Our new PCR test for an early Q Fever diagnosis

At Sullivan Nicolaides Pathology we have developed a new real-time PCR test for Q fever that delivers an early diagnosis to help you with a timely start to treatment.

Dr Jenny Robson, Head of Microbiology

Q fever is one of the most important zoonotic infections in Australia, with about 300 cases notified annually, particularly from rural Queensland and New South Wales (Figure 1). Domestic ruminants—cattle, sheep and goats—are the main reservoirs of human infection. Massive multiplication of the organism in infected animal placentas and fetal fluids results in environmental contamination. Organisms are also shed in faeces, urine, milk and other body fluids, such as vaginal mucus and saliva.

Coxiella burnetii, the causative bacterium, is resistant to desiccation, allowing prolonged survival in the environment. Fewer than 10 organisms are needed to establish infection via the respiratory route. Rural and abattoir workers are most at risk, however new reservoirs for human infection identified include cats, dogs and wildlife (e.g. marsupials) accounting for the increasing recognition of sporadic urban cases without classic risk factors.

Q fever can be broadly divided into acute and chronic forms. Acute infection can be totally asymptomatic in 50 to 60% of cases. There are two main symptomatic presentations of acute disease that follow an incubation period of 14–21 days. The first is a self-limiting flu-like illness with high fever, rigors, profuse sweats, extreme fatigue, muscle and joint pain, severe headache and photophobia. There is usually evidence of hepatitis ranging from isolated increase in serum transaminases to clinically-apparent hepatomegaly without jaundice. Community acquired pneumonia is the other manifestation. Cases range from being mild to having acute respiratory distress syndrome.

Diagnosis of acute infection

The clinical diagnosis of acute Q fever is challenging because of its non-specific manifestations; hence laboratory confirmation is important. Leucopaenia, thrombocytopenia, the presence of atypical lymphocytes and moderately raised hepatic transaminases suggest the diagnosis. Hyponatraemia due to inappropriate secretion of antidiuretic hormone (SIADH), hypocholesterolaemia and a raised CRP are also commonly noted non-specific laboratory features. Specific Q fever antibodies are often absent during the first 10 to 14 days of infection, making early diagnosis by serology difficult.

Antigenic variation and Q fever serology

C. burnetii displays biphasic variation of its cell wall antigens resulting in different degrees of virulence. This antigenic variation is referred to as Phase 1 and Phase 2. After acute infection, antibodies to Phase 2 antigens are the first to develop, followed by antibodies to Phase 1 antigens. As with other infections, Phase 2 IgM develops before Phase 2 IgG. Phase 2 IgG seroconversion, a four-fold rise in Phase 2 IgG titres or Phase 2 Complement Fixing antibodies (Phase 2 CFT) in convalescent serum is diagnostic of acute Q fever (Figure 2).
Diagnosis of chronic infection

About 1–4% of infections develop chronic disease, particularly in patients with predisposing factors – preexisting valvular surgery, vascular prosthesis, aneurysms, renal insufficiency and older age (> 60 years). Chronic Q fever diagnosis relies on a combination of clinical parameters, development of persistently high Phase 1 IgG ≥ 1:1280 ± elevated Phase 1 IgA antibodies and imaging supportive of chronic infection. Q fever PCR may be of value in this setting with involved tissue being Q fever PCR positive. Serum Q fever PCR positivity in this condition is less sensitive, being of the order of 60% or less.

Algorithms for monitoring patients after acute Q fever depend on the presence of risk factors for chronic infection and remain controversial. Serology is generally recommended at 9 months, or 3, 6 and 12 months, with optional PCR and imaging if risk factors are identified.

GPs have a role in prevention by promoting Q Fever vaccination. Prevaccination screening includes both serology and skin testing in order to prevent hypersensitivity reactions.

Correction – rebate for plasma metanephrines

Please be advised that plasma metanephrines are eligible for Medicare rebate. The article ‘Preferred screening test for phaeochromocytoma’, published in Syzygy July 2013, incorrectly stated that this test was not Medicare rebateable.

So, what’s new?

All requests for Q fever serology will also generate a Q fever PCR in combination with serology.

Benefits

• earlier diagnosis
• identification of more clinical cases where convalescent serology is not collected

Request forms – clinical notes

In order to interpret the results of combined PCR testing and serology it is very useful to record the duration of symptoms in clinical notes.

Test name: Zoonoses PCR
Specimen: SST (0.5 mL)
Transport: Ambient
Medicare: Rebate available

We welcome Dr Carl Kennedy

DR CARL KENNEDY BSc MBBS (Hons) FRACP FRCPA

Carl, a graduate of The University of Queensland, trained in Clinical Immunology and Allergy and Immunopathology at the Royal Brisbane Hospital and Princess Alexandra Hospital. In addition to his work in pathology, Carl practises as a consultant immunologist and allergist, which gives him personal insight into difficulties faced by his clinical colleagues in the diagnosis and management of allergy and autoimmune disorders. As a medical student and later a registrar under a Queensland Health rural scholarship, Carl worked in many remote locations as far afield as Mt Isa, Roma, Childers, Bundaberg, Gympie and Gin Gin, which gave him an appreciation of the challenges faced by rural and remote practitioners.