CASE REPORT

Acute kidney injury with hypoxic respiratory failure

Zachary Neubert,¹ Paul Hoffmann,² David Owshalimpur³¹Department of Internal Medicine, Madigan Army Medical Center, Tacoma, Washington, USA²Department of Anesthesia, Walter Reed Army Medical Center, Washington, District of Columbia, USA³Department of Medicine/ Nephrology, Madigan Army Medical Center, Tacoma, Washington, USA

Correspondence to

Dr Zachary Neubert,
zachary.s.neubert.mil@mail.mil

Accepted 31 August 2014

SUMMARY

A 27-year-old Caucasian man was transferred from a remote clinic with acute kidney injury for the prior 7–10 days preceded by gastroenteritis. His kidney biopsy showed non-specific mesangiopathic glomerular changes, minimal tubulointerstitial disease without sclerosis, crescents, nor evidence of vasculitis. On his third hospital day, he developed acute hypoxic respiratory failure requiring intubation and mechanical ventilation. Pulmonary renal syndromes ranked highest on his differential diagnosis. He was extubated after 2 days of mechanical ventilation and after pulse dose steroids. His lung biopsy showed pulmonary capillaritis. Our case describes a patient with clinically appearing renopulmonary syndrome, but found to have pulmonary capillaritis, a rare form of lung disease that may also cause acute kidney injury.

BACKGROUND

Systemic vasculitides are a group of multiorgan diseases predominately affecting the kidneys and lungs. The patient with these conditions often suffers from a variety of pulmonary renal syndromes. These syndromes are a combination of pulmonary haemorrhage and acute renal failure, which can lead to rapid deterioration and even death.¹ Patients may present with severe respiratory failure and/or acute renal failure requiring admission to intensive care unit (ICU).^{2–3} As this disease runs a fulminant course, early detection, treatment and monitoring are crucial for a patient's survival.³ We offer an interesting case of a young active duty male soldier who underwent rapid pulmonary deterioration after onset of acute renal failure.

CASE PRESENTATION

Our patient is a healthy 27-year-old Caucasian man with no medical history, who was seen by his primary care physician (PCP) roughly 10 days prior to arrival to our tertiary care centre for symptoms consistent with gastroenteritis. He was instructed to maintain hydration, and returned to his PCP's office 1 week later as his symptoms had not improved. Laboratory evaluation noted a serum creatinine of 8.8. He was air evacuated from his rural setting to our tertiary care centre. He was admitted to the general medicine floor where he was treated symptomatically over the weekend while awaiting a kidney biopsy on the following Monday. On the third hospital night, during routine vital signs check, the patient developed severe acute respiratory distress with oxygen desaturation to mid-70s on room air. He was placed on a 15 L non-rebreather mask with improvement in his oxygen saturations only to the mid-80s. His

respiratory status declined through the night requiring ICU admission, intubation and mechanical ventilation. He was stabilised and presumptively started on treatment for a pulmonary renal syndrome which included high-dose intravenous steroids and ventilatory support.

INVESTIGATIONS

We began an extensive workup for the aetiology of his initial acute renal failure, and subsequent respiratory failure. He only had a positive lupus anticoagulant antibody. Cardiolipin, glomerular basement membrane, proteinase 3, myeloperoxidase, ribonucleoprotein, DsDNA, chromatin, ribosomal P, SM, Ro/SSA, La/SSB, centromere B, Scl70, Jo antibodies, antinuclear antibodies were all negative. C reactive protein was elevated at 50 and erythrocyte sedimentation rate was elevated at 26 as expected given the patient's acute inflammatory response secondary to injury. C3, C4 were within normal limits. B2 glycoprotein IgA, IgG and IgM were negative. Fungal, sputum and acid-fast bacilli cultures were negative. Urine drug screen was negative for barbiturates, methylphenidates, benzodiazepines, tetrahydrocannabinol, cocaine, amphetamines, tricyclic antidepressants and phencyclidine. The patient was negative for hepatitis B testing. Kidney biopsy showed non-specific mesangiopathic glomerular changes, and the pathology report could not rule out postinfectious glomerulonephritis with minimal tubulointerstitial disease. The kidney biopsy was negative for vasculitis, sclerosis and crescents. Electron microscopy of the biopsy showed mild fibrosis with mild interstitial oedema. On having negative laboratory evidence of common vasculitis, and a relatively unremarkable kidney biopsy, a thoracotomy with wedge resection lung biopsy was performed. Lung biopsy showed acute alveolar haemorrhage with focal capillaritis, macrophages and erythematous changes without evidence of vasculitis. His CT chest was not consistent with diffuse alveolar haemorrhage. He was diagnosed with pulmonary capillaritis.

DIFFERENTIAL

- ▶ Pulmonary Vasculitides (Goodpasture's, granulomatosis with polyangiitis, microscopic polyangiitis, polyarteritis nodosa)
- ▶ Postinfection glomerulonephritis
- ▶ Acute valvular regurgitation
- ▶ Hepatitis
- ▶ Autoimmune (systemic lupus erythematosus (SLE), Sjogren's, Scleroderma, IgA nephropathy, Henoch-Schonlein purpura)
- ▶ Pulmonary embolism
- ▶ Pneumonia

CrossMark

To cite: Neubert Z, Hoffmann P, Owshalimpur D. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2014-206582

- Pulmonary oedema
- Pulmonary capillaritis

TREATMENT

The patient was started on methylprednisolone 1 g intravenous daily with quick improvement in his clinical condition. He was successfully extubated approximately 48 h of steroid therapy. Respiratory and renal function improved with steroids. The patient was discharged from the hospital 1 week after his period of respiratory failure with normal kidney function. He eventually returned to Alaska with close follow-up with his primary care provider and pulmonologist.

