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CLINICAL SPOTLIGHT

18-FDG PET/ CT Scan in the Diagnosis and Follow-up of Chronic Q fever Aortic Valve Endocarditis

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Chronic Q fever endocarditis is a rare but important infection associated with risk of morbidity and mortality. Echocardiography rarely visualises the vegetative lesion. We describe the first Australian report of chronic Q fever aortic valve endocarditis confirmed with the use of 18 –FDG PET/ CT scan. Following valvular replacement, the patient had ongoing high serological titres despite active treatment and he was managed with yearly serial PET/ CT scan to confirm the absence of active infection. The utility of serial PET /CT scan imaging as a follow-up management strategy has not been described in the literature previously and should be investigated further.

Keywords Q fever • Coxiella • 18 FDG • Fluorodeoxyglucose • Positron Emission Topography • Endocarditis

Introduction

Q fever is a worldwide zoonotic infection caused by the bacterium *Coxiella burnetii* which can present as an acute or chronic disease [1]. Chronic Q fever infection has conventionally been defined as infection caused by *Coxiella burnetii* with a clinical evolution of more than six months and most commonly affects the heart, vascular system and bone (osteomyelitis). Recent literature, however, has described the use of serological criteria (ie phase I IgG antigen greater than or equal to 1 / 800) as the definition for chronic Q fever supplanting the notion of clinical chronicity [2].

Chronic Q fever endocarditis should be suspected in those with the relevant exposure risk factor with culture negative endocarditis. However, the diagnostic yield of trans-thoracic or transoesophageal echocardiography to localise a vegetative lesion is low [2,3]. Previous authors have reported positive 18-FDG PET / CT scans with Q fever endocarditis but its role in the diagnosis and follow-up of Q fever endocarditis is still to be determined [4,5].

Case Report

A 60-year-old Caucasian male with a history of bicuspid aortic valve presented to a country hospital in September 2010 with a four-month history of fatigue, fevers, night sweats, and weight loss (6 kg). He had acute Q fever in 2001 following exposure working as a truck driver for a local abattoir. His Q fever serology results then showed a phase II IgG of 1280 (diagnostic range: >200), and a phase I IgG of 80. He was treated with oral antibiotics but his follow-up serology showed evolution to chronic Q fever with a rise in the phase I IgG titre to 5120 (diagnostic range: >800) in January 2003.

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The patient was transferred to a metropolitan hospital with examination findings of splinter haemorrhages (Figure 1) on hands and feet, and a grade 3/6 ejection aortic systolic murmur. There was no evidence of hepatosplenomegaly or lymphadenopathy. His Q fever serology showed a phase I IgG IFA (immunofluorescence assay) of 5120 (endocarditis diagnosis: >800 for minor criterion, >6400 for major criterion), and phase II IgG IFA of 2560. Polymerase Chain Reaction (PCR) of blood sample for *Coxiella burnetti* DNA was negative. He underwent a transoesophageal echocardiogram (TOE) which showed a moderate aortic stenosis with thickened and calcified valve but there was no evidence of vegetation.

An 18-FDG (fluorodeoxyglucose) positron emission topography (PET)/CT scan (Figure 2) showed intense focal activity at the posterior margin of aortic valve with overlapping of anterior wall of left atrium. A diagnosis of Q fever endocarditis was made and the patient was commenced on oral doxycycline 100 mg BD and hydroxychloroquine 600 mg daily. His symptoms improved significantly with improved exercise tolerance, weight gain and resolution of the splinter haemorrhages. After 12 months both phase I and II IgG remained elevated at 8192 and 4096 respectively. Hydroxychloroquine levels were monitored and were in the recommended range. The medications were not without untoward side effects. The patient developed mild but acceptable skin pigmentation due to the hydroxychloroquine, and acute photosensitivity from the doxycycline was experienced if measures to avoid sunburn were not adhered to. He underwent a repeat TOE which showed no evidence of vegetation. The repeated PET/ CT scan still showed persistent FDG uptake at the posterior margin of the aortic valve. Clinically patient had worsening exertional dyspnoea, and mechanical aortic valve replacement was performed November 2011.

Intraoperatively, calcified bicuspid valve leaflets were seen with oedema and discolouration of the leaflets. In addition there was evidence of abscess with tissue necrosis extending from an area of the aortic root to the roof of the left atrium requiring extensive debridement (Figures 3 and 4). Tissue PCR confirmed infection with *Coxiella burnetii*. Following surgery his serological titres improved but remained elevated with phase I IgG of 4096, and phase II IgG of 4096 in August 2015. He underwent serial yearly PET scans which showed no uptake in 2012, minimal in 2013, no uptake in 2014 and minimal in 2015 (Figure 5). Clinically, the patient had good functional capacity with ability to resume full-time work. As of last follow-up, the patient remained on long-term doxycycline and hydroxychloroquine.

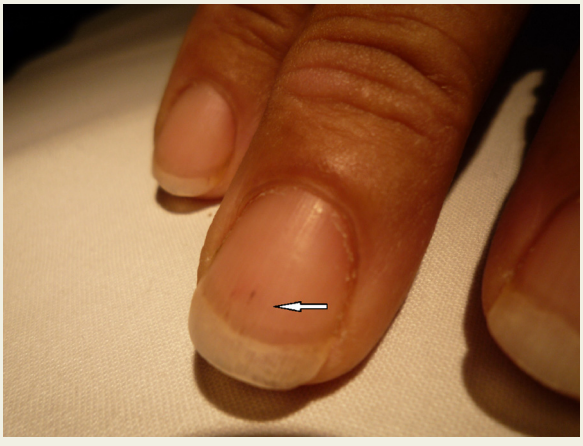

Figure 1 Splinter haemorrhages on fingernail.

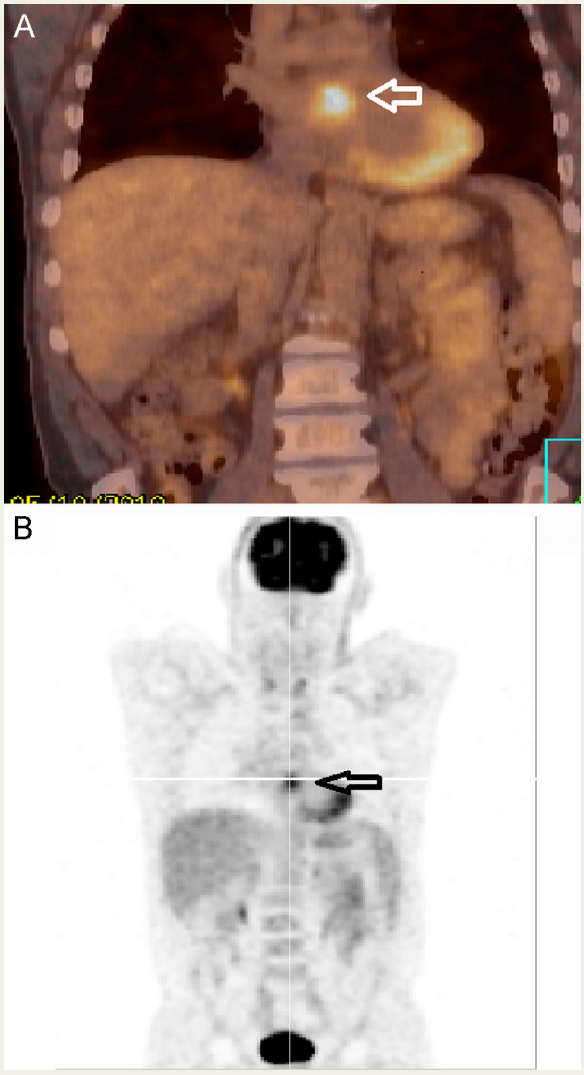

Figure 2 18 FDG PET/CT scan in 2010 with intense uptake at the aortic valve (with extension into the roof of the left atrium) on coronal combined PET/ CT image (A) and coronal MIP (maximum intensity projection) image (B) (Arrow).

Discussion

Q fever endocarditis is a serious manifestation of chronic Q fever which can develop months or years after the acute infection. The major risk factors for the development of Q fever endocarditis are prior acute Q fever, and valvular heart