JoTH Journal of Tropical Health

Perspectives

When Observation Became Evidence: The Enduring Value of Case Reports in Clinical Medicine (Anuradhapura Medical Association Oration 2025)

Sisira Siribaddana

Department of Medicine, Faculty of Medicine and Allied Sciences, Rajarata University of Sri Lanka, Saliyapura 50008, Sri Lanka.

Summary

Case reports and case series have long been the foundation of clinical observation, serving as crucial signals for new diseases, unusual manifestations of known conditions, rare adverse drug reactions, and unforeseen complications. This review illustrates how bedside observations become scientific evidence, highlighting discoveries that shaped medical understanding across infectious, toxicological, nephrological, and genomic domains. One early example described a patient with tuberculosis-associated autoimmune haemolytic anaemia (AIHA), a rare but significant immune-mediated complication. Our systematic review confirmed that while AIHA is uncommon, TB-induced AIHA is even rarer, with distinctive clinical and laboratory profiles. In contrast, tuberculous pericarditis represents another immunopathological spectrum, driven not by antibodies but by delayed-type hypersensitivity, leading to characteristic haemorrhagic, non-clotting pericardial effusions. Recognition of these distinct immunological pathways has direct therapeutic implications, including the use of steroids as adjuvants in pericarditis but not always in AIHA. Another set of bedside observations arose from an environmental disaster at a glove manufacturing plant in Sri Lanka, where workers developed acute systemic symptoms traced to latex transported in barrels previously containing toluene diisocyanate. This investigation uncovered previously unreported muscle involvement in isocyanate toxicity, echoing historical examples of tri-cresyl phosphate–related neuropathy and underscoring the dangers of chemical container reuse. Turning to nephrology, the search for the cause of chronic interstitial nephritis among agricultural communities (CINAC) has been shaped by case reports that helped avoid blind alleys. An exertional heat stroke case in a young military trainee initially suggested that repeated subclinical heat stress might underlie CINAC; however, biomarker studies in children and systematic reviews challenged this hypothesis, pointing instead toward environmental toxins as primary drivers, with heat as an aggravating factor. Further prospective studies, such as the Anuradhapura Snakebite Cohort, clarified that while snake envenoming causes acute kidney injury, it does not contribute to CINAC, with chronic kidney disease in this population more strongly linked to comorbidities and environmental exposures. A series of patients with distal renal tubular acidosis co-existing with Southeast Asian ovalocytosis in Rajarata provided another window into tubular disorders, revealing genetic underpinnings in SLC4A1 mutations but also prompting comparisons with CINAC, where histopathology suggests toxin-induced proximal tubular injury akin to calcineurin inhibitor nephrotoxicity. Finally, observations at the bedside of young patients with necrotising pneumonia led to genomic investigations that traced a highly virulent, Panton-Valentine leukocidin–positive MRSA clone circulating in Sri Lanka and globally. Whole-genome sequencing demonstrated its dominance across both community and healthcare settings, linking clinical severity with molecular epidemiology and highlighting blurred boundaries between hospital- and community-acquired infections. Attentive clinical observation, when combined with systematic follow-up and modern tools, can transform individual case reports into evidence that advances medical understanding. Such reports bridge bedside practice with scientific discovery, shaping research, therapy, and health policy.

***Keywords:** Case report, Clinical medicine, Evidence-based medicine

Funding: None

Competing interests: None
Received: 04 November 2025 **Accepted:** 13 November 2025 **Published:** 24 November 2025

Corresponding author: Email: sisira.siribaddana@gmail.com https://orcid.org/0000-0001-5821-2557

Cite this article as: Siribaddana S. When Observation Became Evidence: The Enduring Value of Case Reports in Clinical Medicine (Anuradhapura Medical Association Oration 2025). Journal of Tropical Health 2025;1 (3): 4-12. DOI: http://doi.org/10.4038/joth.v1i3.38

Introduction

Case reports and series play a foundational role in clinical medicine. They serve as early signals for new diseases, unique presentations of known conditions, rare adverse drug reactions, or unforeseen complications (Table 1).

Case reports contribute to hypothesis generation, alert clinicians to emerging trends, and can rapidly disseminate crucial bedside observations, especially in resource-limited or rapidly evolving clinical settings. By revisiting case reports and series, this review illustrates how observation becomes evidence, fueling discovery, deepening understanding, and refining the practice of medicine.

