PostScript

LETTERS TO THE EDITOR

Fatal perimyocarditis in a migrant worker to the tropics from an unexpected aetiology: a case report

Perimyocarditis is an acute inflammation of the pericardium and myocardium resulting in myocellular damage that may present as acute chest pain, electrocardiographic changes and raised cardiac enzymes. It is most commonly of viral aetiology and is usually asymptomatic with complete resolution expected in most cases. It can however lead to fulminant cardiac failure resulting in death or requiring cardiac transplantation.¹

Perimyocarditis is difficult to distinguish from an acute coronary syndrome without the benefit of a good clinical history and evaluation. Equally difficult and no less important is establishing the underlying aetiological pathogen responsible as it may alter the treatment and outcome for the patients involved.

This case report presents a case of fatal perimyocarditis in a young migrant worker with positive serology for mumps virus, and a review of the literature. It also highlights the importance of an exhaustive diagnostic work up in order to establish the aetiology of the illness.

Case report

The patient is a 29-year-old Indonesian migrant worker who had been in Malaysia for the last two years with no past history of any medical illnesses. He presented acutely to the emergency department with retrosternal chest pain, low grade fever over the preceding 7 days associated with progressive shortness of breath and a reduced effort tolerance. He was otherwise fit and healthy pre-morbidly and worked in the construction industry. He denied any history of intravenous drug use or any risk-taking behaviour. There was no recent history of jungle trekking, water immersion or travel.

On physical examination he was jaundiced, and tachypnoeic despite clear lung fields; blood pressure was 87/42 mm Hg despite being on intravenous dobutamine. On praecordial examination, the apical impulse was not felt and his heart sounds were distant without appreciation of murmurs. His jugular venous pressure extended to the earlobes and he had a hepatomegaly extending 3 cm below the costal margin. The rest of the examination was normal. Table 1 presents his bedside urine and preliminary laboratory studies, and fig 1A–C shows his ECG, chest x ray and echocardiogram.

Pathological findings

A transthoracic echocardiogram showed global hypokinesia with global pericardial effusion (fig 1C). A pericardiocentesis was performed and 1 litre of haemorrhagic fluid was aspirated. A coronary angiogram performed later that same day showed normal coronary angiographic findings (fig 1D,E). He was put on multiple antibiotic treatment to cover for Gram-negative microbes such as leptospira and typhoid which were common among

migrant workers. He was eventually intubated and given assisted ventilation in view of his worsening respiratory effort, poor oxygenation and persistently low blood pressure; however his multi-organ failure deteriorated further and he died on day 3.

Cultures from the blood and pericardiocentesis were negative for microbes and tuberculosis. His thick blood smear for malarial parasites was also negative. Cultures from the urine and stool were also negative for *Salmonella* or any other microbes. Table 1 presents a postmortem review of his serological findings. A postmortem myocardial biopsy revealed borderline myocarditis with lymphocytic infiltrate without myocyte damage. Subsequent viral and bacterial cultures were, however, negative.

Discussion

Perimyocarditis is almost exclusively due to an infective cause and the most common pathogen is viral.¹ It is relatively common, usually asymptomatic and has a spontaneous and complete resolution.² Anecdotal evidence seem to suggest that its prevalence could be as high as 40% of cases admitted for cardiac arrhythmias, congestive cardiac failure or abnormal electrocardiograms.² It is most commonly associated with parvovirus B19, enteroviruses, adenoviruses and cytomegalovirus; it is also known to be associated with mumps virus, although less commonly.^{2–4}

In contrast to western prevalence trends, anecdotally the most common cause of myocarditis in the tropics is bacterial. Examples include *Salmonella typhi*,⁵ *Mycobacterium tuberculosis*,⁶ *Leptospira*,⁷ *Rickettsia*,⁸ and malarial parasites.⁹ The most common viral pathogen appear to be dengue.¹⁰ However, under-diagnosis of viral myocarditis is to be expected in view of its usually asymptomatic nature and the lack of data on the aetiological prevalence of myocarditis in the tropics.

Mumps perimyocarditis is exceedingly rare in Malaysia and to date no data has been published on its prevalence here. This is again to be expected in view of the introduction of mass childhood measles–mumps–rubella (MMR) vaccination since 2002.¹¹ Indonesia however, has never introduced the MMR for mass childhood vaccination.¹² Migrant workers from Indonesia who constitute the largest single group of migrant workers in Malaysia would naturally be more susceptible to more severe manifestations of the mumps virus infection.

With a total migrant worker population of 1.8 million, a myriad of obscure and increasingly severe infectious diseases are becoming more apparent in Malaysia. The onus therefore lies on the medical fraternity to initiate an exhaustive diagnostic work up when assessing the aetiology of illnesses among its migrant population, as is aptly illustrated in this case report.

In conclusion, knowledge of the prevailing trend of perimyocarditis in the tropics, be it anecdotal, does not preclude the institution of sound clinical judgement and the initiation of an exhaustive diagnostic work up in order to establish the underlying aetiology of illnesses, especially in the migrant worker population.

Azarisman Shah Mohd Shah

Department of Medicine, International Islamic University Malaysia, Jalan Istana, Bandar Indera Mahkota, Kuantan, Pahang Darul Makmur, Malaysia

Mohd Daud Sulaiman

Department of Medicine, Hospital Universiti Kebangsaan Malaysia, Jalan Tenteram, Bandar Tun Razak, Kuala Lumpur, Malaysia

Table 1 Biochemical and serological findings

Full blood count	
Haemoglobin	12.7 g/dl
White cell count	12.5×10^9
Neutrophils	9.9×10^9 (79.3%)
Lymphocytes	1.5×10^9 (11.7%)
Monocytes	1.0×10^9 (8.1%)
Platelets	358×10^9
Renal profile	
Sodium	132 mmol/l
Potassium	5.4 mmol/l
Urea	13.2 mmol/l
Creatinine	152 μ mol/l
Liver function tests	
Albumin	38 g/l
Total protein	66 g/l
Bilirubin	53 μ mol/l
Alanine transaminase	49 U/l
Alkaline phosphatase	69 U/l
Creatine kinase	679 U/l
Troponin-T	0.66 μ g/l
Amylase	57 U/l
Urinalysis	
pH	6.5
Specific gravity	1.020
Leucocytes	Negative
Protein	5 g/l +++
Glucose	3 mmol/l +
Blood	250/ μ l +++
Bilirubin	17 μ mol/l +
Ketones	Negative
Arterial blood gases	
pH	7.340
pO ₂	19.2 mm Hg
pCO ₂	76.5 mm Hg
HCO ₃ ⁻	10.1 mmol/l
Postmortem serological findings	
Leptospira IgM	Negative
Widal Weil-Felix	Negative
Cytomegalovirus IgM	Negative
Epstein-Barr virus IgM	Negative
Haantan virus IgM	Negative
Anti-HIV 1 and 2	Negative
Anti-hepatitis A/C IgM	Negative
Hepatitis B surface antigen	Negative
Measles virus IgM	Negative
Dengue virus IgM	Negative
Mumps virus IgM and IgG	Positive
Behring Enzygnost anti-parotitis virus IgM and IgG (ELISA)	IgM 0.96, IgG 1.50*

*Samples with values below 0.10 were regarded as not detectable.

Figure 1 (A) ECG showing deep T-wave inversion in the anterior leads. (B) Globular heart in the upright posteroanterior view indicating a large pericardial effusion. (C) Transthoracic echocardiogram confirms the presence of a large pericardial effusion. (D, E) Coronary angiogram shows normal coronary arteries. LV, left ventricle; LA, left atrium.

Noorfaizan Saidin

Department of Medicine, Hospital Universiti
Kebangsaan Malaysia, Jalan Tenteram, Bandar Tun
Razak, Kuala Lumpur, Malaysia

Oteh Maskon

Department of Medicine, Hospital Universiti
Kebangsaan Malaysia, Jalan Tenteram, Bandar Tun
Razak, Kuala Lumpur, Malaysia

Correspondence to: Dr Azarisman Shah Mohd Shah,
International Islamic University Malaysia, Department of
Medicine, Jalan Hospital Campus, 25150 Kuantan,
Pahang, Malaysia;
risman1973@hotmail.com

doi: 10.1136/jcp.2006.044503

Accepted 26 October 2006

Competing interests: None declared.

References

- 1 **Magnani J, Dec WG.** Myocarditis: current trends in diagnosis and treatment. *Circulation* 2006;**43**:876–90.
- 2 **Cheng TO.** Prevalence of viral myocarditis. *Arch Intern Med* 2003;**163**:1742.
- 3 **Maisch B, Ristic AD, Portig I, et al.** Human viral cardiomyopathy. *Front Biosci* 2003;**8**:39–67.
- 4 **Spodick DH.** Mechanisms of acute epicardial and myocardial injury in pericardial disease. *Chest* 1998;**113**:855–6.
- 5 **Ruben J.** Typhoid fever. *Top Emerg Med* 2003;**25**:44–8.
- 6 **Dada M, Lazarus N, Khorsany A, et al.** Sudden death caused by myocardial tuberculosis: case report and review of the literature. *Am J Forensic Med Pathol* 2000;**21**:385–8.
- 7 **Vinetz JM.** Leptospirosis. *Curr Opin Infect Dis* 2001;**14**:527–38.
- 8 **Cowan G.** Rickettsial diseases: the typhus group of fevers—a review. *Postgrad Med J* 2000;**76**:269–72.
- 9 **Kohli R, Gulati S, Kapoor A, et al.** Cardiac tamponade: an unusual complication of *Plasmodium falciparum*-induced acute renal failure. *Nephrol Dial Transplant* 2003;**18**:1678–9.
- 10 **Wilder-Smith A, Schwartz E.** Current concepts: dengue in travellers. *N Engl J Med* 2005;**353**:924–32.
- 11 **Narimah A.** Rubella immunization in Malaysia—20 years on and the challenges ahead. *Med J Malaysia* 2005;**60**:1–3.
- 12 **Indonesia 2002/2003 Demographic Health Survey Final Report, Chapter 12.** Immunization of children. http://www.measuredhs.com/pubs/pub_details.cfm?ID=439&srchTp=advanced.

Polyarteritis nodosa in hairy cell leukaemia: an autopsy report

Polyarteritis nodosa (PAN) is a form of systemic necrotising vasculitis involving predominantly small- and medium-sized muscular arteries. Aetiological factors are usually unknown in the causation of polyarteritis nodosa, though some cases may be due to hepatitis B virus, cytomegalovirus, drug hypersensitivity, and malignancies. Hairy cell leukaemia (HCL) is characterised by pancytopenia, splenomegaly and inspirable bone marrow. The coexistence of the two diseases is highly unlikely by chance, and it is hypothesised that an aetiological relationship exists between the two conditions.

Case report

A 37-year-old man presented with a one-week history of fever and oliguria. On examination, he had generalised lymphadenopathy and hepatosplenomegaly. Other systemic examination was normal.

Laboratory tests revealed pancytopenia; erythrocyte sedimentation rate (ESR) was 56 mm in the first hour. Bone marrow aspirate was dry-tap. Trephine biopsy showed hypercellular marrow spaces with characteristic features of hairy cell leukaemia. These lymphoid cells expressed CD25 and CD103. He died from the respiratory illness.

An autopsy was performed after obtaining informed consent. Bone marrow and spleen (weight 1470 g) showed morphological features of HCL (fig 1A). Grossly, mesenteric (fig 2A), renal (fig 2B), and coronary arteries showed multiple segmental aneurysmal dilations measuring 0.4–1.8 cm in diameter. Kidneys (weight 190 g) showed geographic infarcts. Microscopically, there were variable changes including neutrophilic infiltration, mononuclear cell infiltrate, fibroblastic proliferation in the intima, fibrinoid necrosis, aneurysm formation, fragmentation of the internal elastic lamina, obliterative lumina, calcification and thrombus formation, indicating changes of acute, sub-acute and healed stages of PAN (fig 1B–D). No granulomatous reaction or glomerulonephritis was noticed. Ultrastructurally, glomeruli did not reveal any electron-dense deposits. Immunostain for hepatitis B surface and core antigen on liver