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Vasculitis and oral and genital ulcers: Behçet´s syndrome or HIV infection?

Sir,

Rheumatic diseases in the course of human immunodeficiency virus (HIV) infection are not common. Septic arthritis and psoriatic arthritis are the most frequent rheumatic syndromes described (1, 2). Vasculitis is being increasingly reported in the setting of HIV infection. Behçet's syndrome is a disease with protean vasculitic manifestations that has been occasionally described in association with HIV infection (3-6). We present a case of Behçet's syndrome and HIV infection.

A 40-year-old Caucasian woman was admitted to hospital with a 15-day history of swelling and pain of the right ankle, right knee and right wrist. She complained of recurrent oral and genital ulceration. Her past medical history revealed that she used to be an intravenous drug abuser (heroin). She abandonned the habit 5 years ago.

On physical examination there were erythematous papules, vesicles and ulcers (0.5 cm in diameter) on both arms and legs. Several ulcers were noted on the buccal mucosa and vagina. Arthritis was present on the right knee, ankle and wrist. The rest of the physical examination was normal.

Laboratory findings included: ESR 60 mm/hr, haemoglobin 99 g/L, and leucocyte count 7.02 x 10^9/L with a normal differential. Electrolytes, BUN, creatinine and a liver function test were within the normal range. Rheumatoid factor (nephelometry) and antinuclear antibodies (rat substrate) were negative. HLA was not determined. Pathergy was not present. A chest x-ray was normal. Knee aspiration yielded a yellow fluid with 1840/mm^3 WBC (6% polymorphonuclear, 15% lymphocytes, 79% monocytes). Crystals were not observed and a culture was negative. A skin biopsy was performed and microscopic examination showed a leukocytoclastic vasculitis. HIV serology (enzyme linked inmunosorbent assay and Western blot analysis) was positive and CD4 lymphocytes were 174 mm^3. Plasma HIV-1 RNA (Amplicor, Roche Molecular System, Inc., Madrid) was of 357.476 copies/ml. Cultures from the genital ulcers were negative. Biopsy of the oral ulcers showed an eroded epidermis and lymphocytic and monocytic inflammatory infiltrates in the dermis. There was no evidence of malignancy or infection. An ophthalmological examination was normal.

Treatment with prednisone (40 mg daily) was begun and her symptons improved quickly. Retroviral treatment with estavudine, amivudine and indinavir was added. When prednisone was tapered to 10 mg, the arthritis and oral and genital ulcers recurred.

The association of Behçet's syndrome and HIV infection is rare. We performed a literature search (Medline 1985-1997) and found 4 other cases (3-6), whose characteristics are summarized in Table I. All patients fulfilled the international criteria for Behçet's disease, except for the one reported in reference 5 (oral and genital ulcers) (7). The association of Behçet's syndrome and HIV infection is controversial. The two diseases share many clinical features, namely arthritis, oral and genital ulcers and vasculitis. Interestingly enough, in a Spanish study HIV serology (ELISA) was performed on a cohort of 23 patients with Behçet's disease and negative results were obtained in all cases (8). There are no epidemiological studies to suggest an association between the two diseases, but considering the cases reported in the literature, an association between HIV infection and Behçet's disease may exist (3-6).

Table I. Characteristics of patients with Behçet's disease and HIV infection.

	Age	Sex	Race	Transmission	Stage
Reference 4	33	male	white	heterosexual	B1
Reference 3	25	male	white	i.v. drug abuser	A2
Reference 5	69	male	white	heterosexual	A2
Reference 6	27	female	black	heterosexual	B3

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Highly positive dsDNA antibodies in minocycline-induced lupus

Sir,

We read with great interest the cases reported by Knights *et al*. dealing with the condition of minocycline-induced arthritis (1). We would like to report the first case of minocycline-induced lupus associated with high titers of dsDNA antibodies.

A 22-year-old female was admitted in August 1997 for a four-week history of jaundice and abdominal pain. Her past medical history included facial acne for which she had been taking minocycline 100 mg daily for 2 years. Two weeks before her hospitalization, she stopped minocycline on her own initiative because her acne was improving.

On admission she had no fever, arthralgia, myalgia, nor cutaneous involvement (except for a slight facial acne). Her physical examination was normal. Laboratory studies revealed acute hepatic cytolysis (ALAT 12N,

ASAT 18N, gamma-GT normal, and alkaline phosphatases 1.5 N); ESR was normal. Tissue-specific antibodies (smooth muscle, mitochondria and liver kidney microsomes) were negative. Alpha-1 antitrypsine was normal. Viral hepatitis screens (HAV, HBV, HCV, and HEV serologies, and HCV PCR) were negative. HIV serology was negative. Antinuclear antibodies were weakly positive (titer 1/80, speckled pattern), and dsDNA antibodies were negative. A hepatobiliary ultrasonography was normal. Her symptoms and abnormal liver test results spontaneously decreased within 2 weeks.

