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Tubulointerstitial nephritis and uveitis post COVID-19 infection in an adult

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Accepted 9 April 2025

SUMMARY

A woman in her 50s contracted COVID-19 and initially presented a few weeks afterwards with left eye pain and redness. She was diagnosed with uveitis and treated with glucocorticoid eye drops. Renal impairment was found on laboratory investigations performed following the diagnosis of uveitis, and urine testing showed proteinuria. Serological testing showed no cause for the new findings, and renal imaging was unremarkable. A renal biopsy was conducted and histology was consistent with tubulointerstitial nephritis. A diagnosis of tubulointerstitial nephritis with uveitis (TINU) was established, and she was given prednisolone, with resolution of proteinuria and improvement in renal function. While TINU is rare but well described in children, it can also uncommonly be a cause of renal impairment in adults post COVID-19 infection, similar to cases in children.

BACKGROUND

Tubulointerstitial nephritis with uveitis (TINU) is a rare disorder characterised by renal and ocular inflammation. TINU has been reported mainly in children and adolescents with a variety of suspected triggers such as infection, medications, vaccinations and supplements.[1–9] However, the pathophysiology is not completely understood.[10 11] The median age was 19 in a case series including 257 cases, while the median age was 25 for another cohort of 48 patients.[10 12] Prognosis has been reported to be good in children with good recovery of renal function in response to immunosuppression.[2 4 8 10 13 14] TINU is typically regarded as a childhood and adolescent disease, although more recent case series in Asia and France have identified cases occurring in both adults and the elderly.[15–22] It has also newly been described as a complication of COVID-19 infection in children and adolescents in the past few years.[23–26] We report a case of TINU post COVID-19 infection in an adult with associated symptoms of long covid.

CASE PRESENTATION

In this case report, we describe a case of a woman in her 50s who was initially referred to our nephrology service with impaired renal function noted by her general practitioner, after a recent diagnosis of uveitis. She had two doses of the Pfizer Comirnaty vaccine in the preceding year. Medical history included hypercholesterolaemia and a history of tubal ligation. She was not prescribed any regular medications prior to her illness. She is an office worker but had not worked for a few months due to significant fatigue post COVID-19 contracted earlier in the year. She had a 22-pack year history of smoking, with cessation 8 years prior to her initial presentation. Her baseline renal function was normal with her serum creatinine measuring at 63 umol/L in 2017, corresponding to an estimated glomerular filtration rate (eGFR) of >90 mL/min/1.73 m^2.

Initial COVID-19 infection was diagnosed in early January via rapid antigen testing. Symptoms that persisted following the diagnosis of COVID-19 included joint pain involving wrists and knees as well as brain fog and fatigue. Her inflammatory markers including C reactive protein (CRP) and erythrocyte sedimentation rate (ESR) remained normal despite her symptoms. Extensive cardiovascular and respiratory testing showed no significant pathological abnormalities. Laboratory investigations to exclude autoimmune disease including anti-cyclic citrullinated peptide (anti-CCP), rheumatoid factor (RF), antinuclear antigen (ANA), extractable nuclear antigen (ENA) and antineutrophil cytoplasmic antibodies (ANCA) were negative. No new medications were commenced other than a short trial of budesonide/eformoterol inhaled therapy for an early postinfectious cough. She was diagnosed with long covid on follow-up with her general practitioner.

In the following month after her COVID-19 diagnosis, she had presented to a private ophthalmology clinic with a painful red left eye with associated photosensitivity. She was reviewed by a private ophthalmologist who identified redness surrounding the cornea, with white cells identified in the anterior chamber. Dexamethasone eye drops at 0.1% concentration and atropine eye drops were prescribed for anterior uveitis. Subsequent review 2 weeks later at ophthalmology clinic noted no significant improvement, and treatment was escalated to prednisolone acetate 1% and phenylephrine hydrochloride 0.12% eye drops. The creatinine level at this time was 85 umol/L which had increased and corresponded to an eGFR of 68 mL/min/1.73 m^2.