OUTCOME AND FOLLOW-UP

The patient was discharged from the hospital 1 week after his period of respiratory failure with normal kidney function and respiratory status. He underwent a slow corticosteroid taper and had complete resolution of symptoms.

DISCUSSION

The case above illustrates the classic rapid deterioration that can occur in patients thought to have pulmonary renal syndrome. The literature has not offered insight to a diagnosis when these classic pulmonary renal diseases have been ruled out. Pulmonary renal syndrome can be classified into three common groups. The first group are the antineutrophil cytoplasmic antibodies (ANCA) positive small vessel vasculitis, consisting of granulomatosis with polyangiitis, microscopic polyangiitis, and Churg-Strauss syndrome.⁴ The second group is the anti-GBM (Goodpasture's disease). The third group is SLE.⁴ Given negative ANCA testing, anti-GBM, anti-dsDNA, and normal complements, we were unable to diagnose the most common causes of pulmonary renal syndrome. A negative urine drug screen on admission made common drugs/toxins that could cause this acute decompensation less likely. Furthermore, our renal biopsy was negative for necrotising glomerulonephritis and negative for vasculitis, commonly found in pulmonary renal syndromes. Studies have noted that pathogenesis of vascular inflammation can be multifactorial with diverse origin in most cases.^{2 5 6} There is a lack of literature available on pulmonary–renal syndromes of unknown aetiology after ruling out the above causes. Herman *et al*⁵ described two cases of pulmonary renal syndrome of unknown aetiology in 1978 and postulated that these were caused by idiopathic rapid progressive glomerulonephritis. The literature has not exhibited further studies of similar presentation in which the commonly known aetiologies of pulmonary renal syndrome have been ruled out.

Postinfectious pulmonary renal syndrome and ANCA negative vasculitis remain possible aetiologies; however, these were not consistent with laboratory and biopsy findings. To provide further mystery to this case, typical vasculitis require high-dose methylprednisolone, cyclophosphamide and sometimes plasma exchange for resolution of symptoms.⁷ Our patient has significant improvement with his first daily dose of methylprednisolone.

One interesting diagnosis that remains a possibility in our patient was uncovered when the lung biopsy showed evidence

of pulmonary capillaritis, an inflammatory disruption of the alveolar interstitium and capillary network.⁷ Franks and Koss⁸ have linked pulmonary capillaritis to many of the common vasculitis, ones in which our patient tested negative. Although pulmonary capillaritis has been commonly linked to granulomatosis with polyangiitis, SLE and microscopic polyangiitis, 25% of patients with pulmonary capillaritis lacked evidence of an associated systemic illness.⁷ Furthermore, nearly all those who are acutely ill with pulmonary capillaritis develop respiratory failure requiring mechanical ventilation and subsequently acute renal failure.⁸ To the best of our knowledge and after an extensive literature search, our case is the first to have acute renal failure before development of respiratory failure.

Limited literature exists on atypical pulmonary–renal syndromes. Pulmonary capillaritis remains the most likely diagnosis for our patient despite having no signs of vasculitis on kidney biopsy.

Learning points

- It is important to keep capillaritis in the differential diagnosis for patients' who develop acute respiratory failure with acute renal impairment.
- Supportive care and anti-inflammatory agents, improve patients' ability to wean from artificial support.
- During an acute decompensation, it is important to re-evaluate for differential diagnosis and consider broadening it when current therapy has failed.
- It might be prudent to check basic laboratory values when flu-like and/or common cold symptoms persist after initial evaluation.

Contributors The contents of this case report were written by ZN, PH and by DO.

Competing interests None.

Patient consent Obtained.

Data sharing statement Not commissioned; externally peer reviewed.

REFERENCES

- 1 Sanders JS, Rutgers A, Stegeman CA, *et al*. Pulmonary: renal syndrome with a focus on anti-GBM disease. *Semin Respir Crit Care Med* 2011;32:328–34.
- 2 Mark EJ, Ramirez JF. Pulmonary capillaritis and hemorrhage in patients with systemic vasculitis. *Arch Pathol Lab Med* 1985;109:413.
- 3 Papis SA, Manali ED, Kalomenidis I, *et al*. Bench-to-bedside review: pulmonary–renal syndromes—an update for the intensivist. *Critical Care* 2007;11:213.
- 4 McCabe C, Jones Q, Nikolopoulou A, *et al*. Pulmonary –renal syndrome: an update for respiratory physicians. *Respir Med* 2011;105:1413–21.
- 5 Herman PG, Balikian JP, Seltzer SE, *et al*. The pulmonary renal syndrome. *AJR Am J Roentgenol* 1978;130:1141–8.
- 6 Jennings CA, King TE Jr, Tuder R, *et al*. Diffuse alveolar hemorrhage with underlying isolated pauciimmune pulmonary capillaritis. *Am J Respir Crit Care Med* 1997;155:1101–9.
- 7 Flanagan F, Glackin L, Slattery DM. Successful treatment of idiopathic pulmonary capillaritis with intravenous cyclophosphamide. *Pediatr Pulmonol* 48:303–5.
- 8 Franks TJ, Koss MN. Pulmonary capillaritis. *Curr Opin Pulm Med* 2000;6:430–5.

Copyright 2014 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow