PERICARDITIS ASSOCIATED WITH TICK-BORNE Q FEVER

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Abstract:

A case of pericarditis associated with Q fever is described. Transmission was probably via an arthropod vector, which was most likely the kangaroo tick *Amblyomma triguttatum*. Complete recovery occurred in association with the use of non-steroidal anti-inflammatory agents only. This is a rare presentation of Q fever implicating transmission by a novel vector. (Aust NZ J Med 1989; 19: 254-256.)

Key words: Pericarditis, Q fever, *Coxiella burnetii*, arthropod vector, *Amblyomma triguttatum*.

We report here a case of acute Q fever with pericarditis which apparently occurred after multiple tick bites. This case is unusual in two respects; firstly, pericarditis as a major manifestation of Q fever is rare, and secondly, this case suggests that direct transmission of *Coxiella burnetii* by a tick vector is possible.

CASE REPORT

A previously healthy 30-year-old management consultant was bitten on the lower limbs and left iliac crest by five ticks while digging a well in scrubland near Toodyay (80 km north east of Perth) on 31 January 1987. A local reaction developed at the iliac crest site and nine days later he suddenly became febrile with malaise, nausea, headache, rigors, night sweats, and generalised pruritus. The day prior to admission he was commenced on amoxycillin and clavulanic acid and that evening experienced generalised arthralgia. The next day 14 February 1987 he developed sharp left sided chest pain which was aggravated by movement and inspiration. He presented to this hospital and was admitted.

On examination he was flushed, sweaty and unwell looking with an oral temperature of 39.5°C. A faint erythematous pruritic rash was present on the back and trunk and immediately posterior to the left iliac crest there was a brown 1 × 0.5 cm indurated lesion at the site of one of the tick bites. He was in sinus rhythm with a pulse rate of 100/min. Blood pressure was 100/60 (supine) and 80/50 (erect). Jugular venous pressure was not elevated, the apex beat was undisplaced but hyperdynamic, and a biphasic pericardial rub was heard at the lower left sternal edge as well as a fourth heart sound. The remainder of the physical examination was normal.

On admission, laboratory investigations revealed the following: hemoglobin 14.6 g/dl, white cell count $5.2 \times 10^9/l$ (3.9 granulocytes, 0.9 lymphocytes, 0.3 monocytes), platelets $170 \times 10^9/l$ and ESR 9 mm/hr. Urea, electrolytes, creatinine, bilirubin and alkaline phosphatase were within normal limits. AST peaked at 285 u/l (reference range 6-42) 18 hours after admission, as did CK at 460 u/l (reference range 30-180) and CK MB at 26 u/l (reference range less than 25). An electrocardiogram showed sinus rhythm, left anterior hemiblock and concave ST elevation in leads I, aVL and V_{1-5} , which evolved with the development of T wave inversion over the next three days (Fig. 1 (i) and (ii)). A chest X-ray was unremarkable and an echocardiogram demonstrated normal ventricular function with fractional shortening of 37%, left ventricular diastolic diameter of 6.2 cm (reference range 3.7-5.3 cm) and systolic diameter of 3.9 cm (reference range 2.5-3.6 cm). Bodysurface area was 1.99 cm². There was no evidence of pericardial effusion. Eight sets of blood cultures yielded no growth. Auto-antibodies were not detected and serology for Ross river virus, influenza virus, coronavirus, enterovirus, chlamydia and mycoplasma did not indicate active infection. Phase 2 antibody titres against *coxiella burnetii* rose from 1:10 on admission to 1:640 eight days later. Phase 1 antibody titres remained undetectable throughout. Weil Felix reaction and immunofluorescence for spotted fever and typhus group rickettsia antibody was negative.

The patient required intravenous morphine on the night of admission for control of his chest pain but subsequently settled with oral paracetamol with codeine and ibuprofen 400 mg t.d.s. His temperature remained elevated until the sixth day of admission. He was discharged on the seventh day after reducing the dose of ibuprofen to 200 mg t.d.s. Two weeks later he noticed a painful swelling of the right testis which spontaneously resolved

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Fig. 1 (i)

Fig. 1 (ii)

Fig. 1 (iii)

Figure 1: Electrocardiograms performed (i) On admission.
(ii) Three days later. (iii) Four months later.

two days later. Four weeks after discharge his ibuprofen was ceased resulting in a recurrence of his chest pain. This resolved with recommencement of ibuprofen which was successfully withdrawn two weeks later. When reviewed four months after discharge the patient was well. The ECG had returned to normal limits (Fig. 1 (iii)), and the *Coxiella burnetii* phase 2 antibody titre remained at 1:640 without detection of phase 1 titre.

DISCUSSION

Q fever in Australia has usually been transmitted by inhalation of infected aerosols associated with abattoirs.¹ Transmission by milk and laboratory aerosols² has been described in cases from other continents but appears uncommon in this country. Q fever is rare in Western Australia accounting for three of 367 cases occurring in Australia in 1986.³ Our case had no history of such exposure and he developed acute Q fever nine days after multiple tick bites. The ticks were most likely *Amblyomma triguttatum* as this is the predominant species of tick in the area.⁴ This species is known to carry *Coxiella burnetii* and transmit Q fever to kangaroos⁵ but has not been directly implicated in transmission of Q fever to humans before. We have been unable to find reference in the literature to other species of tick transmitting Q fever to humans.

Our case had the typical clinical and electrocardiographic features of pericarditis. Four additional cases of pericarditis associated with Q fever occur in the English literature. Three of these cases had acute pericarditis with typical chest pain and pericardial rub which completely resolved following corticosteroid or antibiotic therapy.^{6,7} All of these patients had radiological cardiomegaly and two patients had pneumonitis with elevated ESR. The fourth reported case⁸ presented with myocarditis and subsequently developed chronic pericardial fibrosis despite antibiotic therapy (pneumonitis and pleural effusions were also noted). Chest pain is well described in Q fever⁹ and it is possible that some cases of pericarditis are missed if a pericardial rub is not sought or electrocardiogram is not performed.

Myocarditis associated with Q fever may present with ischemic type chest pain, dyspnea or dysrhythmia.^{10,11} The transient fourth heart sound and left anterior hemiblock in our case suggested some myocardial involvement but at no stage did he exhibit clinical or echocardiographic evidence of left ventricular dysfunction and the chamber dimensions were within the reference range when corrected for surface area. Involvement of sub-epicardial myocardium is, however, common in pericarditis.¹²

Pneumonitis was present in 7% of cases of Q fever in a large series reported by Spellman¹ but was not seen in our case, possibly reflecting the non-respiratory portal of entry. The ESR was not elevated in our case, a finding seen in 13% of cases in Spellman's study. This study also noted rash in 8% of cases and orchitis in 1% of cases, both features being observed in our patient.

Acute Q fever is associated with elevated phase II antibody titres. Chronic Q fever may manifest as endocarditis or hepatitis, and is associated with elevation of phase I antibody titres. The risk of progression from acute to chronic disease with or without specific antibiotic therapy is unknown.²

In summary, we have presented a rare complication of Q fever not previously described in Australia. In addition we have implicated the role of a tick vector in direct transmission to humans for the first time.

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