Could it be Q fever?

Seven lessons for Australia from the recent Dutch epidemic

Q fever (*Coxiella burnetii* infection) is a common zoonosis in Australia, especially in rural Queensland and New South Wales.[1] However, the largest outbreak of Q fever ever identified was in the Netherlands in 2007–2010, with about 4000 people affected.[2] While further research is needed on best practice to control transmission, detect and treat infection, and monitor acute cases for the early detection of chronic Q fever, lessons have already emerged for Australian medical and public health practitioners.

Lesson 1: suspect Q fever, even when there is no direct animal contact. Q fever is often considered an occupational hazard associated with working in agricultural and meat processing industries; however, the Dutch outbreak shows that under certain environmental conditions, Q fever can become a community-acquired infection. The outbreak originated at "intensive" dairy goat farms built close to towns. In the dry conditions, *C. burnetii* was dispersed many kilometres from the infected goats and fields fertilised with infected goat manure, and was transmitted to humans through inhalation of infected droplets or dust. Only 3.7% of affected patients worked in at-risk occupations;[3] the main risk was associated with living within 2 km of an infected farm.[4] Similarly in Australia, there is growing evidence that a high proportion of patients with Q fever do not have any animal contact, although this is still the main risk factor.[5] However, data about mode of acquisition, which could inform vaccination strategies (eg, community versus risk factor-based programs), are not currently collected at a national level.[5]

Lesson 2: consider antibiotic treatment, even if the patient appears to have recovered spontaneously. Whereas acute Q fever usually presents as a self-limiting, flu-like illness (sometimes complicated by pneumonia or hepatitis), chronic infection is more serious, usually manifesting as endocarditis or vascular infection, especially in patients with pre-existing damaged cardiac valves or aneurysms. A case–control study conducted after the Dutch outbreak suggests that treatment of acute Q fever may reduce the risk of developing chronic infection,[6] although this finding needs to be confirmed through larger prospective studies. Treatment is usually with oral doxycycline; *C. burnetii* is not susceptible to β-lactam antibiotics.

Lesson 3: request laboratory tests for Q fever diagnosis that are sensitive and specific. For patients with a recent onset of illness (within the past 10 days), request "*C. burnetii* PCR (polymerase chain reaction)" testing, as PCR results are more likely to be positive than serology early in infection, and request to "store serum" (see Lesson 4). For patients with a later presentation, request "*C. burnetii* serology" testing.

Lesson 4: follow up clinically and with serology, depending on risk-factor profile. Early detection and prompt treatment of chronic Q fever is vital. Experience in the Netherlands suggests that a routine transoesophageal echocardiogram (TOE) is not useful,[7] and the degree of serological follow-up should depend on the risk factor profile.[8] In a patient with pre-existing cardiac or vascular lesions, a TOE and PCR with serial serology at 3 months, 6 months and 12

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months is appropriate.[8] If the patient is clinically well and has falling antibody titres at 12 months, further monitoring is not necessary. In patients without risk factors, serological follow-up at 6 months and 12 months only is recommended. Initial and follow-up serological assays should be performed by the same lab so that changing titres are more likely to reflect clinical change rather than potential variability between laboratories.

Lesson 5: monitor serology in pregnant women with untreated Q fever infection. Although a population-based study in the Netherlands found no evidence of adverse outcomes in pregnancy,[9] there is uncertainty about the consequences of untreated infection in pregnant women. Recommended practice is to monitor serology monthly until delivery. If titres rise, treat with combined trimethoprim and sulfamethoxazole (comtrimoxazole), followed by doxycycline postpartum.[10]

Lesson 6: recognise post-Q fever fatigue. Post-Q fever fatigue syndrome (fatigue persisting for more than 12 months after infection) was reported in 44% of 515 Dutch patients with long-term follow-up.[11] Although the syndrome is recognised in many parts of the world, its pathogenesis remains unclear.

Lesson 7: collaborate in surveillance and disease control. The Dutch outbreak emphasises the importance of Q fever surveillance in both humans and animals, and of established mechanisms for communication between veterinarians and public health practitioners. Abortion waves in dairy goat herds, affecting up to 60% of goats in some herds, were diagnosed up to 2 years before the outbreak in humans, and 3 years before the implementation of mandatory notification of Q fever in dairy goats and of measures to reduce transmission.[2] A collaborative alliance between industry stakeholders, health departments, vaccine manufacturers, general practitioner groups and the community is essential to ensure timely and appropriate public health action.

Australia is the only country with a licensed Q fever vaccine (Q-VAX; CSL Limited). This highly effective vaccine requires prevaccination screening tests (to exclude individuals likely to have hypersensitivity reactions to the vaccine) and is currently not recommended for children aged less than 15 years.[12] Research is required to determine the safety and effectiveness of Q-VAX in children, as the risk of infection rises rapidly in the teenage years,[1,13] and childhood infection can be debilitating.[14] An even better approach would be to develop a new (subunit) vaccine that does not require pretesting. Nevertheless, based on the epidemiological profile of reported cases (see Lesson 1), we would recommend greater use of the current vaccine in adults living in rural Australia.

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