Disseminated gonococcal infection with meningitis: a rare presentation

A 46-year-old male was admitted to a tertiary care hospital in KwaZulu-Natal Province on 30 January 2016 with a one-day history of confusion, inability to communicate and urinary incontinence. He had no history of seizures or head trauma. He was HIV-positive and had been recommenced on first-line ARVs two weeks prior to admission, following a prolonged period of non-adherence. He had completed treatment for pulmonary tuberculosis in 2013. On examination he was cachectic and apyrexial. His vital signs were normal. He had no skin lesions or evidence of arthritis. Cardiovascular and respiratory system examination revealed no abnormalities. There was no neck stiffness. Salient findings on central nervous system examination included a GCS of 13/15, and a right-sided hemiparesis (left upper motor neurone pattern). Preliminary laboratory investigations revealed a neutrophilia, mild anaemia, and a raised CRP (152 mg/dl) and ESR (81 mm/hr). His CD4 count was 154 cells/µl. Syphilis serology was negative. His chest radiograph was normal.

Due to the presence of focal neurological signs, lumbar puncture was deferred. CT brain scan revealed features consistent with a meningitis and vasculitis of the left middle cerebral artery with resultant cerebral infarction. Blood cultures taken on admission grew an oxidase-positive Gram-negative coccus. A panel of biochemical tests (Vitek 2 bioMérieux) identified the organism as Neisseria gonorrhoeae. The patient was commenced on high-dose intravenous ceftriaxone, but his condition deteriorated and he demise approximately two weeks later. The isolate was referred to the STI Laboratory at the Centre for HIV & STIs, NICD, for confirmatory identification. Molecular testing using an in-house multiplex PCR, as well as a real time N. gonorrhoeae specific PCR, verified the phenotypic identification.

In South Africa, periodic aetiological surveillance of STI syndromes conducted by the NICD has revealed that N. gonorrhoeae is the cause of 70-80% of male urethritis syndrome (MUS) and approximately 10% of vaginal discharge syndrome (VDS). Disseminated gonococcal infection (DGI), which results from bloodstream invasion of the organism, typically develops 2-3 weeks after primary mucosal infection. Although most patients will not give a history of recent urogenital disease, the majority (up to 80%) have evidence of asymptomatic mucosal (urogenital, rectal, pharyngeal) infection. Patients usually present with one of two syndromes, with some overlap between the two forms: (1) a triad of tenosynovitis, dermatitis and polyarthritis; or (2) purulent asymmetrical oligo/mono-arthritis without associated skin lesions. Fever is usually (but not invariably) present during the acute bacteraemic stage of infection. However, central nervous system (CNS) infection with N. gonorrhoeae is a rare manifestation of DGI. Disease onset is acute, and includes features of meningo meningism and confusion. Infection may be complicated by cerebral vasculitis and the development of focal neurological signs. In immunocompromised patients, the prognosis may be worsened by the development of overwhelming sepsis and disseminated intravascular coagulation. Laboratory investigations for extra-genital infection should include, where applicable, synovial fluid analysis and at least two sets of blood cultures. Suspected meningitis necessitates lumbar puncture and cerebrospinal fluid analysis. Additionally, mucosal sites (urethral, cervical, rectal, pharyngeal) should be sampled both for bacterial culture and validated molecular testing.

In 2015, the National Department of Health STI management guidelines for primary healthcare centres were formally revised with respect to the syndromic management of MUS and VDS. In response to the increase in N. gonorrhoeae antimicrobial resistance observed worldwide, a pre-emptive strategy of dual antimicrobial therapy was incorporated to curb the emergence of resistance to extended-spectrum cephalosporins. Specifically, oral cefixime was replaced with single doses of injectable ceftriaxone and oral azithromycin. Recommended antimicrobial treatment of DGI consists of dual therapy: intravenous ceftriaxone for 7-14 days and a 1 g stat dose of oral azithromycin. The public health response involves tracing and treating direct sexual contacts with stat doses of intramuscular ceftriaxone and oral azithromycin.

References

Source: Attending nd pathologist. Centre for HIV & STIs, NICD-NHLS (ranminik@nicd.ac.za).