Table 1: Landmark case reports that shaped clinical medicine

Category	Example	Clinical insight	Reference
New diseases	AIDS	First reports of Pneumocystis pneumonia led to discovery of AIDS.	CDC, MMWR, 1981;30(21):250-252
Unique presentation	MI presenting as ear pain	MI was diagnosed in a patient with left ear pain.	Rothwell PM. PMJ 1993;69(810), 300-301. doi:10.1136/pgmj.69.810.300
Rare adverse drug reaction	Thalidomide embryopathy	Early 1960s case series linked thalidomide to severe limb deformities.	Lenz W, Lancet, 1962;279(7219):45-46. doi:10.1016/S0140-6736(62)92401-0
Unforeseen complication	VITT after COVID-19 vaccine	A case series revealed vaccine-induced immune thrombosis	Greinacher A, NEJM, 2021;384(22):2092-2101. doi:10.1056/NEJMoa2104840

AIDS; acute immune deficiency syndrome, MI; myocardial infarction, VITT; vaccine-induced thrombotic thrombocytopenia

Contrasting immunopathogenesis in tuberculosis: Delayed hypersensitivity pericarditis versus autoimmune haemolytic anaemia (AIHA)

A 37-year-old man presented with central chest pain, shortness of breath, anaemia, and icterus. An initial diagnosis of AIHA was made, but the patient requested discharge. One month later, he was admitted with high remittent fever and a palpable, tender submental lymph node. His haemoglobin was 6.6 g/dl, and the reticulocyte count was 10%. Investigations revealed polyclonal gammopathy and negative results for the *Mycoplasma test*, Paul-Bunnell test, and Donath-Landsteiner test. Direct and indirect antiglobulin tests against polyspecific antisera were also negative. Cold agglutination titres were high, at 4°C (1024 anti-i adult, 128 anti-i cord). The biopsy of the submental lymph node was compatible with a diagnosis of tuberculous (TB) lymphadenitis. Fever subsided within four days of starting anti-TB therapy. By December 1993, his haemoglobin had risen to 12 g/dl, and the reticulocyte count decreased to 2%. The patient did not receive any steroids [1]. While this single case highlights the unusual occurrence of TB-associated cold-antibody haemolytic anaemia, a broader review of the literature helps to contextualise its frequency, clinical spectrum, and treatment outcomes.

TB-induced AIHA is a rare complication, with all reported cases showing a positive direct Coombs test. The exact mechanism is unclear, but it is likely to involve an altered immune response triggering IgG or IgM antibodies, leading to warm, cold, or mixed-type AIHA. Although AIHA itself is uncommon, TB-induced AIHA is even rarer, mainly reported from India and Southeast Asia, reflecting the high TB burden. We conducted a systematic review on TB-induced AIHA (Figure 1).

