



## The Changing Face of Syphilis Infection

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### Letter

Syphilis is a resurgent sexually transmitted infection in the UK which is disproportionately diagnosed in patients living with HIV, particularly men who have sex with men. Evidence exists to suggest that syphilis presents differently in patients with HIV, particularly in those with severe immunosuppression. Progression to neurosyphilis is more common in HIV co-infection and can be asymptomatic, often for several years. Symptoms of neurosyphilis vary but can include meningitis, meningo vascular disease, general paresis and tabes dorsalis. Debate exists surrounding in which circumstances to perform a lumbar puncture and the current gold standard diagnostics have inadequate sensitivity. We recommend a pragmatic approach to lumbar punctures, interpretation of investigations, and when to consider treatment with a neuro penetrative antibiotic regimen [1].

Syphilis, caused by the spirochaete bacterium *Treponema pallidum*, has seen a resurgence in high-income countries in recent years, particularly among men who have sex with men. The widespread availability of penicillin in the United States and other industrialized countries following World War Two resulted in rates of syphilis falling from 76 per 100,000 populations in 1945 to 4 per 100,000 in 1955-57. After this period syphilis became concentrated within men who have sex with men and incidence surged during the 1980s HIV/AIDS epidemic. In response to the fear induced by the epidemic, changes in sexual behaviour caused another decline until recently where rates have risen rapidly again. In the United States during 2014-2015 syphilis occurred in 7.5 cases per 100,000, the highest rate since 1994. Similar trends were seen in England where in 2016 the number of cases was 5920, 12% higher than the previous year and the highest number of new diagnoses since 1949, with 80.9% of cases reported in men who have sex with men.

Syphilis infection involves a number of stages. Primary syphilis classically presents 9-90 days after infection with a single, non-tender genital ulcer called a chancre which represents the first site of *T.pallidum* invasion. If untreated, primary infection progresses to secondary syphilis, typically 12 weeks, but sometimes up to 12 months after initial infection. The classic presentation of secondary syphilis is a rash which typically involves the trunk, may involve the hands or feet and may be accompanied by condylomata lata, wart-like lesions around the anogenital region. Latent syphilis results when both primary and secondary syphilis are not treated and is defined by serological proof of infection but no symptoms. It is divided into early and late latent syphilis, with early latent syphilis infectious and late latent syphilis, defined in the UK as more than two years after infection, found to generally be non-infectious [2].

Neurosyphilis is a broad term used to describe the direct invasion of *T pallidum* into the nervous system and can affect the brain, spinal cord and peripheral nerves. Approximately 25-40% of patients will have 'neuro invasion' at some point, typically during the primary or secondary stage of infection, but the majority will spontaneously clear the infection from the cerebrospinal fluid (CSF) without requiring treatment for neurosyphilis and potentially without having any symptoms. In patients whose immune system is unable to clear

the infection, neurosyphilis will occur and this can also present with or without symptoms, the latter form known as asymptomatic neurosyphilis [3].

Syphilis can affect the eyes in a multitude of ways. Conceivably the most well remembered ocular manifestation is the Argyll Robertson pupils which accommodate but do not react to light and thus demonstrate light-near dissociation. This finding is highly specific for syphilis but can also be seen in diabetic neuropathy or as part of a dorsal midbrain syndrome. Anterior or posterior uveitis is perhaps most commonly encountered but papillary conjunctivitis, scleritis, neuro retinitis and retinal vasculitis are also observed, usually in secondary and tertiary stages. Furthermore, syphilis is on the important list of differential diagnoses for presentations of progressive optic neuropathy, and thus should be excluded in patients whose optic neuritis is 'atypical'. Ocular syphilis is considered to be a type of neurosyphilis, although it is not always accompanied by syphilitic meningitis or abnormal CSF results. However, given that the recommended treatment is the same neuro penetrative regimen used for neurosyphilis, it may not be necessary to perform CSF analysis in every case [4].

Further research is required to determine to what extent previous or ongoing syphilis infection in people living with HIV contributes to neurocognitive decline and which patients with HIV and syphilis co-infection should be actively investigated for neurosyphilis. Comparison of serological and clinical outcomes of patients with HIV and early syphilis treated with single dose Benzathine Penicillin G compared to the neuro penetrative procaine penicillin regimen is needed. More research is also required to validate improved diagnostic markers such as CXCL13. Case One: A 42 year old HIV positive man presents to a sexual health clinic with a primary chancre and evidence of *T.pallidum* on dark ground microscopy. His most recent CD4 count is 55 cells/ $\mu$ L and he is not on antiretroviral therapy. Neurological examination including fundoscopy is normal. This man should certainly be offered a lumbar puncture. He is at high risk of neurosyphilis as well as other opportunistic infections that can cause meningoencephalitis. If the lumbar puncture identifies WBC.

Case Two: A 26 year old HIV positive man presents with a 24 hour history of fever, headache and neck stiffness. He is on antiretroviral therapy, his most recent CD4 was 550 cells/ $\mu$ L and he reports having attended a sex party four weeks ago. Syphilis serology identifies positive EIA and TPHA but negative VDRL. Lumbar puncture reveals WBC of

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12 cells/  $\mu\text{L}$ , predominantly lymphocytes, and CSF VDRL is negative. The bloods suggest either primary syphilis or a previously treated infection. Correlating this with previous serology and a good history should help to establish which. If primary syphilis is likely then this event signifies neuro invasion of *T.pallidum* which is often cleared spontaneously by patients but given the symptoms and pleocytosis he should be treated with a neuro penetrative penicillin regimen. If this is not a case of primary syphilis then other causes of his symptoms and pleocytosis, such as a viral meningitis, should be sought [5].

This man has certainly had syphilis in the past and he may well have been treated without remembering. However, his bloods may indicate late-latent infection which may be the cause of his cognitive impairment. A negative CSF-VDRL does not rule out this diagnosis and he should be treated for neurosyphilis whilst continuing other investigations for his cognitive decline.

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