CASE REPORT

Non-infectious aortitis in an immunosuppressed renal transplant recipient with IgA nephropathy

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SUMMARY

A 54-year-old woman presented with atypical chest pain, fever and malaise. She was immunosuppressed with three agents following a living-donor kidney transplant 1 year previously. Her native kidney failure was secondary to biopsy-demonstrated crescentic IgA nephropathy, with systemic involvement. A CT pulmonary angiogram revealed an inflammatory cuff of soft tissue around the descending thoracic aorta suggesting aortitis. Inflammatory markers were elevated. Given her immunosuppression, the patient was screened extensively for infective causes and was empirically commenced on intravenous meropenem. After 72 hours of no clinical or serological response to antibiotic therapy, negative microbiological investigations and worsening inflammation on serial imaging, she was commenced on high-dose methylprednisolone for presumed inflammatory aortitis. Symptoms and inflammatory markers rapidly normalised and the patient was discharged home on oral prednisolone. A clinical diagnosis of IgA-related aortitis was made. Imaging 3 months later showed complete resolution of the aortitis.

BACKGROUND

Aortitis is inflammation of the aorta.¹ It can be caused by various pathogenic mechanisms, broadly classified as infectious and non-infectious (table 1). Infectious aortitis is rare, but should be actively sought and excluded due to the high risk of aortic rupture and death if left untreated.^{1–7} Non-infectious aortitis is most commonly caused by rheumatological conditions, in particular the large vessel vasculitides, including giant cell arteritis and Takayasu arteritis.^{18 9}

IgA nephropathy is the most common type of glomerulonephritis in developed countries and is the leading cause of end-stage renal failure in young adults.^{10 11} In addition to renal disease, IgA nephropathy can be associated with a number of extrarenal manifestations including arthralgia and gastrointestinal vasculitis, but rarely skin involvement.¹⁰ The pathogenesis of IgA nephropathy is poorly understood, though it is widely believed to be a systemic, immune-mediated process.^{10–12} IgA nephropathy can recur post-transplantation,^{11 12} with one retrospective registry analysis showing a 10-year cumulative incidence of graft loss due to recurrent IgA nephropathy of 4.3%.¹³ There is no strong evidence to suggest that immunosuppression slows disease progression,^{14 15} except possibly

in crescentic IgA disease.^{16–18} The STOP-IgAN trial showed that addition of immunosuppression to supportive care in patients with high-risk IgA nephropathy had no effect on the rate of decrease in estimated glomerular filtration rate (eGFR) or on overall patient outcome, but was associated with higher rates of certain adverse events including infection and malignancy.¹⁵ However, individuals with crescentic IgA were excluded from this study.

Here, we report a rare case of non-infectious aortitis in a renal transplant recipient that developed despite triple-agent immunosuppression. To our knowledge, this is the first report of such a case. This case suggests a possible association between IgA nephropathy and inflammatory aortitis.

CASE PRESENTATION

A 54-year-old Caucasian woman presented in October 2016 with an 8-day history of intermittent ‘band-like’ pleuritic chest pain, which was relieved by sitting forward. She also reported shortness of breath on exertion, fevers and malaise. She had no headache, no scalp tenderness, no visual disturbance, no jaw claudication and no proximal muscle pain or tenderness. She had first presented 4 days previously and been prescribed doxycycline for a presumed lower respiratory tract infection; this had had no effect.

The patient had been diagnosed with crescentic IgA nephropathy in 2012 following renal biopsy investigation of rapidly progressing renal failure. She went on to receive a non-directed ‘altruistic’ living-donor kidney transplant in 2015, and at the time of presentation she was on triple-agent immunosuppression with tacrolimus, prednisolone and mycophenolate mofetil.

In addition, prior to transplantation she had previously experienced two episodes of myalgia and episcleritis, which were extensively investigated and in the absence of any immunological evidence of alternative diagnoses were diagnosed as probable extrarenal manifestations of IgA disease. At this time she was treated with mycophenolate mofetil.

Her other medical history included hypercholesterolaemia, gout, impaired glucose tolerance post-transplantation and ocular hypertension.

In addition to her immunosuppression, the patient’s regular medication included alfacalcidol, amitriptyline and Premique hormone replacement therapy (HRT). The patient had a non-anaphylactic penicillin allergy, was a lifelong non-smoker, drank 1–2 units of alcohol per week and worked as a civil servant.

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Table 1 Causes of aortitis^{1 2 7 36–52}

Infective causes	Non-infective causes
Bacterial	Large vessel vasculitis
<i>Staphylococcal</i> spp.	Giant cell arteritis
<i>Streptococcus pneumoniae</i>	Takayasu arteritis
<i>Enterococcus</i> spp.	Rheumatoid arthritis
<i>Salmonella</i> spp.	Systemic lupus erythematosus
<i>Treponema pallidum</i> (syphilis)	Ankylosing spondylitis
<i>Neisseria</i> spp.	Reactive arthritis
Rarer causes: <i>Coxiella burnetii</i> , <i>Pseudomonas</i> , <i>Escherichia coli</i> , <i>Klebsiella</i> , <i>Campylobacter</i> , <i>Yersinia</i> , <i>Brucella</i> , <i>Burkholderia pseudomallei</i>	
Mycobacterial	Other vasculitides
<i>Mycobacterium tuberculosis</i>	Antineutrophil cytoplasmic antibody-associated vasculitides:
Fungal	Granulomatosis with polyangiitis
<i>Aspergillus</i>	Microscopic polyangiitis
<i>Candida</i>	Polyarteritis nodosa
<i>Cryptococcus</i>	Beçhet's disease
Viral	Cogan syndrome
Varicella zoster virus	Sarcoidosis
HIV	Isolated aortitis—for example, isolated idiopathic aortitis

On examination at the time of presentation, the patient had a temperature of 38.2°C and remained febrile during admission. All other vital signs were within normal limits. Cardiovascular and respiratory examinations were unremarkable. There was no radio-radial or radio-femoral delay, no carotid or subclavian bruits and no splinter haemorrhages. Peripheral pulses were strong throughout and no blood pressure difference was detected between the two arms.

Figure 1 CT angiogram aorta showing periaortic cuffing of inflammatory tissue surrounding the descending thoracic aorta (arrow).

INVESTIGATIONS

Initial blood tests revealed raised inflammatory markers, with a C-reactive protein (CRP) of 356, white cell count of $13.79 \times 10^9/L$ and neutrophils of $10.46 \times 10^9/L$. Plasma viscosity was raised at 2.31 mPa/s (normal range for adults being 1.50–1.72 mPa/s). Plasma viscosity is superior to erythrocyte sedimentation rate (ESR) in the diagnosis and monitoring of inflammatory conditions.^{19–21} Plasma viscosity rises more quickly than ESR, correlates better with clinical condition and is subject to fewer false negatives as, unlike ESR, it is not affected by other physiological parameters including anaemia, hypoalbuminaemia and immunoglobulins.²¹ Of particular importance, and in contrast to ESR, plasma viscosity is not affected by corticosteroids, meaning that it remains a diagnostically useful test even if a patient has already commenced treatment.

In terms of other important investigation results for this patient, renal transplant function was excellent and unchanged from baseline, with a creatinine of 73 $\mu\text{mol/L}$ and Modification of Diet in Renal Disease (MDRD) eGFR of 81 mL/min/1.73 m². Haemoglobin, bone profile, liver function, cardiac troponins, ECG and chest X-ray were all unremarkable.

