Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Severe rhabdomyolysis and acute kidney injury associated with probable Legionella pneumophila infection: a case report

Willis Thompson

willis.thompson@health.qld.gov.au

Townsville Hospital

Vikas Srivastava

Townsville Hospital

Andrew Mallet

James Cook University

John Di Giusto

James Cook University

Case Report

Keywords: Legionella pneumophila, rhabdomyolysis, acute kidney injury, haemodialysis, case report.

Posted Date: August 25th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-10672561/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

Abstract

Background

Legionella pneumophila infection is an uncommon but recognised precipitator of severe rhabdomyolysis and acute kidney injury. Although this association has previously been described, additional cases have accumulated since the most recent systematic review in 2020. This report combines an atypical, predominantly extrapulmonary presentation with an updated review of subsequently published cases to highlight the evolving spectrum of *Legionella*-associated muscle and kidney injury and the diagnostic challenges that arise when alternative causes of rhabdomyolysis are plausible.

Case presentation

A previously independent 78-year-old male presented with three weeks of progressive lower-limb weakness, anorexia, weight loss, dark urine, oliguria, and a preceding flu-like illness. Examination revealed clinical dehydration and marked proximal lower-limb myopathy with preserved upper-limb strength. Initial investigations revealed severe acute kidney injury, with a creatinine level of 989 μmol/L, an eGFR of 4 mL/min/1.73 m², a urea level of 40.2 mmol/L, and a creatine kinase level of 33,400 U/L. Nephrotoxins were withheld, and the patient was treated with intravenous fluids and broad-spectrum antibiotics. Despite this resuscitation, he developed worsening oliguria, metabolic acidosis, and hyperkalaemia requiring haemodialysis. Extensive investigations of autoimmune, inflammatory, haematological, obstructive, medication-related, and metal-ion causes were undertaken. Legionella urinary antigen testing was negative; however, serology revealed *L. pneumophila* serogroup 1 IgM and total antibody titres ≥ 1:512. The patient required eight haemodialysis sessions before entering a non-oliguric recovery phase, with creatinine improving to 173 μmol/L and CK normalising.

Conclusions

Severe rhabdomyolysis-associated AKI may be the dominant manifestation of probable *Legionella* infection even when respiratory features are limited and urinary antigen testing is negative. A negative urinary antigen result should not terminate investigation when clinical suspicion persists; respiratory culture or PCR and appropriately interpreted serology should be considered. The accumulating literature since 2020 further demonstrates substantial heterogeneity in renal involvement, ranging from preserved kidney function to dialysis-requiring AKI.

Background

Legionella pneumophila is a recognised cause of atypical pneumonia and systemic illness, with extrapulmonary manifestations, including rhabdomyolysis, acute kidney injury, transaminitis, neurological symptoms, and electrolyte disturbance [1–3]. Although rhabdomyolysis is more commonly

attributed to trauma, exertion, seizures, medications, toxins, or metabolic disturbances, infectious causes account for a small but clinically important proportion of cases [3]. Among these, Legionella infection is an uncommon but well-described bacterial trigger of severe rhabdomyolysis and pigment-associated acute tubular injury [1–3].

The associations among Legionella infection, rhabdomyolysis, and renal failure are clinically important because rhabdomyolysis may be the dominant presenting feature, whereas respiratory symptoms or radiographic pneumonia may be absent, mild, delayed, or initially overlooked [2, 3]. In a retrospective cohort of hospitalised patients with confirmed Legionella infection who underwent creatine kinase testing, hyperCKaemia was identified in 53.9% of patients and was associated with higher rates of acute renal failure and dialysis requirements [1]. A review of published cases revealed recurrent reports of the triad of Legionnaires' disease, rhabdomyolysis, and renal failure, supporting the need to consider Legionella in patients presenting with otherwise unexplained rhabdomyolysis-associated AKI [2].

We report a case that highlights several diagnostic challenges with plausible competing explanations, including high-intensity statin therapy, immune-mediated or inflammatory myopathy, thrombotic microangiopathy, obstruction, and cobalt/chromium exposure from a metal-on-metal hip prosthesis [4–6].