One month following commencement of treatment of her uveitis with an initial clinical response, her ophthalmic symptoms recurred with significant left eye pain and redness, with persistently elevated creatinine at 116 umol/L (eGFR of 47 mL/min/1.73 m^2). She was then referred to the nephrology service as an outpatient. While awaiting outpatient review, her creatinine level further increased to 131 umol/L (eGFR of 41 mL/min/1.73 m^2). Then, she presented to hospital for assessment. She was assessed by the nephrology team and admitted to expedite investigation.

To cite: Lau J, Tan K-S. *BMJ Case Rep* 2025;**18**:e261002. doi:10.1136/bcr-2024-261002

On review, she reported no fever, dysuria, haematuria or frothy urine. Other than ongoing fatigue and bilateral mild flank pain, her symptoms remained unchanged for the past few months.

On initial examination, blood pressure was 119/85 mm Hg, and pulse was 86 beats per minute. There were dual heart sounds with no murmurs, and her jugular venous pulse was not elevated. Her chest was clear and there was no pedal oedema. There was mild renal angle tenderness on strong balloting of her kidneys bilaterally. There was no small joint arthropathy, and no aphthous ulcers were found. There was no evidence of uveitis on examination.

INVESTIGATIONS

Investigations conducted into the cause of her fatigue prior to review in hospital were initially conducted via respiratory and cardiology services. Lung function tests showed normal forced expiratory volume in 1 second (FEV1) at 2.84 L, 105% predicted, and forced vital capacity (FVC) at 3.39 L, 97.8% predicted. FEV1/FVC ratio was 83.67, with normal lung volumes. D-dimer was normal, and CT of her chest showed no abnormalities post COVID-19 infection. Exercise stress echocardiogram showed normal left ventricular size and function and was symptomatically and electrically negative, with no exercise-induced wall motion abnormalities. She was able to exercise for 7 min and 31 s on the accelerated BRUCE protocol to reach 11.4 metabolic equivalent of tasks. The haemoglobin level remained stable at 127 g/L, with a normal white cell count at 4.2×10^9/L. Iron studies were normal with a transferrin saturation of 20% and a ferritin level of 65 ug/L.

Prior to admission, ESR was elevated at 78 mm/hour and CRP was elevated at 26 mg/L. Her baseline creatinine was 63 umol/L. Serum creatinine level peaked at 140 umol/L (eGFR 37) while in hospital, with a normal serum bicarbonate level at 24 mmol/L. Urine protein-creatinine ratio on admission was 84 g/mol and urine albumin-creatinine ratio was 14 g/mol, consistent with primarily tubular dysfunction. Urine microscopy showed leucocyte count of $10x10^6$/L, with no detectable erythrocytes or epithelial cells. A glomerulonephritis panel of tests requested included an ANA, ENA screen, ANCA, antistreptolysin O level, antideoxyribonuclease (anti-DNAse) B, hepatitis serology, HIV antigen and antibody, serum protein electrophoresis, free light chain ratio and Bence Jones protein. ANA, ENA, antidouble-stranded DNA, antistreptolysin O, anti-DNAse B, as well as repeat anti-CCP and RF, were negative. Serum C3 and C4 were normal. Hepatitis and HIV tests were negative. An elevated serum free light chain ratio was at 1.90, and a monoclonal antibody was detected, although no Bence Jones protein or monoclonal immunoglobulins were detected in the urine. Elevated alpha 2 monoclonal protein level at 10 g/L and beta and kappa IgA at 10 g/L were found. No other features of myeloma or organ dysfunction associated with monoclonal gammopathy were found. Renal imaging showed a 12 cm right kidney and 11 cm left kidney, with a simple 5 mm cortical cyst in the right mid-pole region. There was no evidence of hydronephrosis or hydroureter. Postvoid residual volume was normal.

Considering her history of uveitis and significant tubular dysfunction and new renal impairment, the team decided to proceed to renal biopsy. As the sensitivity and specificity of ACE levels in the diagnosis of sarcoidosis are variable, the patient had a normal chest X-ray, and the indication to proceed with a renal biopsy was present, our team elected not to test ACE levels or 1,25-dihydroxycholecalciferol levels.

A renal biopsy was conducted urgently, which showed a diffuse interstitial infiltrate of lymphocytes, plasma cells, eosinophils, neutrophils and macrophages with tubulointerstitial inflammation

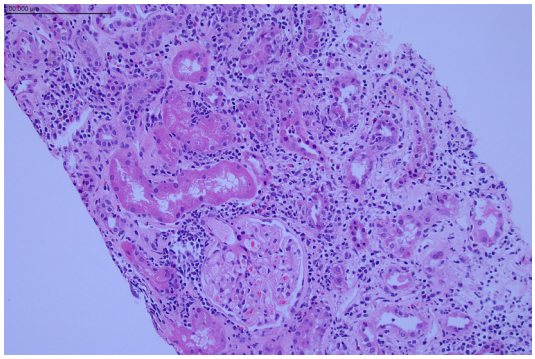

Figure 1 H&E stain showing tubulointerstitial inflammation.

(figure 1). There were eight glomeruli present, with one globally sclerosed and no glomeruli demonstrating crescents, hypercellularity or necrosis. There were associated acute tubular necrosis and moderate lymphocytic tubulitis. Immunostaining demonstrates the CD3 positive lymphocytes (figure 2). Tubulitis is also demonstrated with the periodic acid-Schiff stain (figure 3). There were no significant reactions in glomeruli or tubules with IgG, IgA, IgM, C3, C1q, fibrin, albumin, kappa or lambda. 50% interstitial fibrosis and tubular atrophy were present. This was consistent with acute tubulointerstitial nephritis, with no specific reactions shown by immunofluorescence.

DIFFERENTIAL DIAGNOSIS

TINU was the primary differential as it provided an explanation for the associated ophthalmological symptoms. The significantly elevated protein-creatinine ratio also suggested a primarily tubular pathology which is supportive of TINU. Therefore, a biopsy was considered early in the workup for this patient.

Other differentials for this case included drug-induced interstitial nephritis, IgA nephropathy or glomerulonephritis. All the above can cause a degree of proteinuria in an acute setting. However, the pattern of albuminuria and proteinuria and a lack of haematuria or pyuria, as well as her symptoms, were not as supportive of these other differentials. The negative immunofluoresence and lack of histological evidence of glomerulonephritis also ruled out IgA nephropathy and IgA nephritis. The trajectory of her serum creatinine was inconsistent with drug-induced interstitial nephritis, and she was not taking any medications strongly associated with interstitial nephritis. The lack of haematuria

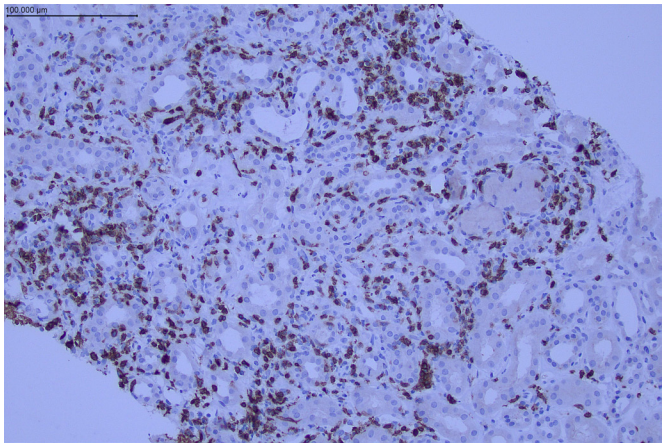

Figure 2 Immunostain showing CD3 positive lymphocytes.

decreased the likelihood of IgA nephropathy and glomerulonephritis. A biopsy was required to confirm which differential diagnosis is regardless. Other differentials were considered, such as Behcet's, although no oral or genital ulceration was present and this patient was of Northern European heritage, with rare prevalences of Behcet's in this demographic.

TREATMENT

The diagnosis was established as TINU following confirmation of the biopsy results showing tubulointerstitial nephritis in conjunction with her ophthalmological diagnosis of uveitis. Prednisolone was commenced at 50 mg per day with trimethoprim-sulfamethoxazole 160/800 mg three times a week for pneumocystis jirovecii pneumonia (PJP) prophylaxis, pantoprazole 40 mg daily for ulcer prevention and calcium and vitamin D for bone health. After 6 weeks of therapy, creatinine improved to 90 umol/L (eGFR 64), and prednisolone was weaned to 37.5 mg for 2 weeks, then 25 mg for 2 weeks. Serum creatinine level subsequently remained static at 89 umol/L and the prednisolone dose was increased again to 37.5 mg.

