Q Fever Endocarditis

Clinical Case Portal

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Abstract

This case illustrates an example of the clinical manifestations of chronic Q Fever leading to Q Fever endocarditis in a patient with known bicuspid aortic valve.
The case involves a 29-year-old male who had been working as a sheep shearer for many years.
The echo findings in this case demonstrate the destructive nature of this disease if left untreated in the acute phase.
The history and nature of the Q Fever bacterium is also discussed.

Introduction

Patient history prior to current observation

This 29 year old male New Zealand sheep shearer had been working remotely both in New Zealand and Australia.

Regular echocardiograms had been carried out in New Zealand over many years to monitor the bicuspid aortic valve, which was known to be significantly regurgitant with a lesser degree of stenosis.
Unfortunately the patient was non-compliant and did not always attend his appointments. This was due to both the nature of his work and the itinerant lifestyle he lead.
The significant aortic regurgitation had lead to a dilated left ventricle however surgery had been put on hold both at the patient's request and due to adequate completion of an exercise stress test which was performed to a high workload with no significant symptoms.
The Bruce Protocol was used. The patient exercised for 15 minutes to reach stage IV. Heart rates and Blood Pressure varied from rest 70bpm, 130/80mmHg to peak exercise 170bpm, 170/80mmHg.
He was regarded as being extremely fit at this time due to the physical nature of his work.
Prior to his current hospital admission he had been working in Western Australia as a shearer for two years. He had been finding the work load harder to manage over this period due to increasing breathlessness and over the last 12 months had to give up the work as he could no longer physically tolerate it.
Despite having developed increasing symptoms and having to cease his job he **still** did not seek medical advice. He felt that he was 'unfit' so began jogging.

Case Report

Clinical findings on admission, evolution and outcome:

On one occasion whilst jogging the patient had a syncopal episode associated with extreme shortness of breath and loss of consciousness. He was admitted to hospital and on examination he was found to be 'fit and muscular', pulse 100bpm, BP 110/66mmHg, RR 30-40 (Tachypnoeic at rest). JVP was not raised, there was marked clubbing of the fingers. Heart sounds were dual with a harsh ejection systolic murmur radiating to the carotids and a 3/6 diastolic murmur. There was a palpable apex, which was displaced laterally. The lungs were clear and the abdomen soft. ECG showed first degree heart block and marked left ventricular hypertrophy. Chest X-ray showed cardiomegaly but no overt heart failure. The patient described symptoms of a 'flu-like illness' 2 months prior with chest pain, discomfort, nocturnal dyspnoea, cough and dizziness.

A transthoracic echocardiogram was performed which showed a markedly thickened and distorted aortic valve with severe regurgitation and a severely dilated and impaired left ventricle. (Figure 1a,1b,2,3,5,6,7,9). In the ascending aorta there was an echocardiographic appearance of what seemed to be an aortic dissection flap. The aorta appeared to have a large false lumen that demonstrated colour Doppler flow through the cavity and around the valve and into the left ventricular outflow tract. (Figure 2, 4). The differential diagnosis was thought to be an abscess cavity(s). An urgent CT scan was performed after the echo. The CT did not demonstrate a dissection flap but there remained some suspicion of aortic dissection. As a result the patient was taken urgently to theatre.

- Surgical report:

The aorta was opened and the aortic valve pathology was consistent with chronic held infective endocarditis. There was a 2cm x 2cm cavity under the non-coronary cusp going on to the roof of the left atrium. The lining of the cavity was quite smooth and there was a thick scar. There was another 5mm x 5mm cavity at the base of the left coronary cusp about 7mm from the left main coronary ostium. There was also a 5mm x 7mm cavity at the non-coronary / right coronary commissure which looked likely to be an active infective cavity. The Aortic valve was excised. All cavities were cleaned with Rifampicin and placated with 4/0 Prolene. A 29mm St Jude Mechanical valve was chosen and successfully implanted in the aortic annulus.

- Histopathology Report:

Conclusion - Aortic valve: Markedly abnormal bicuspid heart valve showing fibrosis, dystrophic calcification and ossification as well as fibrinous exudation on the intimal surface. No acute endocarditis is seen.

Outcome:

This patient has since been back to hospital for a follow-up echocardiogram, which showed the 29mm St Judes aortic prosthesis functioning normally. There was a considerable reduction in left ventricular size. (Figure 8).

He is now working as a brick layers labourer, which is also a physically demanding job. He has improved clinically and remains on antibacterial therapy. He is on oral Hydroxychloroquine 600mg daily and Doxycycline 100mg bd which will be continued for at least 18 months. He is being monitored on a monthly basis with regular blood tests to check serology for the duration of treatment.

Discussion

Q fever is a zoonosis caused by the **Coxiella Burnetti** bacterium. Primary reservoirs are cattle, sheep and goats. Humans are most commonly infected by inhalation of contaminated aerosols such as dust or when in contact with contaminated straw, wool, hair or hides. Most at risk are abattoir workers, **sheep**

shearers and farmers. The bacterium is extremely infectious since one organism is sufficient to cause infection. Infection with **C. Burnetti** can be acute or chronic, and exhibits a wide spectrum of clinical manifestations.

It is prevalent in most places in the world except New Zealand (1).
Presentation of the disease is extremely variable and infection may lead to asymptomatic seroconversion, acute disease (ranging from flu-like syndrome to severe pneumonia requiring intensive care and / or hepatitis), or chronic infection (manifesting mainly as endocarditis 78%) (2).
This case demonstrates chronic infection leading to destruction of the aortic valve by endocarditis.

Q Fever is usually mild and recovery can occur without therapy however patients who have underlying valvular abnormalities will nearly always contract endocarditis. Therefore patients with clinically unknown or mild valve lesions such as bicuspid aortic valve are at high risk. Aortic valves are more commonly affected than mitral. The clinical picture depends very much on the diagnostic delay and in this case there has been a considerable delay, which is likely to span a few years.
Q Fever Endocarditis does not typically resemble acute endocarditis and vegetation's have a differing appearance as seen on the echo images showing a nodular appearance of the valve leaflets. (figure 9)

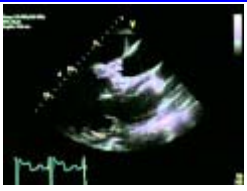
Routine blood cultures will always be negative unless using the modified Duke criteria – which include the C. Burnetti serology as a major criterion. These will help with the diagnosis (2,4).

Treatment for chronic Q fever endocarditis is a bactericidal treatment for 18 months to life. Monthly blood serology should be carried out. This should eradicate any remaining **C. Burnetti** and avoid relapses (2).
Q Fever continues to be an important disease in Australia. Despite the development of an effective vaccine that has been commercially available since 1989, the number of cases has continued to increase (3).

References

1. 'Q Fever is absent from New Zealand', International Journal of Epidemiology 1993, Vol 22, Number 5, Pp.945-949 , F Hilbink, M Penrose, E Kovacova and J Kazar (Central Animal Health Laboratory, Ministry of Agriculture and Fisheries, Upper Hutt, New Zealand.
2. Natural History and pathophysiology of Q Fever, The Lancet, Infectious Diseases Journal, April 2005, Volume 5, Issue 4, Pages 219-226 D Raoult, TJ Marrie, JL Mege (Marseille France and Alberta Canada Universities)
3. A Review of Q Fever in Australia between 1991 – 1994, Australia and New Zealand Journal of Public Health. 1997 Dec; 21(7):677-80. Garner MG, Longbottom HM, Cannon RM, Plant AJ,
4. Diagnosis of Q Fever, Journal of Clinical Microbiology. 1998 July; 36(7): 1823-1834, Fournier P-E, Marrie TJ, Raoult D.

Video 1 :
Transthoracic Parasternal Long axis view



Video 2 :

Parasternal Long axis zoomed view

Video 3 :
Parasternal Long axis zoomed view

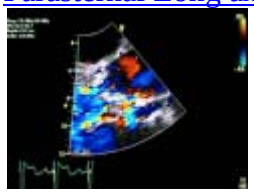
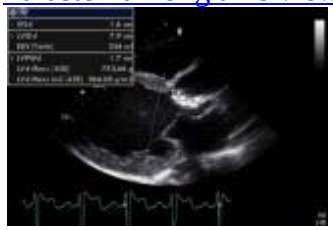
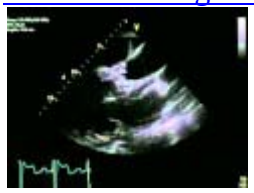


Fig. 1 :
Parasternal long axis view of the left ventricle



Video 4 :
Parasternal long axis view



Video 5 :
Apical 5 chamber view

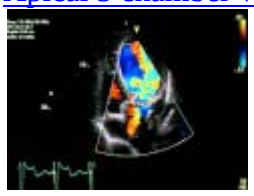


Fig. 2 :
Continuous Wave Doppler

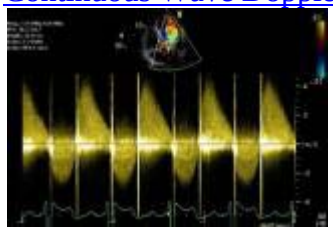
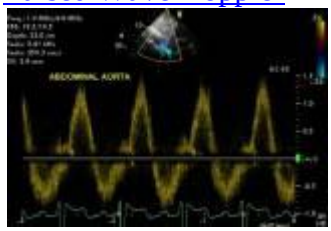
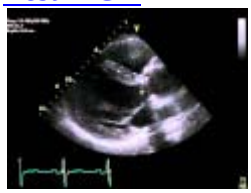


Fig. 3 :
Pulsed Wave Doppler



Video 6 :
Test MGA



Video 7 :
Short axis view