The geographical context is also relevant. Tropical northern Australia has a distinct burden of endemic infections with potentially nonspecific systemic presentations, including leptospirosis, melioidosis, and rickettsial disease [7]. Regional exposure to Legionella species is also well documented, with seroepidemiological data from northeastern Australia demonstrating substantial Legionella seroprevalence and associations between *L. pneumophila* exposure, rainfall and land use [8]. Early consideration of atypical and tropical infections may prompt timely targeted testing and antimicrobial therapy. This case is educational because *Legionella*-associated rhabdomyolysis was not immediately apparent and several plausible medication-related, autoimmune and toxic causes initially competed for the diagnosis. In addition, we provide an updated synthesis of reported *Legionella*-associated rhabdomyolysis cases published since the 2020 systematic review, describing the expanding spectrum of renal involvement and outcomes. Together, the case and literature update emphasise the importance of considering *Legionella* in otherwise unexplained severe rhabdomyolysis-associated AKI even when pneumonia is not clinically dominant.

Case presentation

A 78-year-old man was referred to the hospital after outpatient blood tests demonstrated severe acute kidney injury with an associated increase in troponin. The relevant history included gout, hypercholesterolaemia, aortic stenosis, and previous left metal-on-metal hip resurfacing/prosthesis. His regular medications were allopurinol 300 mg daily, metoprolol 25 mg twice daily, rosuvastatin 40 mg daily, and ezetimibe 10 mg daily. Baseline renal function in February 2026 was a creatinine level of 101 μmol/L with an eGFR of 62 mL/min/1.73 m².

The prehospital history was notable, with approximately 2–3 weeks of progressive symptoms. The patient described early disorientation, reduced appetite, approximately 4.5 kg weight loss, thirst and dry mouth, muscle aches and tightness, muscle spasms, exertional dyspnoea, unsteadiness, urinary frequency, dark urine and reduced urine output. The functional decline was prominent, with increasing difficulty standing, walking, bending, crouching, and carrying objects, indicating the need to manually lift each leg into his vehicle. His wife reported that he appeared to ignore or fail to appreciate the severity of his deterioration despite usually being proactive about his health.

Potential exposure history included several hours of outdoor work on a rural property in windy conditions without a mask and with uncertain hydration. He had also been working near a usually dry creek depression that had flooded approximately one to two weeks earlier. He reported a preceding flu-like/upper respiratory illness with cough, coryzal symptoms, chills and sore thighs. He reported no recent falls, seizures, excessive exercise, diarrhoea, nonsteroidal anti-inflammatory use, illicit drug use, new medication, or recent statin dose escalation.

On admission, he was alert and oriented, afebrile at 36.4°C, had a blood pressure of 112/50 mmHg, had a pulse of 68 beats/min, had a respiratory rate of 18 breaths/min, and had an oxygen saturation of 95% on room air. He was clinically dehydrated with dry mucous membranes, no elevated jugular venous pressure, clear lung fields, a soft nontender abdomen, and no peripheral oedema. Cardiac examination revealed a systolic murmur. Neurological examination revealed preserved upper-limb strength and predominant proximal lower-limb weakness: hip flexion 3/5 bilaterally, hip abduction/adduction 4/5 bilaterally, knee flexion/extension 3/5 bilaterally, and ankle dorsiflexion/plantarflexion 4/5 bilaterally. Ankle clonus was not sustained at 1–2 beats bilaterally.

Initial investigations revealed a haemoglobin level of 80 g/L, a normal white-cell count, a potassium level of 4.3 mmol/L, a bicarbonate level of 18 mmol/L, a urea level of 40.2 mmol/L, a creatinine level of 989 μmol/L, an eGFR of 4 mL/min/1.73 m², and a creatine kinase level of 33,400 U/L. Liver enzymes were elevated, with ALT 490 U/L and AST 925 U/L. Investigations also revealed an elevated troponin level of 386 ng/L and a C-reactive protein level of 63 mg/L. ECG revealed no changes in the ST segment. CT KUB revealed a normal non-contrast renal appearance with no evidence of urinary tract obstruction. The chronology of the patient's clinical course, diagnostic investigations, haemodialysis and subsequent renal recovery is summarised in Fig. 1.

The initial impression was severe oliguric acute kidney injury, likely secondary to rhabdomyolysis and reduced oral intake. Despite intravenous fluids and withdrawal of potential nephrotoxins, oliguria persisted and renal function deteriorated, with creatinine rising to approximately 1060 μmol/L, eGFR remaining at 4 mL/min/1.73 m², potassium increasing to 5.1 mmol/L, worsening metabolic acidosis, and fever to 39°C. Urgent haemodialysis was commenced via a non-tunnelled vascular catheter. He underwent eight haemodialysis sessions between 26 April and 4 May, after which the renal course entered a non-oliguric recovery phase.

