrated and had a GCS of 14/15. His serum lithium level was 1.18 mmol/L; therapeutic range 0.6–1.2 mmol/L, toxic range >1.5 mmol/L. Total calcium was 3.2 mmol/L; reference range 2.1–2.55 mmol/L. Phosphate was 0.98 mmol/L; reference range 0.81–1.45 mmol/L. PTH was 197.8 pg/mL; reference range 15–65 pg/mL. Serum creatinine was 78.9 μ mol/L; reference range 65–120 μ mol/L. ALP was 513.3 IU/L; reference range 30–120 IU/L. Serum 25-hydroxyvitamin D was 9.3 ng/mL, indicating deficiency. Following omission of lithium and adequate hydration, neurological symptoms and polyuria gradually improved. Ultrasound scan revealed a focal lesion at the left lower pole of the thyroid gland, while there were no calculi or nephrocalcinosis in either kidney. DXA scan revealed osteoporosis, with a forearm T-score of –2.1.

The patient was started on vitamin D. After three months, serum corrected calcium was 2.82 mmol/L, phosphate was 1.0 mmol/L, PTH was 76.1 pg/mL, and 25-hydroxyvitamin D was 58.5 ng/mL, indicating sufficiency. The possibility of lithium-induced hyperparathyroidism was supported by hypocalciuria and a significant drop in PTH following vitamin D correction. The patient is being managed with adequate hydration, vitamin D, and intravenous zoledronic acid.

Discussion

Biochemical investigations play a major role in the assessment of lithium-related complications and hypercalcaemia. Lithium may cause parathyroid adenoma or hyperplasia, resulting in persistence of the disease even after cessation of treatment. Therefore, biochemical evaluation is required to assess the prognosis of the condition.

Keywords

bipolar disorder, nephrocalcinosis, hyperparathyroidism

ABSTRACTS OF ORAL PRESENTATIONS

CASE REPORTS AND CASE SERIES

OP-CR 03

Beyond the Textbook: When Calcium Tells a Different Story – A Diagnostic Twist in Gitelman Syndrome

Krishanthi MP¹, Liyanagunawardana JE¹, Arooran T¹, Siddiqa OMOM¹, Prashanthan S¹, Sumanatilleke M², Katulanda GW¹

¹Department of Chemical Pathology, National Hospital of Sri Lanka

²Diabetes and Endocrinology Unit 1, National Hospital of Sri Lanka

mpkrishanthi@gmail.com

Introduction

Gitelman syndrome (GS) is an inherited disorder caused by mutations in the *SLC12A3* gene. It is classically characterised by hypokalaemic metabolic alkalosis, hypomagnesaemia, and hypocalciuria. Despite its well-defined biochemical profile, phenotypic variability may obscure the diagnosis, particularly when patients present with features typically associated with other renal tubulopathies.

Case Presentation

A 36-year-old man presented with a two-month history of progressive generalised weakness, culminating in an acute inability to walk. He reported a similar episode five years earlier, which had been managed as refractory hypokalaemia without further evaluation.

On examination, he was normotensive, with a blood pressure of 100/60 mmHg, and had symmetrical flaccid quadriplegia, with preserved deep tendon reflexes and intact sensation.

Biochemical evaluation demonstrated persistent hypokalaemia, hypomagnesaemia, and metabolic alkalosis. Urinary indices indicated inappropriate renal potassium loss with concomitant magnesium wasting: transtubular potassium gradient 10, potassium-to-creatinine ratio 0.05 mmol/mg, 24-hour urinary potassium 72.9 mmol/24 h, and urinary magnesium 148.8 mg/24 h. Contrary to expectations, renal imaging revealed nephrocalcinosis, while urinary calcium excretion was low, with a calcium-to-creatinine ratio of 0.07.

Elevated plasma renin activity with normal aldosterone concentration excluded primary aldosteronism. A definitive diagnosis was established by identifying biallelic inactivating mutations in the *SLC12A3* gene. Targeted therapy with potassium and magnesium supplementation, in combination with spironolactone, resulted in significant neurological recovery.

Discussion

This case challenges conventional diagnosis by demonstrating GS with nephrocalcinosis, an atypical association more commonly observed in Bartter syndrome or distal renal tubular acidosis. The coexistence of marked hypomagnesaemia and relative hypocalciuria served as important discriminating features.

Clinicians should maintain a high index of suspicion for GS in adults presenting with recurrent hypokalaemic paralysis and renal electrolyte wasting, even in the presence of potentially misleading radiological findings. Early recognition, supported by comprehensive biochemical evaluation and genetic confirmation, is essential to prevent recurrence and guide long-term management.

Key words

Gitelman syndrome, hypokalaemia, nephrocalcinosis, *SLC12A3* gene

ABSTRACTS OF ORAL PRESENTATIONS

CASE REPORTS AND CASE SERIES

OP-CR 04

Biochemical Pattern Recognition of Cisplatin-Induced Renal Tubular Electrolyte Wasting Using Fractional Excretion Analysis in Pancreato-Biliary Adenocarcinoma: A Case Report

Senevirathna DCL¹, Dissanayake M¹, Samarasinghe R²

¹Department of Chemical Pathology, National Hospital Galle, Sri Lanka.

²Department of Chemical Pathology, National Cancer Institute, Maharagama, Sri Lanka.

chamila626@gmail.com

Introduction

Cisplatin is a widely used chemotherapeutic agent whose major dose-limiting toxicity is nephrotoxicity resulting from renal tubular injury. This injury may cause significant electrolyte wasting involving magnesium, potassium, sodium, and phosphate. In oncology patients, electrolyte disturbances have multiple potential causes, including syndrome of inappropriate antidiuretic hormone secretion, poor intake, and gastrointestinal losses. Systematic biochemical evaluation is therefore essential to identify the underlying mechanism.

Case presentation

A 56-year-old woman with seropositive rheumatoid arthritis presented with chronic cough and recurrent haemoptysis and was diagnosed with differentiated adenocarcinoma involving the right lung, with suspected pancreato-biliary origin. She was commenced on cisplatin and gemcitabine. Two months after treatment initiation, she developed hyponatraemia of 118–124 mmol/L, hypokalaemia of 1.9–2.0 mmol/L, hypomagnesaemia of 0.26 mmol/L, hypophosphataemia of 0.62 mmol/L, and hypocalcaemia of 1.98 mmol/L. Paired serum and urine electrolyte analysis demonstrated increased fractional excretion of sodium at 4.39%, potassium at 29.62%, and magnesium at 11.43%, indicating significant renal electrolyte wasting with relative calcium sparing. Findings were consistent with cisplatin-induced renal tubular dysfunction. Electrolyte replacement resulted in biochemical improvement, while chemotherapy was temporarily withheld.

Discussion

This case illustrates the characteristic biochemical pattern of cisplatin-induced renal tubular dysfunction, where hypomagnesaemia plays a central pathophysiological role and contributes to refractory hypokalaemia and secondary hypocalcaemia. Recognition of multi-electrolyte renal wasting through fractional excretion analysis enabled differentiation from other causes of hyponatraemia. This pattern contrasts with gastrointestinal losses or poor intake, where the kidney typically conserves electrolytes, resulting in low urinary excretion. SIADH was also excluded, as hyponatraemia is the key feature, with inappropriately elevated urinary sodium but without associated magnesium and potassium wasting.

The prominent renal magnesium wasting observed in this patient is a recognised biochemical hallmark of cisplatin-induced tubular injury. This case highlights the critical role of chemical pathology in integrating biochemical data with clinical context to identify drug-induced tubulopathy and guide targeted electrolyte correction.

Key words

cisplatin nephrotoxicity, renal tubular dysfunction, electrolyte wasting, fractional excretion

ABSTRACTS OF ORAL PRESENTATIONS

CASE REPORTS AND CASE SERIES

OP-CR 05

The Renin Paradox in Cushing's Syndrome: Resolution Following Adrenal Tumour Resection

Sumathipala RPAP¹, Basnayake BMAM¹, Kumarathunga D², Jayawardana RDP¹

¹Department of Chemical Pathology, National Hospital Kandy, Sri Lanka

²Department of Endocrinology, District General Hospital, Nuwara Eliya, Sri Lanka
ayomisumathipala@gmail.com

Introduction

ACTH-independent Cushing's syndrome results from autonomous cortisol secretion by the adrenal cortex, commonly due to adenoma or hyperplasia. Large adrenal tumours may exert local mass effects on adjacent structures. Renal artery compression is a rare but plausible mechanism leading to activation of the renin–angiotensin–aldosterone system due to reduced renal perfusion.

Case presentation

A 45-year-old female with a 10-year history of resistant hypertension on five antihypertensives, including spironolactone, presented with rapid weight gain and classical cushingoid features over six months. Biochemical evaluation confirmed ACTH-independent Cushing's syndrome, with non-suppressed cortisol following ODSST at 371 nmol/L and LDDST at 631 nmol/L, with suppressed ACTH of 4.7 pg/mL; reference range 6–48 pg/mL.

CECT adrenal protocol revealed a 50 × 50 × 45 mm left adrenal mass compressing the upper pole of the kidney. The mean cortisol burden in the day curve was elevated at 701.6 nmol/L. Associated findings included impaired glycaemia, with HbA1c of 6.3%, and osteopenia on DEXA scan. Androgen profile and urinary metanephrines were normal.

Evaluation for mineralocorticoid excess revealed a markedly elevated renin level of >1000 μ IU/mL; reference range 4.2–45.6 μ IU/mL, with an elevated aldosterone level of 61.57 ng/dL; reference range 7–30 ng/dL. These tests were performed while she was on five different antihypertensives.

She underwent left laparoscopic adrenalectomy. Histology confirmed an adrenocortical adenoma with myelolipomatous change. At six months, biochemical remission was achieved, with ODSST cortisol of 14.49 nmol/L. Hypertension improved significantly, enabling reduction to a single antihypertensive, and ARR parameters normalised.

Discussion

This case illustrates a rare dual-pathophysiological mechanism contributing to severe hypertension: cortisol-mediated effects of Cushing's syndrome alongside secondary RAAS activation due to tumour-related renal vascular compression. The markedly elevated renin level, despite potential pharmacological interference, and its normalisation following surgery support a mechanical aetiology rather than co-secretion.

Recognition of this interplay is crucial when interpreting ARR in adrenal masses and highlights the importance of considering structural causes in atypical biochemical profiles.

Key words

Cushing's syndrome, aldosterone-renin ratio, adrenal tumour, renin–angiotensin–aldosterone system

Oral Presentation Award Winners



ORAL PRESENTATION WINNERS

RESEARCH AND AUDIT

1ST Place



OP-RP 01 | Cardiovascular Risk Misclassification with Non-Fasting Lipid Testing: A Paired Study in a Sri Lankan Population

Dr. Kavindya Fernando

Registrar in Chemical Pathology,
Medical Research Institute, Colombo, Sri Lanka
Lecturer (Probationary), Department of Biochemistry and Clinical Chemistry, Faculty of Medicine, University of Kelaniya, Sri Lanka

2ND Place



OP-RP 02 | Derivation of a Laboratory-Specific Corrected Calcium Equation and Comparison with Payne's Equation in Hospitalized Patients at the Teaching Hospital, Jaffna

Mr AGS Lakmin

MLS Graduate
University of Jaffna

3RD Place



OP-AR 01 | Sustainability Audit in Biochemistry and Molecular Biology Research Laboratories: Opportunities for Carbon Reduction in a Sri Lankan Academic Setting

Ms JATS Jayaweera

Postgraduate Student (MSc. Biochemistry and Molecular Biology)
Department of Biochemistry and Molecular Biology, Faculty of Medicine, University of Colombo

ORAL PRESENTATION WINNERS

CASE REPORTS AND CASE SERIES

1ST Place



OP CR 03 Beyond the Textbook: When Calcium Tells a Different Story—A Diagnostic Twist in Gitelman Syndrome

Dr M P Krishanthi

Postgraduate Trainee in Chemical Pathology
National Hospital, Sri Lanka

2ND Place



OP CR 05 The Renin Paradox In Cushing's Syndrome: Resolution Following Adrenal Tumour Resection

Dr R P A P Sumathipala

Registrar in Chemical Pathology
National Hospital, Kandy

3RD Place



OP CR 01 Biochemical Evaluation of Persistent Hypophosphatemia — Diagnostic Utility of Fibroblast Growth Factor-23 Measurement

Dr N U W Jayasuriya

Registrar in Chemical Pathology
National Hospital, Sri Lanka

Abstracts of Posters Research and Audits



ABSTRACTS OF POSTERS

RESEARCH AND AUDITS

- RP 01** Effectiveness of an Intervention to Improve Knowledge on the Preanalytical Phase in Clinical Chemistry in a Cohort of Pre-Intern Medical Graduates in Sri Lanka
- RP 02** Association of Carcinoembryonic Antigen, Gamma-Glutamyl Transferase, and Uric Acid with Disease Severity in a Cohort of Colorectal Cancer Patients at the National Cancer Institute, Maharagama, Sri Lanka
- RP 03** Analytical Agreement and Clinical Reliability of Gel-Derived Albumin Reported on Serum Protein Electrophoresis Compared with Bromocresol Green Measurement
- RP 04** Serum Ferritin as a Correlate of Proteinuria Burden among Anaemic Chronic Kidney Disease Patients: Findings from Medical and Nephrology Clinics, Teaching Hospital Jaffna
- RP 05** Can the Transtubular Potassium Gradient Replace 24-Hour Urinary Potassium in Hypokalaemia Evaluation? Evidence from a Single-Centre Diagnostic Performance Study
- AP 01** A National Survey on the Use of Cardiac Biomarkers in the Diagnosis of Myocardial Infarction in Sri Lanka

ABSTRACTS OF POSTERS

RESEARCH AND AUDITS

RP 01

Effectiveness of an Intervention to Improve Knowledge on the Preanalytical Phase in Clinical Chemistry in a Cohort of Pre-Intern Medical Graduates in Sri Lanka

Fernando SCJ¹, Fernando ISIP², Premadasa MGTW³, Seneviwickrama KLMD^{4,5}, Dilanthi HW⁶, Jayawardana RDP³, Vidanapathirana DM⁷

¹Regional Blood Centre, Base Hospital Mahaoya, Mahaoya, Sri Lanka

²Department of Paediatrics, Base Hospital Mahaoya, Mahaoya, Sri Lanka

³Department of Chemical Pathology, National Hospital Kandy, Kandy, Sri Lanka

⁴Department of Community Medicine, Faculty of Medical Sciences, University of Sri Jayewardenepura, Sri Lanka

⁵Centre for Cancer Research, University of Sri Jayewardenepura, Sri Lanka

⁶Department of Biochemistry and Molecular Biology, University of Colombo, Sri Lanka

⁷Department of Pathology, Faculty of Medical Sciences, University of Sri Jayewardenepura, Sri Lanka
maduri@sjp.ac.lk

Introduction

Errors in the preanalytical phase constitute the largest proportion of laboratory errors and can adversely affect diagnostic accuracy and patient safety. Intern doctors play a key role in this phase of clinical chemistry testing. However, limited reinforcement of clinical biochemistry knowledge during the transition from paraclinical to clinical training may lead to knowledge gaps regarding proper preanalytical procedures. This study aimed to evaluate the effectiveness of an educational intervention in improving knowledge of the preanalytical phase in clinical chemistry among pre-intern medical graduates in Sri Lanka.

