College of Chemical Pathologists of Sri Lanka

CCPSL NEWSLETTER

2025/2026

Issue 07

COVER STORY

10th Annual Academic Sessions (AAS) 2025
College of Chemical Pathologists of Sri Lanka
UNRAVELING CHALLENGES: QUALITY SAGA FOR EXCELLENCE

IN THIS ISSUE

Editors

Dr Rasangika Gunasekara

Dr Fasilaas Kiyamudeen

Message from the President (2025/2026)

Dr. Thushara Hewageegana
MBBS, Dip Path, MD (Chemical Pathology)
Consultant Chemical Pathologist
TH Anuradhapura
Sri Lanka

Dear Esteemed Members of the College of Chemical Pathologists of Sri Lanka (CCPSL),

We are proud to bring the 7th edition of the CCPSL newsletter to you. The newsletter of the CCPSL provides a salubrious platform for the members to share interesting cases, research findings and current updates in chemical pathology. I express my heartfelt gratitude to all contributors.

With warm regards,
Dr. HTN Hewageegana
President, College of Chemical Pathologists of Sri Lanka

Email: colchempath@gmail.com Tel: +94 (0) 76 704 4871 Website: ccpsrilanka.com

CCPSL NEWS

Induction of the 10th President of CCPSL and Inauguration Ceremony of Annual Academic Sessions of CCPSL 2025

The induction of the 10th President of the College of Chemical Pathologists of Sri Lanka (CCPSL) and the inauguration of the 10th Annual Academic Sessions for 2025 took place on the 10th July 2025 at the Monarch Imperial, Kotte, Sri Lanka.

The inauguration ceremony was highly successful, with numerous participants in attendance. The chief guest was Hon. Dr. Nalinda Jayatissa, Minister of Health and Mass Media.

Dr Thushara Hewageegana was inducted as the 10th President of CCPSL by the immediate Past President, Dr. Dulani Jayawardana. In his presidential address, Dr. Hewageegana highlighted the role of a Chemical Pathologist and the services provided in managing patients.

Dr. Abhaya Illeperuma, Consultant Histopathologist, was awarded the CCPSL Fellowship in recognition of his exceptional contribution to the field of pathology and his passionate services as a medical professional, eminent scholar and exemplary teacher.

At this prestigious event, two experienced Senior Medical Laboratory Technologists, Mrs. Wasanthamala Abeywickrama and Mr. Gamini Pathirana, were felicitated for their remarkable achievements and valuable contributions to the field of Medical Laboratory Technology.

In continuation of the awarding ceremony, the winners of the oral presentation, e-poster presentation, inter-medical faculty chemical pathology quiz competitions, and best publication by a chemical pathologist during the academic year of 2024 were also honoured by the President of CCPSL.

The prestigious CCPSL Oration for the fourth time titled “Partnership in Prevention: Unique Model to Promote Preventive Services in Community” was delivered by Prof. Devaki Nair, Consultant Chemical Pathologist, Speciality Lead for Clinical Biochemistry, Health Services Laboratories, London, United Kingdom.

The inauguration was concluded with a Sri Lankan cultural musical event and a grand reception.

Email: colchempath@gmail.com Tel: +94 (0) 76 704 4871 Website: ccpsrilanka.com

CCPSL NEWS

10th Annual Academic Sessions of CCPSL

The 10th Annual Academic Sessions of the College of Chemical Pathologists of Sri Lanka (CCPSL) were successfully conducted at the Monarch Imperial in Colombo on the 11th and 12th of July 2025. The event, held under the theme **"Unraveling Challenges: Quality Saga for Excellence"**, offered two days of fresh insights and expertise in chemical pathology and laboratory management.

An academic program and a Medical Laboratory Science (MLS) program were conducted simultaneously in the scientific program. The academic program included seven plenaries and six symposia covering a wide spectrum of important subjects and challenges in Chemical Pathology.

The 10th Annual Academic Sessions of CCPSL featured thirty-one resource professionals, including ten international faculty, who shared their expertise in the fields of renal, metabolic, lipids, endocrine, pharmacogenomics, calcium and bone, and laboratory management.

The event also served as an excellent platform for chemical pathology trainees and researchers to showcase their work and share their experiences. Out of 65 submitted abstracts, 12 were selected for oral presentations, while the remainder were displayed as e-posters. The oral presentation competition, judged by a distinguished panel, was conducted on a pre-scheduled date prior to the main program. The e-posters and the relevant video presentations were judged by a panel of foreign speakers. The competition aims to develop the research interest and the scientific presentation abilities among chemical pathology and MLS trainees. Winners of the oral presentation competition and e-poster competitions were announced and awarded at the inauguration ceremony.

The 2025 AAS-CCPSL Clinical Lab Expo ran concurrently with the sessions, open to all attendees and non-registered laboratory professionals interested in the most recent advancements in laboratory medicine and diagnostic technologies. It provided an opportunity for attendees to interact with exhibitors and find solutions to their laboratory-related needs.

Email: colchempath@gmail.com Tel: +94 (0) 76 704 4871 Website: ccpsrilanka.com

CCPSL NEWS

Winners of the awards - 10th Annual Academic Sessions of CCPSL

Oral Presentations (Research and Audits Category)

First place

Comparative Assessment of Lipid Parameters and Atherogenic Indices in Risk Identification of Acute Coronary Syndrome: A Descriptive Study

Fernando KM

Department of Chemical Pathology, Medical Research Institute, Colombo
Department of Biochemistry and Clinical Chemistry
Faculty of Medicine, University of Kelaniya, Sri Lanka

kavindyam@kln.ac.lk

Second place

Navigating the Optimal Equation for Low Density Lipoprotein Cholesterol Estimation in Lipid Profiles with Triglycerides >400 mg/dL: Insights from a Tertiary Care Center

Rammuthupura KD

Department of Chemical Pathology
Sri Jayawardenepura General Hospital, Nugegoda

kishanjalee@gmail.com

Third place

Evaluating the Diagnostic Accuracy of Serum Ferritin as a Marker for Iron Deficiency Anaemia in Paediatric Patients

Kulasinghe MSN

Department of Chemical Pathology
National Hospital of Sri Lanka, Colombo

kmihilie@gmail.com

Oral presentations (Case Reports and Case Series Category)

First place

Uric Acid Analysis in Body Fluids, an Important Screening Tool to Detect Purine Pathway Defects in a Neonate with Intractable Seizures

Kulasekera RCS

Department of Chemical Pathology
Apeksha Hospital,Maharagama

chamisulok@gmail.com

Second place

A Case Report of Chorea-Hyperglycaemia-Basal Ganglia Syndrome as the First Presentation of Type 2 Diabetes Mellitus in a 65-Year-Old Male

Weerathunga DN

Department of Neurology
Teaching Hospital, Karapitiya

dulmini.dn@gmail.com

Third place

A Case Report: Secondary Amenorrhea with Virilization: A Clue to the Underlying Endocrine Disorder

Dissanayake CS

Department of Chemical Pathology
National Hospital of Sri Lanka

Sachinthani34@gmail.com

E-Poster Presentations (Research and Audits Category)

First place

Distribution of Serum Homocysteine Levels among Patients with Young-Onset Cardiovascular and Cerebrovascular Events

Panapitiya NP

Department of Chemical Pathology
Medical Research Institute
Colombo

neisali@yahoo.com

Second place

Assessing Aldosterone and Renin for Primary Aldosteronism Screening: A Tertiary Care Center Experience in Sri Lanka

Galmangodage NE

Department of Chemical Pathology
National Hospital of Sri Lanka

ne.galmangodage@gmail.com

Third place

Evaluating the Predictive Value of Biomarkers in ST-elevation Myocardial Infarction Diagnosis: Comparing High-sensitivity Cardiac Troponin I, De Ritis Ratio, and HEART Score

Fernando KM

Department of Chemical Pathology,
Medical Research Institute,
Colombo
Department of Biochemistry and Clinical Chemistry
Faculty of Medicine, University of Kelaniya, Sri Lanka

kavindyam@kln.ac.lk

E-poster presentation (Case Reports and Case Series Category)

First place

Thoracic Origins of Cushing's Syndrome: A Clinicopathological Case Series of Two Carcinoid Tumors

Galmangodage NE

Department of Chemical Pathology
National Hospital of Sri Lanka

ne.galmangodage@gmail.com

Second place

Hypercalcemic Storm: A Case Report of Milk-Alkali Syndrome, Thyrotoxicosis, and Pancreatic Cancer

Galmangodage NE

Department of Chemical Pathology
National Hospital of Sri Lanka

ne.galmangodage@gmail.com

Third place

The Role of Chemical Pathology in Establishing Etiology of Hyperinsulinemic Hypoglycemia: A Case Series

Galmangodage NE

Department of Chemical Pathology
National Hospital of Sri Lanka

ne.galmangodage@gmail.com

Award for the Best Publication by a Chemical Pathologist

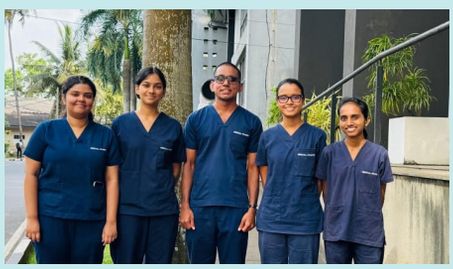
The award for the best publication by a Chemical Pathologist for the year 2024 was awarded to Dr. Deepani Siriwardhana, Consultant Chemical Pathologist, Senior Lecturer, Technical Assessor for SLAB, Department of Biochemistry and Clinical Chemistry, Faculty of Medicine, University of Moratuwa, for the research paper titled “Optimization of 25% Sufosalicylic Acid Protein-to-Creatinine Ratio for Screening of Low-Grade Proteinuria”

Winners of Inter-Medical University Quiz Competition

First place – Faculty of Medicine, University of Colombo

Wijewardena S/ Tharshikan S
Theekshanie D / Doluweera M
Amarasinghe G

Second place – Faculty of Medicine, Kothalawela Defence University (KDU)



Kavindya S / Premanath S
Rathnayake H / Fernando D
Vivekananthan K

Third place - Faculty of Medicine, University of Kelaniya

Nisansani TA / Hansika PH
Amarasinghe KGC /
Madurapperuma MKJK
Wijesinghe HJG

Pre-Analytical Quality Indicators: Strengthening the Forgotten Frontline of Laboratory Medicine:

"Quality begins before the sample reaches the analyser."

Dr. Thamara Herath
Consultant Chemical Pathologist
Department of Biochemistry,
Medical Research Institute, Colombo 08, Sri Lanka

Why Pre-Analytical Quality Matters

Nearly half to two-thirds of laboratory errors (46–68%) arise before analysis begins, during test ordering, patient preparation, specimen collection, labelling, transport, or storage (1,2,3). By the time a sample reaches the analyser, much of its quality has already been determined by these early steps (4,5). This is why the pre-analytical phase is often described as the first critical link in the "brain-to-brain" laboratory loop, where every action from test request to result interpretation matters (4,6).

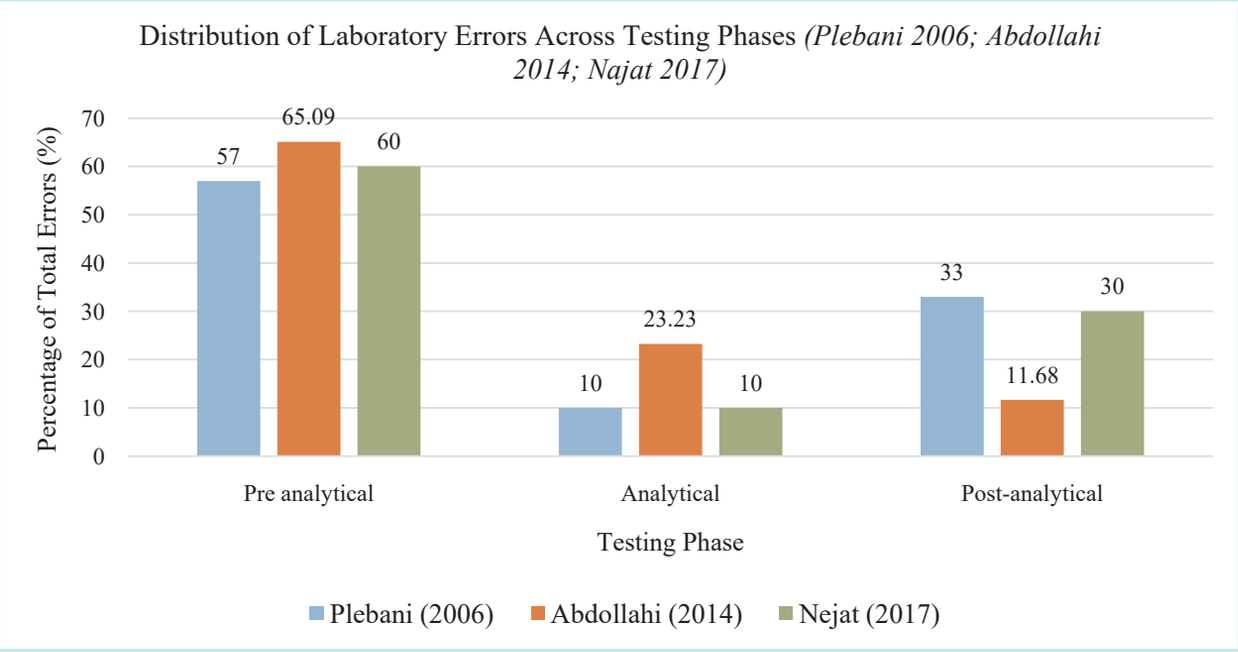


Figure 1: Comparison of the distribution of laboratory errors across the pre-analytical, analytical, and post-analytical phases in three published studies (Plebani 2006; Abdollahi 2014; Najat 2017). All studies highlight the predominance of pre-analytical errors.

Yet, this stage remains the most overlooked, largely because most pre-analytical activities occur outside the laboratory, in wards, clinics, or collection centres, beyond the lab's direct supervision and routine quality checks (1,6). While the analytical phase has benefitted from automation, rigorous quality control (QC), and accreditation standards, pre-analytical processes still depend heavily on human performance, communication, and manual documentation.

Neglecting this early phase has direct clinical consequences. A mislabelled or haemolyzed specimen can trigger repeat sampling, delayed reports, misdiagnosis, or even inappropriate treatment. In high-volume hospitals, such errors translate into lost time, wasted resources, and avoidable patient

distress (6). In Sri Lanka, where laboratories often function under resource constraints and limited automation, pre-analytical lapses frequently go unnoticed or unreported, even though they directly affect the validity and reliability of every result issued.

Simply put, a laboratory result is only as accurate as the sample it is based on. Recognizing and managing the pre-analytical phase as a quality-critical process is therefore essential for improving patient safety and laboratory efficiency.

Measuring What Matters: Understanding Pre-Analytical Quality Indicators (QIs)?

In laboratory quality management, quality indicators (QIs) are measurable parameters used to evaluate whether a process meets defined standards (5). They transform "quality" from an abstract goal into something quantifiable and actionable. According to ISO 15189, accredited laboratories must establish QIs for the pre-analytical , analytical, and post-analytical phases, ensuring that every part of the testing cycle is monitored (1,7).

The IFCC Working Group on Laboratory Errors and Patient Safety (WG-LEPS) recently proposed a simplified set of six 'Essential Quality Indicators' to support global harmonization (8) including, misidentified requests/samples, sample rejection rate, haemolysis rate, unacceptable EQA performance, troponin turnaround time, and corrected reports. Although concise, this essential panel is derived from the broader IFCC Model of Quality Indicators (MQI), which still includes additional pre-analytical measures, such as wrong container type, clotted samples, delayed transport, and incomplete request forms, that collectively influence sample rejection rates (5,8,9,10).

In the present article, we describe six pre-analytical quality indicators that remain highly relevant for Sri Lankan laboratory settings.

- Specimen misidentification rate (target = 0%)
- Haemolyzed sample rate (target < 0.5%)
- Clotted anticoagulated samples (target < 0.1%)
- Samples received in wrong containers
- Samples transported at an inappropriate temperature
- Incomplete request forms

Each indicator expresses both numerator (number of errors) and denominator (total samples), allowing labs to calculate error rates and monitor trends.Tracking QIs turns invisible weaknesses into measurable data, guiding corrective action, whether retraining staff, improving transport, or introducing barcode labelling.

Instead of managing errors reactively, laboratories can use QIs to detect problems before they compromise patient results. As the saying goes: "You can't fix what you don't measure."

The Sri Lankan Reality: A Hidden Gap in the Quality Chain

Even in large, well-established laboratories such as the Medical Research Institute (MRI) and tertiary hospital centres, pre-analytical quality receives less systematic attention than analytical performance. Most government and teaching hospital laboratories still rely on paper-based request forms and manual specimen logs. When a sample is rejected, the incident may be recorded in a notebook but rarely analysed as part of a continuous quality improvement cycle.

As a result, labs often operate in the dark regarding the true scope of pre-analytical errors. Some of the most frequent issues observed in Sri Lankan setting include:

1. **Inadequate or incorrect labelling:**
   Samples from peripheral centres sometimes arrive without patient identifiers or with mismatched labels. A local audit found mislabelling accounted for around one-third of all sample rejections in a hospital haematology unit (11).

2. **Improper sample transport:**
   Many laboratories depend on manual courier systems without temperature control or standardized time tracking. Exposure to heat, leakage, or delays in transport can degrade analytes (12).

3. **Incomplete request forms**:
   Handwritten forms missing essential details, patient age, and test requested, or clinical notes, are a chronic source of inefficiency. One Sri Lankan study showed that incomplete request forms accounted for 54% of sample rejections in the biochemistry section of a hospital lab (11). Essentially, over Half of the rejected specimens could be traced to clerical deficiencies.

4. **Absence of LIS and barcoding:**
   Without Laboratory Information Systems (LIS), it is difficult to trace a specimen's journey or identify recurring problem sources (for instance, a particular ward with frequent labelling mistakes). The lack of data visibility makes targeted improvement nearly impossible.

Collectively, these challenges reveal a systemic blind spot. Sri Lanka has achieved significant gains in analytical accuracy through External Quality Assessment (EQA) participation, but those efforts risk being undermined if the samples themselves are suboptimal. For example, haemolyzed and insufficient-volume samples are leading causes of rejections worldwide (12), and Sri Lanka is no exception, yet without measuring, labs may not realize how often this is happening.

Building a Framework of Pre-Analytical Quality Improvement

How can Sri Lankan laboratories begin to systematically improve the pre-analytical stage? International models (such as the IFCC's WG-LEPS recommendations) and local experts suggest a stepwise approach. Below is a proposed framework to establish Pre-Analytical Quality Indicators and monitoring in Sri Lanka:

Step 1 – Define a core set of QIs:

A national consensus led by the College of Chemical Pathologists of Sri Lanka (CCPSL) should identify a concise core set of 8–10 QIs, such as haemolysis rate, misidentification, insufficient volume, and wrong container type, each with clear definitions, denominators, and performance targets.

Step 2 – Collect standardized data routinely:

Even without LIS integration, Excel or Google Sheets can record sample rejection causes. Monthly summaries enable simple trend analysis and early identification of problem sources.

Step 3 – Benchmark and share results nationally:

Data gains value only when compared. A Pre-Analytical QI Pilot Program, perhaps coordinated by the MRI, could invite participating hospital labs to submit monthly statistics confidentially.
Aggregated results would establish baseline national performance, for instance, the average haemolysis rate or proportion of incomplete request forms, and enable peer benchmarking, fostering healthy competition and mutual learning.

Step 4 – Provide feedback and targeted training:

Regular feedback to wards, nurses, and phlebotomy staff is crucial.If haemolysis rates rise, phlebotomy technique retraining may be needed; if certain wards submit unlabelled tubes, focused awareness sessions should follow. Pre-analytical quality is a shared responsibility, not the lab's alone (6).

Step 5 – Leverage technology:

In the long term, LIS implementation and barcode-based labelling will be key to sustainability. Electronic request systems can enforce mandatory fields, flag duplicate orders, and capture timestamps automatically. Even partial automation, such as barcode printers for sample labels or temperature-logged transport boxes, significantly reduces human error. Where resources allow, automated pre-analytical systems (centrifugation, sorting) have been shown internationally to cut specimen handling errors by up to 60% (13).

These five steps can be piloted in tertiary-care centres and gradually extended to peripheral laboratories. What gets measured gets improved; what gets improved inspires further measurement.

Challenges on the Road Ahead

Implementing a pre-analytical QIs in Sri Lanka will face practical barriers, but none are insurmountable.

- **Limited digital infrastructure:**
  Many government labs still lack LIS integration. Manual data collection is labour-intensive but can serve as a stepping-stone until electronic systems are feasible. Demonstrating tangible benefits, like reduced repeat testing, can help secure administrative support.

- **Inconsistent Training & Practices:**
  Pre-analytical processes involve multiple stakeholders: ward nurses, medical officers, phlebotomists, and couriers.Variations in training lead to inconsistent practices. Regular workshops, SOPs, and refresher programs are vital to standardize procedures.

- **Weak communication loops:**
  Currently, when a sample is rejected, feedback rarely reaches the person who collected it, meaning the same error may recur.
  Establishing simple feedback mechanisms, monthly ward-wise summary reports or visual dashboards, closes the learning loop.

- **Lack of benchmarks and accreditation requirements:**
  Although criteria for acceptance and rejection of samples are mentioned in ISO15189:2022 (E) (7), pre-analytical QIs are not yet mandatory in Sri Lankan accreditation schemes. Advocacy through professional bodies can help integrate them into ISO assessments and EQA programs.

- **Resource constraints:**
  Quality initiatives require time and staff. Management should recognize quality monitoring as essential work, not an optional extra.

The Way Forward

To uplift laboratory medicine quality nationally, stakeholders must acknowledge that excellence begins at the point of collection, not at the analyzer.

The following priorities can guide the next phase:

1. **National Consensus on QIs:**
   CCPSL and the Sri Lanka College of Haematologists should collaborate to publish standardized pre-analytical QI definitions and acceptable performance limits.

2. **Integration into External Quality Assessment (EQA):**
   The MRI's national EQA scheme can expand to include a Pre-Analytical module, where participating labs report QI data quarterly. This would normalize monitoring and provide comparative national insight.

3. **Continuous education:**
   Annual Continuous Medical Education (CME) sessions for nurses, phlebotomists, and laboratory staff should emphasize specimen integrity, labelling, and request form completion. Recognizing high-performing wards or teams can build motivation and accountability (6).

4. **Digital and Process Innovations:**
   Seek funding for LIS and barcode systems. Even simple mobile-app-based specimen tracking or QR codes can modernize workflows and minimize errors.

5. **Cultivate a Data-Driven Culture:**
   Quality must become a continuous conversation. Regular QI dashboards reviewed in management meetings, along with internal bulletins highlighting improvements, keep the momentum alive.

When laboratories commit to measuring, communicating, and learning from pre-analytical data, quality improvement becomes self-sustaining.

Conclusion

The pre-analytical phase of laboratory testing, long regarded as the “forgotten frontline”, is finally gaining the recognition it deserves. True laboratory excellence begins not with calibration curves, but with correctly identified, properly handled specimens.

Sri Lanka’s laboratories have already advanced in analytical quality; the next leap forward is extending that culture of accuracy to the pre-analytical process. By adopting measurable QIs, sharing data transparently, and fostering a feedback-oriented mindset, laboratories can reduce sample rejections, prevent errors, and strengthen clinician confidence.

Ultimately, every correctly collected sample represents a safer patient journey. When we safeguard quality before analysis, we safeguard the accuracy and credibility of everything that follows.

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Essential Basic Laboratory Tests in Diagnosing Inborn Errors of Metabolism

Dr. Dilinika Perera
Consultant Chemical Pathologist
Teaching Hospital Kurunegala

Introduction

Inborn errors of metabolism (IEM) are inherited disorder that develop as a consequence of mutations that affect the functions of proteins (1).The diagnosis of inborn errors of metabolism (IEM) takes many forms. Appropriate and careful test selection must be determined by a combination of the patient's clinical presentation and the results of routine first tier biochemical tests. This will guide more specific testing in cost effective way (1,2).

General approach to diagnosing IEM

Clinical phenotypes of IEM are extensive and often non-specific, reflecting more common conditions (1,2). When evaluating a patient for a possible IEM, routine biochemical tests can identify underlying patterns suspicious for a metabolic defect. Common findings include hypoketotic hypoglycemia, lactic acidosis, metabolic acidosis, ketosis and hyperammonemia (2,3).

Essential Laboratory Investigations

Assessing blood and urine test results in combination with the clinical presentation can narrow the focus toward a particular subset of metabolic disorders (1,2).

Table 1 Routine biochemical tests that should be always ordered when an IEM is suspected (1).

Test
Liver function tests
Renal function tests
Plasma Ammonia
Blood gas Analysis
Anion gap
Plasma Glucose
Plasma Lactate
Urine ketones
Urine Reducing Substances

This table represents the most suitable routine laboratory investigations when suspecting an IEM in a patient during a metabolic stress.

Liver Function Tests (LFT)

Jaundice and other features of liver dysfunction, is one of the most commonpresenting feature of IEM. LFTs are crucial first line biochemical investigation in suspected IEM. One example is classic galactosaemia, which ischaracterized by severe conjugated hyperbilirubinaemia, and raised serum aminotransferases (2,3,4).

Table 2 shows some inherited metabolic disorders presenting with liver failure (1).

Tyrosinaemia type 1
Galactosaemia
Neonatal Haemochromatosis
Fatty acid Oxidation Defects
Respiratory Chain disorders

Renal Function Tests (RFT)

In IEM, toxic accumulates, energy failures and substrate deficiency can affect renal functions (1,4). **Table3** shows some inherited metabolic disorders presenting with renal impairment (1,3,5).

Lysosomal Storage Diseases: Fabry Disease, Cystinosis
Amino acid Disorders: Phenylketonuria(PKU), Methylmalonic aciduria
Organic Acidaemias: Maple Syrup Urine Disease (MSUD)
Carbohydrate Disorders : Glycogen Storage Diseases
Mitochondrial Disease: Affecting energy production

Serum creatinine is low it indicates creatine synthetic or transporter defect (4).

Plasma ammonia

It is essential to carry out plasma ammonia levels in children with acute or chronic encephalopathy, recurrent vomiting or hyperventilation (2).

Hyperammonemia may be missed especially in neonatal period when the clinical presentation is nonspecific. Preanalytical error prevention during sample handling and separation is essential (1)

Hyperammonemia can be caused by many non-metabolic conditions for an example liver disease. However, a marked elevation of ammonia, typically 10–100 times the upper limit of normal, can be associated with urea cycle disorders (1)

The finding of metabolic acidosis in a hyperammonemic patient is indicative of an organic acid disorder (1). In older children and adults, plasma ammonia should be measured in those with unexplained encephalopathy, vomiting, drowsiness or Reye- like syndrome (2)

Blood Gas Analysis and Anion Gap

This is a vital investigation in a patient with a suspected IEM, essentially in acute episodes of illness. Metabolic acidosis in paediatric population is commonly as a reason of infections, catabolic state or severe dehydration (2)

Calculation of the anion gap may assist in the interpretation of the blood gas analysis: metabolic acidosis with an increased anion gap is observed in IEMS, however metabolic acidosis with a normal anion gap is more likely to be due to diarrhea or renal tubular acidosis (2)

Measurement of Ketones

Ketosis is normal physiological response to fasting. Ketosis not associated with acidosis or hypoglycemia in children can be considered as physiological (1,2).

In patient with a metabolic acidosis suspected IEM it is important to test urine for ketone. Metabolic acidosis with ketonuria in neonatal, period is nearly always pathological and should prompt consideration of branch chain amino acid disorders: propionic, methyl malonic or isovaleric acidemias and maple syrup urine disease (MSUD) (2,5).

Gluconeogenetic and glycolytic defects should be considered when ketotic
patients hashypoglycaemia (2,3).
The absence of ketones can give clue to underlying IEM, the most common example associated with hypoketonuria with hypoglycemia is fatty acid oxidation defects (1).

Plasma and CSF Lactate

Secondary causes of lactate accumulation should be excluded before an inherited disease of lactate/ pyruvate metabolism is sought (2).

Lactic acidosis is commonly present in the acutely ill, when circulatory collapse results in tissue hypoxia (2).

Lactic acidosis associated IEM are organic acid disorders such as propionic and methyl malonic aciduria (2).

Increased CSF lactate is an important biochemical marker in diagnosis of children with suspicion of mitochondrial disorders (2).

Plasma Glucose

Hypoglycaemia is a presenting feature of many metabolic disorders. It could be due to primary block in glucose metabolism for example glycogen storage disease, or when glucose metabolism is secondarily affected such as tyrosinaemia (2,5).

Each laboratory or hospital should have its own protocol to investigate hypoglcaemia. This protocol should be carried out if laboratory blood glucose of < 2.6mmol/L (2).

Disorders of mitochondrial fatty acid oxidation, ketone body metabolism, carbohydrate metabolism and organic acidaemias can all cause hypoglycemia (6).

When evaluating hypoglycemia, first consider whether the patient is ketotic or nonketotic (2).

Disorders of mitochondrial fatty acid oxidation and ketogenesis including HMG-CoA lyase deficiency and HMG-CoA synthase deficiency are associated with hypoketotic hypoglycemia. Ketotic hypoglycemia typically caused by disorders such as organic acidemias, defect of ketone body metabolism and less commonly MSUD (2,4,6).

Urinary Reducing Substances

The detection of reducing substances in urine, usually simple but historically mainstay of first line test in investigating Item. This test has poor specificity (2,4).

IEM that detect urine reducing substances are galactosaemia, fructose intolerance and tyrosinaemia (2,4).

Serum Uric Acid

High serum uric acid levels indicate disorders of carbohydrate disorders such as glycogenolysis or gluconeogenesis. High levels with intellectual disability and self-mutilation is highly suggestive of Lesch-Nyhan Syndrome (HPRT deficiency– purine recycling disorder) (2,4).

A low level may point towards xanthine/hypoxanthine disorder or a molybdenum cofactor deficiency (MoCD) (2,4).

Creatine Kinase

It is elevated in IEM affecting the muscle causing glycogenolysis, or rhabdomyolysis, and in disorders of energy production such as fatty acid oxidation defects, gluconeogenesis defects or a mitochondrial disorder (1,5).

Plasma total homocysteine level

This is elevated in disorders of vitamin B12 or folate metabolism, and in classic homocystinuria due to deficiency of cystathionine beta-synthase enzyme that utilizes pyridoxine as a cofactor. Low homocysteine level may be indicative of a methionine disorder such as methyl adenosyl transferase deficiency (4,5).

Lipid profile

This is elevated in disorders of vitamin B12 or folate metabolism, and in classic homocystinuria due to deficiency of cystathionine beta-synthase enzyme that utilizes pyridoxine as a cofactor. Low homocysteine level may be indicative of a methionine disorder such as methyl adenosyl transferase deficiency (4,5).

Prolactin levels

This may be elevated in neurotransmitter disorders (dopamine synthesis).

Copper levels (in plasma)

This may be decreased in Wilson disease, Menkes, aceruloplasminemia, and MEDNIK syndrome; and increased in peroxisomal disorders (4).

Serum Iron studies

Increased serum iron/ferritin is observed in hemochromatosis and peroxisomal disorders (4)

Table 4 shows basic biochemical tests for diagnosis of IEMs (1,2,4,5)

Disease group	Basic Biochemical Test
Disorders of amino acid, organic acids and peptide metabolism	GELAK (glucose, electrolytes, lactate, acid-base, ammonia, ketones), homocysteine
Disorders of fatty acid and ketone body metabolism	Glucose, lactate, ammonia, ketones, acid-base, creatine kinase, uric acid
Disorders of carbohydrate metabolism	Glucose, other sugars, insulin, acid-base, lactate, ketones, LFT
Disorders of energy metabolism	Glucose, lactate, Pyruvate, ketones, organic acids
Disorders in the metabolism of purines , pyrimidines	Uric acid in serum, urine crystals
Disorders of sterol and bile acid synthesis	Cholesterol in serum , lipid profile, liver function tests
Disorders of lipid and lipoprotein metabolism	Lipid profile, insulin, liver enzymes
Congenital disorders of glycosylation and other disorders of protein modification	Liver function test
Lysosomal disorders	Liver function, renal function, serum creatine kinase
Peroxisomal disorders	Liver function tests, Renal functions, Lipid profile
Disorders of neurotransmitter metabolism	GELAK, Prolactin, uric acid, homocysteine, folic acid, vit B12 levels

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Ethics in Chemical Pathology: Responsible Management of Patient Information, Research Activities and Artificial Intelligence Applications

Dr. Maduri Vidanapathirana
Senior Lecturer/Consultant Chemical Pathologist
Department of Pathology
Faculty of Medical Sciences University of Sri Jayewardenepura

Introduction

In an era of accelerating technological progress, the field of chemical pathology encounters both promising opportunities and notable obstacles. The growing accumulation of patient information, broadening research capabilities, and incorporation of artificial intelligence (AI) and machine-learning technologies provide meaningful enhancements to diagnostic precision, laboratory productivity, and clinical applications. Nevertheless, these advances also present intricate ethical questions. Chemical pathologists must balance the stewardship of laboratory data, responsibilities as researchers and educators, and the evolving relationship with AI-enabled systems. This article outlines key ethical domains relevant to chemical pathology, including data governance, research ethics, and algorithmic applications, potential problems and practical considerations.

1. Patient Information: Ownership, Confidentiality, and Secondary Use

The laboratory is a major generator of patient data including biochemical test results, quality-control logs, sample metadata, patient identifiers, and increasingly, linked datasets across biochemistry, genetics, and omics. The ethical principles of respect for persons, beneficence, and justice underpin all handling of patient data (1). Even when data are de-identified, the risk of re-identification increases in large linked datasets and when AI tools are applied (2).

Key issues to consider:

Consent and transparency: Patients usually consent to testing with the expectation that their data will be used solely for clinical care. Using these data for research, AI development, or commercial purposes often extends beyond that original agreement. Clear communication and the option to opt out are essential to maintain trust and uphold ethical practice (3).

Anonymisation vs de-identification: De-identified data are frequently considered low risk, but large datasets can be re-linked to reveal identities. For example, a research group may use de-identified thyroid-function test data (TSH, free T4, and free T3) from a tertiary-care laboratory to train an AI model predicting subclinical hypothyroidism. Although names and hospital numbers are removed, the combination of age, gender, clinic location, and testing date could still allow re-identification when matched with outpatient appointment or pharmacy data (4).

Ownership and control: Questions about who actually owns laboratory data -whether it is the patient, the hospital, or a commercial partner remain ethically complex. Chemical pathologists must advocate for patients' rights to their data and transparent policies for sharing or commercial use. Laboratory-derived AI-based decision-support systems raise ethical questions about data ownership and patient understanding (5).

Equity and justice: Large datasets used for AI model training must represent diverse populations. If Sri Lankan data are excluded or under-represented in global datasets, bias and inequity arise when tools are applied locally (6).

Consultants and educators help shape institutional practices that protect patient information by supporting consent for secondary use, strong anonymisation measures, and secure systems with proper oversight. By keeping patients informed about how their data may be used or shared, they strengthen trust and ethical stewardship of health information.

2. Research Ethics in Chemical Pathology

Chemical pathology departments increasingly engage in assay validation, method comparisons, population studies, and translational work on metabolic disorders. Ethical oversight must be robust and tailored to these domains. When the chemical pathology laboratory performs more than routine testing it is the duty of the lab to maintain strong but transparent ethical standards.

Considerations:

Human-subject research: Foundational ethical frameworks such as the Declaration of Helsinki, the Belmont Report, and Council for International Organisations of Medical Sciences (CIOMS) guidelines remain relevant (1,7). Researchers must ensure appropriate risk–benefit assessment, informed consent, privacy protection, and special consideration for vulnerable groups (e.g., children, issues related to parental consent, and genetic data).

Secondary data and reuse: Retrospective studies often rely on laboratory databases. Researchers must verify that original consent permits reuse, data are securely stored and anonymised, and ethical review acknowledges reuse implications.

Methodological integrity and reproducibility: Poor methodology or bias wastes participant effort and may mislead clinical practice. Good laboratory governance, clear protocols, and sample integrity are ethical imperatives.

Publication and authorship ethics: Responsible conduct includes appropriate authorship(on actual contributions), avoidance of duplicate publication, transparent reporting, disclosure of conflicts of interest, and open discussion of limitations (8).

Commercial collaborations: Collaborations with reagent manufacturers or analyser vendors are common. Researchers must declare support, maintain independence, and safeguard patient data from misuse.

Benefit translation: Ethical research should ultimately benefit patients through assay harmonisation or improved metabolic disease detection and include plans for local implementation, particularly in resource-limited settings (9).

Embedding these principles in postgraduate training fosters critical, reflective scientists who consider data integrity and societal impact, not just data output.

3. AI and Algorithmic Tools in the Chemical Pathology Laboratory

AI applications in chemical pathology ranging from instrument flagging to decision-support and metabolic-risk prediction introduce new ethical challenges.

Key domains:

Transparency (Explainability): Many AI models are "black boxes." Lack of explainability undermines accountability and trust (10). If an AI algorithm flags a patient as "high-risk metabolic syndrome" without understandable reasoning, it weakens professional responsibility and clinician confidence.

Accountability and liability: When AI misclassifies samples, determining liability is complex. Kadakia et al highlight regulatory and legal gaps in laboratory-developed AI tests (11). Pathologists must validate such tools locally and define responsibility structures.

Bias and fairness: Algorithms trained on non-representative data may misclassify results for certain ethnic groups or populations (6,12). Local validation is ethically required before adoption.

Data quality and integrity: AI systems in laboratory medicine depend on accurate, consistent, and complete data to function reliably. In chemical pathology, pre-analytical variations can distort test results before analysis. Missing data from incomplete records or selective testing and data noise from analytical imprecision or transcription errors further weaken model performance. Ensuring high-quality,standardised datathrough robust laboratory governance is therefore essential for trustworthy AI outputs and safe clinical application (13).

Professional role and autonomy: Excessive automation risks reducing the pathologist's interpretative role. Ethical frameworks emphasise maintaining professional judgement and human oversight (14).

4. Practical Ethical Framework for Chemical Pathology Labs and Trainees

A practical ethical framework in chemical pathology should integrate robust governance, education, and continuous quality improvement to ensure responsible data use, research integrity, and fair AI application.

4.1 Governance and Policy

Create or revise laboratory data-governance policies with provisions for data retention, secure destruction, anonymisation, and patient opt-out.
Establish Aloversight committees including pathologists, data scientists, and ethicists.
Ensure ethicscommittee review of all research proposals involving data reuse or AI.

4.2 Education and Training

Incorporate data ethics, AI ethics, and research integrity into postgraduate curricula (15).
Encourage reflection on beneficence, harm, representativeness, and autonomy in every project.
Engage trainees in practical exercises such as auditing data flows, evaluating anonymisation processes, and validating AI tools. These activities help them understand how ethical principles are applied in real laboratory operations and foster a culture of accountability and ethical awareness in daily practice.

4.3 Research & Publication Ethics

Ensure informed consent, data privacy, and ethical approval are obtained before initiating any project. Clearly disclose conflicts of interest and provide transparent details of data governance and model validation within the methods section. Publish negative or inconclusive findings as well, since withholding such results can bias the scientific record and hinder evidence-based progress (8).

4.4 AI Implementation in Laboratory Practice

AI tools should be validated locally before clinical use, with developers providing full transparency about algorithm inputs, logic, and bias assessment. Continuous human oversight is essential while AI should complement, not replace and clinical judgement. Laboratories must monitor performance, ensure fairness across patient groups, and maintain secure vendor contracts to safeguard patient data and prevent misuse.

4.5 Professional Bodies and Continuous Quality Improvement

National colleges should draft AI ethics guidelines.
Conduct continuous professional development sessions on AI evaluation, algorithmic bias, and research integrity.
Audit anonymisation, consent processes, and secondary-use approvals regularly.

5. Ethical Challenges in Resource-Limited Settings

In Sri Lanka and other low-resource contexts, additional ethical issues arise:

Data export and collaborations: Safeguard patient interests and ensure equitable benefit sharing.
Applicability of AI tools: Validate imported algorithms locally to prevent diagnostic bias (12).
Cost–benefit: Weigh AI implementation costs against essential quality-assurance needs.
Data sovereignty: Protect national ownership and recognition in global datasets.
Consent literacy: Ensure culturally appropriate, comprehensible consent procedures (16).

6. Illustrative Case Example

Context: A tertiary hospital laboratory in Sri Lanka agrees to share one year of anonymised biochemistry data (glucose, lipids, liver enzymes, uric acid) and demographics with a commercial AI vendor to train a metabolic-risk model.

Ethical reflections:

- Was specific consent obtained, or is waiver justified?
- Is anonymisation adequate, or could linkage reveal identities (4)?
- Will the algorithm be validated locally to ensure fairness and accuracy for the Sri Lankan population?
- How are benefits returned - capacity-building or profit extraction?
- Does a governance framework exist for oversight, contracts, and audits of such collaborations?
- Are patients aware their data may be used by a commercial entity?
- Does this divert staff time from core services?

This scenario illustrates how closely intertwined patient data, research, AI, and commerce have become and why ethical reflection is essential.

In conclusion, Chemical Pathologists must combine analytical expertise with ethical responsibility in managing patient data. In an era of big data and AI, ethical governance, research integrity, and transparency form the foundation for responsible innovation. Upholding these principles ensures that laboratory medicine progresses equitably while maintaining patient trust. The true advancement of the field will depend not only on technology but on the integrity with which it is applied.

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Research

Comparison of Dried Blood Spot Sample Volumes Between TSH-Positive and TSH-Negative Cases in a Newborn Screening Program: A Case–Control Study

Panapitiya N P, Jayasinghe A U, Aththanayake A M I S,
Jainulabdeen F, Herath T P K
Medical Research Institute, Colombo

Introduction

Newborn screening for congenital hypothyroidism depends on accurate measurement of Thyroid Stimulating Hormone (TSH) from dried blood spot (DBS) samples. Current recommendations specify the use of well-formed, fully penetrated DBS with a diameter of 7–8 mm (1,2). For TSH measurement, 3mm diameter piece of the sample is punched out. While the analytical performance of the TSH assay is routinely monitored, pre-analytical factors—particularly DBS quality—receive far less attention (3). An audit conducted at the Medical Research Institute (MRI) in 2022 showed that 88.2% of DBS samples received had inadequate blood volumes (4).

Comparing DBS quality between TSH-positive and TSH-negative samples therefore helps illustrate the potential impact of suboptimal specimens and raises awareness of this critical pre-analytical issue.

All DBS samples collected from term neonates (gestational age 37- 40 weeks) on the second or third day of life were eligible for inclusion. Samples with incomplete records were excluded. From all eligible TSH-positive samples, 100 were selected using simple random sampling. For each selected case, one gestational-age and sex matched TSH-negative sample was chosen as a control.

Basic demographic and clinical details were extracted from the request forms, and the DBS characteristics were assessed manually. For each card, the number of DBS spots and the appearance of the primary punched spot (categorized as uniform, layered, multiple-drop, or unpenetrated) were recorded. The diameter of the penetrated area of the punched spot was measured at two perpendicular angles using a calibrated ruler, and both minimum and maximum values were documented.

Data were analysed using IBM SPSS Statistics software. Comparisons between cases and controls were done using independent sample t-test and chi squared test. A p-value <0.05 was considered statistically significant.

Results

A total of 200 dried blood spot (DBS) samples were analysed, consisting of 100 TSH-positive cases and 100 TSH-negative controls. Each group comprised 51 females and 49 males. The distribution of GA was similar across the two groups as demonstrated in Chart 1.

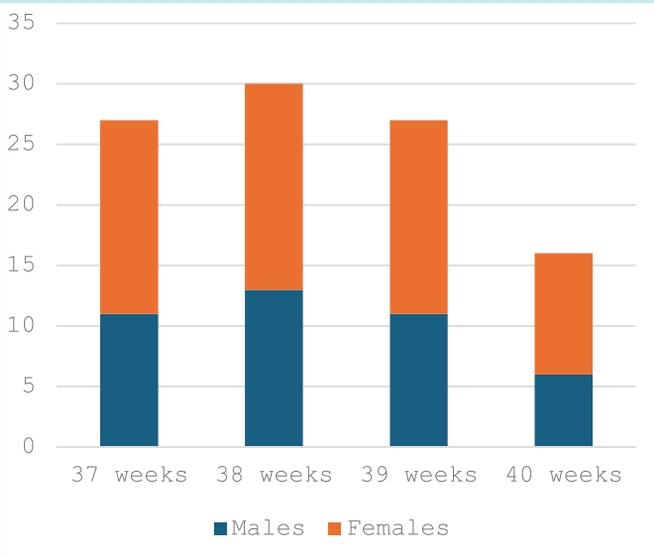


Chart: 1 Gender Distribution According to the Gestational Age

Majority of samples (89%) contained 3–5 DBS spots. Among the positive samples, most (59%) showed uniform, well-penetrated spots, whereas 47% of negative samples were unpenetrated (Table 1).

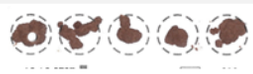
Appearance	Front Side Appearance	Back Side Appearance	Cases (Positives)	Controls (Negatives)
Uniform	 *Figure 1a*	 *Figure 1b*	59 (62.8%)	35 (37.2%)
Layered	 *Figure 2a*	 *Figure 2b*	11 (44%)	14 (56%)
Multiple Drops	 *Figure 3a*	*Figure 3b*	1 (20%)	4 (80%)
Unpenetrated	 *Figure 4a*	*Figure 4b*	29 (38.2%)	47 (61.8%)

Table 1: Appearance of DBS

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The proportion of well-penetrated spots was significantly higher among positives (83.3%) than negatives (16.7%) (p = 0.000).

The mean penetrated DBS diameter was significantly higher in the TSH-positive group (6.47 mm) compared to the TSH-negative group (4.05 mm), (p = 0.000).

Diameter-based quality categories (ideal ≥8 mm, acceptable ≥5 mm, unacceptable <3 mm) are summarised in Table 2. Number of samples in the ideal and acceptable ranges were significantly higher among the positive group while unacceptable samples were more prevalent in the negative group.

Minium DBS diameter	Front Side Appearance	Back Side Appearance	Cases (Positives)	Controls (Negatives)	p value
≥8mm (Ideal)			25 (83.3%)	5 (16.7%)	0.000
>5mm (Acceptable)			71 (68.3%)	33 (31.7%)	0.000
<3mm (Unacceptable)			1 (6.7%)	14 (93.3%)	0.000

Table 2: Diameter Based Assessment

Overall, the percentage of samples fulfilling the acceptable criteria for both penetration and diameter was significantly higher in the TSH-positive group, as demonstrated in Chart 2 (p = 0.000).

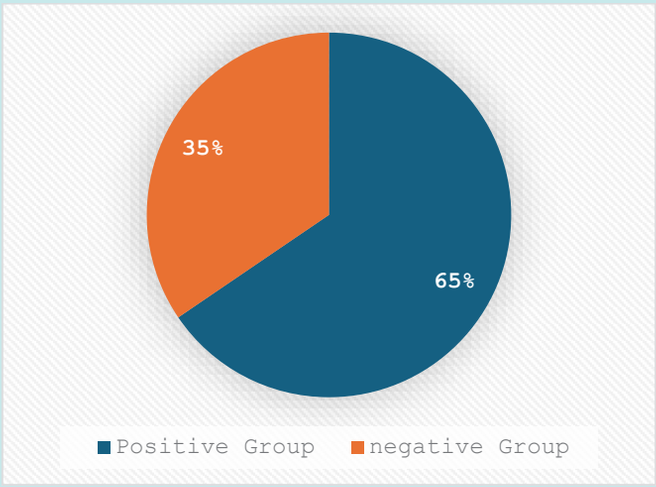

Chart 2: Percentages of Overall Acceptable Samples

Discussion and Conclusion

TSH concentrations in DBS samples are derived using a calibration curve, which assumes that patient specimens resemble calibrators—uniformly saturated, fully penetrated blood spots (5). In this study, however, 53% of samples did not meet this standard. As illustrated in Figures 4a and 4b, unpenetrated spots (38% of all samples) may appear adequate on the front surface but show no blood on the reverse side. When such spots are punched, the resulting disc contains insufficient blood, leading to marked underestimation of TSH levels and increasing the likelihood of false-negative screening results—undermining the fundamental purpose of the NSP.

Despite poor quality, nearly 90% of samples contained 3–5 blood spots. Given that the programme currently screens only for TSH, a single well-formed, fully penetrated spot is sufficient, emphasizing the need to priorities quality over quantity.

Recommendations

To improve the reliability of the Newborn Screening Programme, we recommend:

- Conducting hands-on training for healthcare staff on proper heel-prick technique and DBS application.

- Emphasizing the importance of collecting at least one well-formed and fully penetrated spot, rather than multiple inadequate ones.

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ESTIMATION OF SERUM FREE LIGHT CHAIN CONCENTRATIONS AND ITS RATIO IN PATIENTS WITH IMPAIRED RENAL FUNCTION

Thusynathy P, Rajitha Samarasinghe
Department of biochemistry,
National Cancer Institute Maharagama, Sri Lanka

Introduction

Serum free light chains (sFLCs), comprising kappa (κ) and lambda (λ) chains, play a key role in the diagnosis and monitoring of patients with light-chain myelomas, non-secretory myeloma and light-chain diseases like light-chain (AL) amyloidosis and light-chain deposition disease (1,2).According to *Katzmann et al* studies normal reference range for sFLC assay established, which included 282 healthy donors and established reference range for κ (3.3–19.4mg/L) and for λ (5.7–26.3 mg/L) free light chains and κ/λ ratio to be around 0.55 (range: 0.26–1.65) (3). However, their concentrations are significantly influenced by renal function, creating challenges in differentiating chronic kidney disease (CKD)-related abnormalities from monoclonal gammopathies. This study evaluates the relationship between sFLC concentrations, κ/λ ratio, and CKD progression (2,4).

Methods

A hospital-based cross-sectional study was conducted at the National Cancer Institute Maharagama, in patients who had impaired renal function (eGFR < 90 ml/min/1.73m^2). sFLC concentrations were measured using immunoturbidimetry, and statistical analysis was performed using SPSS 17 and SAS 9.4. Log-transformed data were analyzed using a general linear mixed model, ANOVA, and Welch's t-test.

Results

The sample consisted of 139 patients. λ FLC concentrations were significantly influenced by CKD stage ($p < 0.01$), with the highest levels observed in Stage 5 (189.01 mg/L) and the lowest in Stage 2 (30.82 mg/L) (Table 01). κ FLC levels, however, did not show statistically significant differences across CKD stages ($p > 0.01$), though higher mean values were noted in advanced stages (Table 02). The κ/λ ratio did not significantly vary among CKD stages ($p > 0.01$), but when compared to the reference value of 0.955, significant deviations were observed across all stages ($p < 0.0001$) (Table 03). Gender differences in sFLC concentrations were not statistically significant.

Table 01: Means and confidence intervals - lambda

Stage	No of observation	Mean	95% Confidence interval for mean
2	8	30.8	14.1 – 47.5
3a	21	83.5	58.4 – 108.7
3b	35	73.9	41.9 – 105.9
4	44	68.1	59.9 – 76.4
5	31	189	130.5 – 247.5

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Table 02: Means and confidence intervals - Kappa

Stage	No of observation	Mean	95% Confidence interval for mean
2	8	68.7	27.2 – 110.3
3a	21	80.2	61.1 – 99.4
3b	35	133.9	17.8 – 249.9
4	44	103.7	61.2 – 146.1
5	31	162.3	116.6 – 208.1

Table 03: Means and confidence intervals – K/L ratio

Stage	No of observation	Mean	95% Confidence interval for mean
2	8	3.8	-0.5 – 8.1
3a	21	1.2	0.8 – 1.6
3b	35	2.6	0.2 – 4.9
4	44	1.5	1.1 – 2.0
5	31	1.0	0.9 – 1.2

Discussion

The results of this study highlight the complex interplay between serum free light chains (sFLC), renal function, and monoclonal gammopathies. The significant variation in λ FLC concentrations with here CKD stages, while κ FLC levels remained relatively stable, suggests differential handling of light chains by the kidney. This aligns with previous research indicating that λ FLC forms dimers with a higher molecular weight, leading to slower renal clearance compared to κ FLC (2,5). Furthermore, the observed significant deviation of the κ/λ ratio from the reference range across all CKD stages emphasizes the need for CKD-specific reference intervals (8,9). Despite the increasing trend in mean kappa concentrations with CKD severity, the large variability within stages may explain why no statistically significant differences were detected between CKD stages. Additionally, the correlation between sFLC levels and creatinine observed in this study supports the proposition that sFLCs may serve as independent markers of renal function decline (6,7). Future studies should focus on refining diagnostic thresholds for CKD patients and exploring the mechanistic role of sFLCs in renal pathology to improve early detection and management strategies. Study had some potential confounders such as inflammation, medication use, and dietary protein intake were not controlled for, yet these could influence sFLC concentrations and κ/λ ratios, particularly in CKD patients.

Conclusion

The study confirms that λ FLC levels are strongly influenced by CKD progression, whereas κ FLC levels remain relatively stable. The κ/λ ratio deviations highlight the need for CKD-specific diagnostic reference ranges to improve accuracy in detecting monoclonal gammopathies.

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Email: colchempath@gmail.com Tel: +94 (0) 76 704 4871 Website: ccpsrilanka.com

Descriptive Evaluation of Serum Immunofixation Electrophoresis Results in Tertiary Care Patients: A Retrospective Single Center Experience

Madurangi DWDD, De Silva DDS, Samarasinghe R
Department of Chemical Pathology, National Cancer Institute, Maharagama, Sri Lanka

Introduction

Multiple myeloma (MM) is a malignancy of plasma cells resulting its clonal proliferation. MM constitutes 1% of all malignancies and represents 10–15% of hematological cancers, making it the second most prevalent hematological malignancy (1–3). Its incidence increases with advancing age and is consistently higher among men (1). The highest incidence rates are reported in individuals of African ancestry, whereas those of Asian ancestry exhibit the lowest rates. However, the epidemiological profile of MM in Sri Lanka remains uncertain due to the paucity of published data.

As per the latest criteria defined by international myeloma working group diagnosis of multiple myeloma should be confirmed by clonal bone marrow plasma cells >10% or bony or extramedullary plasmacytoma proven by biopsy and any one or more of the CRAB features (hypercalcemia, renal insufficiency, anemia and bone lesions) and myeloma-defining events including 60% or greater clonal plasma cells on bone marrow examination, serum involved/ uninvolved free light chain ratio of 100 or greater, provided the absolute level of the involved light chain is at least 100mg/L and more than one focal lesion on MRI that is at least 5mm or greater in size (3).

Monoclonal plasma cells produce excessive heavy and/or light chain immunoglobulins, which will appear on electrophoresis as a sharp, narrow peak known as the M protein. Detection and quantification of M protein is vital in the diagnosis and follow-up of MM. Immunofixation further characterizes this protein by identifying its specific heavy and light chain components.

Materials and Methods

This retrospective, cross-sectional analysis aimed to identify the M protein subtype pattern in the Sri Lankan MM community.

All the patients who were evaluated with serum protein immunofixation at the Department of Chemical Pathology, National Cancer Institute, Maharagama, during the period from January 2025 to May 2025 were included in the study.

Serum Immunofixation Electrophoresis (IFE) was done using mammalian immunoglobulin G, A, M, D, E, kappa and lambda by Sebia gel electrophoresis analyzer.

Results

Total of 96 patients were evaluated during the specified period. Our cohort contained 54 males and 32 females with a male: female ratio of 1.69. Despite having a M protein band in the serum protein electrophoresis (SPE), 10 of these patients did not exhibit monoclonal protein on IFE. Whereas IFE confirmed the presence of M protein in the remaining 86 patients.

Out of that 86 patients, 73 patients (84.88%) had monoclonal gammopathy and 12 patients (13.95%) and 1 patient (1.16%) had bi-clonal and tri-clonal gammopathy respectively (Figure 1).

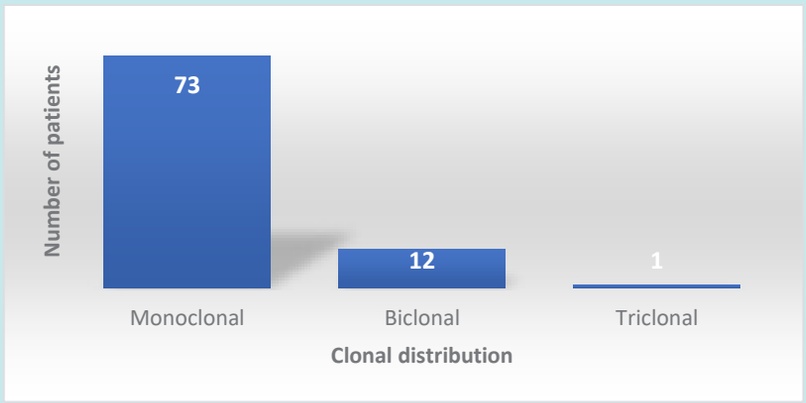

Figure 1
Clonal Distribution of the patient cohort

Isotyping of the monoclonal proteins revealed intact immunoglobulin in 89.53%, light chain without the corresponding heavy chains in 8.13% and combined intact immunoglobulin with free light chain in 2.33%. (Figure 2)

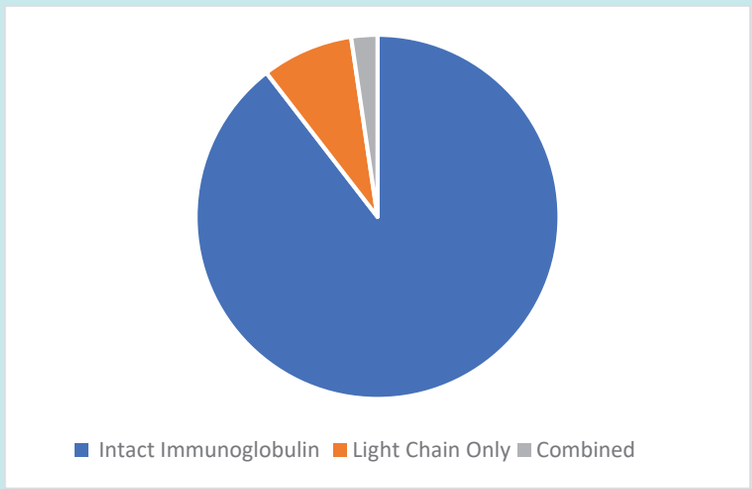

Figure 2
Distribution of subtype of multiple myeloma

IFE further demonstrated as IgG (55.69%) is the most commoniso type of heavy chain, followed by IgA (26.58%) and IgM (17.72%) in patients with intact immunoglobulin. Moreover, free kappa light chain disease was more common (85.71%) than free lambda light chain disease (14.28%).

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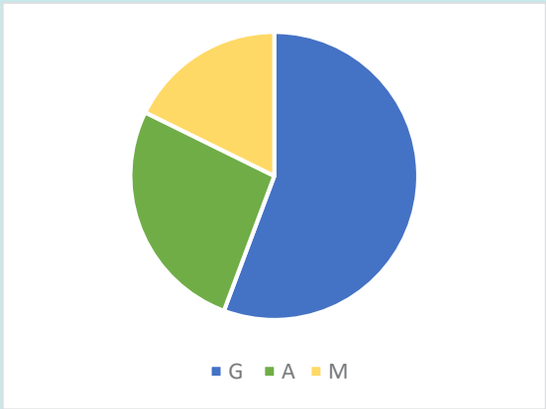

Figure 3
Distribution of heavy chain disease

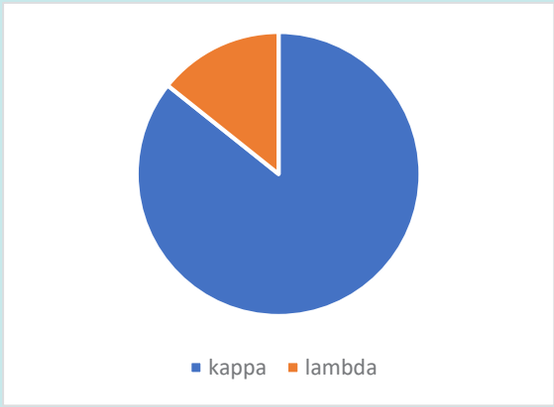

Figure 4
Distribution of light chain disease

Most prevalent subtype in intact immunoglobulin and combined heavy chain-light chain disease was Ig G kappa (32.91%), followed by IgG lambda (22.78%) as demonstrated in figure 5. Both IgA kappa and IgM kappa were similar in prevalence (15.18%). Patients with IgM lambda were least prevalent in our cohort (2.53%). There were no patients with IgD or IgE subtypes.

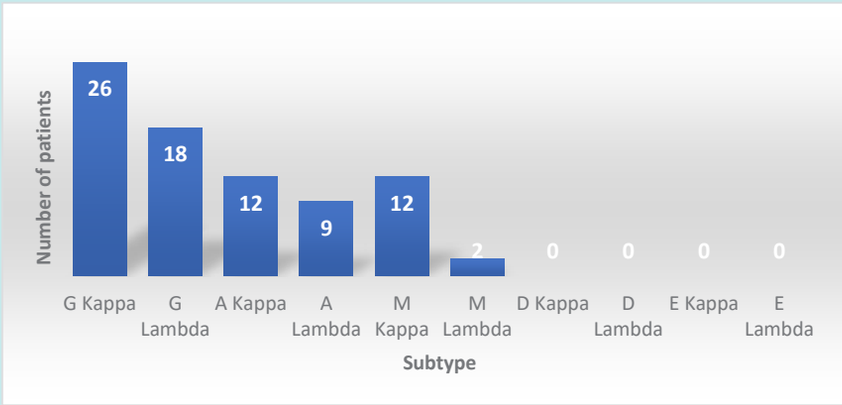

Figure 5
Distribution of subtypes of immunoglobulins

Discussion

Serum immunofixation electrophoresis is known to has improved sensitivity compared to serum protein electrophoresis (4). Therefore, immunotyping of M-proteins has been widely used as a biomarker for the diagnosis of MM.

This isotypic analysis demonstrated that the most predominant heavy chain isotype was IgG, followed by IgA. Furthermore, the most prevalent light chain subtype was free kappa light chains. Our results were consistent with global data regarding isotype distribution in multiple myeloma patients, where IgG accounts for 52%, IgA for 21% and only light chain secretion for 16%; IgD, IgM and IgE phenotypes are infrequent (5).

IFE is performed with antisera to IgG, IgA, IgM, κ and λ. Thus, when a monoclonal light chain is seen without a corresponding heavy chain, an additional immunofixation should be performed with antisera to D and E heavy chains. If there is no corresponding heavy chain, Light-chain MM is likely and should be further evaluated with Serum free light chain assay and urine immunofixation.

In conclusion,using IFE along with SPE will drastically improve the diagnostic accuracy.

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A Sweet Finding That isn't Diabetes

Dr. Kavindya Fernando
Registrar in Chemical Pathology
Medical Research Institute, Colombo
Lecturer, Department of Biochemistry and Clinical Chemistry
Faculty of Medicine, University of Kelaniya

Case Presentation

An 8 year and 8 month old boy, the youngest of four children born to healthy, non-consanguineous parents, was investigated following an episode of urinary tract infection during which urine glucose was incidentally detected. He had no prior illnesses and normal growth and development and no polyuria or polydipsia.

Family history revealed that two elder sisters also had persistent glycosuria with normal blood glucose, suggesting a hereditary renal tubular transport disorder, most consistent with an autosomal recessive or incompletely dominant inheritance pattern.

On examination, he was clinically well and euhydrated, with no signs of dehydration or metabolic acidosis. Plasma glucose, renal function, and electrolytes were within reference limits, and he was admitted for further evaluation to exclude an inherited proximal tubular defect.

Basic Investigations

Test	Result	Unit	Reference Range
Serum sodium	133	mmol/L	135–145
Serum potassium	4.0	mmol/L	3.5–5.1
Serum urea	5.03	mmol/L	1.8–6.4
Serum creatinine	33	umol/L	30–60
Serum albumin	34	g/L	34–50
Serum calcium	2.21	mmol/L	2.2–2.7
Serum uric acid	162	umol/L	140–360
Fasting plasma glucose	5.0	mmol/L	3.9–6.0
Whole blood pH	7.38		7.35-7.45
Whole blood bicarbonate	23	mmol/L	22-26
Urine osmolality	972	mOsm/kg	500–1200
Urine pH	6.5		5.0–7.0
Urine reducing substances	++++		—
Urine albumin	Nil		—
Fractional Excretion Na^+	0.44	%	1–3
Fractional Excretion K^+	2.68	%	4–16
Fractional Excretion PO_4^{3-}	3.92	%	15–20
Fractional Excretion uric acid	7.95	%	6–12
Urine Ca/Cr ratio	0.41		0.04–0.7

Questions

1. What are the possible causes of glycosuria with normal blood glucose?
2. What biochemical findings suggest an isolated tubular defect rather than a generalized proximal tubular disorder?
3. What is the most likely diagnosis in this child?
4. How can familial renal glycosuria (FRG) be confirmed and differentiated from other causes?
5. What are the clinical implications and management strategies?
6. What is the underlying genetic mechanism of FRG?

Answers

1. **Causes of normoglycaemic glycosuria:**
   - Familial (isolated) renal glycosuria (FRG)
   - Generalized proximal tubular disorders (Fanconi syndrome, cystinosis, Lowe syndrome)
   - Secondary renal glycosuria from drugs (e.g., SGLT2 inhibitors) or tubular injury (1,2).

2. **Evidence for isolated tubular defect:**
   - The child's normal renal function, electrolytes, phosphate, uric acid, and acid–base balance exclude generalized proximal tubular dysfunction.
   - Serum bicarbonate was normal, confirming the absence of metabolic acidosis, which is typical of Fanconi syndrome.
   - Fractional excretion indices within or below normal limits (Na^+, K^+, PO_4^{3-}, uric acid) indicate preserved reabsorptive capacity for other solutes.
   - Together, these findings strongly support an isolated glucose reabsorption defect in the proximal tubule (3).

3. **Most probable diagnosis:**
   - The findings are consistent with Familial Renal Glycosuria (FRG), a benign inherited defect of proximal tubular glucose transport.
   - The family history involving two siblings supports a hereditary pattern, most likely autosomal recessive or dominant with incomplete penetrance (4,5).

4. **Diagnostic confirmation:**
   - Persistent urinary glucose despite normal plasma glucose, negative ketones, and preserved tubular functions are diagnostic.
   - Molecular testing for SLC5A2 (SGLT2) mutations confirms the disorder, though clinical-biochemical correlation alone is often sufficient (3,4,6)

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5. **Clinical implications and management:**
   - FRG is generally benign; no specific therapy is required.
   - The key priority is to avoid misdiagnosis as diabetes mellitus, which may lead to unnecessary dietary restrictions or therapy.
   - Adequate hydration and periodic monitoring for urinary tract infections or rare electrolyte abnormalities are advised.
   - Family members may be screened, especially if glycosuria is incidentally detected (2,5).

6. **Genetic basis:**
   - FRG results from mutations in the SLC5A2 gene on chromosome 16p11.2, which encodes the sodium–glucose cotransporter 2 (SGLT2) in the renal proximal tubule.
   - The mutation leads to reduced glucose reabsorption, causing glycosuria de spite normoglycemia.
   - Phenotypic expression varies: heterozygotes may have mild glycosuria, while homozygotes exhibit marked, lifelong glycosuria.
   - Compensatory reabsorption via SGLT1 (SLC5A1) in the late proximal tubule explains normal glucose tolerance (5–7).

Learning Highlights

- Isolated renal glycosuria mimics diabetes but is non-pathological.
- Family history is crucial when evaluating paediatric glycosuria.
- Normal acid–base and electrolyte balance distinguishes FRG from generalized proximal tubular disorders.
- The chemical pathologist plays a key role in integrating clinical data with tubular function indices
- Genetic confirmation is useful for family counselling but often not essential for management.

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Hypocholesterolemia, Cause or Consequence of Liver Disease

Dr. Nuwani Jayasuriya
Registrar in Chemical Pathology
National Hospital Colombo

Case Presentation

A 66-year-old male, awaiting liver transplantation due to Non-Alcoholic Fatty Liver Disease, presented to the Emergency Treatment Unit (ETU) with headache and faintness. He had no history of diabetes or hypertension. Also, he was not on lipid-lowering therapy. On examination of the patient, well built, the pulse rate was 88/min, and the blood pressure was 180/100 mmHg.

Investigations:

	Value	Reference range
Total Cholesterol	57mg/dL	<200 mg/dL
Triglycerides	70mg/dL	<150 mg/dL
HDL	19mg/dL	>40 mg/dL
LDL	22mg/dL	<100 mg/dL
Non-HDL	38mg/dL	< 130 mg /dL
VLDL	16mg/dL	-
TC/HDL	3.0	< 3.5
LDL/HDL	1.16	< 2.5
AST	253U/L	0-40U/L
ALT	169U/L	9-48 U/L
TSH	2.55mIU/L	0.4 – 4.05 mIU/L

Questions for Discussion:

1. What are the secondary causes for hypocholesterolemia?

2. How does chronic liver cell disease cause hypocholesterolemia?

3. What are the primary causes leading to hypocholesterolemia?

4. What is the mechanism of NASH in a person with a congenital disorder of hypocholesterolemia?

5. How to differentiate congenital from acquired causes of hypocholesterolemia?

Answers:

1.

- Severe liver disease
- Malnutrition / cachexia
- Hyperthyroidism
- Chronic infections / inflammation
- Chronic illness / critical illness
- Drug-induced

2. A hepatocellular injury reduces HMG-CoA reductase and ApoB/ApoA1 synthesis, causing decreased hepatic synthesis of cholesterol and apolipoproteins. Also, damaged hepatocytes have defective MTP function, causing poor VLDL export.

3.

- Familial hypobetalipoproteinemia ApoB mutation
- Abetalipoproteinemia (ABL) MTP mutation
- Familial chylomicron retention disease Defective chylomicron secretion

4.

The liver synthesizes triglycerides and exports them as VLDL. The defects in ApoB or MTP prevent export, leading to triglyceride accumulation. This causes oxidative stress, inflammation, and fibrosis, resulting in histological non-alcoholic steatohepatitis (NASH). Thus, low cholesterol may be causal, not just secondary, to liver injury.

5. Differentiating congenital vs acquired:

Congenital: Early-onset, family history, genetic mutation confirmation, lifelong low cholesterol
Acquired: Secondary to liver disease, malnutrition, hyperthyroidism, or systemic illness; onset later in life; may improve with treatment of the underlying cause

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New-Onset Bony Lesion in a Patient with Chronic Kidney Disease

Dr. B.G.D. Pradeepa
Registrar in Chemical Pathology
National Hospital of Sri Lanka

Case Presentation

A 59-year-old male was found to have elevated serum creatinine during a routine medical check-up two years ago. He was diagnosed with hypertension and dyslipidemia and was referred to the renal clinic for follow-up of stage 3a chronic kidney disease (CKD).

In April 2025, he was admitted to the local hospital with a perianal abscess. During the hospital stay, he developed acute kidney injury (AKI) on top of pre-existing CKD. Once he complained of left upper chest pain, examination revealed a 2.5–4 cm lump over the left clavicle, and a chest X-ray showed a lytic lesion in that area. Biopsy of the clavicular mass was taken.

Haematology	Results	Unit	Reference range
White cell count	6.58	10^9/L	4 - 11
Neutrophils	5.67	10^9/L	2 -7
Lymphocytes	0.74	10^9/L	0.8 - 4.0
Red cell count	3.22	10^{12}/L	3.5 – 5.5
Hemoglobin	9.7	g/dL	11 – 16
Hematocrit	29.8	%	37– 54
Mean corpuscular volume	92.7	fL	80 – 100
Mean corpuscular Hb	30.1	pg	27 – 34
Platelets	229	10^9/L	150 – 450
Erythrocyte Sedimentation Rate	116	mm/hour	
Blood Picture	Normochromic normocytic red cells with moderate rouleaux formation.		
Biochemistry			
Serum sodium	141. 2	mmol/L	135 – 145
Serum potassium	4.39	mmol/L	3.5 – 5.1
Albumin	3.46	g/dL	3.8 – 5.5
Corrected calcium	3.03	mmol/L	2.15 - 2.57
Magnesium	1.02	mmol/L	0.70 – 0.99
Phosphorus	0.78	mmol/L	0.87 – 1.45
Alkaline phosphatase	111	U/L	40 – 129
Serum creatinine eGFR	1.65 46	mg/dL ml/min/1.73 m^2	0.7 – 1.2 56 – 130

Blood Urea	20.26	mg/dL	
Urine full report	Protein +++		
Urine Protein/ Creatinine Ratio	2954.81	mg/g creatinine	< 150: Normal
ALT	14.2	U/L	0 – 45
AST	19.3	U/L	0 – 40
Total bilirubin	0.601	mg/dL	0.3 – 1.2
Total protein	9.16	g/L	6.0 – 8.0
FBS	91.79	mg/dL	75 - 115
LDH	205	U/L	0 - 200

Questions

1. What are the significant laboratory findings in this patient?
2. What further investigations should be performed?
3. What is the most probable diagnosis in this patient?
4. What are the diagnostic criteria for multiple myeloma according to the International Myeloma Working Group?
5. Which investigations are used to assess prognosis in this condition?

Answers

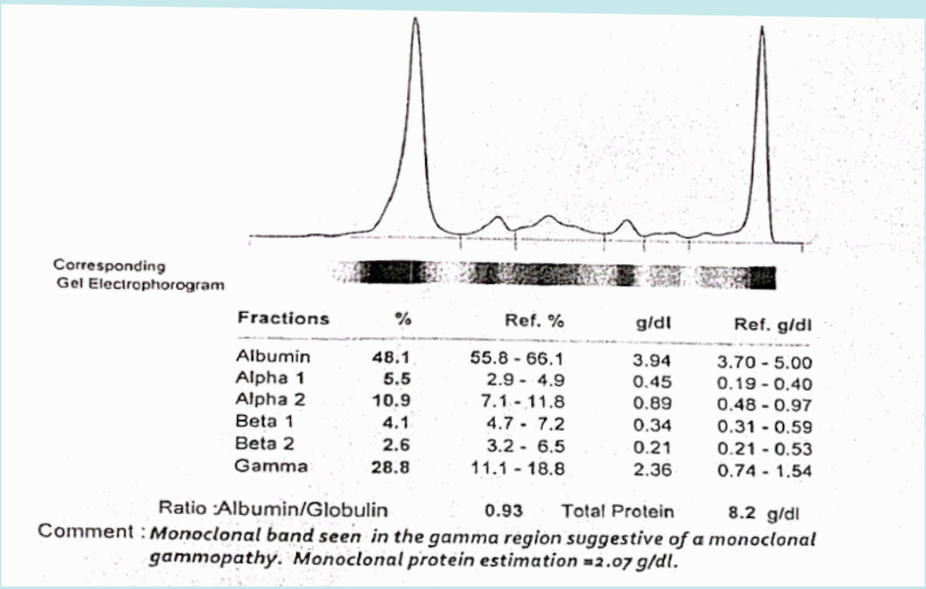
1. What are the significant laboratory findings in this patient?
Normocytic normochromic anemia and rouleaux formation in peripheral blood smear
Renal impairment with Proteinuria
Hypercalcemia, hyperproteinemia and hyperglobulinemia
Markedly elevated ESR

2. What further investigations should be performed?
Serum protein electrophoresis and immunofixation
Serum free light chain assay

Urine protein electrophoresis and immunofixation
Bone marrow aspiration and trephine biopsy
Skeletal survey / whole body low-dose CT / PET-CT or MRI

His Further investigations are as follows.

1. Serum Protein Capillary Electrophoresis



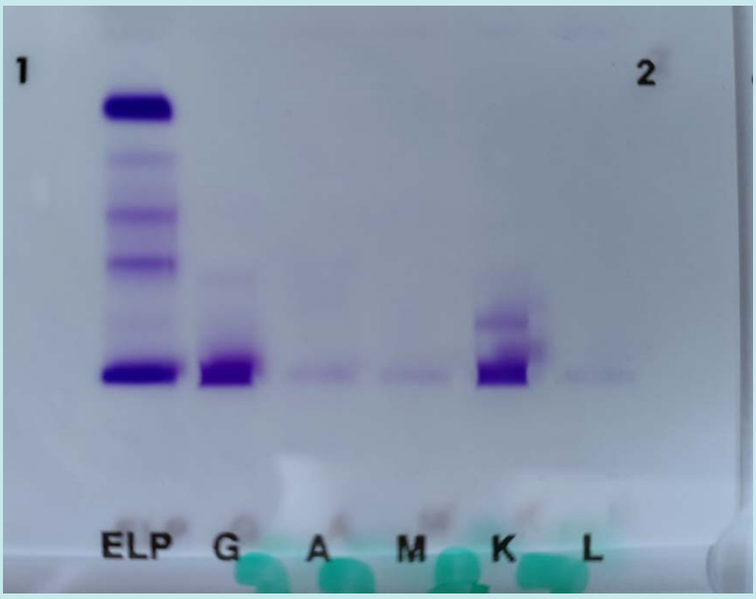
Corresponding Gel Electrophorogram

Fractions	%	Ref. %	g/dl	Ref. g/dl
Albumin	48.1	55.8 - 66.1	3.94	3.70 - 5.00
Alpha 1	5.5	2.9 - 4.9	0.45	0.19 - 0.40
Alpha 2	10.9	7.1 - 11.8	0.89	0.48 - 0.97
Beta 1	4.1	4.7 - 7.2	0.34	0.31 - 0.59
Beta 2	2.6	3.2 - 6.5	0.21	0.21 - 0.53
Gamma	28.8	11.1 - 18.8	2.36	0.74 - 1.54

Ratio :Albumin/Globulin 0.93 Total Protein 8.2 g/dl

Comment : *Monoclonal band seen in the gamma region suggestive of a monoclonal gammopathy. Monoclonal protein estimation =2.07 g/dl.*

Serum immunofixation



Free Light Chain Assay

Test	Result	Unit	Flag	Reference Range
Kappa(K) Light Chains	3420.00	mg/L	H	6.7 – 22.4
Lambda(L) Light Chains	17.20	mg/L	H	8.3 – 27.0
K/L Ratio	198.84			0.26 – 1.65
A:UA >100				

Histopathology report left clavicle lump: plasmacytoma.

Bone marrow biopsy and bone marrow aspirate

Plasma cells – 35% of marrow nucleated cells are plasma cells. Atypical plasma cells including few binucleated forms and plasma blasts seen.

Flowcytometry report

These plasma cells are CD19 negative, CD56 positive, CD27 negative and cytoplasmic Ig Kappa restricted.

HRCT of Chest: multiple lytic lesions.

3. What is the most probable diagnosis in this patient?
   Multiple Myeloma presenting with a solitary plasmacytoma of the left clavicle.

4. What are the diagnostic criteria according to the International Myeloma Working Group?

   A. Clonal bone marrow plasma cells ≥ 10% OR biopsy-proven plasmacytoma,
   AND
   B. At least one myeloma-defining event, which includes:

CRAB features:

C – Hypercalcemia: serum calcium > 2.75 mmol/L or > 0.25 mmol/L above upper normal limit

R – Renal failure: serum creatinine > 2.0 mg/dL or eGFR < 40 mL/min

A – Anemia: hemoglobin< 10 g/dL or > 2 g/dL below normal

B – Bone lesions: one or more lytic lesions on imaging

OR

Myeloma-defining biomarkers:

   ≥ 60% clonal plasma cells in the bone marrow
   Serum involved/uninvolved free light chain ratio ≥ 100
   1 focal lesion ≥ 5 mm on MRI

5. Which investigations are used to assess prognosis in this patient?

   Serum β_2-microglobulin level

   Serum albumin

   Cytogenetic and FISH studies

   LDH level

   Plasma cell labelling index or proliferation markers

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The Image tells a story

Strengthening Diagnostic Accuracy: The Laboratory's Role in Uncannulated AVS

Siddiqa Ozaal[1], Thayani Arooran[1], Janeesha Liyanagunawardane[1], Sivatharshana Prashanthan[1], Pradeepa Gamage[1], Manilaka Sumanatilake[2], Gaya Katulanda[1]

[1] Department of Chemical Pathology, National Hospital of Sri Lanka
[2] Endocrinology Unit, National Hospital of Sri Lanka

A 54-year-old man with hypertension since 2022 presented with hypokalemic periodic paralysis (K^+ 1.8 mmol/L). His overnight dexamethasone suppression test showed appropriate suppression (33.25 nmol/L). Laboratory evaluation demonstrated elevated aldosterone (36.5 ng/dL) with low renin activity (0.6 ng/mL/hr), giving an aldosterone-renin ratio of 730, strongly suggestive of primary aldosteronism. CT imaging identified a left adrenal adenoma, and adrenal venous sampling (AVS) was peformed for lateralization.

ADRENAL VENOUS SAMPLING - STIMULATED

Site	Aldosterone (ng/dL)	Cortisol (nmol/L)	Aldo/Cor Ratio	Selectivity Index
R/Adrenal Vein	188.7 [2]	17 508 [1]	0.011	42.8
L/Adrenal Vein	4 545.0 [3]	1 928	2.357	4.7
Femoral vein	47.0	409	0.115	

[1] 1: 10 Dilution
[2] 1: 50 Dilution
[3] 1: 100 Dilution

Questions

1. What is the rationale for using cosyntropin-stimulated AVS?
2. Comment on the adequacy of adrenal vein cannulation.
3. How can issues with cannulation be addressed?

Answers

Cosyntropin-stimulated AVS offers several advantages: it stabilizes aldosterone secretion, minimizes stress-related hormonal fluctuations, and creates a stronger adrenal-to-peripheral vein cortisol gradient. These effects provide a clearer biochemical contrast between the glands and increase confidence that each catheter has reached the adrenal vein.

Despite these benefits, the selectivity index (SI)-the ratio of adrenal vein cortisol to Peripheral vein cortisol—revealed an important limitation in this study. Under stimulation, an SI ≥5:1 confirms adequate cannulation. The right adrenal vein achieved this threshold; however, the left adrenal vein did not, indicating unsuccessful cannulation and preventing direct interpretation.

When SI fails to provide bilateral selectivity, the contralateral suppression index (CSI- Aldosterone: cortisol in the cannulated adrenal vein / Aldosterone: cortisol in the peripheral vein) becomes an invaluable interpretive tool. A CSI <0.5 strongly supports unilateral aldosterone excess. This patient's CSI of 0.095 demonstrates marked suppression of the right adrenal, confidently indicating a left-sided aldosterone-producing lesion-fully consistent with imaging.

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Chemical Pathology Crossword Puzzle - 2025

Authors

Kavindya Fernando[1,2], Charuni Rathnayaka[1], Miflar Meera Mohideen[1], Waruni Dahanayaka[1], Thamara Herath[1]

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

[2]Department of Biochemistry and Clinical Chemistry, Faculty of Medicine, University of Kelaniya, Sri Lanka

Instructions

Match each clue to a biochemical concept. Good luck!

Clue List

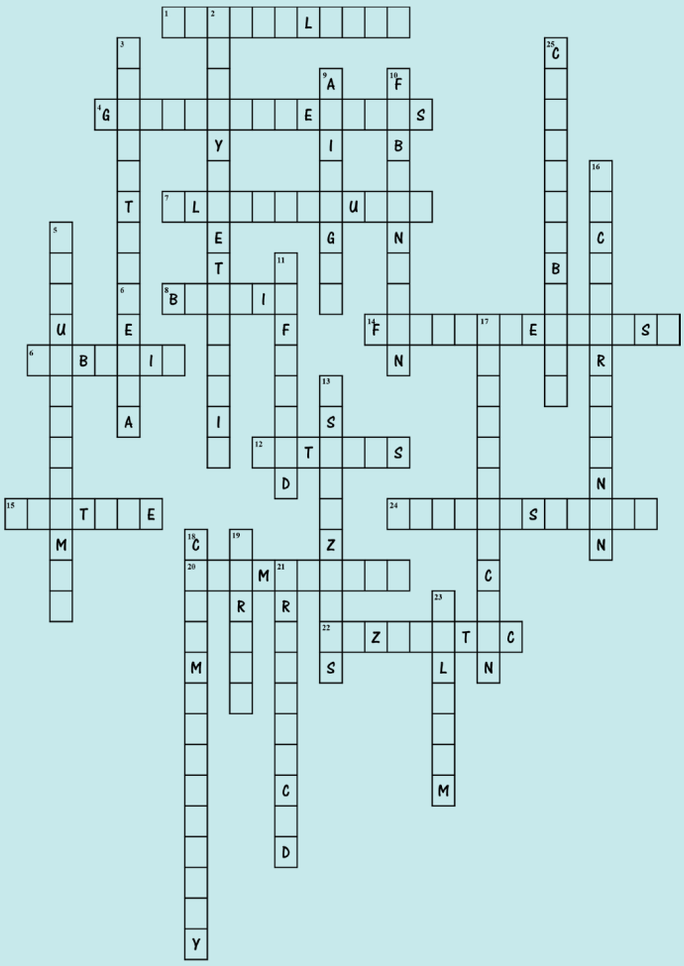
ACROSS

1. The acute-phase protein that falls when red cells explode.
4. When glycogen quits, this pathway clocks in
6. The protein that hides the real number until you correct for it.
7. The disease that leaves its mark in black.
8. The vitamin that can turn positive results negative, and vice-versa.
12. Three acids walk into DKA, and only two shows up on nitroprusside.
14. Haem synthesis choke-point famously vulnerable to lead.
15. Hexagons in the urine sediment.
20. A potassium-raising disaster you can create with one rough venepuncture.
22. Preferred type of methodology for creatinine in icteric samples.
24. Reflects glycaemia over roughly 2–3 weeks via ketoamine formation.

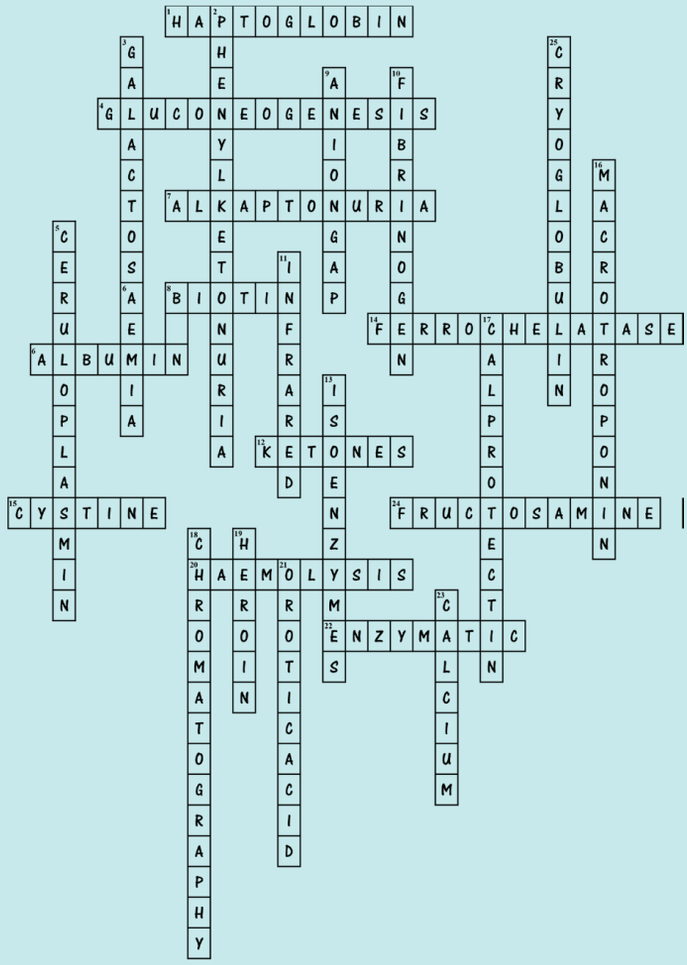
DOWN

2. Blonde hair, blue eyes, but not always Scandinavian.
3. Neonate with “sugar” in urine but a perfectly normal glucometer.
5. Low levels of this protein support a diagnosis of copper overload disorder.
9. Index revealing unmeasured anions
10. The ghost band that shows up when your “sample” came from an EDTA tube.
11. The spectrum that reveals what your kidney stone is made of.
13. Same job, different uniforms and ALP knows this well.
16. When high troponin is real, but the injury isn’t.
17. The neutrophil’s calling card in stool.
18. The “organiser” that tidies your sample before mass spectrometry arrives.
19. The opioid with a 6-monoacetylmorphin fingerprint.
21. When urea cycle traffic jams, this metabolite escapes into urine.
23. Ionised form rises when pH goes down and vice versa
25. Protein “snowfall” at low temperatures causing spurious results unless samples are kept warm.

The Crossword Grid



Answer Keys



Email: colchempath@gmail.com Tel: +94 (0) 76 704 4871 Website: ccpsrilanka.com

Activities in Brief (2025/ 2026)

Webinar

On 12th February 2025, a webinar was conducted by Mrs W.D.V.Karunaratne, government analyst, on "Toxicological Analysis", which identifies harmful substances in body fluids (blood, urine) to diagnose poisoning, manage overdoses, monitor therapeutic drugs, and support legal cases.

On 19th March 2025, a webinar was hosted by the College of Chemical Pathologists in Sri Lanka on"Stool Examination-How can the Pathologist help the Clinician?" The session was conducted by Dr Sanath Senanayake, Consultant Parasitologist and Senior Lecturer, Faculty of Medicine, University of Colombo.

Hybrid event

On 21st March 2025, a hybrid event took place at the conference hall, OPD complex of the National Hospital, Sri Lanka (NHSL). The event featured Dr Bryan Shine, Consultant Chemical Pathologist, Department of Clinical Biochemistry, Oxford University Hospitals, United Kingdom, who delivered an insightful lecture titled "Electrolyte Disorders". The lecture covered a wide range of electrolyte disorders and their clinical implications.

Workshops

An Immunoassay workshop was held on 24th April 2025, which consisted of multiple essential aspects of immunoassay, including the overview of immunoassay, standardization and free hormone assays. It further included 17OHP assay, free light chain assay, Tacrolimus assay and Aldosterone: Renin Ratio (ARR).

On 25th April 2025, Laboratory Quality Management was organized by the College of Chemical Pathologists of Sri Lanka, which took place at the Academic centre of Postgraduate Institute of Medicine. The workshop included essential topics on day-to-day practice, such as laboratory instrument calibration, clinical waste management, CEA regulations, laboratory grade water testing and post-collection clinical waste management.

On 09th June 2025, a workshop on sample collection took place at the Nephrology auditorium of National Hospital, Galle, which covered the important topics on accuracy of chemical pathology testing, risk management in the preanalytical phase, sample collection and handling of special tests and the role of quality indicators in reducing pre-analytical errors.

On 29th August 2025, a workshop was held at the Academic centre of Postgraduate Institute of Medicine, titled "From Manual to Fully Automated Urine Analysis". The keynote speaker was Prof. Surgio Bernadini, IFCC secretary, full Professor in Clinical Biochemistry and Clinical Molecular Biology at the University of Tor Vergata Hospital, Rome, Italy. The workshop started with the fundamentals of urine analysis and bridging from manual to fully automated urine analysis.

Educational workshop on Portfolio Writing was held on 24th Oct 2025 at the Academic centre of Postgraduate Institute of Medicine. The workshop covered the precise way of writing the portfolio.

Medical Laboratory Science programme

College of Chemical Pathologists of Sri Lanka successfully hosted a Medical Laboratory Science program on 7th March 2025 on sample collection at the auditorium of Sri Lanka College of Obstetricians & Gynaecologists, No 112, Model Farm Road. Colombo 8. It provided advanced professional knowledge and skills in the field.

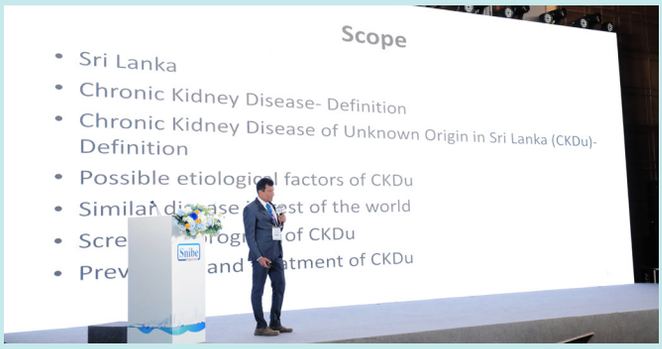
Inter University Quiz
The College of Chemical Pathologists of Sri Lanka organized an Inter university Quiz on Chemical Pathology for the first time. It's a great commencement in the history of the College of Chemical Pathologists' activities. Nine Medical faculties of Sri Lanka participated in the event. The winners of the quiz, University of Colombo (1st place), Kothalawala Defence University (2nd place) and University of Kelaniya (3rd place) were honoured at the Inauguration ceremony of the Annual Academic Sessions of the College of Chemical Pathologists.

Sri Lankan Representation at Overseas Forums:
Dr. Manjula Dissanayake was invited as a distinguished guest speaker twice to present at international forums. On 1st February 2025, he presented on"Pitfalls in interpretation of Endocrine Tests" at the Medical Biochemistry Conference (MBCON 2025) in Bangladesh, organized by the Bangladesh Society of Medical Biochemists.On 21st June 2025, at the Global LabTalk Forum in China, he delivered an insightful lecture on the topic "17OHP: Current Status and Future Perspectives in Sri Lanka".

Dr. Thushara Hewageegana was invited as a distinguished guest speaker to present at the International Symposium on Laboratory Medicine in Kunming, China, on 13th April 2025, where he delivered an enriching lecture on "Chronic Kidney Disease of Unknown Origin in Sri Lanka".

International Achievements:
The Chemical Pathology team of the National Hospital of Sri Lanka (NHSL) won first place at the IFCC Global MedLabWeek 2025 under the theme Labs Save Lives - video competition and again third place at APFCB, Global MedLabWeek 2025.

Dr. Nesali Panapitiya's podcast, "The Silent Mission of Biochemistry," won second place in the IFCC Global MedLab Week 2025 podcast competition.



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All teams:
Endocrine Medicine Surgery Chem Path වැනි..
Hospital එකේ එකටම වැඩ කරනා අපි..
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Clinical team:
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casualty, clinic , ward round පැය හතරක් යති..
emergency එකක් ඕන වෙලේ වෙන්නහැකි..
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Chem path team:
පැය හතරක් ward round යනකොට හරිහැටි,
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electrolytes , acid base cases හරිහැටි..
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electrolytes , acid base cases හරිහැටි..

Clinical team
බේත් දෙන්නෙ නෑ..
Casualty ඇත්තෙනෑ..
Emergency නෑ..night duty ඇත්තෙ නෑ…

Chem path team
ඒත්
Chem path lab නැත්තම් hospital දුවන්නෑ…//

Clinical team:
Endocrine ලෙඩ්ඩු දවසට සියක් එනව..
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Chem path team:
Endocrine පොත් පිරෙන්න reports තියෙනවා..
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Important patients ලාහොදට දන්නවා

Clinical team
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Casualty ඇත්තෙනෑ..
Emergency නෑ..night duty ඇත්තෙනෑ…

Chem path team
ඒත්
Chem path lab නැත්තම් hospital දුවන්නෑ…//

Clinical team:
ඉර අව්වක් දකින් නැතුව surgery කරනවා,
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Chem path කරන අපේ wife shape කරනවා…
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Chem path team:
උදේ අට්ට වැඩට ඇවිත් හතර වෙනතුතුරුත් ඉන්නවා..
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Clinical team:
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Casualty ඇත්තෙනෑ..
Emergency නෑ..night duty ඇත්තෙනෑ…

Chem path team:
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Chem path lab නැත්තම් hospital දුවන්නෑ…//

Chem path team:
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Authors
Dr. Dilini Jayasekara Senior Registrar National Hospital Colombo
Dr. Pavithra Samarakoon Senior Registrar National Hospital Colombo

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