Molecular diagnosis of Acanthamoeba infection

Acanthamoeba spp. are a family of free-living protozoans, that have two stages to their life cycle, a trophozoite stage that feeds on small algae, bacteria and other protozoa and dormant cysts that survive in air, soil, dust and water (Dart et al 2009).

At Micropathology Ltd we perform a test for Acanthamoeba sp. based on molecular amplification with a next day target turnaround time.

Acanthamoeba have recently made national news headlines as one of the perils of wearing contact lenses (http://www.bbc.co.uk/news/health-32791627). In the cornea Acanthamoebas are thought to feed on Keratocytes (Dart et al 2009). The pathogenicity is linked to their ability to cause host cell death by interfering with host cell signalling, secreting toxins and an ability to phagocytose host cells (Khan, 2006). The BBC article mentions 60 new cases of this particularly infection. In the same period at Micropathology we detected 79 cases out of 741 specimens. At around 10% of suspected specimens being positive this is in agreement with the French study of Maubon et al (2012). Common specimen types include corneal swabs, scrapes, contact lens fluid and contact lenses themselves.

Acanthamoeba encephalitis, first described in 1972 is an extremely rare infection of the central nervous system that is seen in both healthy and immunocompromised individuals. Primary amoebic meningoencephalitis (caused by Naegleria fowleri) and granulomatous amoebic encephalitis (caused by Acanthamoeba spp and Balamuthia mandrillaris) are the two clinical manifestations of CNS amoebic infection. Of the over 400 cases that have been reported in the literature, only two to three percent of those affected survived (Kaushal, 2008). Dissemination occurs when acanthamoebae spread haematogenously from the upper respiratory tract or skin lesions into the brain parenchyma (Anderson et al, 2012).

Acanthamoeba meningoencephalitis is a slowly progressing infection with typical symptoms including headache, fever, neck stiffness, seizures, altered mental status and neurological symptoms leading to coma and death within one week to several months after onset. Treatment is difficult due to poor rates of diagnosis and a lack of antimicrobial therapy, resulting in the high mortality rate of the disease (Anderson et al, 2012). There are reports of successful treatment of patients using a combination of trimethoprim-sulfamethoxazole, rifampicin and ketoconazole (Singhal et al, 2001).

In the case of the far more common Acanthamoeba keratitis, for a good prognosis, early detection followed by prompt treatment is essential (Dart et al 2009). Traditional culture techniques involve inoculating the specimen onto a lawn of
*Escherichia coli* on non-nutrient agar for 3-6 days with occasionally up to 3 weeks (Dart et al 2009; Maubon et al 2012). This is not in keeping with the urgency of the situation as the disease develops. It has also been suggested that detection by molecular amplification can also aid in the detection of extra-corneal spread of Acanthamoeba since accurate histological confirmation is often difficult (Dart et al 2009).

**References**