Serial biochemical trends are presented in Fig. 2. The creatine kinase level declined rapidly from 33,400 U/L on admission to 1,260 U/L by 1 May and subsequently normalised (Fig. 2A). Despite this early improvement, severe acute kidney injury persisted, requiring eight haemodialysis sessions between April 26th and May 4th. The patient tolerated all eight haemodialysis sessions without significant complications. Renal recovery occurred more gradually, with creatinine decreasing to 173 μmol/L and the eGFR increasing to 32 mL/min/1.73 m² by follow-up on 5 June (Fig. 2B).

(A) Creatine kinase activity declined rapidly following admission. (B) Serum creatinine and estimated glomerular filtration rate trends with haemodialysis sessions indicated by shaded columns. CK, creatine kinase; eGFR, estimated glomerular filtration rate; HD, haemodialysis.

The differential diagnosis for this presentation included viral myositis/rhabdomyolysis, statin-associated myopathy, immune-mediated necrotising myopathy, other inflammatory myopathies, including inclusion body myositis/autoimmune myositis, malignancy-associated or paraneoplastic myopathy, acute tubular necrosis in the context of illness, glomerulonephritis, thrombotic microangiopathy/atypical haemolytic uremic syndrome, and toxic metal exposure related to a metal-on-metal hip prosthesis. Urine myoglobin was markedly elevated at 259,000 μg/L, supporting pigment-associated tubular injury. Autoimmune and glomerulonephritis testing, including antinuclear antibodies, extractable nuclear antigens, antineutrophil cytoplasmic antibodies, anti-glomerular basement membrane antibodies, complement, and confirmatory anti-double-stranded DNA testing, was unrevealing. The myositis antibody profile and anti-HMG-CoA reductase antibody results were negative. The haemolysis evaluation did not support thrombotic microangiopathy, and serum protein electrophoresis and flow cytometry revealed no monoclonal process.

Given that the patient previously underwent metal-on-metal hip arthroplasty (Fig. 3), systemic metal ion toxicity was considered an alternative. The serum cobalt and chromium concentrations were elevated at 0.31 μmol/L and 0.23 μmol/L, respectively. Although mild non-sustained ankle clonus was present, there were no other neurological, cardiac, endocrine, or haematological features of systemic cobalt toxicity. Interpretation of the metal-ion concentrations is also complicated by severe renal impairment. Metal-ion exposure was therefore considered a possible contributing factor but was not regarded as the primary cause of the acute presentation [6].

Respiratory viral polymerase chain reaction (PCR) and blood cultures were negative. Legionella testing was initiated during the later diagnostic work-up. Urinary antigen testing for *L. pneumophila* serogroup 1 was negative on May 5th. Serology reported on May 13th demonstrated *L. pneumophila* serogroup 1 IgM and total antibody titres of at least 1:512, whereas *L. pneumophila* serogroup 2 and L. longbeachae titres were less than 1:64. In the context of compatible systemic prodrome, preceding respiratory illness, severe rhabdomyolysis, and the exclusion of major competing diagnoses, the final clinical interpretation was probable Legionella-associated rhabdomyolysis with pigment-associated acute tubular injury. Following the positive *L. pneumophila* serology, 500 mg of azithromycin orally once daily was

administered for 7 days. The treatment was well tolerated, with no identified antimicrobial-related adverse effects.

The CK concentration decreased from 33,400 U/L at admission to 1,260 U/L by May 1st and 249 U/L by May 7th. Renal recovery lagged behind the biochemical resolution of muscle injury. The urine output improved to approximately 1.4 L/day on May 4th and 2.3 L/day on May 5th, allowing dialysis to be withheld and tunnelled catheter insertion to be cancelled. Creatinine was 552 μmol/L at discharge on 14 May, 362 μmol/L on 18 May, and 173 μmol/L with an eGFR of 32 mL/min/1.73 m² on 5 June. CK, aminotransferases, and albuminuria had normalised or substantially improved. The selected laboratory findings from baseline to follow-up are summarised in Table 1.

Table 1
Selected laboratory findings from baseline to follow-up.

Variable	Baseline Feb 2026	Admission 24 Apr	HD commencement 26 Apr	Discharge 14 May	Follow-up 5 Jun
Creatinine (μmol/L)	101	989	1,060	552	173
eGFR (mL/min/1.73 m²)	62	4	4	8	32
CK (U/L)	—	33,400	—	—	84
Urine myoglobin (μg/L)	NR	259,000	51,700	—	—
AST/ALT (U/L)	31/21	925/490	932/487	39/50	26/14

ALT, alanine aminotransferase; AST, aspartate aminotransferase; CK, creatine kinase; eGFR, estimated glomerular filtration rate; HD, haemodialysis; NR, not recorded.