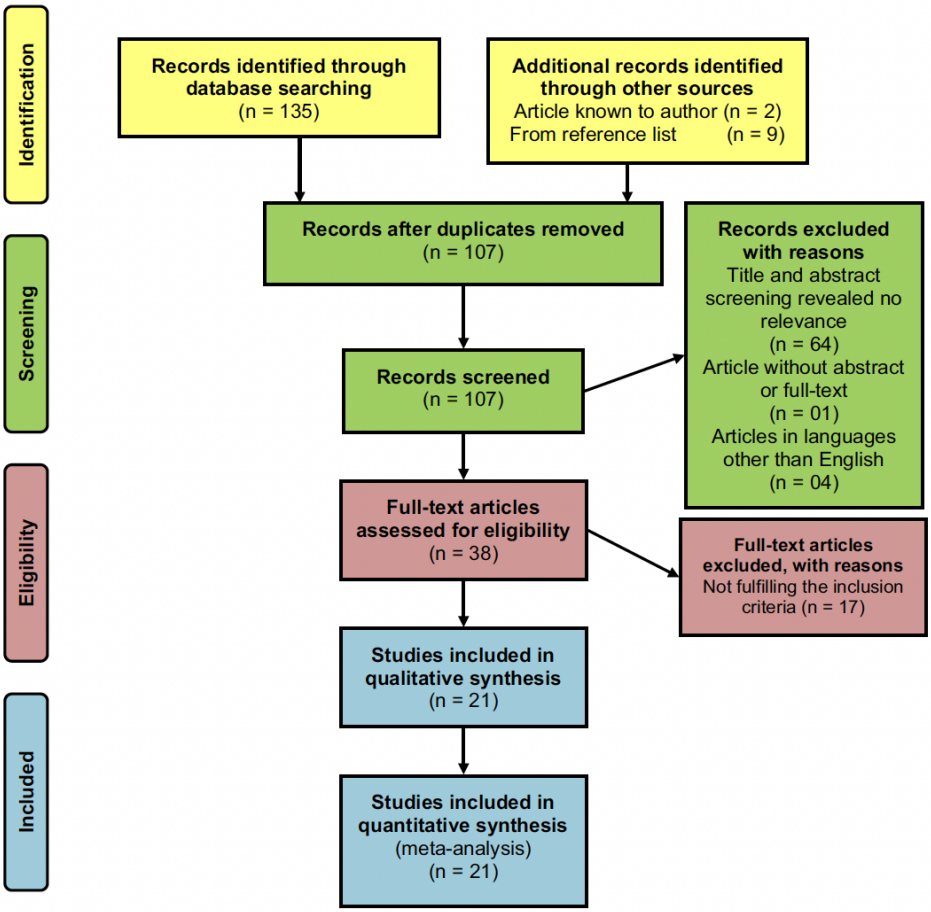

Figure 1: PRISMA flow diagram illustrating the article selection process.

Clinically, most patients present with fever (81%), pallor (62%), and symptoms like chronic cough (48%) or weight loss (38%). Unlike typical TB, hepatosplenomegaly may suggest AIHA. Lab findings reveal severe anaemia (mean haemoglobin 5.77 g/dL), elevated reticulocytes, LDH, and bilirubin, along with low haptoglobin. Radiologically, chest X-rays often show TB-related abnormalities (82%), while CT scans may reveal lymphadenopathy (71%). Pulmonary TB is the most common site (43%), followed by disseminated TB (29%). Treatment involves anti-TB drugs, with nearly half requiring blood transfusions. Steroids are used in 38% of cases, particularly for disseminated TB-induced AIHA (67%). Surprisingly, cold-type AIHA required steroids more often (33%) than warm-type (14%) [2].

These antibody-mediated mechanisms contrast sharply with another significant immune-mediated complication of TB, namely tuberculous pericarditis, where tissue damage arises predominantly through a delayed-type hypersensitivity reaction rather than direct autoantibody production. The pericardial fluid in tuberculous pericarditis is typically blood-stained and nonclotting, due to fibrinolytic activity and immune-mediated mechanisms.

Tuberculous pericarditis is a common cause of pericardial effusions in Sri Lanka. Diagnosis can be challenging, as pericardial aspirates for acid-fast bacilli and Mantoux yield negative results. In such cases, antituberculosis drugs can serve as a diagnostic aid. Steroids are a vital adjuvant therapy, typically initiated after the fever has responded to antituberculosis drugs. Clinical trials have shown that adding steroids significantly increases the recovery rate, reduces the risk of death, and decreases the need for pericardiectomy [3]. Taken together, these observations underscore two distinct but clinically meaningful immune pathways in TB: the antibody-mediated cytotoxicity driving AIHA and the T-cell-mediated delayed hypersensitivity driving pericarditis (Table 2).

Table 2: Contrasting immunopathogenic mechanisms in tuberculous pericarditis and tuberculosis-associated autoimmune hemolytic anaemia

Feature	TB pericarditis	TB-associated AIHA
Immune reaction type	Type IV (cell-mediated, delayed hypersensitivity)	Type II (antibody-mediated cytotoxicity)
Main players	Th1 cells, macrophages, IFN-γ, granulomas	B cells, plasma cells, IgG or IgM autoantibodies, and complement
Pathogenesis	Persistent mycobacterial antigens trigger $CD4^+$ T-cell activation, macrophage recruitment and finally granulomas & fibrosis	TB infection triggers loss of tolerance or molecular mimicry, with autoantibodies binding RBC surface antigens & complement activation or splenic macrophage-mediated destruction
Evidence	Pericardial fluid is haemorrhagic and does not clot	Positive direct antiglobulin (Coombs) test

An environmental disaster: A case series

In early 1994, a quiet surgical glove manufacturing plant in Homagama became the scene of an unexpected health crisis. On January 6, 32 employees were rushed to Sri Jayewardenepura General Hospital, presenting with headache, burning sensations in their eyes, and muscle pain. Over the next few days, eight more workers were admitted, raising concerns about an occupational cause for their sudden illness.

We suspected a problem within the factory. Forty patients, aged 17 to 41 years, all working in the manufacturing and storage sections, were affected. Interestingly, 11 employees from the accounts department and two workers on leave remained unaffected, pointing strongly to an issue on the factory floor.

Inspection of the factory revealed a crucial detail: the barrels used to transport latex from the estates had been recently changed. These barrels had previously contained toluene diisocyanate (TDI). Despite clear warning labels advising against reuse, these barrels had been washed and repurposed for transporting latex. The workers had even noticed a change in the latex itself, a different smell, colour, and delayed drying, though they couldn't identify the reason. Chemical analysis of the latex was performed at the Ceylon Institute of Scientific and Industrial Research using infrared spectroscopy, based on the N = C = O stretching vibration. A mixture of 2,4 TDI and 2,6 TDI in the latex was extracted with o-dichlorobenzene, and the gas extract was analysed by gas chromatography with a flame ionisation detector. The affected workers exhibited mucosal irritation of the gastrointestinal and respiratory tracts, skin inflammation, and conjunctival irritation. Some even experienced neurological symptoms such as euphoria, ataxia, and memory loss. A notable finding was that 32 of the 40 patients experienced muscle pain, and seven showed elevated creatine phosphokinase activity, indicating striated muscle involvement. Muscle involvement had not been previously reported in TDI toxicity. Production at the glove plant resumed with fresh latex transported in new barrels; no workers subsequently fell ill. The highly reactive nature of isocyanates, due to their N=C=O group, is responsible for their intense irritative action, rather than that of hydrogen cyanide [4].

In a chilling parallel, Methylene diphenyl diisocyanate gas, another isocyanate, accidentally escaped from a storage in the Bhopal pesticide manufacturing plant, causing around 8000 deaths [5].

This highlights the importance of preventing the reuse of containers that have previously held toxic substances. In the late 1970s, a series of patients in Sri Lanka's Central Province developed toxic polyneuropathy after consuming sesame seed oil that had been stored in containers contaminated with tri-cresyl phosphate [6].

Avoiding blind alleys: Case reports guiding the search for the cause of chronic interstitial nephritis among agricultural communities (CINAC) in rajarata

1. Heat stroke, acute kidney injury, and the debate on CINAC aetiology

Heat stroke is a life-threatening condition defined by a core body temperature exceeding 40 °C with central nervous system dysfunction. It occurs in two forms: classical heat stroke and exertional heat stroke. We describe a case of a 23-year-old military trainee who developed early renal failure, liver failure, and rhabdomyolysis after participating in a 9-mile ruck march in hot, humid conditions (30 °C, 76% humidity) [7]. Despite adhering to preventive measures, he

collapsed near the finish line with convulsions, delirium, loss of consciousness, and reduced sweating. Initial investigations suggested a possible asymptomatic bacterial infection that may have compromised physiological resilience, although this remains unproven. The case highlights that heat stroke and its complications, including acute kidney injury (AKI), can occur even in young, otherwise healthy individuals despite preventive strategies, underscoring the need for education on early recognition and first aid.

This case prompted us to consider whether repeated subclinical heat stress and dehydration could similarly explain the CINAC, seen in Sri Lanka and elsewhere. However, two of our papers cast significant doubt on the heat-stress hypothesis. The first is a review that critically examines the evidence and finds little support for the idea that recurrent, mild AKI from occupational heat exposure drives CINAC [8]. We noted that global warming has not caused comparable epidemics of kidney disease in other hot climates, that AKI among manual workers is rarely severe enough to progress to chronic kidney disease, and that alternative etiologies, such as agrochemicals, heavy metals, and fluoride, warrant greater scrutiny.

Further undermining the heat stress theory, we studied schoolchildren in Sri Lanka's dry zone, comparing those with high versus low outdoor heat exposure [9]. They found no significant differences in urinary markers of kidney injury (kidney injury molecule one — KIM-1, albumin creatinine ratio — ACR). However, neutrophil gelatinase-associated lipocalin (NGAL) levels varied, possibly due to exercise-induced muscle stress. Importantly, urinary KIM-1 was consistently higher in children from CINAC-endemic areas, regardless of heat exposure, suggesting environmental or geographic factors unrelated to heat stress.

Recent methodologically sound meta-analysis comes to the following conclusions "There was no consistent evidence from our systematic review to support the association between chronic kidney disease and heat stress–dehydration. Although heat stress and dehydration as causes of chronic kidney disease have some biological plausibility, to the best of our knowledge, there are no studies that indisputably show that they are the single or preponderant cause of the onset of the so-called chronic kidney disease of non-traditional aetiology" [10].

Taken together, our case report demonstrates how acute heat stress can clearly damage the kidneys in susceptible settings; however, the epidemiological and biomarker evidence from Sri Lanka argues against heat exposure as the central driver of CINAC. Instead, heat may act as a secondary aggravating factor, with environmental nephrotoxins more likely playing a primary causal role. This linkage from an individual case of exertional heat stroke to population-level investigations illustrates how bedside observation can stimulate broader inquiry and how careful research can help exclude an initially plausible but ultimately insufficient hypothesis.

2. *Does snakebite-associated acute kidney injury contribute to CINAC or chronic kidney disease (CKD)? Evidence from the Anuradhapura snakebite cohort*

Russell's viper and Merrem's hump-nosed pit viper are known to cause AKI. Four patients presented to THA with renal manifestations following snake envenomation: Patient 1 with immediate and prolonged anuria, Patient 2 with delayed anuria, both requiring dialysis and with one leading to fatality, Patient 3 and Patient 4with clear signs of CKD maintained normal urine output without progressing to AKI, despite elevated creatinine and other complications. All four had previously documented high creatinine or contracted kidneys in ultrasound [11].

We investigated the long-term kidney effects of snake envenoming to explore the possibility of developing CKD [12]. While previous reports suggested that AKI from snake envenoming could progress to CKD [13,14], particularly from viperine snakes in Sri Lanka and India, there were no long-term follow-ups. Rajarata has a high incidence of both snakebites and CINAC. We aimed to determine whether snakebite-associated AKI leads to CKD and whether snakebite is associated with CINAC by prospectively following two groups of confirmed snakebite patients from the Anuradhapura Snakebite Cohort: 199 patients in Group I, reviewed after four years, and 168 patients in Group II, reviewed after one year. The study found no significant association between snakebite-associated AKI and the development of CKD. Among the patients who experienced AKI after snakebite,12 (6%) in Group I and 9 (5%) in Group II, all had a normal eGFR at their one and four-year follow-ups. Importantly, all nine patients across both groups who did have a low eGFR (consistent with CKD stages 3–5) at the time of review were found to have been diagnosed with CKD before the snakebite or were receiving treatment for comorbidities like hypertension or diabetes. Although microalbuminuria was common, affecting 25% of Group I and 26% of Group II patients, it was strongly associated with known comorbidities for CKD rather than snake envenoming or snakebite-associated AKI. The observed renal abnormalities, including microalbuminuria, were more likely attributed to

background conditions like hypertension, diabetes mellitus, and the endemic CINAC in this rural farming population, rather than being a direct consequence of snakebite or snakebite-associated AKI. The study found no evidence supporting an association between snakebite-associated AKI and CINAC.

3. Is it due to aristocholic acid nephropathy?

Following the emergence of CINAC, the WHO recommended regulating herbal medicines that contain aristolochic acid (AA), an established nephrotoxin, as a control measure. This recommendation is based on speculation, as the available information is inadequate to justify restricting the use of Complementary and Alternative Medicine (CAM), which has existed in Sri Lanka for over 2500 years. The use of the native species, *Aristolochia indica* ("Sapsanda"), is sparse in Sri Lankan Ayurveda, often appearing as a minor ingredient in polyherbal compounds, many of which are used externally. Crucially, the cumulative doses and duration of use in Sri Lankan CAM are much lower than the intake levels (e.g., cumulative oral intake over 150 g) known to be associated with Aristolochic acid nephropathy (AAN). Furthermore, no clinical report exists in Sri Lanka linking CKD to the consumption of Aristolochia from CAM treatment. Hence, regulating such medicines without proper data risks undermining traditional medical practices and requires rigorous research before making policy decisions. [15]

4. *Linking renal tubular disorders and CINAC: shared or non-shared pathways*

Case series and several case reports of distal renal tubular acidosis (dRTA) co-existing with Southeast Asian ovalocytosis (SAO) have been reported, all from Rajarata [16-18]. These patients present with severe hypokalemia and paralysis, with dRTA confirmed by normal anion gap, hyperchloremia, high urine pH, and a positive urine anion gap. The presence of stomatocytes and abundant ovalocytes identified SAO, a condition more commonly described in Southeast Asian countries but rarely outside Rajarata in Sri Lanka. Complications observed included CKD, medullary nephrocalcinosis, and metabolic bone disease. Familial dRTA and SAO can be co-inherited through mutations in the SLC4A1 gene, while the characteristic erythrocyte rigidity in SAO is believed to confer protection against malaria. Clinically, recurrent or chronic hypokalemia should prompt investigation for dRTA, as early diagnosis and correction of acidosis to prevent CKD progression.