Methods

A before-and-after interventional study was conducted among pre-intern medical graduates from eight medical faculties recognized by the University Grants Commission in Sri Lanka. A pre-tested, self-administered online questionnaire consisting of 15 questions, with a maximum score of 75, was used to assess knowledge of preanalytical procedures and was distributed via a digital platform. An educational webinar on minimizing preanalytical errors was conducted as the intervention. Post-test knowledge was assessed immediately after the intervention.

Data were analysed using SPSS, and the paired-sample t-test was used to compare changes in knowledge scores. The level of significance was set at $p < 0.05$.

Results

Among the 22 participants, 59% were female. The mean pre-test score was 49.6 (SD = 8.9), while the mean post-test score increased to 65.4 (SD = 7.3). The increase in mean knowledge score after the webinar was statistically significant: paired t-test, $t = 6.60$, $p < 0.001$. The mean increase was 15.75, with a large effect size: Cohen's $d = 1.41$.

ABSTRACTS OF POSTERS

RESEARCH AND AUDITS

Conclusion

Pre-intern medical graduates in Sri Lanka demonstrated suboptimal knowledge regarding the preanalytical phase of laboratory testing. A short, targeted educational webinar significantly improved their knowledge level. Integrating structured training on preanalytical procedures into undergraduate medical curricula may help reduce laboratory errors and improve patient safety.

Keywords

preanalytical phase, clinical chemistry, laboratory errors, medical education, Sri Lanka

ABSTRACTS OF POSTERS

RESEARCH AND AUDITS

RP 02

Association of Carcinoembryonic Antigen, Gamma-Glutamyl Transferase, and Uric Acid with Disease Severity in a Cohort of Colorectal Cancer Patients at the National Cancer Institute, Maharagama, Sri Lanka

Gunasekara RASR¹, Amarasekara MHK¹, Samarasinghe R¹, Siriwardhana ID²

¹National Cancer Institute, Maharagama, Sri Lanka

²Faculty of Medicine, University of Moratuwa, Sri Lanka

rasangi.gunasekara@gmail.com

Introduction

Colorectal cancer (CRC) represents a significant global health burden and is among the five most common cancers in Sri Lanka, with a rising incidence. The association of biochemical markers such as carcinoembryonic antigen (CEA), gamma-glutamyl transferase (GGT), and uric acid with different stages of CRC is valuable for assessing disease severity, thereby facilitating effective treatment and follow-up.

Methods

A hospital-based descriptive cross-sectional study was conducted among 100 patients with colorectal cancer from February 2021 to February 2023. Patients with renal insufficiency, defined as an estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m², heavy alcohol consumption, defined as >30 g of alcohol per week per person, gout, or cirrhosis were excluded.

Pretreatment serum levels of CEA, GGT, and uric acid were measured. CRC staging from I to IV was determined using the TNM classification based on biopsy and imaging findings. Data were analysed using SPSS version 21, with a two-sided p-value <0.05 considered statistically significant. Associations between biomarkers and CRC stages were assessed using one-way ANOVA, while correlations between CEA, GGT, and uric acid were evaluated using Pearson's correlation.

Results

Serum CEA and GGT levels were significantly associated with CRC stages I–IV, with p-values of 0.045 and 0.003, respectively. GGT levels were elevated in metastatic CRC, stage IV, compared with non-metastatic CRC, stages I–III. CEA levels were significantly associated with GGT levels, with a strong positive correlation: $r = 0.925$, $p < 0.001$. Uric acid did not show a significant association with the CRC stage.

Conclusion

In this study, serum GGT levels showed a progressive increase across CRC stages I–IV and were significantly associated with disease severity. CEA levels were also significantly associated with CRC stages. These findings suggest that GGT may be a useful marker for assessing disease severity in CRC. However, large-scale studies are required to validate these observations.

Keywords

colorectal cancer stages, CEA, GGT, uric acid

ABSTRACTS OF POSTERS

RESEARCH AND AUDITS

RP 03

Analytical Agreement and Clinical Reliability of Gel-Derived Albumin Reported on Serum Protein Electrophoresis Compared with Bromocresol Green Measurement

Fernando K¹, Harshanee T¹, Rathnayaka C¹, Mohideen MM¹, Dahanayaka W¹, Herath T¹

¹Department of Biochemistry, Medical Research Institute, Borella, Sri Lanka

kavindyamjn14@gmail.com

Introduction

Serum protein electrophoresis (SPEP) reports routinely provide albumin and other protein fractions derived by densitometric analysis using total protein values. In clinical practice, these reported albumin values may be interpreted as equivalent to routinely measured serum albumin. However, as these values are calculated rather than directly measured, their analytical agreement and clinical reliability remain insufficiently evaluated. This study evaluated the agreement between gel-derived albumin reported on SPEP and serum albumin measured by the bromocresol green (BCG)

Methods

A retrospective analytical study was performed on 158 serum samples. The albumin fraction (%) obtained from gel-based SPEP was converted to absolute concentration using biuret total protein. Serum albumin was independently measured using the BCG assay. Agreement was evaluated using Pearson's correlation and linear regression. Proportional bias was assessed by correlating the inter-method difference with mean albumin values. Clinical acceptability was defined as an absolute inter-method difference within ± 5 g/L.

Results

Mean albumin concentrations were comparable between the BCG and gel-derived methods: 35.45 ± 6.61 g/L and 35.72 ± 7.61 g/L, respectively. A very strong correlation was observed: $r = 0.956$, $p < 0.001$; $R^2 = 0.914$. Bland-Altman analysis demonstrated minimal bias of -0.28 g/L, with narrow limits of agreement from -4.83 to $+4.28$ g/L. More than 97% of measurements fell within clinically acceptable limits.

Mild proportional bias was present: $r = -0.435$, $p < 0.001$, with slightly greater negative deviation at higher albumin concentrations. Clinically significant disagreement occurred in only 2.5% of samples.

Conclusion

Gel-derived albumin demonstrated excellent analytical agreement and high clinical reliability compared with BCG-measured albumin, with minimal systematic bias and a very low rate of clinically significant disagreement. These findings suggest that SPEP-reported albumin may provide clinically useful estimates in appropriate laboratory settings, while recognising methodological differences between derived and directly measured albumin values. The retrospective, single-centre design may limit broader generalisability.

Keywords

serum albumin, gel electrophoresis, method agreement, clinical reliability, Bland-Altman analysis

ABSTRACTS OF POSTERS

RESEARCH AND AUDITS

RP 04

Serum Ferritin as a Correlate of Proteinuria Burden among Anaemic Chronic Kidney Disease Patients: Findings from Medical and Nephrology Clinics, Teaching Hospital Jaffna

Thiruselvanathan S¹, Mayilvaganam K³, Jegatheesar K³, Thangarajah BR², Arasaratnam V¹

¹Department of Biochemistry, Faculty of Medicine, University of Jaffna, Sri Lanka

²Department of Medicine, Faculty of Medicine, University of Jaffna, Sri Lanka

³Department of Medical Laboratory Sciences, Faculty of Allied Health Sciences, University of Jaffna, Sri Lanka

thirusangavi2016@gmail.com

Introduction

Chronic kidney disease (CKD) is often diagnosed at later stages due to its slow progression. Anaemia is common among patients with CKD and may include iron deficiency. Serum ferritin acts as a biomarker of iron stores, while proteinuria is a marker of CKD severity. The relationship between the degree of proteinuria and serum ferritin remains poorly characterized in the Sri Lankan CKD population. This study evaluated the association between serum ferritin levels and the degree of proteinuria in anaemic patients with CKD.

Methodology

This descriptive cross-sectional study recruited 137 patients with CKD by convenience sampling from the nephrology clinic (n = 123) and medical clinic (n = 14) at Teaching Hospital, Jaffna. Haemoglobin, measured by the cyanmethaemoglobin method, serum ferritin, measured by immunoassay, and creatinine levels were measured. CKD stages were determined using estimated glomerular filtration rate (eGFR) among anaemic patients. Urine protein-creatinine ratio (UPCR) values were obtained from clinic reports. Data were analysed using SPSS software.

Results

The prevalence of anaemia among patients from the nephrology and medical clinics was 44.7% (n = 55) and 78.6% (n = 11), respectively. The two groups had similar median serum ferritin values: 76.98 ng/mL, IQR 39.77–173.50, and 67.84 ng/mL, IQR 22.38–163.88, respectively; Mann–Whitney U = 326.0, p = 0.692.

Among the 66 anaemic patients, nephrotic-range proteinuria was the most prevalent category, observed in 27 patients (40.9%). The mean ranks for serum ferritin increased across the different degrees of proteinuria, although this was not statistically significant: Kruskal–Wallis H = 8.947, p = 0.062. Spearman's correlation showed a significant positive association between UPCR and serum ferritin levels: r = 0.359, p = 0.003.

CKD Stage 3a and Stage 4 had higher mean ranks for serum ferritin: 40.23 and 45.18, respectively; Kruskal–Wallis H = 20.646, p < 0.001

Conclusion

A significant positive correlation between UPCR and serum ferritin levels highlighted that serum ferritin is positively associated with proteinuria burden among patients with CKD.

Keywords

anaemia, chronic kidney disease, serum ferritin, proteinuria

ABSTRACTS OF POSTERS

RESEARCH AND AUDITS

RP 05

Can the Transtubular Potassium Gradient Replace 24-Hour Urinary Potassium in Hypokalaemia Evaluation? Evidence from a Single-Centre Diagnostic Performance Study

Siddiqa OMOM¹, Arooran T¹, Liyanagunawardana JE¹, Prashanthan S¹, Krishanthi MP¹, Weerakkody WAAT¹, Katulanda G¹

¹Department of Chemical Pathology, National Hospital of Sri Lanka

siddiqa17@gmail.com

Background

Accurate differentiation between renal and extrarenal potassium loss is crucial in the evaluation of hypokalaemia. Twenty-four-hour urinary potassium excretion (24UKE) is considered the standard method for assessing renal potassium loss (RKL); however, it is time-consuming and highly dependent on complete and accurate urine collection. The transtubular potassium gradient (TTKG), calculated from a spot urine sample, offers a simpler and more convenient alternative. This study aimed to evaluate the correlation, diagnostic performance, and agreement between TTKG and 24UKE in patients investigated for hypokalaemia.

Methodology

A retrospective analytical study was conducted at the NHSL Biochemistry Laboratory from April to September 2025. Thirty patients referred for hypokalaemia evaluation were included. Patients with incomplete 24-hour urine collections, chronic kidney disease, or serum osmolality exceeding urine osmolality were excluded. Statistical analysis was performed using SPSS 2025.

Results

Patients were aged 16–66 years, with a female predominance of 60%. Both TTKG and 24UKE demonstrated positive skewness, which was more pronounced for TTKG, indicating higher values in a subset of patients. Therefore, due to the skewed distribution of both variables, correlation was assessed using Spearman's rank correlation coefficient.

The prevalence of RKL, based on 24UKE, was 50.0% in this study population. TTKG showed a moderately strong positive monotonic correlation with 24UKE (Spearman's $\rho = 0.75$, $p < 0.001$).

Using 24UKE as the reference method, the diagnostic performance of TTKG for detecting renal potassium loss was assessed with 95% confidence intervals. TTKG demonstrated a sensitivity of 80.0% (95% CI: 54.8–93.0%) and a specificity of 73.3% (95% CI: 48.0–89.1%). The positive predictive value was 75.0% (95% CI: 50.5–89.8%), while the negative predictive value was 78.6% (95% CI: 52.4–92.4%). The positive likelihood ratio was 3.00 (95% CI: 1.25–7.21), and the negative likelihood ratio was 0.27 (95% CI: 0.09–0.78).

Agreement analysis demonstrated moderate concordance between TTKG and 24UKE classifications ($\kappa = 0.53$, $p = 0.003$). Chi-square analysis also showed a statistically significant association between the two methods ($\chi^2 = 8.571$, $p = 0.003$).

Conclusion

TTKG showed a moderately strong correlation and moderate agreement with 24UKE in hypokalaemia evaluation. Although these findings do not establish complete interchangeability, TTKG may be considered a practical alternative or adjunct to 24UKE assessment, particularly when 24-hour urine collection is difficult, provided results are interpreted within the clinical and biochemical context.

Keywords

hypokalaemia, TTKG, 24-hour urinary potassium excretion, renal potassium loss

ABSTRACTS OF POSTERS

RESEARCH AND AUDITS

AP 01

A National Survey on the Use of Cardiac Biomarkers in the Diagnosis of Myocardial Infarction in Sri Lanka

Abeynayake GPN, Abeywickrama PDM, De Silva YSS, Wijethilaka LPCG, Gunasekara DGCN
Teaching Hospital Kurunegala, Kurunegala, North Western Province, 60000, Sri Lanka
gawri_abey@yahoo.com

Introduction:

The International Federation of Clinical Chemistry and Laboratory Medicine and the Association for Diagnostics and Laboratory Medicine recommend the use of high-sensitivity cardiac troponin assays in the diagnosis of myocardial infarction, while the European Society of Cardiology recommends their use in rapid diagnostic algorithms. This survey was conducted to assess the use of cardiac biomarkers in the routine diagnosis of myocardial infarction in Sri Lanka.

Methods:

An email containing a link to a Google Form was distributed to 89 biochemistry laboratories in government hospitals in Sri Lanka through the Department of Clinical Biochemistry, Teaching Hospital Kurunegala. The questionnaire consisted of 30 questions developed by the EFLM TG-CM (European Federation of Clinical Chemistry and Laboratory Medicine Task Group on Cardiac Markers), with minor modifications made prior to distribution. The questions were related to cardiac marker testing, analytical methods, decision limits, reporting units, and the use of diagnostic pathways. The data were extracted into an Excel sheet and analysed.

Results:

Seventy-four responses were received, giving a response rate of 83%. Responses were obtained from 14 teaching hospitals, 20 district general hospitals, 38 base hospitals, and 2 special institutions. The preferred cardiac biomarker was troponin I, and none of the responding laboratories offered troponin T. Nearly 40%, 15%, and 12% of laboratories offered creatine kinase, creatine kinase-MB isoenzyme, and myoglobin, respectively, as part of their cardiac biomarker profile. The majority of laboratories (65%) measured high-sensitivity cardiac troponin, while 16% used contemporary troponin assays. Approximately one-third of laboratories (30%) used point-of-care troponin testing. Among respondents using high-sensitivity cardiac troponin, 40% reported results in ng/L, while 33% used sex-specific cut-offs. Only a few hospitals (20%) used documented diagnostic pathways.

Conclusion:

High-sensitivity cardiac troponin is widely used in Sri Lanka. Education of laboratory professionals and clinicians is needed to improve the appropriate use of high-sensitivity cardiac troponin and to maximise its clinical benefits. However, as responses were received from different categories of staff, including consultants, trainees, medical officers, and medical laboratory technologists, the findings may have been influenced by individual understanding of cardiac biomarker testing.

Keywords:

national survey, cardiac biomarkers, troponin

Abstracts of Posters Case Reports and Case Series



ABSTRACTS OF POSTERS

CASE REPORTS AND CASE SERIES

- CR 01** Classic SIADH Superimposed on a Reset Osmostat: Diagnostic Value of the Water-Load Test in Severe Hyponatremia - A Case Report
- CR 02** Refractory Hyponatremia in Pregnancy: A Case Report of Acute Intermittent Porphyria
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- CR 16** Apparent Triclonal Pattern in IgA κ Multiple Myeloma: Diagnostic Challenges in Interpreting Complex Monoclonal Protein Profiles
- CR 17** Unexplained Vertigo Leading to the Diagnosis of Possible Gitelman Syndrome: a Case Report
- CR 18** Role of Biochemical Investigations in Diagnosing Differentiation Syndrome (ATRA Syndrome): A Case Report
- CR 20** Biochemical Pattern Recognition of Cisplatin-Induced Renal Tubular Electrolyte Wasting Using Fractional Excretion Analysis in Pancreato-Biliary Adenocarcinoma: A Case Report
- CR 21** A Case Report of Unexpected Monoclonal Signature in a Young Adult with Extensive Lytic Bone Disease: A Laboratory Perspective of Plasma Cell Neoplasia
- CR 23** A Rare Clinical Manifestation of Hyperthyroidism: Thyrotoxic Periodic Paralysis
- CR 24** Immunoglobulin Light Chain(AL) Amyloidosis Presenting with Renal Involvement: Diagnostic and Monitoring Utility of Serum Free Light Chain Analysis
- CR 25** Biochemically silent pheochromocytoma in a child: A diagnostic challenge
- CR 26** Alkaptonuria: Analytical Interference in Urine Protein Assays
- CR 28** Chemical Pathology–Driven Differentiation of Amyloidosis in End-Stage Renal Disease Diagnostic Pitfalls of Serum Free Light Chain Interpretation in Dialysis Patients
- CR 29** A Case of Citrullinemia Type I in an Infant with Hyperammonemia, Recurrent Convulsions and Dystonia

ABSTRACTS OF POSTERS

CASE REPORTS AND CASE SERIES

CR 01

Classic SIADH Superimposed on a Reset Osmostat: Diagnostic Value of the Water-Load Test in Severe Hyponatraemia – A Case Report

Basnayake BMTK¹, Siddiq OMOM¹, Arooran T¹, Pradeepa BGD¹, Thushiyanthi N¹, Katulanda GW¹, Karunatilake SH²

¹Department of Chemical Pathology, National Hospital of Sri Lanka

²Medical Unit, National Hospital of Sri Lanka

kanchanabasnayake@gmail.com

Introduction

Classic syndrome of inappropriate antidiuresis is characterised by unregulated antidiuretic hormone secretion, leading to impaired free water excretion and hyponatraemia. In contrast, a reset osmostat is a condition in which the osmotic threshold for antidiuretic hormone release is lowered but remains physiologically regulated, preserving the ability to excrete a water load. This is a case of acute SIADH in the background of a reset osmostat, verified through detailed biochemical evaluation and dynamic testing.

Case presentation

A 59-year-old man with a history of pulmonary tuberculosis complicated by post-tuberculous bronchiectasis and cavitary pneumonia, with reinfection and on anti-tuberculous therapy, presented with vomiting, chest pain, unsteadiness, and a generalised convulsion. Examination revealed herpes zoster infection over the left chest wall.

Investigations showed severe hyponatraemia with serum sodium of 103 mmol/L, low serum osmolality of 219 mOsm/kg, inappropriately elevated urine osmolality of 359 mOsm/kg, and urine sodium of 71 mmol/L. Thyroid, adrenal, renal, and liver function tests were normal, and the patient was clinically euvolaemic. Fractional excretion of uric acid was elevated at 20.9%, supporting classic SIADH. Hyponatraemia improved with fluid restriction. Two weeks later, serum sodium was 130 mmol/L, with serum osmolality of 270 mOsm/kg and normalised fractional excretion of uric acid of 12%.

Persistent mild hyponatraemia prompted a standardised water-load test of 20 mL/kg. Excretion of 72.5% of the water load within four hours, with appropriate urine dilution, indicated preserved free water clearance, which was more suggestive of a reset osmostat than persistent classic SIADH.

Discussion

Classic SIADH was likely triggered by severe pain due to herpes zoster infection and anti-tuberculous drugs. The reset osmostat may be attributed to repeated tuberculosis infection. Routine biochemical tests, in combination with the water-load test, identified acute-on-chronic hyponatraemia and allowed appropriate management.

Keywords

reset osmostat, water-load test, classic SIADH

ABSTRACTS OF POSTERS

CASE REPORTS AND CASE SERIES

CR 02

Refractory Hyponatraemia in Pregnancy: A Case Report of Acute Intermittent Porphyrria

Suraweera SADA¹, Piyasiri KHDS¹, Jayasekara LMDT¹, Katulanda GW¹

¹Department of Chemical Pathology, National Hospital of Sri Lanka

anudyasuraweera@gmail.com

Introduction

Acute intermittent porphyria is a rare metabolic disorder that can present with complex clinical features. This case illustrates the diagnostic challenge of acute intermittent porphyria presenting during pregnancy with refractory hyponatraemia.

Case presentation

A 24-year-old woman, at 28 weeks of gestation, presented with acute-onset generalised body weakness, hyperemesis, and hyponatraemia. Further investigations revealed refractory hyponatraemia with low serum osmolality. A diagnostic possibility of transient tubulopathy in pregnancy was suspected, and postpartum evaluation was planned.

Five months after delivery, she presented again with excessive vomiting and abdominal pain associated with menstruation. She also had a history of passing dark-coloured urine. The Hoesch test for urinary porphobilinogen was positive, followed by a positive total urine porphyrin scan by fluorometry. A diagnosis of acute intermittent porphyria was made, with identification of a pathogenic heterozygous mutation in the *HMBS* gene, c.517C>T; p.Arg173Ter.

Discussion

Although rare, porphyria should be considered an important differential diagnosis in patients presenting with acute neurological symptoms and hyponatraemia.

Keywords

porphyria, hyponatraemia

ABSTRACTS OF POSTERS

CASE REPORTS AND CASE SERIES

CR 03

When Clinical Suspicion Misleads: Decoding Hypokalaemia through Comprehensive Biochemical Evaluation – A Case Report

Siddiqua OMOM¹, Arooran T¹, Liyanagunawardana JE¹, Rajalingham T², Fernando A², Katulanda G¹

¹Department of Chemical Pathology, National Hospital of Sri Lanka

²Department of Neurology, National Hospital of Sri Lanka

siddiqua17@gmail.com

Introduction

Hypokalaemia is a common electrolyte abnormality with a broad differential diagnosis, encompassing gastrointestinal, renal, and endocrine causes. Differentiating among these aetiologies can be clinically challenging, particularly when initial impressions are misleading. A structured laboratory approach integrating serum electrolytes, urinary indices, acid-base analysis, and targeted hormonal evaluation is essential for accurate diagnosis. This case illustrates how systematic biochemical assessment can uncover an uncommon underlying disorder.

Case presentation

A 61-year-old woman with diabetes and hyperthyroidism underwent hysterectomy for endometrial cancer. On postoperative day one, she developed bilateral lower limb weakness and was found to have severe hypokalaemia of 1.8 mmol/L. Given the history of hypokalaemia and hyperthyroidism, thyrotoxic hypokalaemic periodic paralysis was initially suspected by the treating clinician. However, persistent hypokalaemia despite normalisation of thyroid function made this diagnosis unlikely.

She was referred to the biochemistry department due to this diagnostic dilemma. Further biochemical evaluation revealed hypomagnesaemia of 1.4 mg/dL, compensated metabolic alkalosis with pH 7.5 and HCO₃⁻ 5.5 mmol/L, increased renal potassium excretion with TTKG 7.24, increased urinary magnesium excretion of 37.4 mg/24 h, and hypocalciuria of 50.6 mg/24 h. Renal function and cortisol levels were normal. Plasma renin concentration was elevated at 50 mIU/L; reference range 4.4–46.1 mIU/L.

Based on persistent hypokalaemia with hypomagnesaemia, inappropriate renal potassium and magnesium wasting, hypocalciuria, metabolic alkalosis, elevated renin levels, and normal renal function, Gitelman syndrome was diagnosed in accordance with KDIGO recommendations.

Discussion

This case highlights the importance of a structured laboratory approach in evaluating complex electrolyte disturbances. Sequential interpretation of serum electrolytes, urinary indices, acid-base status, and hormonal parameters enabled accurate diagnosis without reliance on genetic testing. In resource-limited settings where molecular studies are not readily available, systematic biochemical evaluation remains essential for identifying rare inherited tubulopathies such as Gitelman syndrome.

Keywords

hypokalaemia, thyrotoxic hypokalaemic periodic paralysis, Gitelman syndrome

ABSTRACTS OF POSTERS

CASE REPORTS AND CASE SERIES

CR 05

Secondary Myelofibrosis, a Rare Presentation of Primary Hyperparathyroidism – A Case Report

Ekanayake CK¹, Sampath WGP¹, Samaratunge EPA¹, Premadasa MGTW¹, Athurugiriya AAID², Narampanawa WMC³, Gunathilake S², Antonypillai CN², Jayawardana RDP¹

¹Department of Chemical Pathology, National Hospital Kandy, Sri Lanka

²Department of Diabetes and Endocrinology, National Hospital Kandy, Sri Lanka

³Department of ENT, National Hospital Kandy, Sri Lanka

chandakushlan@gmail.com

Introduction

Hyperparathyroidism is a rare cause of secondary myelofibrosis. Excess PTH stimulates bone marrow fibroblasts and activates the release of inflammatory cytokines such as IL-6, leading to bone marrow fibrosis.

Case presentation

A 66-year-old female presented with shortness of breath and palpitations of two weeks' duration. She had a history of backache for one year. On examination, she was pale, dyspnoeic, and tachycardic. Investigations revealed pancytopenia. Bone marrow aspiration showed hypoplasia and megaloblastic changes, while myelofibrosis was noted in the trephine biopsy.

Incidentally, her calcium level was found to be elevated at 2.78 mmol/L; reference range 2.10–2.55 mmol/L. Further investigations revealed phosphate 0.81 mmol/L; reference range 0.81–1.45 mmol/L, ALP 453 U/L; reference range 30–120 U/L, vitamin D <4.0 ng/mL, PTH 2430.6 pg/mL; reference range 15–65 pg/mL, creatinine 87 µmol/L; reference range 65–120 µmol/L, and high 24-hour urinary calcium excretion of 6.83 mmol/day; reference >2.5 mmol/day.

The patient was supplemented with vitamin D. After an adequate interval, repeat PTH levels remained high, confirming the diagnosis of primary hyperparathyroidism. DXA scan demonstrated severe osteoporosis. Ultrasound scan of the neck revealed a lesion inferior to the lower pole of the left thyroid lobe, which was further supported by CECT neck findings suggestive of a parathyroid adenoma.

The patient underwent excision of the left parathyroid adenoma with intraoperative PTH assessment. Pre-operative, 10-minute postoperative, and 20-minute postoperative PTH values were 2534 pg/mL, 334 pg/mL, and 224 pg/mL, respectively, showing more than a 50% postoperative drop compared with the preoperative value and fulfilling the Miami criteria for successful removal of the parathyroid tumour. Histology confirmed a left parathyroid adenoma. In the immediate postoperative period, she developed hungry bone syndrome and recovered with adequate supplementation. She was later discharged once her anaemic symptoms and haemoglobin levels improved.

Discussion

Hyperparathyroidism should be considered as a possible cause of secondary myelofibrosis in elderly patients presenting with anaemia. Intraoperative PTH monitoring is a very useful tool for confirming successful removal of the lesion while awaiting histological confirmation.

Keywords

hyperparathyroidism, myelofibrosis, intraoperative PTH

ABSTRACTS OF POSTERS

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CR 06

From Radiological Haemangioma to a Probable Solitary Skull Plasmacytoma: Diagnostic Redirection through Laboratory Evaluation – A Case Report

Premadasa MGTW¹, Samarathunga EPA¹, Gunarathne KN², Jayawardana RDP¹

¹Department of Chemical Pathology, National Hospital Kandy, Sri Lanka

²Department of Haemato-Oncology, National Hospital Kandy, Sri Lanka

thiliniwathsala110@gmail.com

Introduction

Solitary plasmacytoma is a rare, localised plasma cell neoplasm that can radiologically mimic benign skull lesions such as haemangioma, leading to potential misdiagnosis and unnecessary surgical intervention.

Case report

A 44-year-old woman presented with a two-year history of a slowly enlarging, mildly painful scalp lump. MRI of the brain reported a skull vault haemangioma, while CT demonstrated a 7.5×5×4 cm expansile lesion causing complete parietal bone erosion with intracranial extension. Laboratory studies revealed a monoclonal IgG lambda paraprotein of 14 g/L on serum protein electrophoresis and serum immunofixation electrophoresis, markedly elevated serum lambda free light chains of 132 mg/L, an abnormal ratio of 0.20, and elevated -microglobulin of 2.199 mg/L. Bone marrow biopsy showed 2–3% plasma cells, with occasional CD138 positivity of <5%.

A probable diagnosis of solitary plasmacytoma of the skull was made based on laboratory findings. Histological confirmation of the skull lesion was not performed due to the high surgical risk associated with a large vascular neoplasm. The patient received localised external-beam radiotherapy over 25 fractions, resulting in a marked reduction in lump size, pain resolution, and a decrease in paraprotein level to 9 g/L at three-month follow-up.

Conclusion

Even in the absence of histological confirmation, integrated laboratory and imaging evaluation can establish a probable diagnosis of solitary skull plasmacytoma, guiding appropriate management and avoiding unnecessary surgical procedures.

Keywords

solitary skull plasmacytoma, haemangioma mimic, monoclonal gammopathy, serum protein electrophoresis

ABSTRACTS OF POSTERS

CASE REPORTS AND CASE SERIES

CR 08

Primary Adrenal Insufficiency Possibly Following Chikungunya Infection – A Case Report

Sampath WGP¹, Ekanayake CK¹, Samaratunge EPA¹, Premadasa MGTW¹, Jayawardana RDP¹, Gunathilake S², Antonypillai CN²

¹Department of Chemical Pathology, National Hospital Kandy, Sri Lanka

²Department of Diabetes and Endocrinology, National Hospital Kandy, Sri Lanka

Priyankarasampath1989@gmail.com

Introduction

Primary adrenal insufficiency is a rare but potentially life-threatening endocrine disorder. The most common causes include autoimmune adrenalitis and infections such as tuberculosis. Viral infections have rarely been implicated in the development of adrenal insufficiency through immune-mediated adrenal damage. We report a rare case of primary adrenal insufficiency possibly triggered by recent chikungunya infection.

Case Presentation

A 41-year-old previously well male presented with an eight-month history of progressive weight loss, fatigue, and postural dizziness following chikungunya infection nine months earlier. He had no history of tuberculosis, malignancy, trauma, autoimmune disease, chronic steroid use, or family history of adrenoleukodystrophy. Examination revealed marked hyperpigmentation of the buccal mucosa and palmar creases. His blood pressure was 90/60 mmHg with postural hypotension, and his body mass index was 18.5 kg/m².

His serum sodium was 129 mmol/L; reference range 137–145 mmol/L, and serum potassium was 5.5 mmol/L; reference range 3.5–5.1 mmol/L. Primary adrenal insufficiency was confirmed by a low 9 am serum cortisol level of 26.5 nmol/L; reference range 123–626 nmol/L, and an elevated adrenocorticotrophic hormone level of 1019 pg/mL; reference range 7.2–63.5 pg/mL. Chikungunya IgM antibody testing performed nine months earlier had been positive.

Other investigations revealed normal random plasma glucose, thyroid, and renal function. Normal chest radiography, negative Mantoux test, and normal abdominal ultrasound helped exclude tuberculosis and structural abnormalities. Very-long-chain fatty acid analysis was not performed due to unavailability.

In the absence of other identifiable causes, the clear temporal association with recent chikungunya infection suggested it as the most likely post-viral aetiology. The patient was commenced on hydrocortisone replacement therapy and showed significant clinical improvement following treatment.

Discussion and Conclusion

Primary adrenal insufficiency may rarely occur as a complication following chikungunya infection. This temporal association suggests possible immune-mediated molecular mimicry and delayed autoimmunity, direct cytopathic damage, microvascular endothelial injury, or bilateral adrenal infarction or haemorrhage, paralleling post-viral adrenal damage described with SARS-CoV-2 infection. A limitation of this case was the unavailability of very-long-chain fatty acid testing. Early recognition and prompt initiation of glucocorticoid replacement are essential to prevent life-threatening adrenal crisis.

Keywords

primary adrenal insufficiency, chikungunya, post-viral adrenal insufficiency, hydrocortisone

ABSTRACTS OF POSTERS

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CR 10

When Mood Stabilisation Disrupts Calcium Homeostasis: Lithium-Induced Hyperparathyroidism – A Case Report

Samaratunga EPA¹, Antonypillai CN², Premadasa MGTW¹, Ekanayaka CK¹, Sampath WGP¹, Basnayaka AM¹, Jayawardana RDP¹

¹Department of Chemical Pathology, National Hospital Kandy

²Diabetes and Endocrine Unit, National Hospital Kandy

m31359@pgim.cmb.ac.lk

Background

Lithium is used in the management of bipolar disorder and may result in hyperparathyroidism as a long-term complication. Lithium alters the calcium-sensing receptor set point, resulting in inappropriate PTH secretion and hypercalcaemia, which may persist for years or become permanent even after cessation of treatment.

Case Presentation

A 68-year-old male, a known patient with bipolar affective disorder and dementia, presented with altered behaviour of one week's duration. He had recent-onset polyuria and constipation. He had been on lithium carbonate, risperidone, quetiapine, and benzhexol for five years.

On examination, he was mildly dehydrated and had a GCS of 14/15. His serum lithium level was 1.18 mmol/L; therapeutic range 0.6–1.2 mmol/L, toxic range >1.5 mmol/L. Total calcium was 3.2 mmol/L; reference range 2.1–2.55 mmol/L. Phosphate was 0.98 mmol/L; reference range 0.81–1.45 mmol/L. PTH was 197.8 pg/mL; reference range 15–65 pg/mL. Serum creatinine was 78.9 μ mol/L; reference range 65–120 μ mol/L. ALP was 513.3 IU/L; reference range 30–120 IU/L. Serum 25-hydroxyvitamin D was 9.3 ng/mL, indicating deficiency. Following omission of lithium and adequate hydration, neurological symptoms and polyuria gradually improved.

Ultrasound scan revealed a focal lesion at the left lower pole of the thyroid gland, while there were no calculi or nephrocalcinosis in either kidney. DXA scan revealed osteoporosis, with a forearm T-score of –2.1.

The patient was started on vitamin D. After three months, serum corrected calcium was 2.82 mmol/L, phosphate was 1.0 mmol/L, PTH was 76.1 pg/mL, and 25-hydroxyvitamin D was 58.5 ng/mL, indicating sufficiency. The possibility of lithium-induced hyperparathyroidism was supported by hypocalciuria and a significant drop in PTH following vitamin D correction. The patient is being managed with adequate hydration, vitamin D, and intravenous zoledronic acid.

Discussion

Biochemical investigations play a major role in the assessment of lithium-related complications and hypercalcaemia. Lithium may cause parathyroid adenoma or hyperplasia, resulting in persistence of the disease even after cessation of treatment. Therefore, biochemical evaluation is required to assess the prognosis of the condition.

Key words

bipolar disorder, nephrocalcinosis, hyperparathyroidism

ABSTRACTS OF POSTERS

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CR 11

Insulin Autoimmune Syndrome Masquerading as Insulinoma: A Case Report with Biochemical and Radiological Discordance

Krishanthi MP¹, Liyanagunawardana JE¹, Arooran T¹, Siddiqi OMO¹, Prashanthan S¹, Sumaiya IF², Sumanatilleke M², Katulanda GW¹

¹Department of Chemical Pathology, National Hospital of Sri Lanka

²Diabetes and Endocrinology Unit 1, National Hospital of Sri Lanka

mpkrishanthi@gmail.com

Introduction

Insulin autoimmune syndrome, also known as Hirata disease, is a rare cause of endogenous hyperinsulinism leading to hypoglycaemia in the presence of high titres of insulin autoantibodies. It mimics insulinoma and poses a significant diagnostic challenge.

Case presentation

A 54-year-old woman presented with recurrent neuroglycopenic symptoms fulfilling Whipple's triad and denied the use of exogenous insulin or sulfonylurea. Symptomatic fasting hypoglycaemia with glucose of 29 mg/dL, in the presence of hyperinsulinaemia of $>1000 \mu\text{IU/mL}$ and elevated C-peptide of 12.8 ng/mL, suggested endogenous hyperinsulinism. An insulin-to-C-peptide molar ratio greater than 1 was more suggestive of insulin autoimmune syndrome than insulinoma.

However, imaging revealed a 4 mm lesion in the pancreatic tail, suggestive of insulinoma. Selective arterial calcium stimulation testing failed to localise an insulin-secreting lesion in any part of the pancreas, creating clinical and biochemical discordance. Polyethylene glycol precipitation testing was inconclusive. Due to high suspicion of insulin autoimmune syndrome, she was started on methylprednisolone. This led to rapid clinical stabilisation and a significant reduction in insulin levels to $80 \mu\text{IU/mL}$, confirming Hirata disease and a non-functional pancreatic mass.

Discussion

This case highlights the importance of biochemical investigations over imaging in endocrine tumour evaluation. When extreme insulin levels accompany non-localising selective arterial calcium stimulation testing, insulin autoimmune syndrome should be strongly suspected, even if polyethylene glycol precipitation testing is inconclusive. In resource-limited settings where insulin autoantibody testing is unavailable, a high index of clinical suspicion remains essential. The positive biochemical response to steroids supported a retrospective diagnosis and prevented unnecessary surgery for a non-functional pancreatic incidentaloma.

Key words

insulin autoimmune syndrome, hyperinsulinaemic hypoglycaemia, insulinoma, corticosteroids, Hirata disease

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CR 12

The Role of Pharmacogenetics in Personalised Oncology: A Case Report Emphasising the Role of *DPYD* Gene Analysis

Madurangi DWDD¹, De Silva DDS¹, Samarasinghe R¹, Bowatte A², Kularathnam GAM²

¹Department of Chemical Pathology, National Cancer Institute, Maharagama, Sri Lanka

²Genomics Laboratory, National Cancer Institute, Maharagama, Sri Lanka

dinidenipitiya@gmail.com

Introduction

Pre-treatment pharmacogenetic testing enables clinicians to identify at-risk patients and adjust therapeutic strategies accordingly, thereby enhancing treatment safety and supporting the implementation of personalised medicine. The *DPYD* gene encodes dihydropyrimidine dehydrogenase, the principal enzyme responsible for the metabolism of fluoropyrimidine chemotherapeutic agents such as 5-fluorouracil and capecitabine. Genetic variants that reduce enzyme activity may lead to impaired drug metabolism and severe, potentially life-threatening toxicity.

Case presentation

A 63-year-old female presented with an incidentally detected left breast lump of three months' duration. Tru-cut biopsy confirmed invasive breast carcinoma. Fine needle aspiration cytology of the left axillary lymph node demonstrated metastatic involvement. Contrast-enhanced computed tomography revealed multifocal carcinoma in the left breast with metastatic adenopathy in the left axilla. The patient was treated with neoadjuvant chemotherapy followed by surgical resection.

Following surgery, adjuvant chemotherapy including 5-fluorouracil was planned. As fluoropyrimidine-related toxicity is associated with variation in dihydropyrimidine dehydrogenase activity, *DPYD* gene analysis was performed prior to initiating therapy. Genetic analysis identified a heterozygous *DPYD* c.1905+1G>A; *DPYD**2A; rs3918290 splice-site mutation, associated with reduced dihydropyrimidine dehydrogenase enzyme activity and increased risk of fluoropyrimidine toxicity. Therefore, the clinician opted against starting 5-fluorouracil due to the increased risk of toxicity. The patient was started on endocrine therapy with anastrozole for tumour receptor positivity, which was well tolerated. She is currently tumour-free and being followed up as an outpatient.

Discussion

Identification of the heterozygous *DPYD* mutation enabled personalised treatment planning with appropriate treatment modification and careful monitoring, highlighting the importance of pharmacogenetic testing before initiating fluoropyrimidine-based chemotherapy. This case also emphasises the need to establish pharmacogenetic testing in Sri Lanka.

Key words:

pharmacogenetics, *DPYD* gene, fluoropyrimidine toxicity

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CR 13

Acute Disseminated Encephalomyelitis with Intrathecal Oligoclonal Bands in CSF Isoelectric Focusing

Madurangi DWDD¹, De Silva DDS¹, Abeygunawardena A², Wijerathne AID², Jogaprajahpan G², Samarasinghe R¹

¹Department of Chemical Pathology, National Cancer Institute, Maharagama, Sri Lanka

²Department of Chemical Pathology, Lady Ridgeway Hospital for Children, Sri Lanka

dinidenipitiya@gmail.com

Introduction:

Acute disseminated encephalomyelitis is an immune-mediated demyelinating disorder of the central nervous system, commonly occurring in children following infections. It typically presents with multifocal neurological deficits and encephalopathy. Early recognition is essential to initiate immunotherapy and improve outcomes.

Clinical Presentation:

A 4-year-and-1-month-old girl was admitted for evaluation of progressive right-sided limb weakness for one month, associated with sudden-onset speech loss and behavioural changes. This presentation was preceded by an upper respiratory tract infection. Neurological examination revealed reduced tone in the right upper and lower limbs, with mild weakness in the right upper limb. Reflexes were brisk in the lower limbs, with an upgoing plantar response on the right side. Cranial nerve examination was normal.

Laboratory investigations revealed normal biochemical and haematological findings. CSF analysis was unremarkable, with negative CSF culture. CSF isoelectric focusing revealed multiple oligoclonal bands. Contrast-enhanced CT brain demonstrated focal hypodensity in the left frontoparietal white matter, suggestive of focal cerebritis. MRI revealed multiple focal high T2/FLAIR lesions suggestive of acute disseminated encephalomyelitis.

The clinical picture of acute-onset multifocal neurological deficits following a recent infection, along with radiological findings and a monophasic course of illness, raised suspicion of acute disseminated encephalomyelitis. The patient was treated with intravenous immunoglobulin and high-dose corticosteroids, followed by oral prednisolone. She showed gradual clinical improvement, although a right hemiplegic gait persisted.

Discussion:

This case highlights the variable presentation of acute disseminated encephalomyelitis, including initial neuropsychiatric symptoms and speech regression, emphasising the importance of early diagnosis and prompt immunomodulatory therapy to optimise neurological recovery.

Although CSF oligoclonal bands are an important discriminator between multiple sclerosis and acute disseminated encephalomyelitis, they may occasionally be present in acute disseminated encephalomyelitis. Therefore, accurate diagnosis requires careful clinical and radiological evaluation.

Key words

acute disseminated encephalomyelitis, isoelectric focusing, neuropsychiatric illness

ABSTRACTS OF POSTERS

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CR 14

Falsely Elevated High-Sensitivity Troponin I Levels: Insight into Antibody Interference

Chinthanie KKS¹, Senevirathna DCL¹, Ushari RGPS¹, Dissanayake M¹

¹Department of Chemical Pathology, National Hospital Galle

kksathyachinthanie@gmail.com

Introduction

High-sensitivity troponin I is a key biomarker for myocardial injury and is widely used in the diagnosis of myocardial infarction. It is measured by non-competitive immunoassays on automated immunoassay analysers and point-of-care testing devices. We report a case of a 25-year-old man with carbimazole-induced perinuclear antineutrophil cytoplasmic antibody-positive vasculitis, who presented with acute epigastric pain and was evaluated for falsely elevated high-sensitivity troponin I.

Case presentation

The patient initially presented with thyrotoxicosis, with TSH <0.01 mIU/L, and was started on carbimazole. After a few weeks, he developed low-grade fever, generalised skin rash, lymphadenopathy, and small joint pain and swelling. Following extensive investigations through a multidisciplinary approach, he was diagnosed with pANCA-positive vasculitis. Carbimazole was stopped, and prednisolone was started.

A few days later, he presented with epigastric pain of one day's duration. His high-sensitivity troponin I level was markedly elevated at 863.9 ng/L, despite persistently normal ECG and 2D echocardiogram findings. He was referred to the Chemical Pathology team due to discordant troponin results. Serial dilutions of the sample showed non-linear recovery. Repeat high-sensitivity troponin I measurements on two different platforms gave normal results of <1.5 ng/L and 4.52 ng/L. Troponin I levels in the supernatant after sample pretreatment with polyethylene glycol 6000 showed low recovery of <40%. Upper gastrointestinal endoscopy revealed antral gastritis with a gastric ulcer.

Discussion

pANCA-positive vasculitis is a known but rare side effect of carbimazole. Antibody interference is a recognised pitfall in immunoassays. Investigation findings were compatible with antibody-mediated assay interference. Symptoms improved with treatment for vasculitis. During follow-up, high-sensitivity troponin I levels on the original analyser gradually reduced from 863.9 ng/L on day 1 to 453.3 ng/L on day 24 and 273.8 ng/L on day 61. Since the troponin interference improved with resolution of vasculitis, a causal relationship between autoantibodies involved in vasculitis and assay interference was suspected.

Key words

high-sensitivity troponin I, immunoassay interference, autoantibodies, carbimazole-induced vasculitis

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CR 15

Biclonal IgD Multiple Myeloma with Concurrent Lambda Light Chain Disease: A Rare Myeloma Variant

Heenatigala C, Madurangi DWDD, De Silva DDS, Kulasekera RCS, Samarasinghe R
Department of Chemical Pathology, National Cancer Institute, Maharagama, Sri Lanka
chathubhashini88@gmail.com

Introduction

Multiple myeloma is a plasma cell malignancy characterised by clonal proliferation of plasma cells and production of monoclonal immunoglobulins causing end-organ damage. Most cases involve immunoglobulin G or A paraproteins. In contrast, immunoglobulin D myeloma is rare, accounting for less than 2% of cases. Biclonal gammopathies, defined by two independent monoclonal proteins from separate plasma cell clones, are also uncommon, comprising about 1–2% of plasma cell dyscrasias. The coexistence of an intact immunoglobulin D heavy chain clone with a separate free light chain-producing clone is exceptionally rare and poses diagnostic challenges in routine laboratory evaluation.

Case Presentation

A 50-year-old patient presented with severe back pain and progressive renal impairment. Investigations showed anaemia, hypercalcaemia, markedly elevated ESR, high total protein with reversed albumin-globulin ratio, and prominent rouleaux formation on blood film. Serum protein electrophoresis revealed a distinct monoclonal band in the gamma region with a paraprotein concentration of 17.6 g/L.

Routine serum immunofixation electrophoresis using antisera against immunoglobulin G, A, M, kappa, and lambda demonstrated biclonal lambda light chain restriction without a corresponding heavy chain band. This discordance between electrophoresis and immunofixation raised suspicion of rare heavy chain myeloma. Extended immunofixation subsequently demonstrated a monoclonal immunoglobulin D band. Urine electrophoresis confirmed Bence Jones proteinuria, and serum free light chain analysis showed markedly elevated lambda free light chains. Bone marrow examination revealed approximately 60% clonal plasma cells, supporting a diagnosis of rare biclonal plasma cell myeloma consisting of immunoglobulin D lambda intact immunoglobulin with a concurrent lambda light chain clone. Due to cost constraints and limited availability, flow cytometry and molecular studies were not performed, and treatment was initiated based on available findings.

Discussion

This case highlights an exceptionally rare biclonal gammopathy where immunoglobulin D multiple myeloma coexists with an independent light chain-secreting clone. Recognition of discordant laboratory findings and the use of extended immunofixation are crucial to identify such rare variants and avoid misclassification.

Key words

immunoglobulin D myeloma, biclonal gammopathy, rare myeloma variant

ABSTRACTS OF POSTERS

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CR 16

Apparent Triclonal Pattern in IgA Multiple Myeloma: Diagnostic Challenges in Interpreting Complex Monoclonal Protein Profiles

De Silva DDS¹, Madurangi DWDD¹, Gunarathna KKSK², Samarasinghe R¹

¹National Cancer Institute, Maharagama

²District General Hospital, Trincomalee

dinudesilva88828@gmail.com

Introduction

Multiple myeloma typically presents as a single monoclonal immunoglobulin. However, complex electrophoretic patterns may mimic biclonal or triclonal gammopathy, posing diagnostic challenges in chemical pathology. Careful integration of serum protein electrophoresis (SPEP), immunofixation (IFE), and serum free light chain (sFLC) analysis is essential to accurately characterise clonal plasma cell disorders.

Case Presentation

A 55-year-old male presented with severe lower back pain. Full blood count showed pancytopenia with rouleaux formation. Biochemistry revealed hypoalbuminemia (29 g/L), low calcium (1.87 mmol/L), elevated LDH (480 U/L), and β 2-microglobulin of 6.2 mg/L.

SPEP demonstrated a dominant monoclonal band in the beta region (40.2 g/L) with two additional faint bands in the gamma regions (3.9 g/L and 0.9 g/L). IFE identified IgA κ restriction in the dominant and one minor band, while the third component corresponded to free κ light chains. sFLC analysis showed markedly elevated free κ (1290 mg/L), suppressed λ (6.3 mg/L), and an abnormal κ/λ ratio (204.8). Bone marrow examination revealed >90% κ -restricted CD138/CD56-positive plasma cells with diffuse infiltration on imaging.

Discussion

Although SPEP suggested a triclonal pattern, integrated laboratory assessment supported a single κ -restricted plasma cell clone with heterogeneous secretory products, including intact IgA κ and excess free κ light chains. Apparent multiple bands likely reflect monoclonal protein heterogeneity, polymerization, or fragmentation rather than true multiple independent clones. This case highlights the importance of combining electrophoretic, immunochemical, and sFLC data to avoid misclassification of complex monoclonal gammopathies.

Conclusion

Apparent multiband monoclonal patterns require cautious interpretation. Integrated chemical pathology evaluation is critical for accurate clonality assessment in multiple myeloma.

Keywords

multiple myeloma, triclonal gammopathy, free light chains, serum protein electrophoresis

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CR 17

Unexplained Vertigo Leading to the Diagnosis of Possible Gitelman Syndrome - A Case Report

Basnayake BMAM¹, Sumathipala RPAP¹, Dissanayake P², Gunasekara RASR³, Jayawardana RDP¹

¹Department of Chemical Pathology, National Hospital, Kandy, Sri Lanka

²Department of Diabetic and Endocrinology, District General Hospital, Pollonnaruwa, Sri Lanka

³Department of Chemical Pathology, District General Hospital, Pollonnaruwa, Sri Lanka

malinthabasnayake@gmail.com

Introduction

Gitelman syndrome is an autosomal recessive salt wasting tubulopathy caused by molecular defect in the sodium chloride cotransporter in the distal convoluted tubule. This is a case of a possible Gitelman syndrome of a female from a consanguineous family who was evaluated for vertigo and found to have hypokalemia and hypomagnesemia.

Case presentation

A 42 year old woman, product of a consanguineous marriage, presented with one-week history of vertigo. She was previously well and denied having any muscle weakness, cramps, salt craving, palpitations, polyuria, vomiting, growth delay, use of diuretics or laxatives, or eating disorders. Her blood pressure was normal, and physical examination was unremarkable. ECG findings were normal.

Laboratory investigations revealed hypokalemia-serum potassium 3.2 mmol/L(3.5–5.5) and hypomagnesemia-serum magnesium 0.4 mmol/L(0.7–1.0). Serum sodium, chloride, corrected calcium, creatinine and osmolality were within normal limits. Arterial blood gas demonstrated metabolic alkalosis.

Elevated urine sodium(114 mmol/L), potassium(76 mmol/L), magnesium(1.3 mmol/L) with low calcium(<0.2 mmol/L) levels were noted. Renal potassium wasting was confirmed with elevated fractional excretion of potassium of 22%, transtubular potassium gradient of 13.3 and urine potassium to creatinine ratio of 2.4 mmol/mmol. Both plasma renin activity[61.9 μ IU/mL;(4.2–45.6)] and aldosterone[40.7 ng/dL;(7–30)] were elevated. She was commenced on oral potassium and magnesium supplementation.

Discussion

Unlike classical Gitelman syndrome presenting in adolescence with cramps and weakness, our patient remained asymptomatic until adulthood. But the combination of hypokalemic metabolic alkalosis, hypomagnesemia, hypocalciuria, kaliuresis, hyperreninemic hyperaldosteronism with normal blood pressure indicated a most possible clinical diagnosis of Gitelman syndrome. Although Genetic testing confirms the diagnosis, it is not always required for a clinical management. Early recognition allows appropriate treatment and genetic counseling. This case highlights the importance of considering Gitelman syndrome in an adults presenting with unexplained hypokalemia and hypomagnesemia even in the absence of classical symptoms.

Key words

Gitelman syndrome, hypokalemia, hypomagnesemia, metabolic alkalosis, hypocalciuria.

ABSTRACTS OF POSTERS

CASE REPORTS AND CASE SERIES

CR 18

Role of Biochemical Investigations in Diagnosing Differentiation Syndrome (ATRA Syndrome) A Case Report

Leelarathna LHPS¹, Samarasinghe R¹

¹Department of Chemical Pathology, National Cancer Institute Maharagama, Sri Lanka
sandali508@gmail.com

Introduction

Differentiation syndrome (ATRA syndrome) is a life-threatening complication seen in acute promyelocytic leukemia patients during induction therapy with all-trans-retinoic acid and/or arsenic trioxide. The etiology is believed to be the massive cytokine release from differentiating promyelocytic leukocytes leading to a systemic inflammatory response. PETHEMA group criteria for diagnosis include fever, weight gain, peripheral edema, dyspnea with pulmonary infiltrates, pleuro-pericardial effusion, hypotension and acute renal failure.

Case presentation:

A 47-year-old newly diagnosed patient with acute promyelocytic leukemia developed shortness of breath and bleeding from venipuncture sites on the seventh day of induction therapy with all-trans-retinoic acid. Initial biochemical investigations showed hyponatremia (132mmol/L), elevated AST (224U/L), ALT (105U/L), elevated blood urea (89mg/dL), elevated LDH (2718U/L) and elevated CRP (170.5mg/L) levels. Serum creatinine, serum potassium and serum phosphate levels remained normal. Features of disseminated intravascular coagulation were also present together with high white cell counts ($37.75 \times 10^3/\mu\text{L}$).

Initial differential diagnoses were differentiation syndrome, tumor lysis syndrome and sepsis. Pulmonary infiltrates on chest X-ray, peripheral edema, elevated CRP, blood urea, AST, ALT and LDH favored the diagnosis of differentiation syndrome. Absence of hyperkalemia and hyperphosphatemia ruled out tumor lysis syndrome. Sepsis was excluded based on negative blood and urine cultures and persistent afebrile status throughout the period. Patient was started on intravenous dexamethasone and frusemide. Induction therapy was continued.

Discussion:

Biochemical investigations have a significant place in the diagnosis of differentiation syndrome and differentiating it from tumor lysis syndrome. Main biochemical abnormalities seen in differentiation syndrome are elevated CRP, blood urea, serum creatinine and LDH. AST and ALT are often elevated due to treatment induced hepatotoxicity. Serum creatinine and blood urea are elevated as a result of acute renal failure due to capillary leakage induced by cytokine release. LDH levels are elevated due to rapid cell turnover.

Key words:

differentiation syndrome, ATRA syndrome, acute promyelocytic leukemia

ABSTRACTS OF POSTERS

CASE REPORTS AND CASE SERIES

CR 20

Biochemical Pattern Recognition of Cisplatin-Induced Renal Tubular Electrolyte Wasting Using Fractional Excretion Analysis in Pancreato-Biliary Adenocarcinoma: A Case Report

Senevirathna DCL¹, Dissanayake M¹, Samarasinghe R²

¹Department of Chemical Pathology, National Hospital Galle, Sri Lanka.

²Department of Chemical Pathology, National Cancer Institute, Maharagama, Sri Lanka.

chamila626@gmail.com

Introduction

Cisplatin is a widely used chemotherapeutic agent whose major dose-limiting toxicity is nephrotoxicity resulting from renal tubular injury. This injury may cause significant electrolyte wasting involving magnesium, potassium, sodium and phosphate. In oncology patients, electrolyte disturbances have multiple potential causes including syndrome of inappropriate antidiuretic hormone secretion (SIADH), poor intake and gastrointestinal losses. Systematic biochemical evaluation is therefore essential to identify the underlying mechanism.

Case Presentation

A 56-year-old woman with seropositive rheumatoid arthritis presented with chronic cough and recurrent haemoptysis was diagnosed with differentiated adenocarcinoma involving the right lung with suspected pancreato-biliary origin. She was commenced on cisplatin and gemcitabine. Two months after treatment initiation, she revealed hyponatraemia (118–124 mmol/L), hypokalaemia (1.9–2.0 mmol/L), hypomagnesaemia (0.26 mmol/L), hypophosphataemia (0.62 mmol/L) and hypocalcaemia (1.98 mmol/L). Paired serum and urine electrolyte analysis demonstrated increased fractional excretion of sodium (4.39%), potassium (29.62%) and magnesium (11.43%), indicating significant renal electrolyte wasting with relative calcium sparing. Findings were consistent with cisplatin-induced renal tubular dysfunction (CIRTD). Replacement of electrolytes resulted in biochemical improvement, while chemotherapy was temporarily withheld.

Discussion

This case illustrates the characteristic biochemical pattern of CIRTD, where hypomagnesaemia plays a central pathophysiological role and contributes to refractory hypokalaemia and secondary hypocalcaemia. Recognition of multi-electrolyte renal wasting through fractional excretion analysis enabled differentiation from other causes of hyponatremias. This pattern contrasts with gastrointestinal losses or poor intake, where the kidney typically conserves electrolytes resulting in low urinary excretions. SIADH also excluded as hyponatraemia is the key feature with inappropriately elevated urinary sodium with no associated magnesium and potassium wasting. The prominent renal magnesium wasting observed in this patient is a recognized biochemical hallmark of cisplatin-induced tubular injury. The case highlights the critical role of chemical pathology in integrating biochemical data with clinical context to identify drug-induced tubulopathy and guide targeted electrolyte correction.

Key words

cisplatin nephrotoxicity, renal tubular dysfunction, electrolyte wasting, fractional excretion

ABSTRACTS OF POSTERS

CASE REPORTS AND CASE SERIES

CR 21

A case report of unexpected monoclonal signature in a young adult with extensive lytic bone disease: A laboratory perspective of plasma cell neoplasia

Senevirathna DCL¹, Dissanayake M¹, Samarasinghe R²

¹Department of Chemical Pathology, National Hospital Galle, Sri Lanka.

²Department of Chemical Pathology, National Cancer Institute, Maharagama, Sri Lanka.

chamila626@gmail.com

Introduction

Plasma cell dyscrasias predominantly affect older adults and are uncommon in the youth. Atypical biochemical and Pathological findings may obscure the diagnosis and delay appropriate management. We describe a diagnostically challenging case of plasma cell neoplasia in a 30-year-old male with discordant electrophoretic and immunochemical findings.

Clinical Presentation

A previously healthy 30-year-old male presented with a six-month history of intermittent thoracic pain, progressive back pain and episodic numbness in the left axilla. ESR was 77 mm/hr. Chest imaging showed anterior mediastinal widening, while contrast-enhanced CT demonstrated para and pre vertebral soft tissue masses with multiple expansile lytic lesions involving the spine, ribs, sternum, and left scapula. Differential diagnoses included plasma cell neoplasm with extramedullary disease, lymphoma, and malignant neurogenic tumors with metastases. Corrected calcium, serum creatinine, and hemoglobin were 2.6 mmol/L (2.15–2.57), 0.7 mg/dL, and 12.4 g/dL (13–17) respectively. Bone marrow aspiration and trephine biopsy were unremarkable. Serum protein electrophoresis and immunofixation demonstrated an IgA-kappa monoclonal band (7.4 g/L). Urine immunofixation revealed both intact kappa and free kappa light chain bands. Serum free light chain assay showed markedly elevated kappa chains (661 mg/L) with a kappa/lambda ratio of 68.9. Needle biopsy of the sternum and associated soft tissue demonstrated plasma cell proliferation with kappa light chain restriction, consistent with plasma cell neoplasia.

Discussion and Conclusion

Young age, extensive extramedullary involvement, normal marrow morphology, modest M-protein concentration, and discordance between serum and urine immunofixation findings created initial diagnostic uncertainty. Efficient urinary clearance of free kappa chains, explains the absence of a distinct serum free light chain band despite advanced disease. Histopathological confirmation of plasma cell proliferation, together with radiological and protein studies, supported plasma cell neoplasia and effectively excluded alternative differentials. This case highlights the importance of integrating biochemical, radiological, and histopathological findings when evaluating atypical plasma cell dyscrasias in young adults.

Key words

plasma cell neoplasia, multiple myeloma, free light chains, serum protein electrophoresis, immunofixation

ABSTRACTS OF POSTERS

CASE REPORTS AND CASE SERIES

CR 23

A Rare Clinical Manifestation of Hyperthyroidism: Thyrotoxic Periodic Paralysis

Weerakkody WAAT¹, Siddiqi OMO¹, Lyanarachchi G², Katulanda GW¹

¹Department of chemical pathology, National Hospital of Sri Lanka

²Medical Unit, National Hospital of Sri Lanka

amavi.tharuka@gmail.com

Introduction

Thyrotoxic periodic paralysis (TPP) is a rare manifestation of hyperthyroidism. Although uncommon globally, it is more frequently observed among the Asian males. TPP is characterized by hypokalemia induced acute reversible symmetrical muscle weakness.

Case Presentation

A 51-year-old man presented with bilateral lower limb weakness following heavy alcohol consumption. Laboratory investigations revealed severe hypokalemia (2.0 mmol/L), hypomagnesemia (1.5 mg/dL) suppressed Thyroid-stimulating hormone (TSH <0.008 mIU/L) with elevated free thyroxine (fT4 -5.46 ng/dL). Ultrasonography demonstrated diffuse enlargement of the thyroid gland with increased echogenicity. He was treated with intravenous potassium chloride and started on carbimazole. His muscle strength improved rapidly following treatment, with complete resolution of symptoms.

Discussion

Excess thyroid hormones activate Na⁺/K⁺-ATPase and leads to intracellular shift of potassium. This shift results in hypokalemia and subsequent muscle weakness. Common precipitating factors include high carbohydrate meals, strenuous exercise, stress, and alcohol consumption.

Prompt diagnosis is crucial, as delayed treatment may lead to life threatening complications such as cardiac arrhythmias.

Conclusion

Thyrotoxic periodic paralysis is a rare but reversible complication of hyperthyroidism. Early diagnosis and prompt treatment with potassium replacement and antithyroid therapy are essential to prevent fatal complications. This case highlights the importance of assessing serum potassium levels and thyroid function tests in patients presenting with acute neuromuscular weakness, even in the absence of overt signs of hyperthyroidism.

Keywords

thyrotoxic periodic paralysis; hyperthyroidism; hypokalemia

ABSTRACTS OF POSTERS

CASE REPORTS AND CASE SERIES

CR 24

Immunoglobulin Light Chain(AL) Amyloidosis Presenting with Renal Involvement: Diagnostic and Monitoring Utility of Serum Free Light Chain Analysis

Kulasekera RCS, Heenatigala C, De Silva DDS, Madurangi DWDD, Samarasinghe R
Department of Chemical Pathology, Apeksha Hospital, Maharagama, Sri Lanka
chamisulok@gmail.com

Introduction

Immunoglobulin light chain (AL) amyloidosis is a systemic plasma cell disorder characterized by extracellular deposition of misfolded monoclonal light chains forming amyloid fibrils that cause progressive organ dysfunction. Renal involvement is common, but early diagnosis remains challenging due to non-specific clinical manifestations and frequently low levels of circulating monoclonal protein. Sensitive laboratory investigations therefore play a critical role in detection and monitoring.

Case Presentation:

A 40-year-old female presented with a one-year history of backache and lower abdominal pain. Initial investigations revealed normocytic normochromic anemia (hemoglobin 10 g/dL) and markedly elevated erythrocyte sedimentation rate (105 mm/hour). Renal and liver function tests were within reference limits. However, 24-hour urine protein excretion was 2 g per day, indicating sub-nephrotic range proteinuria, while ultrasonography showed normal-sized kidneys.

Serum protein electrophoresis demonstrated a monoclonal band in the gamma region with a paraprotein concentration of 17.7 g/L. Immunofixation electrophoresis confirmed a monoclonal IgG lambda component, and urine immunofixation revealed monoclonal heavy chain fragments with lambda light chains. Serum free light chain (sFLC) analysis showed kappa 14 mg/L and lambda 126 mg/L with a markedly abnormal κ/λ ratio, indicating monoclonal lambda light chain excess. Bone marrow examination revealed 20% plasma cells.

Renal and lymph node biopsies demonstrated Congo-red–positive amyloid deposits with apple-green birefringence under polarized light, confirming amyloidosis. In the absence of CRAB (hemoglobin was 12 g/dL at the time of diagnosis) or myeloma defining events, findings were consistent with AL amyloidosis.

Discussion:

This case highlights the importance of integrating serum free light chain analysis with electrophoretic techniques in the diagnostic evaluation of unexplained proteinuria and suspected plasma cell disorders. sFLC measurement provides a sensitive tool for detecting monoclonal light chain production and plays a pivotal role in disease staging and therapeutic monitoring in AL amyloidosis.