Discussion

This case describes severe rhabdomyolysis-associated acute kidney injury in a previously independent older man who presented predominantly with progressive proximal lower-limb weakness, dark urine, oliguria, systemic symptoms, and transaminitis, with minimal respiratory findings. The severity of muscle and kidney injury was marked, with an admission creatinine of 989 μmol/L, an eGFR of 4 mL/min/1.73 m², and a CK of 33,400 U/L, followed by progressive oligo-anuric AKI requiring temporary haemodialysis. This case is clinically important because Legionella infection may present predominantly through extrapulmonary manifestations and may be overlooked when respiratory symptoms are mild, resolved, or not prominent at presentation.

Rhabdomyolysis is an uncommon but recognised extrapulmonary manifestation of Legionella infection. The strongest available evidence derives from observational studies, a systematic review, and a focused

narrative review, whereas much of the remaining literature consists of individual case reports. Hill et al. examined 267 hospitalised patients with confirmed Legionella infection who underwent CK testing and reported hyperCKaemia in 53.9% of these patients [1]. CK elevation was associated with greater illness severity, including higher rates of acute renal failure, dialysis, neurological complications, and intensive care admission. More recently, Wyler et al. reported rhabdomyolysis in 23.8% of patients with laboratory-confirmed *Legionella* infection, with rhabdomyolysis associated with AKI, longer hospitalisation and increased intensive care unit admission [9]. These findings suggest that skeletal muscle involvement is clinically relevant in *Legionella* infection and that rhabdomyolysis, when present, may identify a more severe disease phenotype, although hyperCKaemia should not be equated with clinically significant rhabdomyolysis in every patient.

Simoni et al. systematically reviewed cases of atypical bacterial pneumonia complicated by both myositis and acute kidney injury [10]. Among the 42 identified cases, Legionella accounted for 27, making it the most frequently reported pathogen associated with this combined muscle–kidney syndrome. Affected patients were predominantly male, and the clinical spectrum ranged from moderate CK elevation to severe rhabdomyolysis requiring renal replacement therapy. Soni and Peter similarly identified more than 20 published cases of the triad of Legionnaires' disease, rhabdomyolysis, and renal failure, with renal dysfunction frequently severe enough to require dialysis [2].

To update the evidence base following the systematic review by Simoni et al., PubMed/MEDLINE and Embase were searched for reports published from 1 April 2020 to 24 July 2026 describing microbiologically supported *Legionella* infection with rhabdomyolysis or myositis and reported renal outcomes. Eligible reports are summarised in Supplementary Table S1 and demonstrate considerable heterogeneity in the severity of muscle injury, renal involvement, and diagnostic approach. For example, Du et al. described severe rhabdomyolysis and AKI requiring continuous renal replacement therapy in a patient whose Legionella infection was identified only through metagenomic next-generation sequencing after conventional testing was unrevealing [11]. Conversely, Sangam et al. reported a CK concentration exceeding 240,000 U/L without AKI, illustrating that the magnitude of CK elevation alone does not reliably predict renal involvement [12]. Collectively, these and other subsequent reports further document the broad spectrum of *Legionella*-associated rhabdomyolysis since the previous systematic review, ranging from preserved kidney function to severe dialysis-requiring AKI.

Against this evolving literature, the contribution of the present case lies in its diagnostic complexity rather than in establishing a novel association between *Legionella*, rhabdomyolysis and AKI. Rhabdomyolysis and renal failure dominated the presentation despite limited respiratory manifestations, urinary antigen testing was negative, and several plausible alternative causes of muscle injury were present. This type of presentation may be underrepresented in the existing evidence base, as Simoni et al. excluded cases involving potentially myotoxic medications and those with extensive multisystem involvement. Although the accumulated literature reinforces the clinical relevance of *Legionella*-associated muscle and kidney injury, it remains largely case-based and susceptible to publication bias, limiting reliable estimates of the incidence and prognosis of clinically significant rhabdomyolysis.

