CD4 lymphocyte count which was 277/mm³ at the time of diagnosis. He was evaluated by two ophthalmologists, both of whom confirmed CMV retinitis with a retinal detachment. In addition, he had a vitreal biopsy which was positive by PCR for CMV and negative for herpes simplex, herpes zoster and Toxoplasma gondii. His retinitis failed to respond to intravenous ganciclovir, but subsequently responded to induction with 3 weeks of intravenous foscarnet therapy and remains quiescent on IV foscarnet maintenance therapy. Following diagnosis of CMV infection his CD4 count fell to 90/mm³ within 6 months, although it improved with combination antiretroviral therapy (zidovudine and zalcitabine). Interestingly, in two of the other non-splenectomised patients CD4 counts fell precipitously soon after diagnosis (from 255/mm³ to 15/mm³) over 8 months in one patient and 235/mm³ to 32/mm³ over 7 months in the other) reinforcing the importance of aggressive antiretroviral therapy once an opportunistic infection associated with immunocompromise develops.

A non HIV related case with a normal CD4 count has highlighted the importance of CD4 cell function in the prevention of CMV retinal infection. 5 HIV infection causes a great heterogeneity of immunological dysfunction. The CD4 count acts as a surrogate marker for the level of immune dysfunction but may hide functional as well as other subtle abnormalities of the immune system. CD4 counts are useful for predicting patients at risk of CMV retinitis and, therefore, those who may benefit from screening. However, although rare, this case further demonstrates that CMV retinitis can occur at CD4 counts greater than 200/mm³ and should serve to caution HIV physicians that the diagnosis of CMV retinitis is not excluded by a relatively high CD4 count.

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Acute cytomegalovirus prostatitis in AIDS

Genitourinary tract disorders are common in the acquired immunodeficiency syndrome (AIDS) including a wide spectrum of abnormalities due to an AIDS-related neoplasm or infection due to typical microorganisms or opportunistic pathogens. 1 Recent papers have emphasised the importance of prostatic disease as an emerging problem for patients with HIV infection. Indeed, involvement of prostatic gland is becoming more prevalent in patients with AIDS than in the general population. 2

Several clinical and pathologic surveys have described cases of prostatic disease caused by typical bacteria, including Escherichia coli and other Gram-negative bacteria, Staphylococcus aureus, Haemophilus parainfluenzae and Salmonella typhi, Mycobacterium tuberculosis and atypical mycobacteria, and fungal pathogens such as Histoplasma capsulatum and Cryptococcus neoformans. 3 HIV itself alone, in the absence of other infecting agents, also has been implicated as a cause of prostatic abnormalities. 2

In the current literature we rarely find reports of prostatitis caused by cytomegalovirus (CMV) in HIV-positive persons. 4 We here report a rare case of prostatitis due to CMV infection in a patient with AIDS in whom the diagnosis was established after death and who received antiviral chemotherapy with ganciclovir.

A 34 year old intravenous drug abuser with AIDS was hospitalised complaining of lower abdominal pain, urinary frequency without dysuria, and fever. Seven months before he had come to our attention because of CMV retinitis that had been treated with ganciclovir with a successful response, but the patient had not continued with a maintenance therapy. At the moment of the admission he appeared severely ill. The laboratory studies revealed a CD4 + lymphocyte cells count below 50/µL, a white blood cells count (WBC) of 3400/mm³ with 60% neutrophils, and 18% lymphocytes, an erythrocyte sedimentation (ESR) rate of 78 mm/hour, and a lactate dehydrogenase (LDH) value of 650 U/L. Urinalysis revealed one to five WBC per high power field without casts and trace amounts of protein. Repeated urine culture grew CMV. Microscopic and cultural examinations of blood, sputum and stool specimens were unremarkable. An ultrasound evaluation of the lower abdomen was normal. The examination of the prostate was normal. An active CMV retinitis in the right ocular fundus was detected. Reinduction therapy with ganciclovir was started in association with cotrimoxazole. The patient’s clinical condition continued to deteriorate, and he died after two weeks of hospitalisation. Post mortem examination reported a disseminated CMV infection, with prominent localisation in the prostatic gland, the lungs and the adrenal glands. Macroscopic evaluation revealed a normal sized prostate, while microscopically there was evidence of a large inflamed prostatic epithelium with multiple areas of tissue necrosis containing CMV intranuclear and intracytoplasmic inclusions.

Although infection with CMV is commonly benign in the immunocompetent person, the virus remains a major cause of morbidity and
mortality among immunocompromised patients with HIV infection or AIDS.

Chorioretinitis is the most common clinical manifestation of infection with CMV in persons with AIDS, followed by gastrointestinal disease. Less common manifestations of CMV include interstitial pneumonitis, subacute encephalitis, polyradiculopathy, and involvement of the adrenal glands, liver and biliary tract.

The first reported case of CMV infection of the prostate involved a 37 year old homosexual man with AIDS who died of disseminated CMV infection. pathological examination showed evidence of multiple organ CMV involvement, and prominent invasive CMV infection of the prostate. Our case represents the second reported case of prostatic infection by CMV in a patient with AIDS and, to our knowledge this is the first report of CMV prostatic disease in an intravenous drug abuser. In the report of Miles et al CMV was found at autopsy in the prostate from an AIDS patient who had no clinical signs and symptoms referable to this autopic finding.1

In other surveys which have documented the pathology profiles of AIDS, male genitourinary CMV infection was common, but none presented demonstrable CMV in the prostate.4

CMV infection of the prostatic gland can present either as local disease or as part of a systemic infection, although in the case reported by Benson et al 5 as in our patient CMV prostatitis appeared as a manifestation of disseminated infection.

However, severe CMV disease of the prostate may occur without the typical features of prostatitis. Thus, the wide range of disease processes observed in patients with AIDS, combined with frequent difficulty of establishing a histologic diagnosis may render the diagnosis obscure, and the selection of the best therapy can be difficult. A possible route for infection includes contiguous infection from surrounding organs, haematogenous or lymphatic spread, or from sexual transmission. For our patient, the mechanism of acquisition of CMV prostatic infection remains undefined.

Several reports support sexual activity as an important role of transmission of CMV infection. Major predisposing conditions for prostatic infection in HIV-positive individuals may include systemic and local immunodeficiency of prostatic tissue and fluid, the presence of concomitant sexually transmitted infections, homosexual practices, and the number of frequency and different sexual partners.

Thus, in addition to subjects engaged in homosexual contacts, subjects with a history of sexually transmitted diseases, and heterosexuals with multiple partners may be at increased risk of becoming CMV infected. CMV is known as an infrequent cause of genitourinary tract involvement, and diagnosis of prostatitis may be difficult to establish, since patients may be asymptomatic or have, as in our case, nonspecific urinary symptoms.

Although prostatitis due to CMV remains an uncommon clinical entity, the diagnosis of CMV prostatitis requires a high degree of clinical suspicion, and should be considered in HIV-positive individuals at risk for invasive CMV infection who present with otherwise unexplained signs and symptoms of genitourinary involvement.

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Systemic lupus erythematosus features in an AIDS patient: diagnostic problems in an African rural hospital

Systemic lupus erythematosus (SLE), as well as other rheumatic diseases, and HIV infection share clinical and laboratory features that can confound the diagnosis especially in hospitals with few laboratory facilities.1 We report a case of a 32 year-old woman from Northern Kenya with a clinical picture of SLE and positive agglutination rapid assay for HIV-1. The patient presented with a four months’ history of fever, malar rash, loss of body weight, headache and arthralgias together with epigastric pain, urinary incontinence and cough.

Physical examination showed poor nutritional status, erythematous maculo-papular butterfly rash, alopecia, tenderness at knees on active and passive movements and mental confusion. The laboratory findings revealed a leucocyte count 2200/mm³ with 26% of lymphocytes (572/mm³), haemoglobin 10-0 g/dl; VDLR and RF were negative. ANA and anti-DNA antibodies testing were not available in our hospital. Urine was positive (1+) for proteins with few pus cells. Chest radiograph and ECG were normal. Following worsening of mental status a lumbar puncture was performed showing proteins 1+, 6 leukocytes/mm³ and glucose 49 mg/dl. Finally the HIV-1 antibodies were tested and found repeatedly positive (twice with the same agglutination rapid assay, Serodia-HIV, Fujirebio Inc).

Although the prevalence of SLE in Kenya is low,2 hoping that the HIV-antibody test result could be a false positive due to SLE, a chloro-

References: