CASE REPORT

The failing heart: a bad case of the 'flu'

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SUMMARY

A 24-year-old Nepali man presented to hospital with a short history of feeling unwell with a flu-like illness. He subsequently went into acute renal failure requiring several sessions of renal replacement therapy by haemofiltration. The underlying aetiology of his renal failure was unclear. His renal function recovered following haemofiltration and he was discharged home with a plan for outpatient follow-up and investigations. He re-presented to hospital 6 days later with severe fluid overload. Echocardiogram was suggestive of impaired left ventricular systolic function; subsequent cardiac MRI confirmed this and was indicative of a dilated cardiomyopathy. A diagnosis of dilated cardiomyopathy with cardiorenal syndrome was made, most likely secondary to viral myocarditis in view of his initial presentation. He was diuresed and treated with prognostic medications for heart failure. His symptoms resolved and on subsequent outpatient review he was feeling well.

BACKGROUND

Cardiomyopathies are defined as diseases of the heart muscle. Subtypes include dilated cardiomyopathy (DCM), hypertrophic cardiomyopathy and restrictive cardiomyopathy.¹

DCM is characterised by dilation of one or both ventricles, leading to impairment of contractility and systolic dysfunction. It can be further subcategorised into ischaemic and non-ischaemic. Non-ischaemic DCM usually presents in young or middle-aged adults, and can present with heart failure, arrhythmia, thromboembolic complications or even sudden death. The prevalence has been estimated at 36 per 100 000, but the actual figure may be much higher, as it is thought that in many cases the condition is asymptomatic.²

DCM has a plethora of causes, the most common of which include alcohol, myocarditis, ischaemic heart disease/hypertension, infiltrative disease and connective tissue disease. In many cases DCM is idiopathic,³ with an increasingly growing body of evidence to suggest a genetic component. It is now felt that over 25% of DCM cases have a genetic basis and several genetic mutations have now been identified.⁴

DCM can have a poor prognosis in a relatively young population and so awareness of its causes and treatments is essential in clinical practice.

of fever, cough, sharp chest pain, shortness of breath, myalgia, and cervical and axillary lymph node enlargement. On presentation he had a low-grade pyrexia (temperature 37.9) and was tachycardic (heart rate 115 bpm, sinus tachycardia on ECG).

His oxygen saturations were 92% on air, with a respiratory rate of 26 breaths/min. On examination, bibasal crackles with decreased air entry were heard, more marked on the left. Chest X-ray was difficult to interpret due to poor inspiration and large body habitus (weight 105 kg, body mass index 35 kg/m²). He was treated with intravenous antibiotics for a presumed chest infection.

Despite a 3-week inpatient stay with prolonged courses of intravenous antibiotics, the patient's symptoms continued, and he subsequently developed acute renal failure, with serum creatinine >300 mmol/L, poor urine output and fluid overload. At this point he was transferred to a tertiary referral centre for haemofiltration, to which he had a good response. A full renal screen was essentially normal (see Investigations section for full details), although a renal biopsy was not possible due to large body habitus. No formal diagnosis for the cause of his renal failure was made at this point. Of note, an echocardiogram performed at this time was reported as showing preserved basal left ventricular (LV) function, but was unable to confirm preserved mid-cavity or apical function because of suboptimal views owing to body habitus and tachycardia. Following haemofiltration, the patient's renal function normalised, his symptoms resolved and he was discharged with a plan for outpatient renal follow-up, after around a month's inpatient stay.

The patient re-presented 6 days after being discharged, with severe dyspnoea, abdominal distension and peripheral oedema. On examination he was grossly fluid overloaded, with a raised jugular venous pressure, ascites, bilateral pleural effusions and pitting oedema to the knees bilaterally. On auscultation of the heart a gallop rhythm was heard. He was tachycardic (heart rate of 130 bpm, sinus tachycardia on ECG with no ischaemic changes) and hypoxic with saturations of 92% on air. His renal function had also deteriorated in the 6 days since his discharge, and he again had an acute kidney injury with a serum creatinine of 135 mmol/L, with an estimated glomerular filtration rate of 57.

INVESTIGATIONS

Investigations have been explained in [tables 1](#) and [2](#).