OUTCOME AND FOLLOW-UP

The patient's creatinine rise began at the time of her diagnosis with uveitis, with her serum creatinine at 85 umol/L (baseline 63 umol/L). Prednisolone was commenced 2 days following the biopsy at 50 mg per day with trimethoprim-sulfamethoxazole for PJP prophylaxis, pantoprazole 40 mg daily for gastroprotection and calcium and vitamin D for bone health. After 6 weeks of immunosuppressive therapy, creatinine level improved to 90 umol/L (eGFR 64), and prednisolone was weaned to 37.5 mg for 2 weeks, then 25 mg for 2 weeks. Serum creatinine subsequently remained improved though static at 89 umol/L and the prednisolone dose was increased back to 37.5 mg for a slower wean to assess for further improvement in renal function. There was no recurrence of uveitis. Renal function then improved to baseline with trial of glucocorticoid-sparing agents. The patient did not tolerate azathioprine as a glucocorticoid-sparing agent, with an increase in her liver transaminases. Mycophenolate was commenced at 360 mg two times per day as a glucocorticoid-sparing agent, and her prednisolone dose continued to be weaned gradually with stable renal function. Following 12 complete months of treatment, including the initial prednisolone treatment with transition to mycophenolate treatment, she was weaned off all immunosuppressive therapy and is under monitoring for recurrence of TINU. Figure 4 shows the trend of her creatinine level over time, with arrows displaying the commencement and cessation of immunosuppression.

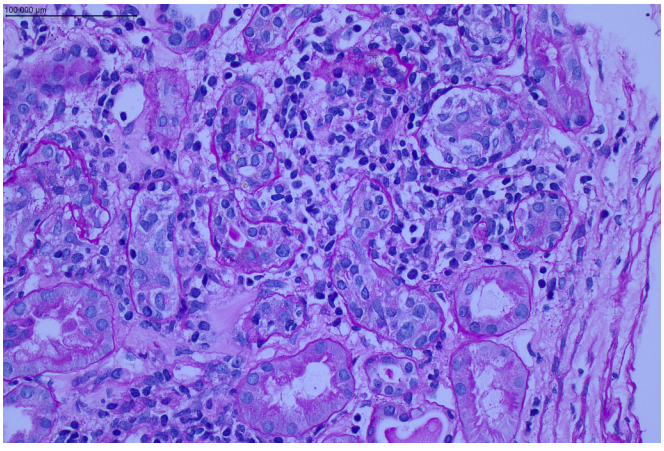

Figure 3 Periodic acid-Schiff Stain showing tubulitis.

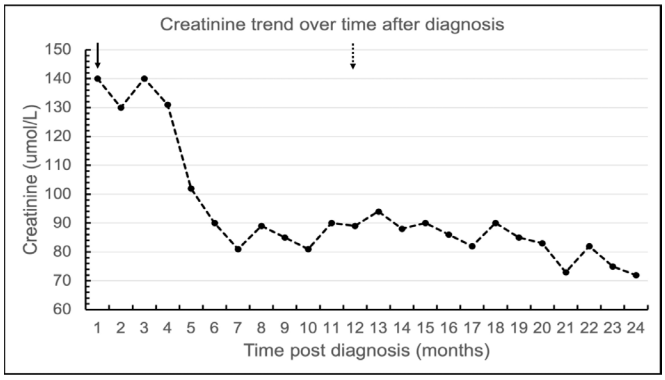

Figure 4 Creatinine trend post diagnosis and commencement of treatment. The solid arrow identifies the time of commencement of prednisolone and the dotted arrow marks the time of cessation of all immunosuppressive therapy.

Further investigation with a bone marrow biopsy was conducted by the local haematology service, and she was diagnosed with IgA monoclonal gammopathy of undetermined significance. This was then monitored by the haematology service with no change over 18 months and she was discharged from this service.

DISCUSSION

To our knowledge, this is the first case of TINU reported in an adult following COVID-19 infection. This remains a rare condition in adults, although TINU can manifest in patients in their 50s, and it can occur post COVID-19 in children. In one recent review, it appears there may be a small female predominance (65% female).[27] The aetiology of this condition remains unclear.[10 11 26] Evidence for the management of TINU continues to be based on case series and retrospective cohort studies. Due to the low prevalence of this condition, there are no randomised controlled trials conducted comparing agents. However, glucocorticoid therapy remains the main treatment option, with various glucocorticoid-sparing agents used in studies to maintain remission.[4 12–14] Prognosis appears to be good with good recovery.[14 26 27]

Although TINU can occur with other triggers, the clear temporal relationship between COVID-19 infection and the development of uveitis and proteinuria is strongly supportive of COVID-19 being the trigger for this case of TINU. A PubMed search was conducted for reported cases of children with TINU post COVID-19 infection as listed below in table 1. Most cases in children were responsive to high-dose steroids with good outcomes and prognosis and recovery of renal function.[23 24 28 29] As COVID-19 is a relatively new virus, this number will likely increase in the coming years. TINU can otherwise be triggered by other infectious causes such as Klebsiella, Chlamydia, herpes

Table 1 Cases reported of tubulointerstitial nephritis post COVID-19 infection

Author	Date of publication	Study type	Age (years)
García-Fernández[24]	2 May 2023	Case report	12
Maggio[23]	1 February 2023	Case report	7
Bilak[28]	2022	Case report	11
Sakhinia[29]	27 February 2023	Case series (4 cases)	13 (median age)

zoster and Epstein-Barr virus.[30–32] Autoimmune associations include rheumatoid arthritis, IgG_4-related autoimmune disease and thrombotic microangiopathy.[33–36] The pathogenesis of TINU remains uncertain, although some data suggest that it may be due to a modified CRP which is an autoantigen targeting renal tubular and uvea cells.[37]

In this patient's case, there was a good outcome with return to baseline renal function in regard to her eGFR. This was surprising considering the degree of fibrosis (50%) reported in the renal biopsy. This may be due to patchy fibrosis as well as sampling of the subcapsular cortex for light microscopy, which is an area that may over-represent global sclerosis secondary to age or non-specific scarring.[32] Renal biopsies are generally conducted with relatively small samples collected with 16 or 18 gauge core biopsy needles, with sample lengths sent for light microscopy ranging from approximately 8 mm to 18 mm depending on local practice. With varying degrees of fibrosis in different areas of the kidney, this may lead to variations in fibrosis depending on the area sampled.

The monoclonal gammopathy identified was unusual in this clinical context. A diagnosis of monoclonal gammopathy of renal significance (MGRS) was considered, although immunofluorescence staining was negative for significant deposition of immunoglobulins in the glomeruli, which suggested that this was not the mechanism of injury. Other features of MGRS were not found. There are case reports of interstitial nephritis associated with Sjogren's disease with monoclonal gammopathy, although our patient had negative ANA and ENA which is inconsistent with Sjogren's.[38–40]

There is evidence that suggests that patients with long covid may have deficiencies in cortisol.[41] Treatments are under investigation at this stage to evaluate the efficacy of glucocorticoid replacement.[42] Unfortunately, this patient's long covid symptoms persisted despite treatment with glucocorticoids and immunosuppression.

Learning points

- Tubulointerstitial nephritis with uveitis (TINU) remains a rare diagnosis, and although it has been extensively described in the paediatric population, it continues to occur occasionally in adults as well.
- TINU requires a histological confirmation for diagnosis and remains a differential for a patient with renal and ophthalmological symptoms.
- Glucocorticoid therapy is the main treatment for TINU.
- Rare complications of COVID-19 continue to emerge.

Contributors The following authors were responsible for drafting of the text, sourcing and editing of clinical images, investigation results, drawing original diagrams and algorithms and critical revision for important intellectual content: JL. The following authors gave final approval of the manuscript: JL and K-ST. Is the patient one of the authors of this manuscript? No. The guarantor is JL.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent for publication Consent obtained directly from patient(s).

Provenance and peer review Not commissioned; externally peer reviewed.

Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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