A CT pulmonary angiogram was performed on presentation to investigate for a possible pulmonary embolism. This revealed a very small peripheral occlusive pulmonary embolus in the right lower lobe pulmonary artery, thought to be an incidental finding that did not explain her symptoms. It also showed a cuff of soft tissue inflammation around the descending thoracic aorta suggestive of aortitis. A CT angiogram of the whole aorta confirmed this finding, demonstrating circumferential soft tissue thickening around the descending aorta with mild enhancement in keeping with periaortic inflammation (figure 1). There was no involvement of the ascending aorta or aortic arch, no extension below the diaphragm, no narrowing, dilatation, aneurysm or ulceration and no evidence of any other arterial involvement. A transthoracic echocardiogram was normal.

The initial priority in this immunocompromised patient was to exclude an infective cause of aortitis; however, a vast array of infective tests were negative (table 2).

C3 was marginally raised at 2.66 g/L, but C4 and IgG4 subclass were normal. Antineutrophil cytoplasmic antibody (ANCA) and antinuclear antibody were negative. It was not possible to acquire a tissue sample for histological examination as the area of inflammation was confined only to the descending aorta. A repeat CT aortic angiogram was performed after 3 days of empirical antibiotic treatment. This showed increased prominence of the cuff of inflammatory tissue surrounding the

Table 2 Results of microbiological investigations

Test	Results
Peripheral blood cultures (five sets)	Negative
Stool microscopy, sensitivity and culture	Negative
Urine for pneumococcal and legionella antigens	Negative
Atypical pneumonia serology (including <i>Mycoplasma pneumoniae</i> , <i>Chlamydia psittaci</i> and <i>Chlamydia trachomatis</i> lymphogranuloma venereum group)	Negative
Lyme, syphilis, <i>Coxiella burnetii</i> and leptospirosis serology	Negative
Rickettsial serology (spotted fever and endemic typhus)	Negative
Respiratory virus serology (including influenza A and B, respiratory syncytial virus and adenovirus)	Negative
Cytomegalovirus IgM and IgG	Negative
Hepatitis A IgG antibody	Positive (immune)
<i>Aspergillus</i> antigen, cryptococcal antigen and β -glucan	Negative

descending thoracic aorta, suggestive of worsening vasculitis. Other features remained similar in appearance and the abdominal aorta remained unaffected.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis in this case was between an infective and an inflammatory cause of aortitis. Table 1 lists the main differentials within each category. Given that this patient was immunosuppressed, an infective cause was initially considered most likely and was essential to exclude.

TREATMENT

The patient was initially treated empirically with intravenous meropenem while awaiting results of microbiological investigations. However, after 3 days of treatment there had been no clinical improvement, inflammatory markers remained raised and a repeat CT aortic angiogram showed worsening radiological features of aortitis. No infective organism had been identified and therefore a diagnosis of inflammatory aortitis was made. Three doses of 500mg intravenous methylprednisolone were given. Evidence suggests that in large vessel vasculitis, initiating steroid treatment with pulsed intravenous methylprednisolone results in higher rates of disease remission and also allows subsequent oral steroids to be reduced more quickly.^{22,23} Pulsed methylprednisolone was followed by a weaning course of high-dose oral prednisolone (1 mg/kg) as per European guidelines.²⁴

Apixaban was initiated to treat the small pulmonary embolus and the patient's HRT was discontinued.

OUTCOME AND FOLLOW-UP

The patient experienced a rapid and dramatic clinical improvement on commencing high-dose corticosteroids. Within 3 days, she was well enough to be discharged from hospital, with regular outpatient follow-up in transplant clinic. After discharge, the patient was reviewed in clinic, 9 days after starting steroids: she felt extremely well and her CRP had fallen to 26. A CT aortic angiogram 3 months later showed complete resolution of

Box 1 American College of Rheumatology 1990 criteria for classification of Takayasu arteritis; three or more criteria gives a sensitivity of 90.5% and a specificity of 97.8%²⁸

- ▶ Age at disease onset less than or equal to 40 years
- ▶ Claudication of the extremities
- ▶ Weak brachial artery pulse
- ▶ Systolic blood pressure difference greater than 10 mm Hg between the two arms
- ▶ Bruit over subclavian arteries or abdominal aorta
- ▶ Angiogram abnormalities (narrowing or occlusion of the entire aorta, its primary branches or large arteries in the proximal upper or lower extremities, usually focal or segmental and not due to atherosclerosis, fibromuscular dysplasia or other similar causes)

the aortitis with no aneurysmal dilatation (figure 2). The patient remains well at the time of writing.

