Due to the ubiquitous nature of latent tuberculosis, and the unpredictability of its transition to active tuberculosis, there will always be a requirement to depart from the principle of diagnostic parsimony (so-called Occam’s razor) by invoking dual infection (in which *Mycobacterium tuberculosis* plays a part) in the aetiopathogenesis of disease in sites such as the lung and meninges even though, at those sites, it is exceptional for disease to be caused by more than one pathogen at a time. A transition from latent to active tuberculosis is to be expected in immunocompromised patients such as those with human immunodeficiency virus/acquired immune deficiency syndrome (HIV/AIDS), with the consequence that community-acquired pneumonia which initially appears to be solely attributable to bacteriologically validated pneumococcal infection may subsequently be shown to co-exist with active pulmonary tuberculosis.

Likewise, in a patient with immunocompromise attributable to sickle cell disease, an initial diagnosis of pneumococcal meningitis does not mitigate against the subsequent documentation of co-existing tuberculosis meningitis. However, even in the absence of immunological compromise, patients from communities with high background prevalence of tuberculosis may also experience co-existence of *M. tuberculosis* and other organisms during the course of meningeal infection. This was exemplified by an otherwise healthy 25-year-old Chinese immigrant (seronegative for HIV) in whom the CD4 count of 299 cells/μl was deemed to be reduced only in proportion to the severity of her underlying invasive tuberculosis infection.

Her CD4 subsequently rose to 399 cells/μl after she responded to treatment. Although cryptococcal meningitis was the sole initial diagnosis, based on a positive antigen test and identification of the organism on direct smear of the cerebrospinal fluid (CSF) and also on CSF culture, coexisting tuberculous meningitis was subsequently validated by CSF culture and by her response to antituberculous chemotherapy. Even in the era antedating the recognition of HIV/AIDS, the background prevalence of tuberculosis was sufficiently high to be a risk factor for the co-existence of bacterial and mycobacterial infection in the meninges, as was the case in a 26-year-old army recruit who had an initial diagnosis of microbiologically validated meningococcal meningitis but an eventual diagnosis of autopsy-validated co-existing tuberculous meningitis.

**Conclusion**

In certain contexts, and in defiance of diagnostic parsimony, clinicians should maintain a high index of suspicion for co-existing active tuberculosis even when a non-mycobacterial organism appears to be the sole culprit pathogen.

**Conflict of interest:** None declared.

**References**