Key words:

AL amyloidosis, proteinuria, serum free light chain assay

ABSTRACTS OF POSTERS

CASE REPORTS AND CASE SERIES

CR 25

Biochemically silent pheochromocytoma in a child: A diagnostic challenge.

Herath HMIU¹, Kiyamudeen F², Suntharesan J³, Jayawardana RDP¹

¹Department of Chemical Pathology, National Hospital, Kandy

²Department of Chemical Pathology, Sirimavo Bandaranayake Specialized Children's Hospital, Peradeniya

³Pediatric and Adolescent Diabetes and Endocrinology Unit, Sirimavo Bandaranayake Specialized Children's Hospital, Peradeniya

iru.herath@gmail.com

Introduction

Pheochromocytoma is a rare catecholamine-secreting tumor in children. Metanephrine measurement is highly sensitive for diagnosing pheochromocytoma, with plasma free metanephrines offering greater sensitivity than urinary metanephrines in high-risk patients. However, false-negative results may occur, particularly with 24-hour urinary assays. Imaging findings may also be non-specific, complicating diagnosis.

Case presentation

A 12-year-3-month-old boy was referred for evaluation of poorly controlled hypertension. He initially presented with headache, blurred vision, palpitations, excessive sweating, fatigue, and weight loss. Examination revealed papilloedema and severe hypertension.

Ultrasound scan of the abdomen showed a right suprarenal mass, raising suspicion of pheochromocytoma. He was started on antihypertensive therapy and referred to the Pediatric Endocrinology clinic for further evaluation.

Biochemical investigations, including renal function, electrolytes, calcium, thyroid-stimulating hormone, cortisol, testosterone, beta-hCG, alpha-fetoprotein, and aldosterone, were normal. Notably, 24-hour urinary metanephrines were below the cutoff value (149.9 µg/day; <350 µg/day). Plasma free metanephrine assay was unavailable. CT abdomen demonstrated a 4.8 × 4.8 × 3.8 cm right suprarenal mass with non-specific features. Despite inconclusive findings, clinical suspicion remained high. The patient underwent laparoscopic right adrenalectomy. Histology confirmed pheochromocytoma confined to the adrenal gland (PASS score 0). Post-operatively, blood pressure normalized and symptoms resolved.

Discussion

Normal urinary metanephrines do not exclude pheochromocytoma. As a screening test, they have lower sensitivity (93%) compared to plasma metanephrines (98%). Intermittent metanephrine secretion, normetanephrine secretion, tumor biology, and pre-analytical factors may contribute to false-negative results. Importantly, this case highlights that biochemical tests should not be interpreted in isolation. When clinical suspicion is strong, negative results should prompt reconsideration, repeat testing, or alternative diagnostic approaches where feasible.

Keywords

pheochromocytoma, urinary metanephrines, suprarenal mass, false-negative

ABSTRACTS OF POSTERS

CASE REPORTS AND CASE SERIES

CR 26

Alkaptonuria: Analytical Interference in Urine Protein Assays

Slegers NB¹, Hathagoda W², Majitha SI¹

¹Department of Chemical Pathology, Lady Ridgeway Hospital for Children

²Department of Paediatrics, University of Colombo

slegersnayoli@gmail.com

Introduction

Alkaptonuria is a rare autosomal recessive disorder caused by homogentisate 1,2-dioxygenase deficiency, leading to accumulation of homogentisic acid. Darkening of urine while standing, due to oxidation of Homogentisic acid is the well-known classic feature of Alkaptonuria. But there are rare other renal manifestations like renal stones, tubular dysfunction and chronic renal failure, where the monitoring of renal functions play a crucial role. This case illustrates how awareness of interference related to these monitoring tools are valuable to avoid misinterpretation results of patients with Alkaptonuria.

Case Presentation

A 2 years and 9month old, diagnosed patient with Alkaptonuria was underwent routine renal workup. Urine dipstick showed only a faint trace of albumin, but urine protein measured by the benzethonium chloride method was markedly elevated (189.9 mg/dL), with urine creatinine by Jaffe method at 5091 μ mol/L giving the protein to creatinine ratio (UPCR) of 373.01 mg/mmol denoting the nephrotic range proteinuria. But of patient didn't have any features of proteinuria. Because of the controversy in the results, urine protein was analyzed by pyrogallol red method. Result was 17.75 mg/dL, giving UPCR of 34.84 mg/mmol which is clinically compatible. With the suspicion of an interference urine protein was analysed by both methods in a sample from another patient who had a massive peak of Homogentisic acid in urine organic acid. Very high urine protein results in benzethonium chloride method and normal results in pyrogallol red method noted in second patient as well. Discrepancy of the test results with the clinical picture was explained by positive interference from homogentisic acid, documented by the particular assay manufacturer as a limitation from Homogentisic >0.6mmol/L.

Discussion

Elevated urine protein levels in patients with Alkaptonuria detected by the benzethonium chloride method are not clinically significant and are attributable to analytical interference from Homogentisic acid.

Keywords

alkaptonuria, homogentisic acid, interference, urine protein analysis, benzethonium chloride method

ABSTRACTS OF POSTERS

CASE REPORTS AND CASE SERIES

CR 28

Chemical Pathology–Driven Differentiation of Amyloidosis in End-Stage Renal Disease: Diagnostic Pitfalls of Serum Free Light Chain Interpretation in Dialysis Patients

De Silva DDS¹, Madurangi DWDD¹, Samarasinghe R¹

¹National Cancer Institute Maharagama, Sri Lanka

dinudesilva88828@gmail.com

Introduction

Differentiating amyloid light-chain (AL) amyloidosis from dialysis-related amyloidosis (DRA) in patients with end-stage renal disease (ESRD) remains diagnostically challenging due to overlapping clinical manifestations and altered serum free light chain (sFLC) kinetics in renal failure. This case highlights the critical role of integrated chemical pathology in resolving such diagnostic uncertainty.

Case Presentation

A 58-year-old male with ESRD secondary to hypertensive nephropathy on maintenance hemodialysis for 10 years presented with progressive dysphagia, macroglossia, and carpal tunnel syndrome. Laboratory investigations showed hemoglobin 10 g/dL and creatinine 596 $\mu\text{mol/L}$. Serum protein electrophoresis revealed biclonal gamma-region peaks. sFLC analysis demonstrated markedly elevated κ (9790 mg/L) with λ 64.2 mg/L, producing a significantly skewed κ/λ ratio of 152.8. Serum and urine immunofixation confirmed monoclonal free κ light chains. Imaging demonstrated a tongue mass and soft tissue lesions suspicious for malignancy. Congo red staining of tissue biopsy confirmed amyloid deposition. Bone marrow examination showed <10% plasma cells without myeloma-defining features.

Discussion

Interpretation of sFLC results in ESRD is complex, as reduced renal clearance typically elevates both κ and λ chains but does not usually produce extreme ratio distortion. The markedly abnormal κ/λ ratio and immunofixation pattern raised concern for an underlying κ -restricted plasma cell clone, suggesting an AL-associated process. However, long-term hemodialysis history and carpal tunnel syndrome supported dialysis-related amyloidosis as an important competing differential. Absence of amyloid typing prevented definitive subclassification, underscoring the necessity of integrating serum protein electrophoresis, immunofixation, and sFLC results with clinical context.

Conclusion

This case emphasizes the essential role of chemical pathology in differentiating AL amyloidosis from dialysis-related amyloidosis in ESRD, particularly when amyloid typing is unavailable, and highlights the importance of integrated laboratory-clinical interpretation to avoid diagnostic misclassification.

Key words

dialysis-related amyloidosis, monoclonal gammopathy, serum free light chains, end-stage renal disease

ABSTRACTS OF POSTERS

CASE REPORTS AND CASE SERIES

CR 29

Case report – A Case of Citrullinemia Type I in an Infant with Hyperammonemia, Recurrent Convulsions and Dystonia

Ushari RGPS¹, Slegers NB², Mohideen MM⁴, Hemika Y², Jayasena S², Jadavan N³, Silva ND³, Majitha SI²

¹Department of Chemical Pathology, National Hospital Galle

²Department of Chemical Pathology, Lady Ridgeway Hospital for Children, Sri Lanka

³Department of Paediatrics, Lady Ridgeway Hospital for Children, Sri Lanka

⁴Department of Chemical Pathology, Medical Research Institute, Colombo

prashanthiushari@gmail.com

Introduction

Citrullinemia is a rare inherited disorder of the urea cycle which causes hyperammonemia. It is divided into type I and type II. Type II is known as citrin deficiency. In type I mutations in the ASS gene cause deficiency in arginosuccinate synthetase enzyme. Citrullinemia can lead to life-threatening neurological manifestations. This is a possible case of citrullinemia type I in an infant.

Case Presentation

A four-month-old baby girl who is the second-born child of non-consanguineous parents with one healthy elder sibling was delivered via elective lower segment caesarian section at the POA of 38 weeks with a birth weight of 4 kg. The pregnancy was complicated with chronic diabetes mellitus. The baby presented with neonatal convulsions on the day five of life. Since then she is being evaluated for recurrent neonatal convulsions, poor feeding, marginal developmental delay, hepatomegaly, microcephaly and dystonia. Plasma ammonia was elevated [$178 \mu\text{mol/L}$ ($0 - 100$)] and plasma lactate was also high [2.91mmol/L ($0.5 - 2.22$)]. The VBG showed compensated respiratory acidosis. The EEG showed multifocal epilepsy and the CT brain revealed mild cerebral atrophy. Amino acid profile was done by Tandem mass spectrometry at an overseas lab and it revealed marked elevation of citrulline $848.66 \mu\text{M}$ ($3 - 75$) with an arginine level at the lower limit of the reference range indicating the possibility of citrullinemia type I.

Discussion

Citrullinemia type II can be differentiated from citrullinemia type I by normal or slightly increased arginine level in citrullinemia type II and it is reduced in citrullinemia type I. In urine organic acid analysis, orotic acid is detectable only in citrullinemia type I between these two disorders. Here urine organic acid profile and genetic testing were not performed due to limited resources. Early screening should be done in infants with hyperammonemia and recurrent seizures for urea cycle disorders.

Key Words

citrullinemia type I, arginosuccinate synthetase deficiency, hyperammonemia, urea cycle