DISCUSSION

This report describes a case of radiologically confirmed aortitis in a renal transplant recipient that was inflammatory rather than infectious in nature, as evidenced by the lack of identifiable organism, lack of response to broad spectrum antibiotics and rapid recovery with high-dose steroids. While there are numerous case reports of infective aortitis in transplant recipients,^{25–27} this, to our knowledge, is the first reported case of non-infectious, inflammatory aortitis. The aortitis described here does not fit with any of the most commonly recognised inflammatory aortitides. Box 1 summarises the American College of Rheumatology 1990 criteria for Takayasu arteritis,²⁸ none of which were satisfied in this case.

Similarly, this case failed to meet the 1988 Ishikawa criteria for Takayasu arteritis.²⁹ The history and examination findings in this case were not in keeping with a diagnosis of giant cell arteritis, and there was no other vessel involvement (in particular temporal artery disease) to suggest this diagnosis. There was no evidence of an alternative vasculitis, as all other immunological bloods test, including ANCA, were negative. Therefore, in the context of a patient with a history of extrarenal IgA disease, the case is in keeping with a diagnosis of IgA aortitis.

There have been few reported cases of possible IgA-related aortitis, and none, to our knowledge, in an individual who is immunosuppressed. In 1999, Kanahara and colleagues described the case of a 51-year-old woman who presented with hypertension, proteinuria, haematuria and deranged renal function shown to be due to IgA nephropathy on renal biopsy.³⁰ Simultaneous investigations revealed an incidental finding of aortitis involving the abdominal aorta, common iliac arteries and renal arteries. The authors postulated that there might be a human leucocyte antigen (HLA) association between the two conditions, because the patient in question had both HLA B52 and HLA DR4,³⁰

Figure 2 CT angiogram aorta 3 months post-diagnosis showing complete resolution of the aortitis.

A:	1,26 (10)	DR:	17(3), 12(5), 52
B:	8,56 (22)	DQ:	2, 7(3)
B _w :	6	DP:	04:01, 04:02
C _w :	1,7		

Figure 3 Patient's human leucocyte antigen type.

shown in previous studies to be associated with aortitis syndrome and IgA nephropathy, respectively.^{31–34} The patient in our case did not have either HLA B52 or HLA DR4: figure 3 shows her full HLA typing. In 2014, a case series was published to look at the possible causes of 21 cases of ascending aortic aneurysm that had histological evidence of aortitis.³⁵ Eight of the 21 patients had a history of some systemic or autoimmune disorder, one of whom had a prior diagnosis of IgA nephropathy.³⁵

IgA nephropathy can recur post-transplantation,³⁶ and this case describes a recurrence of extrarenal disease. However, this case of IgA-associated aortitis is particularly unusual in that it occurred relatively soon (approximately 1 year) post-transplantation, while the patient was significantly immunosuppressed with three agents, including steroids.

Learning points

- This report describes the first known case of non-infectious, inflammatory aortitis in a renal transplant recipient with IgA disease.
- In this patient with IgA nephropathy and a history of extrarenal IgA disease, this likely represents a case of IgA-related aortitis.
- This adds to evidence from limited other case reports that there may be an association between IgA nephropathy and aortitis.
- This is the first reported case of IgA-related aortitis in an immunosuppressed transplant patient.

Contributors CMS, SCC and PKB were part of the clinical team responsible for the patient's care during the reported presentation, conceived and planned the case report. CMS and SCC acquired the data regarding the case and investigation results. SCC and PKB reviewed and summarised the existing relevant medical literature, provided feedback on the first draft and approved the final submitted version. CMS wrote the first draft of the report, edited further versions of the draft and did a further literature review.

Competing interests None declared.

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