One month later the patient's acne worsened and she re-started minocycline 100 mg/day. After 4 weeks she developed inflammatory polyarthralgia affecting the small joints of the hands, the wrists, the shoulders and the ankles, with myalgia and fatigue but no fever. Despite the administration of naproxen, her symptoms became so severe that she was re-admitted 4 months later.

On physical examination, the patient had tenosynovitis of the left hand and tender metacarpophalangeal joints. Laboratory abnormalities included an ESR of 22 mm/hr and CRP 12.5 mg/l. Renal and liver tests were normal. Antinuclear antibodies were 1/800 (speckled pattern), dsDNA antibodies were 64 UI (Farr test) and myeloperoxidase antibodies were 97 U (N < 20). Antibodies to extractable nuclear antigen, cANCA, smooth muscle, mitochondria, and liver kidney microsomes were negative. Anticardiolipin IgG antibodies were weakly positive (19 UGPL, N < 15). Human leukocyte antibody typing was positive for A1, A2, B8, B60, Bw6, DRB1*15, DRB1*04, DRB5*0101, DRB4*01. Hand and wrist X-rays were normal. Minocycline was stopped and her symptoms dramatically improved over one week with no adjuvant therapy.

To the best of our knowledge this represents the first description of highly positive dsDNA antibodies in a typical case of minocycline-induced lupus. Although dsDNA positive antibodies may indicate idiopathic SLE rather than drug-induced lupus, their presence does not eliminate the diagnosis of minocycline-induced lupus.

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Leucocytoclastic vasculitis in a young body builder

Sir,

A 19-year-old male was admitted for severe inability to walk for the last 2 days. There were no systemic complaints. He was previously healthy and the family history was not contributory. Recent previous trauma or infection were ruled out. For the last 4 years he had been engaged in regular and intensive (2 - 3 hrs every day) body building exercises. He denied consuming anabolic steroids or other illicit drugs. During the week before his admission he had been involved in intense body-building activity including stressful exercises to build up the muscles of the thighs and calves.

Physical examination showed a muscular young man. The BP was 130/80, the pulse was regular (80 per minute), and the body temperature was normal. Purpuric lesions and erythema were present on the lower part of the thighs, calves and ankles, but not on the buttocks. All of these areas and the feet were swollen, warm and highly sensitive on palpation. There was no neurological deficit. The patient could not walk.

His ESR was 60 mm/1st hr (Westergren). A complete blood count, serum biochemistry and thyroid function tests, proteins, protein-electrophoresis and urine analysis were normal. Creatinine phosphokinase (CPK) was 713 U/L (30 - 280). A urine examination for myoglobin was negative. ANA, anti-ds DNA antibodies, RF, anti-RNP, anti-cardiolipin, C- and P-ANCA were all negative. The serum levels of C3, C4 and CRP were within normal limits. A chest X-ray was normal.

A skin biopsy from the right ankle area revealed acute leucocytoclastic vasculitis. As anamnesis, physical examination and laboratory investigations failed to provide the etiology of the vasculitis, the possibility of prolonged and intensive physical exercise as the pathogenetic mechanism was considered. During 3 days of complete rest the purpuric lesions, erythema, swelling and limb pain gradually disappeared, and the patient was able to walk again.

Our patient's elevated serum CPK levels were attributed to his intense regimen of physical training. A pre-discharge blood test showed the normalization of all values. The patient was advised to rest for an additional week and to substantially reduce his future physical activities. Nevertheless, a few days later later, a new (but milder) episode of calf and ankle pain and swelling occurred, following the resumption of exercise. They rapidly subsided after rest.

Leucocytoclastic vasculitis has been associated with a cohort of pathological conditions including collagen, infectious and malignant diseases, hypersensitivity to drugs, and other conditions (1). Sporadic cases of exercise-induced purpura (2-4), as well as one case of exercise-induced leucocytoclastic vasculitis in a patient with urticarial vasculitis (5), have been reported. The association of prolonged exercise with skin leucocytoclastic vasculitis was established by a clinical pathological study in subjects who developed purpuric lesions and vasculitis on the lower legs after engaging in long distance walking (6).

The pathogenetic mechanism of exercise-induced leucocytoclastic vasculitis is still unclear. An altered cutaneous microcirculation (7), transient alterations in immunological and/or biochemical parameters (8, 9), and activation of the complement cascade system by endurance exercise (10) are possible explanations. We propose that prolonged and strenuous physical exercise should be included among the various etiologies of leucocytoclastic vasculitis.

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