Interestingly, patients with CINAC in Sri Lanka and Mesoamerica also display hypokalemia and electrolyte disturbances, raising questions about overlapping mechanisms. However, unlike inherited dRTA–SAO syndromes, the aetiology of CINAC is considered multifactorial. Substantial evidence implicates occupational and environmental exposures, particularly agrochemicals and heavy metals, as primary triggers, with heat stress and recurrent dehydration acting as contributory but insufficient factors [19]. A new perspective has emerged from renal histopathology, revealing enlarged, dysmorphic lysosomes with granular content in proximal tubular cells —a lesion highly characteristic of toxin-induced nephropathies. Strikingly, these findings resemble those observed in nephrotoxicity associated with calcineurin inhibitors, suggesting that pesticides that interfere with the calcineurin pathway may cause CINAC [20,21]. This positions CINAC as a toxin-induced proximal tubular nephropathy, distinct from the genetically determined tubular disorders seen with dRTA and SAO, yet sharing the common clinical thread of tubular dysfunction and electrolyte imbalance.

From bedside to genome: Tracking a virulent Panton-Valentine Leukocidin (PVL)-positive methicillin-resistant *Staphylococcus Aureus* (MRSA) clone in Sri Lanka

Between April & May 2013, four young, immunocompetent males presented with pneumonia to Teaching Hospital Anuradhapura (THA) [22]. These patients developed severe respiratory distress, which rapidly progressed to necrotising pneumonia, requiring assisted ventilation. Key clinical features included a preceding "flu-like" illness, acute respiratory distress, and complications such as pleural effusion, pneumothorax, cavitation and pneumatocele. Positive blood cultures confirmed the diagnosis of MRSA. Aggressive therapy with combinations of antibiotics (vancomycin, clindamycin, and linezolid) proved effective. We speculated that Panton-Valentine Leukocidin (PVL) toxin-secreting MRSA was the cause of this severe necrotising pneumonia.

In the second study, we performed genomic analysis of MRSA isolates from Sri Lanka, collected from THA between June and October 2014 [23]. This study aimed to characterise MRSA prevalence, with a particular focus on the presence of mecA and mecC genes (indicating methicillin resistance) and PVL-associated genes, using spa (Staphylococcus protein A) typing and whole genome sequencing (WGS). Key findings revealed that all 94 MRSA

isolates harboured mecA. While most isolates (76.9%) were associated with skin and soft tissue infections, a significant proportion caused invasive infections, including bacteremia (17.6%), empyema (3.3%), and osteomyelitis (2.2%). Crucially, 65.9% (62 out of 94) of the isolates were PVL-positive, and the vast majority of these (90.3%) belonged to the dominant CC5 lineage, specifically PVL-positive ST5-MRSA-IVc. This prevalent lineage was found in both community-acquired and hospital-onset infections, highlighting the increasingly blurred distinction between these settings. WGS analysis further demonstrated that representative PVL-positive ST5-MRSA-IVc isolates from Sri Lanka, England, and Australia formed a single phylogenetic clade, suggesting wide geographical circulation of this clone.

This genomic study explains the severe clinical cases of necrotising pneumonia due to community-acquired, highly virulent, PVL-positive ST5-MRSA-IVc lineage in Sri Lanka [22]. Therefore, the second study provides molecular evidence of the clinical threat posed by CA-MRSA in Sri Lanka, demonstrating the widespread presence of a highly pathogenic clone responsible for the severe outcomes observed in the first study.

The third paper provides genomic and epidemiological analysis of *S. aureus* investigating its circulation between community and healthcare settings [24]. This prospective longitudinal cohort study was conducted at the THA between July and December 2021. A total of 153 participants, including S. aureus-positive index patients and their household contacts, were recruited for the study. The study found a baseline *S. aureus* colonisation prevalence of 11.7% among healthy household contacts, with 30.8% of these isolates being MRSA. WGS of 88 isolates revealed three predominant multilocus sequence types (MLSTs): ST672 (25%), ST6 (18.2%), and ST5 (13.6%), all of which were identified in both community and healthcare environments.

We have identified a small number of strains that appear to be well-established and capable of causing both severe infections and asymptomatic colonisation. Notably, ST5 isolates exhibited a more virulent phenotype, with 100% being PVL-and mecA-positive and typically harbouring SCCmec IVc. In contrast, ST672 strains were predominantly mecA and PVL-negative.

Interestingly, the study did not detect household transmission of the index patient's infecting strain, suggesting that household decolonisation strategies may be less effective in this setting than in others.

This study provides further evidence for the persistent presence of the highly virulent PVL-positive ST5-MRSA-IVc lineage in Sri Lanka, which we identified as a dominant clone in 2014 isolates, linking it to both community- and hospital-onset infections and to its wide geographical circulation. Therefore, the genomic and epidemiological data provide a contemporary molecular basis for the aggressive *S. aureus* infections observed clinically, highlighting that PVL-positive ST5 strains, which are considered highly virulent, remain a concern in the public health landscape.

Conclusion

These examples collectively demonstrate that attentive clinical observation, coupled with systematic follow-up and modern investigative tools, transforms anecdote into evidence. Case reports not only alert us to new diseases and hazards but also refine understanding, prevent misattribution, and guide public health and therapeutic strategies. The enduring value of case reports lies in their ability to bridge bedside observation with scientific discovery, ensuring that what begins as a single patient's story can evolve into evidence shaping medical practice, research priorities, and health policy.

Acknowledgements

Almaguer M, Arias CA, Boldock E, Chandana EP, Chandana EPS, Coleman S, Cook EC, Coombs GW, Corrigan RM, Dahanayaka NJ, Darton TC, Davies S, De Broe ME, De Silva PMCS, de Silva TI, Díaz L, Dissanayake DMDIB, Farah MMF, Fernando R, Gunasekara TDKSC, Gunatilake S, Herath C, Herrera R, Isbister GK, Jayaratne KADS, Jayasinghe S, Jayasumana C, Jayasundara N, Jayaweera JAAS, Kearns AM, Locke T, Lokunarangoda N, Lopez L, McTavish SM, Mendis V, Orantes C, Ordunez P, Pang S, Pichon B, Pilapitiya SD, Premawardhana NP, Rathish D, Samarasinghe R, Sandakumari G, Sarathchandra C, Seevaamirtham R, Silva AKA, Silva LC, Snow SJ, Suligoy CM, Udukala M, Vijitharan V, Waiddyanatha S, Weerakoon K, Weerawansa MRP, Weerawansa P, Wijerathne BTB, Wijesundera A, Wimalasooriya YVM.

References

1. Siribaddana SH, Wijesundera A. Autoimmune haemolytic anaemia responding to anti-tuberculous treatment. Trop Doct. 1997;27:243-4. doi:10.1177/004947559702700424.
2. Rathish D, Siribaddana S. Tuberculosis induced autoimmune haemolytic anaemia: a systematic review to find out common clinical presentations, investigation findings and the treatment options. Allergy Asthma Clin Immunol. 2018;14:11. doi:10.1186/s13223-018-0236-y.
3. Wijesundere A, Siribaddana SH. Tuberculous pericarditis. BMJ. 1994;308:535. doi:10.1136/bmj.308.6927.535b.
4. Siribaddana SH, Wijesundera A, Fernando R. Toluene diisocyanate exposure in a glove manufacturing plant. J Toxicol Clin Toxicol. 1998;36:95-8. doi:10.3109/15563659809162594..
5. Varma DR, Mulay S. Methyl Isocyanate. In: Gupta RC, ed. Handbook of Toxicology of Chemical Warfare Agents. Amsterdam: Elsevier 2015:287-99.
6. Senanayake N, Jeyaratnam J. Toxic Polyneuropathy due to Gingili oil contaminated with tri-cresyl phosphate affecting adolescent girls in Sri Lanka.Lancet. 1981;317:88-9. doi:10.1016/S0140-6736(81)90016-7.
7. Wijerathne BTB, Pilapitiya SD, Vijitharan V, et al. Exertional heat stroke in a young military trainee: is it preventable? Mil Med Res. 2016;3:8. doi:10.1186/s40779-016-0078-1.
8. Herath C, Jayasumana C, De Silva PMCS, et al. Kidney diseases in agricultural communities: A case against heat-stress nephropathy. Kidney Int Rep. 2018;3:271-80. doi:10.1016/j.ekir.2017.10.006.
9. Gunasekara TDKSC, De Silva PMCS, Chandana EPS, et al. Environmental heat exposure and implications on renal health of pediatric communities in the dry climatic zone of Sri Lanka: An approach with urinary biomarkers. Environ Res. 2023;222:115399. doi:10.1016/j.envres.2023.115399.
10. Chapman E, Haby MM, Illanes E, et al. Risk factors for chronic kidney disease of non-traditional causes: a systematic review. Rev Panam Salud Pública. 2019;43:e35. doi:10.26633/RPSP.2019.35.
11. Silva A, Samarasinghe R, Pilapitiya S, et al. Viper bites complicate chronic agrochemical nephropathy in rural Sri Lanka. J Venom Anim Toxins Incl Trop Dis. 2014;20:1–5. doi:10.1186/1678-9199-20-33.
12. Waiddyanatha S, Silva A, Weerakoon K, et al. Does snake envenoming cause chronic kidney disease? A cohort study in rural Sri Lanka. Clin Toxicol. 2023;61:47-55. doi:10.1080/15563650.2022.2147843.
13. Herath HMNJ, Wazil AWM, Abeysekara DTDJ, et al. Chronic kidney disease in snake envenomed patients with acute kidney injury in Sri Lanka: A descriptive study. Postgrad Med J. 2012;88:138-42. doi:10.1136/postgradmedj-2011-130225.
14. Wijewickrama ES, Gooneratne L V., Gnanathasan A, et al. Thrombotic microangiopathy and acute kidney injury following Sri Lankan Daboia russelii and Hypnale species envenoming. Clin Toxicol. 2020;58:997-1003. doi:10.1080/15563650.2020.1717509.
15. Wijesinghe W, Pilapitiya S, Hettiarchchi P, et al. Regulation of herbal medicine use based on speculation? A case from Sri Lanka. J Tradit Complement Med. 2017;7:269-71. doi:10.1016/j.jtcme.2016.06.009.
16. Gunaratne W, Dissanayake D, Jayaratne K, et al. A case series of distal renal tubular acidosis, Southeast Asian ovalocytosis and metabolic bone disease. BMC Nephrol. 2020;21:327. doi:10.1186/s12882-020-01959-7.
17. Seevaamirtham R, Sarathchandra C, Senanayake H, et al. Case report on Southeast Asian ovalocytosis: A patient with type 1 renal tubular acidosis and an asymptomatic patient incidentally found. Jaffna Med J. 2019;31:38-41. doi:10.4038/jmj.v31i2.79.
18. Vidyatilake H, Gooneratne L. Two families in Sri Lanka with Southeast Asian ovalocytosis. Ceylon Med J. 2011;49:27-9. doi:10.4038/cmj.v49i1.3284.
19. Jayasumana C, Orantes C, Herrera R, et al. Chronic interstitial nephritis in agricultural communities: a worldwide epidemic with social, occupational and environmental determinants. Nephrol Dial Transplant. 2016;32:234-41. doi:10.1093/ndt/gfw346.
20. Vervaet BA, Nast CC, Jayasumana C, et al. Chronic interstitial nephritis in agricultural communities is a toxin-induced proximal tubular nephropathy. Kidney Int. 2020;97:350-69. doi:10.1016/j.kint.2019.11.009.
21. Vervaet BA, Nast CC, Schreurs G, et al. Chronic interstitial nephritis in agricultural communities: a new perspective on etiology, diagnosis and mechanism. Nephrol Ther. 2021;17:S45-50. doi:10.1016/j.nephro.2020.02.010.

22. Sandakumari G, Pilapitiya S, Dahanayaka N, et al. A case series of necrotising pneumonia due to community acquired MRSA. Anuradhapura Med J. 2014;8:40-4. doi:10.4038/amj.v8i2.7528.

23. McTavish SM, Snow SJ, Cook EC, et al. Genomic and epidemiological evidence of a dominant Panton-Valentine Leucocidin-positive methicillin resistant *Staphylococcus aureus* lineage in Sri Lanka and presence among isolates from the United Kingdom and Australia. Front Cell Infect Microbiol. 2019;9:123. doi:10.3389/fcimb.2019.00123.

24. Locke T, Siribaddana S, Jayaweera JAAS, et al. A prospective cohort study to investigate the transmission and burden of *Staphylococcus aureus* in Sri Lanka. Microb Genom. 2024;10:001336. doi:10.1099/mgen.0.001336.