DIFFERENTIAL DIAGNOSIS

When the patient initially presented to his local hospital, he was treated unsuccessfully with

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CASE PRESENTATION

We describe the case of a 24-year-old Nepali man who moved to the UK in 2010. He had no known medical history, and initially presented to his local accident and emergency department with a history

Table 1 Results of renal, haematology and infective screens

Renal	
Autoimmune screen	Normal
Complement levels	Normal
Immunoglobulins	Normal
Urine PCR	Normal
Serum electrophoresis	Normal
Haematology	
β-2 microglobulin	Raised initially, then normal when checked subsequently (6 weeks later)
DAT	Positive initially, then negative when checked subsequently (6 weeks later)
LDH	Raised initially, then normal when checked subsequently (6 weeks later)
Urinary Bence-Jones protein	Normal
Bone marrow biopsy	No evidence of lymphoproliferative disease
Blood film	Red cell anisopoikilocytosis with target cells present. No spherocytes or polychromasia. White cell morphology within normal limits. No evidence of haemolysis
Microbiology	
Blood culture	No growth
Urine culture	No growth
Pleural fluid culture	No growth
Ascitic fluid culture	No growth
Viral serology (hepatitis/CMV/HIV/VZV/EBV/parvovirus B19/enterovirus/adenovirus)	Negative
Parasitic/toxoplasma serology	Negative
Leishmaniasis/ <i>Brucella</i> / <i>leptospira</i> / <i>Bartonella</i>	Negative
Syphilis	Negative

CMV, cytomegalovirus; DAT, direct antiglobulin test; EBV, Epstein-Barr virus; LDH, lactate dehydrogenase; VZV, varicella-zoster virus.

intravenous antibiotics for sepsis, presumed to be of chest origin, in view of his cough, shortness of breath and bibasal crackles on auscultation. After the development of acute renal failure and his transfer to the tertiary referral centre, it was still felt that his symptoms may have an infective cause, with the renal failure being due to sepsis-related acute kidney injury. He was discussed by the infectious diseases team, and had blood/urine/ascitic/pleural fluid cultures sent, all of which showed no growth. Full viral screen and parasitic serology were also negative (see [table 1](#), above). A primary renal cause for the patient's acute kidney injury and subsequent fluid overload was also suspected. However, urine analysis showed no significant proteinuria, and seven separate samples sent for urine protein-creatinine ratio were within normal range. A full renal screen was normal, although renal biopsy was not possible due to the patient's body habitus.

A diagnosis of lymphoproliferative disease was also considered, based on the initial presentation with lymphadenopathy, low-grade fever and systemic symptoms. A number of abnormalities were noted on his initial haematology work-up, some of which were in keeping with haematological malignancy, including raised β-2-microglobulin and lactate dehydrogenase (LDH; see [table 1](#)). A positron emission tomography (PET) scan was conducted for further assessment, and showed appearances consistent with a lymphoproliferative disease. However, the patient subsequently went on to have a normal bone marrow biopsy, and a repeat PET scan performed 6 weeks after the original scan showed resolution of the lymphadenopathy and splenomegaly previously seen, with no steroid or other specific treatment having been given between the two scans. The serum electrophoresis and immunoglobulin analysis did not fit with immune paresis.

The haematological workup also showed some results suggestive of an autoimmune haemolytic process (direct antiglobulin test (DAT) positive), with other results inconsistent with haemolysis (raised haptoglobin, low reticulocyte count). Repeat blood tests performed when the patient had recovered from his acute illness

showed normal haptoglobin, negative DAT and normal reticulocyte count, and subsequent blood film was suggestive of a mixed iron and folate deficiency picture as a cause of his anaemia. Unfortunately, a thiamine level was not sent, but wet beriberi would be another potential differential in a patient with nutritional deficiencies presenting with symptoms of cardiac failure.

Interestingly, the initial echocardiogram did not pick up the dilated LV or impaired systolic function. This was due to the fact that it was not possible to obtain good images owing to the patient's large body habitus and tachycardia. The report was therefore unable to comment on LV function other than to say that basal function appeared preserved. In retrospect, and in view of subsequent echocardiogram (shown in [figures 1 and 2](#)) and cardiac MRI findings, it seems likely that our patient did have a degree of acute heart failure at presentation, which the initial echocardiogram was not able to visualise. It is therefore likely that his acute kidney injury was caused by cardiorenal syndrome secondary to the acute heart failure, which in turn is thought to have been caused by viral myocarditis. This is in keeping with his initial presentation with generalised lymphadenopathy and a flu-like prodrome. Subsequent serial echocardiograms and cardiac MRI were consistent with DCM. Imaging results are summarised in [table 2](#), above.

An endomyocardial biopsy was not performed as this would not routinely be considered in the absence of significant gadolinium enhancement to suggest giant cell or sarcoid myocarditis.

