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Coxiella burnetii (Q-Fever)

Q-fever is a globally distributed zoonosis and caused by the Gram-negative intracellular bacterium *Coxiella burnetii*. It is found most commonly amongst goats, sheep, and cattle, with human infection primarily occurring through direct contact with animals (Bielawska-Drozd *et al.*, 2013). Routes of transmission include inhalation of contaminated aerosols, and ingestion of contaminated animal products including raw milk. Those most at risk of infection include farmers, abattoir workers, meat packers and vets. Direct human-to-human transmission is thought to be relatively rare. Only a single organism is required to establish infection in humans (Brooke *et al.*, 2013), and a resistant, infectious spore-like form can persist in the environment for considerable lengths of time.

In humans, acute infection with *C. burnetii* is typically asymptomatic (~60% of cases) and is thus likely to go under-reported. Other presentations of acute illness include flu-like symptoms including headaches, fever, dry cough and atypical pneumonia. In up to 4% of cases, the patient can experience neurological disorders including meningitis and encephalitis.

Rarely, a more serious chronic infection can develop which can lead to endocarditis (in ~70% of cases), granulomatous hepatitis, chronic fatigue syndrome, chronic hepatitis, osteomyelitis and death (Fournier *et al.*, 1998; Picchi *et al.*, 1960). The acute illness will usually clear quickly with appropriate antibiotic therapy, whilst the chronic illness can require antibiotic treatment over several years (Kersh, 2013). The number of Q-fever cases reported in humans in the UK are relatively few (less than 100 per year), although there have been a number of notable outbreaks (Hawker *et al.*, 1998).

Requirements for intracellular multiplication together with the necessity for biosafety level 3 facilities restrict the cultivation of *C. burnetii* to specialized laboratories and so culture diagnosis is not possible (Mori *et al.*, 2007). Diagnosis of Q-fever using serological methods is unreliable since it can take several weeks before antibodies reach detectable levels, and these can persist after infection has cleared.

Our assay:

At Micropathology Ltd, we use a qualitative single-round traditional PCR assay which targets the *superoxide dismutase* gene for the detection of *C. burnetii*. Currently, UKAS accredited specimen types for this assay are EDTA whole blood, bronchoalveolar lavages (BALs) and tissues. Other sample types may be tested, and

are reported alongside a caveat to state the assay is not UKAS accredited for testing alternate sample types.

Turnaround times are stated in the user manual (http://www.micropathology.com/customer-downloads-handbooks.php) with results usually available in practice much sooner than the given time frame. Where there is a delay, we are usually confirming a result and addressing clinical data given with the specimen.

References:

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