The mechanisms underlying muscle and kidney injury in Legionella infection are incompletely understood. The proposed mechanisms of myonecrosis include direct bacterial invasion, toxin-mediated injury, and systemic inflammatory effects [2, 10]. The resulting kidney injury is probably multifactorial. Myoglobin may cause tubular obstruction, oxidative tubular toxicity, and intrarenal vasoconstriction, whereas dehydration, aciduria, systemic inflammation, sepsis-related hypoperfusion, and haemodynamic instability may amplify renal injury. Pathological reports of myoglobin cast nephropathy provide direct evidence of pigment-associated tubular injury, although acute tubular necrosis and tubulointerstitial nephritis have also been described in Legionella infection [2, 13].

These mechanisms are consistent with the clinical course observed in this patient. Reduced oral intake, clinical dehydration, dark urine, marked myoglobinuria, severe CK elevation, oliguria, and the absence of urinary tract obstruction support pigment-associated acute tubular injury as the dominant renal process. CK declined rapidly following admission, whereas renal recovery was delayed, a pattern described in other severe Legionella-associated cases and consistent with cessation of muscle injury followed by slower tubular recovery [13, 14].

A central feature of this case was the presence of several plausible competing diagnoses. Long-term high intensity rosuvastatin therapy was relevant, particularly at a dose of 40 mg daily; however, there was no clear recent dose change, and the associated systemic and infectious prodrome made isolated statin toxicity less convincing. Statin exposure may nevertheless increase susceptibility to muscle injury. Immune-mediated necrotising myopathy and other inflammatory myopathies were considered but were not supported by myositis antibody testing or anti-HMG-CoA reductase antibodies, while the acute course and intercurrent systemic illness were also atypical for these conditions. Post-renal obstruction was excluded by CT KUB and subsequent renal ultrasonography. Thrombotic microangiopathy and atypical haemolytic uremic syndrome were considered because of the combination of AKI, anaemia, thrombocytopenia, and elevated lactate dehydrogenase but were not supported by peripheral blood film or haemolysis investigations. Cobalt and chromium toxicity related to the patient's metal-on-metal hip resurfacing was also considered because metal-ion concentrations were elevated, although the absence of a characteristic neurological, cardiac, endocrine, or haematological syndrome made systemic metal toxicity a less convincing primary explanation.

The infectious syndrome, strongly positive *L. pneumophila* serology, marked myoglobinuria, compatible muscle–kidney phenotype, and exclusion of major competing diagnoses supported Legionella infection as the most likely precipitating factor. Nevertheless, microbiological evidence requires careful interpretation. Unlike the predominantly pneumonia-associated presentations described in the published literature, respiratory disease was not the dominant clinical feature in this case [2]. Furthermore, urinary *Legionella* antigen testing was negative, which initially reduced clinical suspicion for *Legionella* infection. However, a negative urinary antigen does not exclude Legionella infection because the test has imperfect sensitivity and primarily detects *L. pneumophila* serogroup 1[15]. Conversely, a single elevated serological titre is less definitive than culture, respiratory polymerase chain reaction, or paired serology demonstrating a fourfold rise in antibody titre [15]. Interpretation of a single elevated titre also requires

caution in northeastern Australia, where background Legionella seroprevalence is substantial [8]. The diagnosis is therefore best described as clinically supported, probable Legionella-associated rhabdomyolysis rather than microbiologically proven Legionnaires' disease. Dehydration, statin exposure, systemic inflammation, and pigment-associated acute tubular injury may also have contributed to the severity of the presentation.

The management implications are practical. In patients presenting with severe unexplained rhabdomyolysis and AKI, early nephrology involvement; close monitoring of urine output; serial assessment of kidney function, electrolytes and acid–base status; withdrawal of potential nephrotoxins and myotoxins; and timely initiation of renal replacement therapy when indicated are essential. Legionella testing should be considered when rhabdomyolysis-associated AKI occurs alongside systemic symptoms, transaminitis, hyponatraemia, gastrointestinal symptoms, or respiratory illness, even when pneumonia is not clinically dominant. Where suspicion remains high despite a negative urinary antigen test, respiratory culture or polymerase chain reaction and paired serology should be considered.

This report is limited by its single-patient design, the absence of culture or respiratory molecular confirmation, and reliance on a single elevated serological titre. The potential contribution of several concurrent factors, including dehydration and statin exposure, also prevents definitive causal attribution. Nevertheless, the observed severe muscle–kidney phenotype is consistent with previous reports of *Legionella*-associated rhabdomyolysis, in which AKI is frequently severe and may require dialysis, with renal injury considered multifactorial [1, 9, 10, 13]. Accordingly, the diagnosis is best regarded as probable *Legionella*-associated rhabdomyolysis with multifactorial AKI rather than microbiologically proven Legionnaires' disease.