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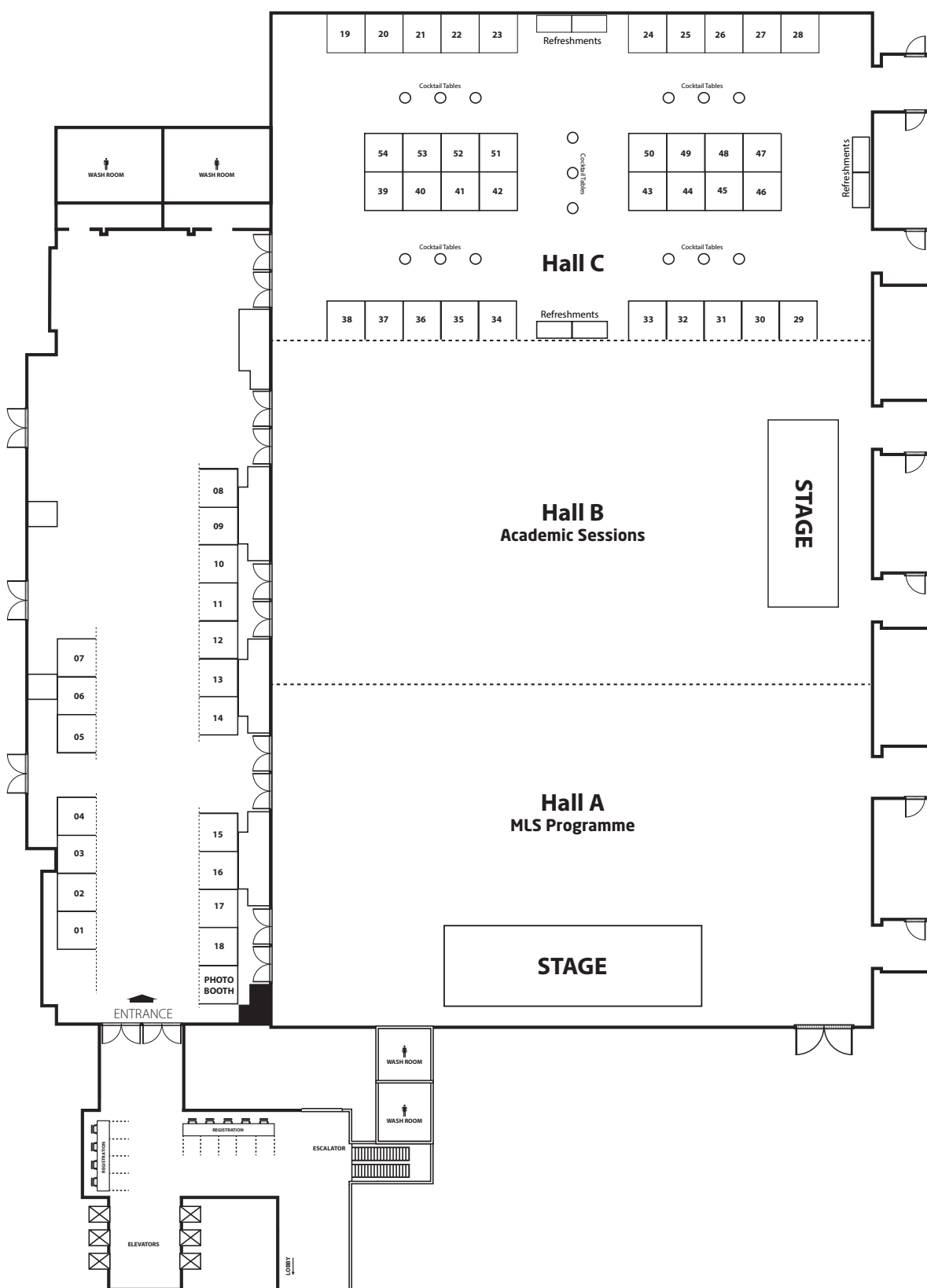
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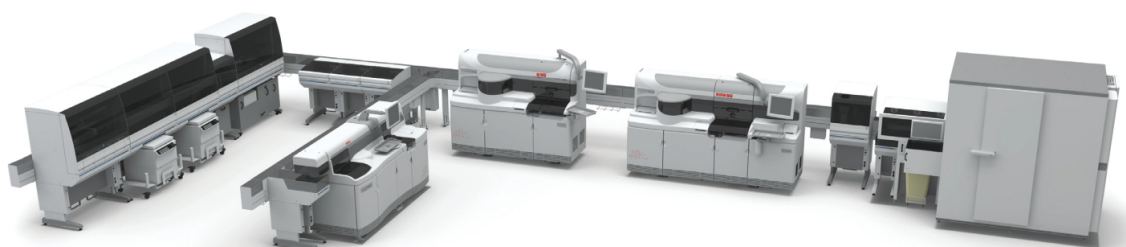
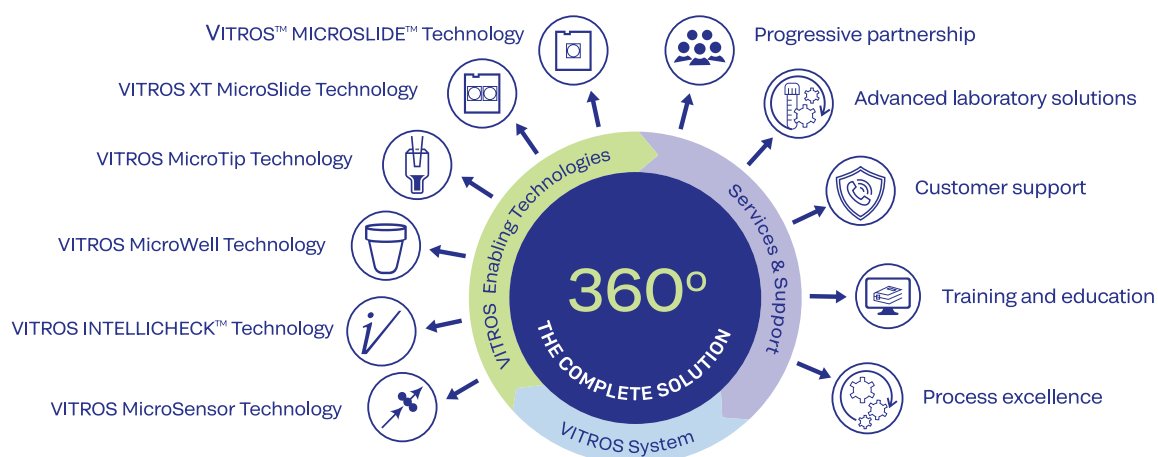
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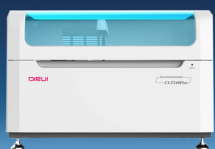
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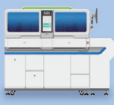
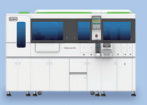
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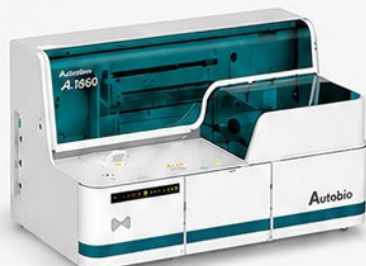


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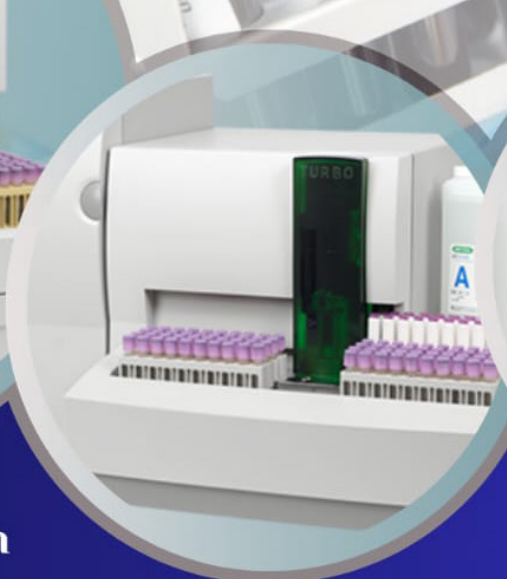


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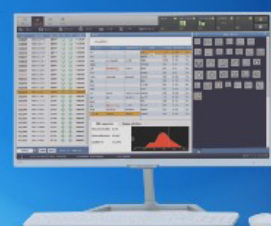
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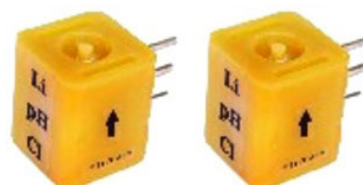
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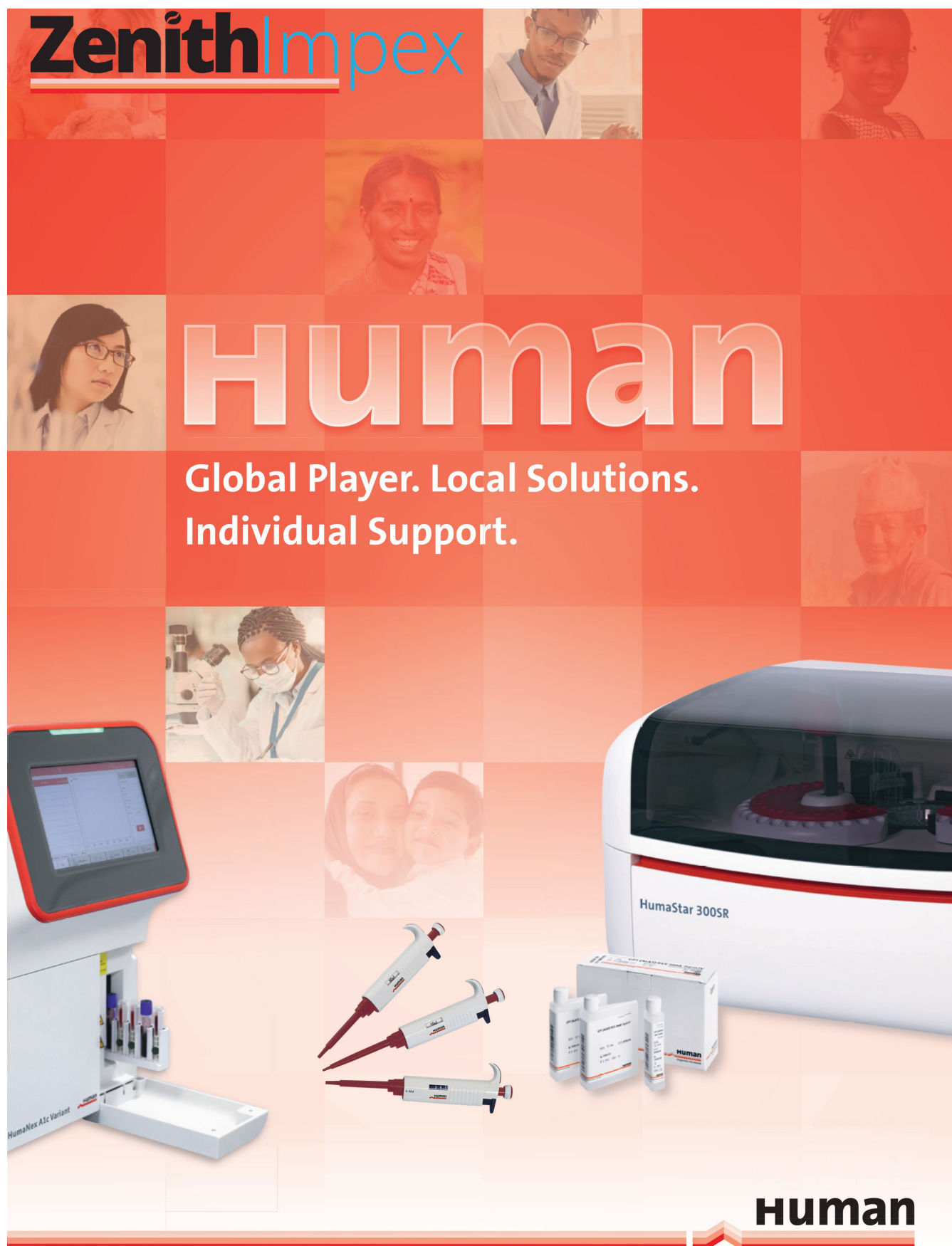
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Recent results from the Advantageous Predictors of Acute Coronary Syndromes Evaluation study (APACE) using the TriageTrue hsTnI Test suggests patients with suspected MI may be triaged within one hour using the concept of the current ESC hs-cTnT/Q1-h algorithm.^{1,2} The results of this algorithm demonstrated high overall efficacy:

- Nearly three-fourths of patients assigned to either the rule-in or rule-out category within one hour. (The remaining patients were moved to the observe zone).²
- 43% of patients triaged without the need for serial hs-cTnI sampling which was higher than hs-cTnT-Elecsys (25%) and hs-cTnI-Architect (22%).²

Performance of the Point-of-Care High Sensitivity Cardiac Troponin I TriageTrue Assay in Patients With Suspected Myocardial Infarction²

1,261 Patients

With Suspected Non-ST-Segment Elevation Myocardial Infarction (NSTEMI)

Point-of-Care High-Sensitivity Cardiac Troponin I Measured at 0 h and at 1 h

Triage by Single Cut-Offs

Direct Rule-Out	Direct Rule-In
At 0 h ≤ 3 ng/l	At 0 h ≥ 60 ng/l
45%	12%
NPV: 100% (99.4%-100%)	PPV: 76.8% (68.3%-83.6%)
Sens: 100% (98.0%-100%)	Spec: 97.2% (95.3%-98.0%)

Triage by Q1-Hour Algorithm

Rule-Out	Observe	Rule-In
At 0 h ≤ 5 ng/l AND Delta 1 h ≤ 2 ng/l	Others	At 0 h ≥ 60 ng/l OR Delta 1 h ≥ 8 ng/l
55%	26%	18%
NPV: 100% (98.8%-100%)		PPV: 76.8% (67.2%-84.7%)
Sens: 100% (98.9%-100%)	NSTEMI: 8%	Spec: 95.0% (92.5%-96.8%)

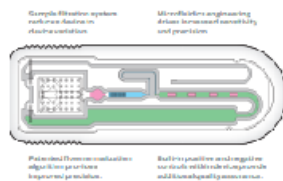
All-Cause Death of Patients Ruled-Out by the Q1-h-Algorithm
0% at 30 Days and 1.6% at 2 Years of Follow-up

² All chest pain onset < 6h before presentation to the emergency department.

The availability of TriageTrue hsTnI Test could allow extending the use of the hs-cTnI Q1-h-Algorithm to settings without a central laboratory including smaller hospitals and general practices when properly validated within these sites.²

True Innovation

High sensitivity rapid testing at the point of care is now possible with TriageTrue hsTnI Test. This innovative test delivers unprecedented sensitivity and precision when used with the Triage MeterPro.



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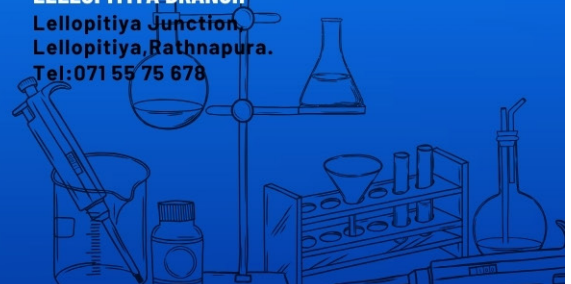
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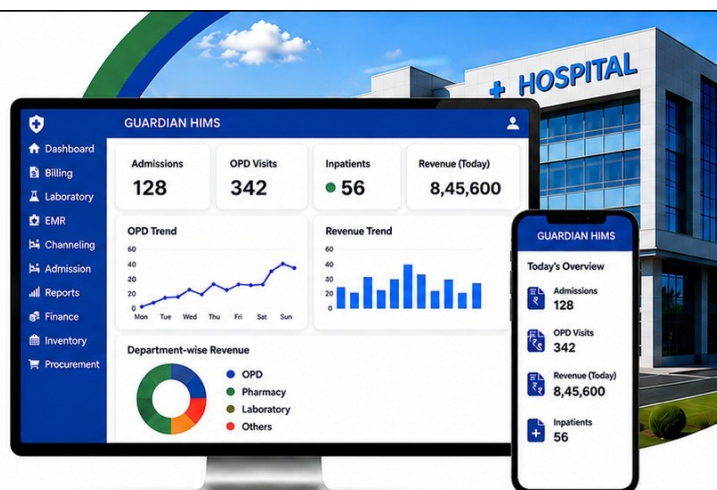
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