TREATMENT

The patient was treated with fluid restriction, and a furosemide infusion was given to achieve diuresis. Despite this, his ascites and pleural effusions were slow to resolve, and an ultrasound-guided pleural drain was inserted for symptomatic relief of breathlessness. Unfortunately, the procedure was technically difficult due to body habitus, and the fluid drained was heavily blood stained, making analysis of LDH and protein unreliable, and differentiation between transudate and exudate not possible ([figure 3](#)).

Table 2 Imaging results*Imaging**Echo:*

The initial echocardiogram, performed shortly after the patient's transfer from his local hospital to the tertiary referral centre, was difficult to comment on due to fluid overload, tachycardia and body habitus. It was reported as showing preserved basal LV function but was unable to display mid-cavity or apical function because of suboptimal views

Following his readmission with symptoms of fluid overload (taken as the index admission), the patient had several repeat echocardiograms, which again were difficult to interpret but were suggestive of some degree of systolic function and LV dilation

One of these repeat echocardiograms is shown in [figure 1](#). The image shows a dilated LV with severely impaired LV systolic function, ejection fraction 22% by Mod Simpsons. Again, it is worth noting that there was poor endocardium definition and suboptimal image quality due to body habitus and fluid overload status

A subsequent follow-up echo is shown in [figure 2](#). This was performed as an outpatient after the resolution of his symptoms, around 3 months after the echo shown in [figure 1](#). The image shows a considerable improvement in LV systolic function with a non-dilated LV consistent with the findings on cardiac MRI

*NM whole body PET with FDG:**Initial PET scan*

The initial PET scan was performed shortly after the patient's transfer from his local hospital to the tertiary referral centre. It showed FDG avid nodes above and below the diaphragm, reversed hepatosplenic ratio and splenomegaly, and was reported as likely to represent a lymphoproliferative disorder

Repeat PET scan (6 weeks later)

The second PET scan was performed during the index admission. It showed no evidence of metabolically active disease; previously demonstrated metabolically active lymph nodes and splenic metabolic activity returned within normal limits

Cardiac MR

[Figure 3A, B](#) show images from cardiac MRI performed during the index admission. It was reported as follows:

Thoracic anatomy:

Normal arrangement of the cardiac chambers and the great vessels
There are large bilateral pleural effusions (right>left) with areas of lung atelectasis
No significant lymphadenopathy (poor resolution because of large pleural effusions)
There is ascites

Resting cardiac function:

Dilated LA (area: 27 cm²). RV size and systolic function is preserved. Dilated LV with severe global systolic dysfunction. No LVH. Mild MR. Mild AR through a trileaflet aortic valve (regurgitant fraction 9%)

LV function:

EDV	295.53 mL (range 102–235 mL)
ESV	183.34 mL
Stroke volume	112.2 mL
Ejection fraction	38.0%

Tissue characterisation:

No significant delayed gadolinium enhancement seen

Conclusion:

Dilated LV with severe global systolic dysfunction. No LVH. Normal RV size with preserved systolic function. Dilated LA. No previous infarct or fibrosis. Mild MR and TR
Large bilateral pleural effusions and ascites

The findings suggest a diagnosis of dilated cardiomyopathy

[Figure 4A, B](#) show images from the second cardiac MRI (performed 3 months later as an outpatient in cardiology follow-up clinic when the patient's symptoms had improved). Images show LV 2-chamber view with end diastole and end systole, respectively, with the improved LV systolic function. It was reported as follows:

EDV	256.76 mL
ESV	111.95 mL
Stroke volume	144.8 mL
Ejection fraction	56.4%
Cardiac output	10.6 L/min
Heart rate	73 bpm

Although the LV remains dilated, there has been considerable improvement in systolic function and a reduction in EDV as well as ESV. The EF is now normal-range. RV size and systolic function appear normal. There is no LVH. The LA is mildly dilated (28 cm² in 4-chamber area). The aortic valve is tricuspid and competent

AR, aortic regurgitation; EDV, end-diastolic volume; EF, ejection fraction; ESV, end-systolic volume; FDG, fluorodeoxyglucose; LA, left atrium; LV, left ventricle; LVH, left ventricular hypertrophy; MR, mitral regurgitation; NM, nuclear medicine; PET, positron emission tomography; RV, right ventricle; TR, tricuspid regurgitation.

Similarly, the patient's ascites was slow to improve, and it was felt that it was sufficiently tense to be compromising his renal function. An ascitic drain was therefore inserted, and analysis of ascitic fluid showed that ascitic protein was less than 65% of serum protein, in keeping with a transudative process as would be seen in heart failure.

Following the presumptive diagnosis of DCM, the patient was treated with ongoing diuresis and usual prognostic

treatment for heart failure (ACE inhibition (ACE-I), β -blockers and spironolactone).

His renal function rapidly improved with diuresis and drainage of ascites.

OUTCOME AND FOLLOW-UP

The patient achieved a good diuresis and was subsequently discharged. He was followed up in a cardiology outpatient clinic,

Figure 1 Repeat echo (as initial echo non-diagnostic due to poor views) showed dilated left ventricle (LV) with severely impaired LV systolic function. It is worth noting that there was poor endocardium definition and suboptimal image quality due to body habitus and fluid overload status.

where he reported feeling well, with no limitation in exercise tolerance and no symptoms of fluid overload. He has had a repeat cardiac MRI as an outpatient (see [figure 4A, B](#)), which showed considerable improvement in systolic function and a reduction in end-diastolic as well as end-systolic volumes, with a normal range ejection fraction. His renal function also returned to normal. He was followed up by the renal physicians, who maintained that his acute renal failure was likely due to acute tubular necrosis secondary to cardiac/viral illness.

DISCUSSION

It now seems likely that the patient's initial presentation was due to a viral infection, which caused a viral myocarditis, leading to secondary DCM and acute kidney injury due to acute tubular necrosis. As mentioned in the background section, there are a number of causes of DCM, including alcohol and drug

misuse, connective tissue disease and myocarditis. The patient did not have any significant drug or alcohol history, nor did he have any significant medical history; he had a negative autoimmune screen. This background, together with his initial presentation with acute onset of fever and lymphadenopathy, is consistent with viral myocarditis as the cause of his DCM.

DCM as a consequence of myocarditis is well described. A study looking at the final diagnoses in 1230 patients with initially unexplained cardiomyopathy showed myocarditis to be the underlying cause in 9% of cases.³ Several viruses are known to be causative agents, including the Coxsackie and influenza viruses, adenovirus, cytomegalovirus, varicella-zoster virus, Epstein-Barr virus, herpes simplex virus⁵ and parvovirus B19.⁶

Myocarditis can range from incidental findings of asymptomatic ECG changes on one end of the spectrum, to onset of cardiogenic shock on the other. This patient had a case of acute

Figure 2 Outpatient follow-up echo (after resolution of symptoms) showed considerable improvement in left ventricle (LV) systolic function with a non-dilated LV, consistent with the findings on cardiac MRI.

Figure 3 (A; diastolic view) Cardiac MRI acquired on index presentation reveal a dilated left ventricle (LV) with severely impaired LV systolic function with minimal change in end-systolic versus end-diastolic LV volume. (B; systolic view) Cardiac MRI acquired on index presentation reveal a dilated LV with severely impaired LV systolic function with minimal changes in end-systolic versus end-diastolic LV volume.

myocarditis. Interestingly, there was no late gadolinium enhancement suggestive of an acute myocarditis pattern in the subepicardial region on his cardiac MRI. Given the resolution in LV parameters and the return to normal LV function, it seems likely that the patient had an episode of acute myocarditis, which then improved spontaneously resulting in resolution of changes.

Lieberman *et al*⁷ classified myocarditis into four different categories including acute myocarditis, chronic active myocarditis, chronic persistent myocarditis and fulminant myocarditis, based on clinical course, histological findings and response to therapy. Fulminant myocarditis involved patients who became acutely unwell, usually after a well-defined viral prodrome, and either develop cardiogenic shock and spontaneously improve, or deteriorate and die from cardiac impairment. Histological analysis will show several foci of active inflammation.

Fulminant myocarditis is the most severe form of myocarditis, where there is significant myocardial injury and impairment resulting in the onset of cardiogenic shock or pump failure. Patients become hypotensive with signs of impaired peripheral perfusion (confusion or reduced Glasgow Coma Scale due to impairment of cerebral perfusion, decreased urine output, and acute kidney injury owing to renal hypoperfusion and hyperlactatemia due to anaerobic metabolism peripherally). Patients with

fulminant myocarditis may go on to require inotropes, vasopressors or even mechanical support with intra-aortic balloon pump counterpulsation or LV assist devices to help support the heart during the acute phase. In contrast, patients with acute myocarditis present with myocarditis that is less severe and may spontaneously improve or respond to immunosuppressive therapy or progress to DCM.⁷

The current data on the role of steroids in the treatment of myocarditis are limited; however, immunosuppressive therapy may be used in biopsy-proven, viral negative inflammatory cardiomyopathy, or in chronic active myocarditis or in cases of autoimmune mechanisms of myocarditis.

In one randomised trial of 111 patients, some were empirically given steroids based on a histopathological diagnosis of myocarditis with LV ejection fraction (LVEF) <45. This study showed that there was no significant change in recovery of LVEF in patients who were given steroids compared with those who did not receive steroids or other immunosuppressive therapy.⁸

The incidence of myocarditis is unknown, as it is thought that the majority of cases are asymptomatic and go undetected. In symptomatic cases, common presenting symptoms include chest pain on a background of fever and a flu-like illness. Besides progression to DCM, complications of myocarditis can include

Figure 4 (A; diastolic view) Images from second cardiac MRI (during follow-up) show left ventricular two-chamber view with end diastole and end systole, respectively, with the improved left ventricular systolic function. (B; systolic view) Images from second cardiac MRI (during follow-up) show left ventricular two-chamber view with end diastole and end systole, respectively, with the improved left ventricular systolic function.

acute heart failure, systemic or pulmonary thromboembolism, tachyarrhythmias and, rarely, heart block.⁹ Myocarditis and DCM overlap significantly with debate over the point at which a myocarditic picture can be considered to progress to DCM. An echocardiogram with LV systolic dysfunction is not sufficient to make the diagnosis of DCM, but it is generally felt that the diagnosis can be made after cardiac MRI, as MRI is able to exclude other causes of impaired ventricular function, such as ischaemic or infiltrative cardiomyopathy.

Much work has been carried out to uncover underlying mechanisms by which viral infection causes myocardial damage and subsequent progression to DCM. It seems likely that direct cytotoxic and immune-mediated damage both play a role. The immune response to viral myocarditis is thought to involve natural killer (NK) cells and NK-like cells in the first instance, accompanied by cytokines including interleukin 1 β (IL-1 β), tumour necrosis factor α , interferon- γ and IL-2 β , followed by T lymphocytes, peaking at around 7–14 days after initial infection. Mechanisms of immune-mediated damage may include

molecular mimicry and release of perforin molecules, which contribute to myocardial cytoskeletal disruption.¹⁰

In view of this, studies have been made to assess the potential for immunosuppressive treatment in patients with myocarditis, but results have been mixed.^{8 11}

The prognosis of patients with DCM due to acute myocarditis is variable, but there is the potential for recovery of LV function. A study looking at the use of intravenous immunoglobulins (IVIG) in patients with recent-onset DCM (defined as onset within the previous 6 months) found no benefit of IVIG, but found favourable short-term prognosis.¹² Patients should be treated aggressively for fluid overload with standard heart failure prognostic medications such as β -blockers, ACE-I and spironolactone.

This case is also interesting for the fact that acute kidney injury was a prominent feature. Acute cardiac failure is known to be a cause of acute kidney injury in a disorder known as cardiorenal syndrome. The pathophysiology of this condition is complex and several mechanisms have been proposed. Haemodynamic effects

of acute heart failure, including hypoperfusion of the kidneys due to a low output cardiac state, together with raised venous pressure leading to renal congestion, are likely to be important in the development of cardiorenal syndrome.¹³ Arterial underfilling also leads to activation of the renin-angiotensin-aldosterone system and the sympathetic nervous system, causing renal vasoconstriction, and sodium and water retention, thus worsening renal hypoperfusion and congestion.¹⁴ Five types of cardiorenal syndrome have been identified, and in this case the presentation is in keeping with cardiorenal syndrome type-1, with the kidney injury occurring as a result of an abrupt deterioration in cardiac function. There was no evidence of hypotension or glomerulonephritis to suggest type-3, and the acute onset rules out type-2 cardiorenal syndrome.

Learning points

- ▶ Viral myocarditis should be considered in a patient presenting with a non-specific viral prodrome and fever.
- ▶ Acute kidney injury of unknown aetiology may be due to acute hypoperfusion owing to an acute deterioration in left ventricular function.
- ▶ Standard heart failure treatment with drugs shown to have prognostic benefit should be used.
- ▶ Dilated cardiomyopathy of recent onset should be investigated and managed promptly, as recovery of ventricular function may be possible depending on the underlying aetiology.
- ▶ Echocardiogram may be difficult to interpret in patients with very high body mass index, so should be interpreted with caution, particularly if not in keeping with the clinical picture. Cardiac MRI offers greater diagnostic accuracy and, with the availability of cardiac MRI at tertiary centres, such patients should perhaps be investigated at tertiary cardiac centres.

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