Conclusion

Probable Legionella infection should be considered in patients presenting with otherwise unexplained severe rhabdomyolysis-associated AKI, particularly when systemic symptoms, transaminitis, dark urine, oliguria, or a preceding respiratory illness are present. Subtle respiratory manifestations and negative urinary antigen tests should not prematurely exclude the diagnosis. Prompt recognition permits appropriate antimicrobial treatment, while withdrawal of potential nephrotoxins and myotoxins and timely renal replacement therapy remain important components of supportive management.

Patient perspective

"I had always been conscious of my health, but during this illness, I did not seem to appreciate how unwell I was. Looking back, I remember struggling to lift my legs into the car and finding normal activities such as walking, bending, and carrying objects increasingly difficult." – Patient

Abbreviations

AKI
acute kidney injury
ALT
alanine aminotransferase
AST
aspartate aminotransferase
CK
creatine kinase
CT KUB
computed tomography of kidneys, ureters, and bladder
eGFR
estimated glomerular filtration rate
HD
haemodialysis

Declarations

Ethics approval and consent to participate

Ethics approval was not required for this single-patient case report according to local institutional policy. Written informed consent for publication was obtained from the patient.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying clinical details/images. A copy of the written consent is available for review by the Editor if requested.

Availability of data and materials

Data sharing is not applicable to this article, as no datasets were generated or analysed. All relevant clinical information is included in this published article.

Competing interests

The authors declare that they have no competing interests.

Funding

No funding was received for this case report.

Acknowledgements

The authors used the Springer Nature/AJE AI-assisted language editing tool for English-language editing and readability. All suggested changes were reviewed and approved by the authors.

Authors' contributions

WT conceived and drafted the case report and prepared the initial manuscript. VS, JD and AM supervised the report, contributed to the clinical interpretation, and critically revised the manuscript for important intellectual content. All the authors read and approved the final manuscript.

References

1. Hill J, Li J, Li Y. Incidence and influence of hyperCKemia in Legionella infection. J Neurol Sci. 2022;436:120252.
2. Soni AJ, Peter A. Established association of legionella with rhabdomyolysis and renal failure: A review of the literature. Respir Med Case Rep. 2019;28:100962.
3. Ma H, Bavishi A, Jain B. Legionella associated rhabdomyolysis: a case report. J Med Case Rep. 2023;17(1):258.
4. Mendes P, Robles PG, Mathur S. Statin-induced rhabdomyolysis: a comprehensive review of case reports. Physiother Can. 2014;66(2):124–32.
5. Sridharan K, Sivaramakrishnan G. Ezetimibe-associated rhabdomyolysis: A comprehensive assessment of the USFDA adverse event reporting system using disproportionality analysis, case reviews, and meta-analysis of randomized clinical trials. J Clin Lipidol. 2025;19(2):327–36.
6. Devlin JJ, Pomerleau AC, Brent J, Morgan BW, Deitchman S, Schwartz M. Clinical features, testing, and management of patients with suspected prosthetic hip-associated cobalt toxicity: a systematic review of cases. J Med Toxicol. 2013;9(4):405–15.
7. Fairhead LJ, Smith S, Sim BZ, Stewart AGA, Stewart JD, Binotto E, Law M, Hanson J. The seasonality of infections in tropical Far North Queensland, Australia: A 21-year retrospective evaluation of the seasonal patterns of six endemic pathogens. PLOS Glob Public Health. 2022;2(5):e0000506.
8. Lethbridge J, Freppel W, Ho MF, Skinner EB, Rustanti L, Roulis E, Rudd PA, Faddy HM, Young MK, Herrero LJ. Seroprevalence of Antibodies Against Legionella Species in Northeastern Australian Blood Donors, 2016 and 2023. J Infect Dis. 2026;233(1):e239–49.
9. Wyler B, Mayur J, Canavan J, Branstetter A, Dai E, Dempsey A. Frequency of rhabdomyolysis in Legionella infection and associated outcomes: a retrospective cross-sectional study. Postgrad Med. 2026;138(1):28–35.
10. Simoni C, Camozzi P, Faré PB, Bianchetti MG, Kottanattu L, Lava SAG, Milani GP. Myositis and acute kidney injury in bacterial atypical pneumonia: Systematic literature review. J Infect Public Health.

2020;13(12):2020–4.

11. Du R, Feng Y, Wang Y, Huang J, Tao Y, Mao H. Metagenomic next-generation sequencing confirms the diagnosis of Legionella pneumonia with rhabdomyolysis and acute kidney injury in a limited resource area: a case report and review. Front Public Health. 2023;11:1145733.
12. Sangam S, Malik S, Hodo F, Patel B, Bengualid V, Sharma S. A Case of Legionella Pneumonia with Rhabdomyolysis, with Extremely high Creatinine Kinase without Acute Kidney Injury in an Adult. Eur J Case Rep Intern Med. 2024;11(8):004539.
13. Fujisawa Y, Miyanaga T, Takeji A, Shirota Y, Ueda Y. A Lethal Combination: Legionnaires' Disease Complicated by Rhabdomyolysis, Acute Kidney Injury, and Non-Occlusive Mesenteric Ischemia. Am J Case Rep. 2023;24:e940792.
14. Gopalakrishna H, Al-Abdouh A, Nair GB, Solipuram V, Bekele A. Immensely High Creatine Kinase Levels in a Case of Rhabdomyolysis Due to Legionnaires' Disease in a Patient on Tofacitinib: A Case Report and Literature Review. J Community Hosp Intern Med Perspect. 2022;12(2):45–9.
15. Pierre DM, Baron J, Yu VL, Stout JE. Diagnostic testing for Legionnaires' disease. Ann Clin Microbiol Antimicrob. 2017;16(1):59.

Figures

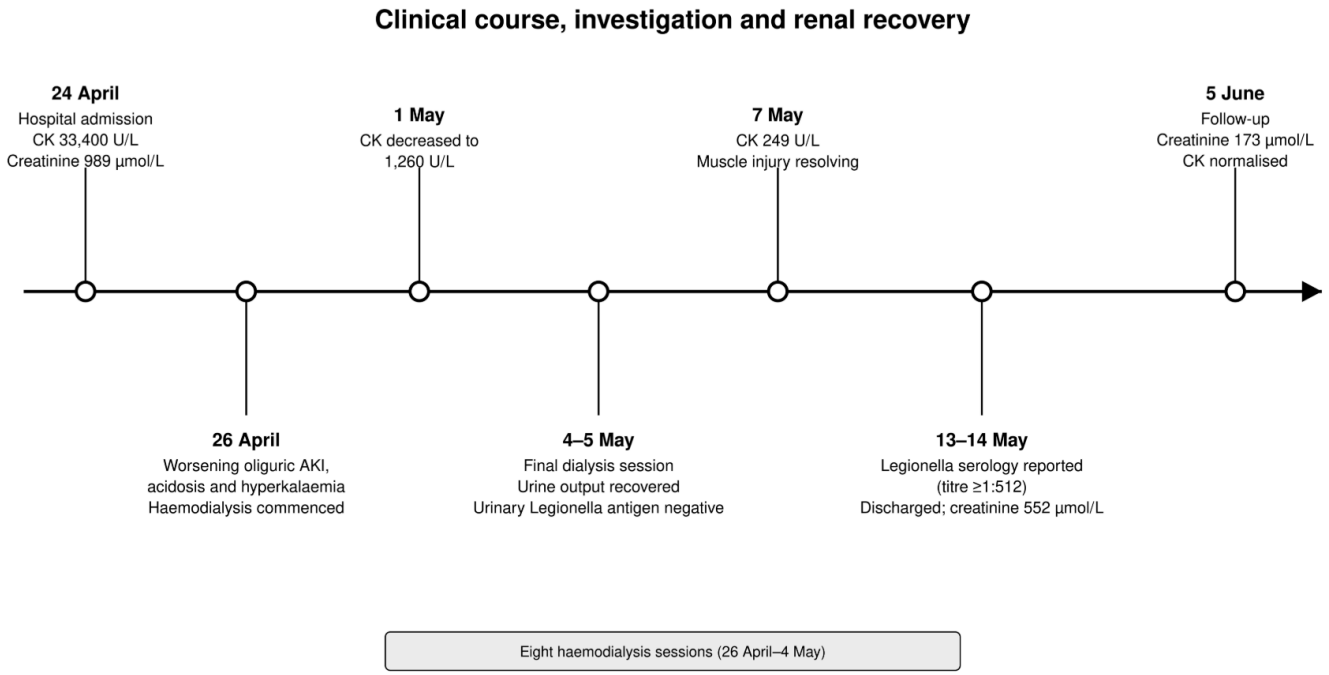


Figure 1

Timeline of the clinical course, diagnostic investigations, treatment and renal recovery.

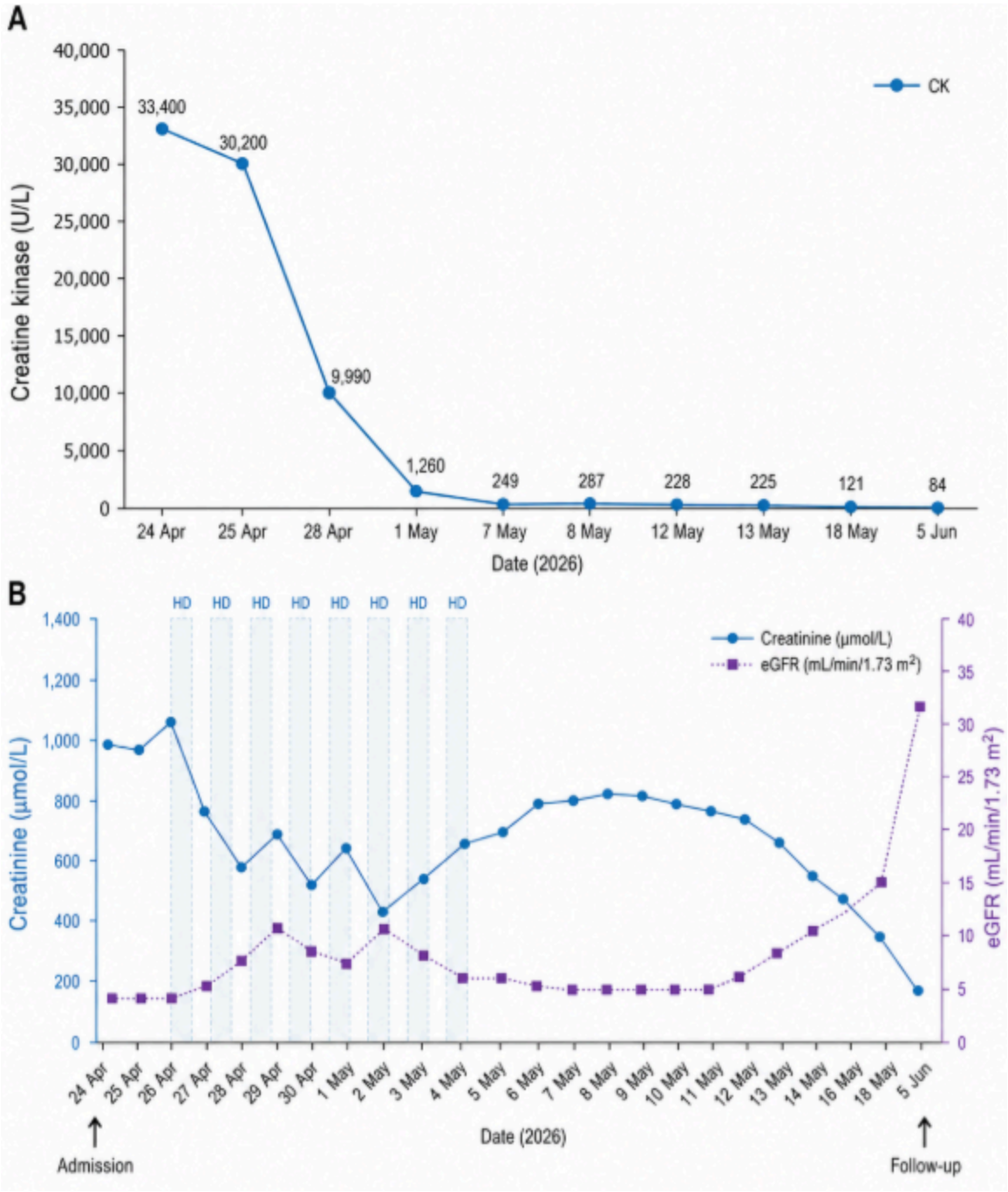


Figure 2

Serial creatine kinase and renal function data were collected during admission and follow-up.

(A) Creatine kinase activity declined rapidly following admission. (B) Serum creatinine and estimated glomerular filtration rate trends with haemodialysis sessions indicated by shaded columns. CK, creatine kinase; eGFR, estimated glomerular filtration rate; HD, haemodialysis.



Figure 3

CT scout image demonstrating left metal-on-metal hip resurfacing arthroplasty.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- SupplementaryTableS1.docx