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Sex. Transm. Inf. 2005;81;133-134
doi:10.1136/sti.2004.011668

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CASE REPORT

Early syphilis presenting as a painful polyradiculopathy in an HIV positive individual

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Neurosyphilis and neurological complications from syphilis may be commoner in HIV disease. With outbreaks of early syphilis in HIV positive individuals being observed over recent years, rare neurological manifestations of secondary syphilis will be observed more commonly. We describe a case of an HIV positive individual whose first presenting feature of early syphilis was a polyradiculopathy.

Over the past 5 years there has been a dramatic rise in the number of cases of early syphilis seen in Western Europe, North America, and Australia.¹ This has largely been due to a number of outbreaks predominantly in men who have sex with men (MSM).² The outbreaks have included high numbers of HIV co-infected individuals.

Case reports and retrospective studies suggest that HIV may alter the clinical presentation and natural history of syphilis³; however, this remains controversial.⁴ Observations suggest early neurosyphilis and neurological complications from syphilis may be commoner. Recent clinical observation data have shown that individuals with early syphilis are more likely to present with secondary syphilis in HIV co-infection.⁵

To date there are two reports of early syphilis causing a painful polyradiculopathy in HIV positive individuals.^{6,7} In these cases the diagnosis of secondary syphilis was established before the development of the polyradiculopathy. We describe a case of an HIV positive MSM whose first presenting feature of early syphilis was a polyradiculopathy.

CASE REPORT

A 50 year old white MSM, diagnosed HIV-1 positive in 1995, presented in September 2003 with a 5 day history of lower back pain and right leg weakness. There was no history of recent trauma or recent travel. There was no associated fever, bowel symptoms or urinary symptoms. He had a history of *Pneumocystis jiroveci* pneumonia (PcP) twice in 1996 and 1998 with no other relevant medical history and no history of syphilis. He was antiretroviral experienced, having been on numerous regimens, often changing as a result of intolerance and toxicity. Three months before this presentation he commenced highly active antiretroviral therapy (HAART) with a regimen consisting of abacavir, tenofovir, lamivudine, and didanosine. At this time CD4 lymphocyte count was 100 cells $\times 10^6/l$ and HIV RNA viral load 30 000 copies/ml. His only other medication was co-trimoxazole as PcP prophylaxis. He had no regular sexual partner and had intercourse with one casual male partner in the past 3 months.

Neurological examination of his legs revealed normal tone in both legs, power 3/5 in right leg (predominantly in hip flexors, less marked in hip adductors, and abductors) and 5/5 in left leg, normal knee reflexes, absent ankle reflexes in both legs and bilateral down-going plantar responses. The remainder of the neurological examination was normal with

no sensory abnormalities. There was no tenderness over the thoraco-lumbar spine. General examination revealed no rashes, lymphadenopathy, or organomegaly. He was afebrile.

The following investigations were performed and found to be normal: urea, electrolytes, liver function tests, random blood glucose, creatine kinase, serum lactate and vitamin B12 level, full blood count, chest x ray, Magnetic resonance imaging (MRI) of brain and lumbosacral spine (including conus views), computed tomography (CT) of pelvis, cerebrospinal fluid (CSF) protein, glucose, cell count, Gram stain, cryptococcal antigen, polymerase chain reaction (PCR) for herpes simplex virus (HSV) 1 and 2, varicella zoster virus, and cytomegalovirus (PCRs all qualitative assays). CSF VDRL and TPPA were negative including VDRL after dilution. Repeat CD4 lymphocyte count was 9 cells $\times 10^6/l$ and HIV RNA viral load 250 000 copies/ml.

Four days after admission he developed fevers, headache, and a rash. The rash was a widespread maculopapular rash affecting the palms of his hands and soles of feet. Pyrexia of 39°C was the first documented fever since admission. No new medication had been administered since admission. A diagnosis of secondary syphilis was suspected with a differential diagnosis of a drug reaction or abacavir hypersensitivity although he had received over 1 year of abacavir therapy previously. A full septic examination was performed in addition to syphilis serology and a skin biopsy.

Forty eight hours later there had been no change in the rash. Serum syphilis EIA (Murex ICE Syphilis) and blood cultures were negative. The only positive finding was a transaminitis with an alanine aminotransferase (ALT) of 148 U/l (normal <30 U/l).

Because of the negative syphilis serology, the most likely diagnosis considered was painful HSV polyradiculopathy with a false negative HSV PCR⁸ and a drug related rash. A 10 day course of high dose intravenous aciclovir was commenced (10 mg/kg aciclovir 8 hourly).

After 4 days, there was no change in the clinical picture. He remained febrile. Skin biopsy result revealed a lymphocytic infiltrate, in keeping with a drug reaction. The only new medication was tenofovir. This agent is unlikely to cause a rash.⁹ However, the other medication he had been on for over a year (antiretrovirals and co-trimoxazole). In keeping with recent HIV genotypic resistance testing HAART regimen was changed to abacavir, didanosine, lamivudine, and Kaletra (lopinavir/ritonavir).

On day 10 of intravenous aciclovir he was afebrile with some improvement in the rash and moderate increase in power of the right leg (4/5). Liver function tests had

Abbreviations: ALT, alanine aminotransferase; CT, computed tomography; CSF, cerebrospinal fluid; HAART, highly active antiretroviral therapy; HSV, herpes simplex virus; MRI, magnetic resonance imaging; MSM, men who have sex with men; PcP, *Pneumocystis jiroveci* pneumonia; PCR, polymerase chain reaction; RPR, rapid plasma reagent

Key messages

- Neurosyphilis and neurological manifestations of syphilis may be commoner in HIV disease
- Early syphilis may cause a radiculopathy which can occur before the classic manifestations of syphilis
- Syphilis serology may be unreliable in the context of immunodeficiency

improved (ALT 65 U/l). Hepatitis A, B, and C serologies were negative. At this time the patient was discharged and syphilis serology rechecked.

Serum syphilis serology now showed a positive enzyme immunoassay with a rapid plasma reagent (RPR) of 1:8. The most unifying diagnosis was secondary syphilis with a painful polyradiculopathy. He was readmitted and treated with 15 days of benzyl penicillin 1.8 g 4 hourly. After the first dose of treatment he developed fevers and rigors, consistent with the Jarisch-Herxheimer reaction. This was treated with intravenous hydrocortisone 100 mg for 3 days.

On completing this course the leg weakness and rash had resolved, CD4 lymphocyte count was 60 cells $\times 10^6/l$ and HIV RNA viral load 880 copies/ml.

The initial serum syphilis EIA was retested and again found to be negative. The reference laboratory (Westmead Hospital, NSW) reported this sample as VDRL negative and TPPA positive.

DISCUSSION

This is the first reported case of early syphilis presenting as a polyradiculopathy before the development of the other clinical signs of secondary syphilis.

The diagnosis is strongly supported by the positive serology, the Jarisch-Herxheimer reaction, and the resolution of symptoms and signs with penicillin treatment.

Acute lumbosacral polyradiculopathy in HIV disease has many aetiologies, with commoner causes such as CMV radiculopathy excluded by CSF analyses.¹⁰ The absence of CSF abnormalities including pleocytosis is unusual but may be related to the severe immunodeficient state or syphilis involving the nerve roots remote from the CSF.

This case illustrates three important points: (i) early syphilis may cause a radiculopathy, (ii) this may occur before the more classic manifestations, and (iii) syphilis

serology tests can be unreliable in the context of immunodeficiency.¹¹ Given the rising prevalence of syphilis, clinicians should maintain clinical vigilance to these issues.

CONTRIBUTORS

AW wrote manuscript and performed literature review; DM assisted in writing of manuscript and assisted in interpretation of serology results; BB substantial input in writing of manuscript.

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Accepted for publication 29 July 2004

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