Ocular myositis as first presenting symptom of human immunodeficiency virus (HIV-1) infection and its response to high-dose cortisone treatment

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Abstract
A 30-year-old male presented with signs of ocular inflammation and motility disturbances in an early stage of HIV infection. The provisional diagnosis of an ocular myositis was confirmed by orbital echograms. A general check up revealed positive anti-smooth-muscle antibodies and antinuclear antibodies as well as a raised erythrocyte sedimentation rate. Oral steroid treatment in addition to steroid eyedrops achieved complete resolution of clinical and sonographic symptoms within 15 weeks. Autoimmune phenomena are well known presentations of HIV infection. In this case oral cortisone proved to be an effective therapy even in the setting of an HIV infection.

Various forms of myositis are known in the course of an HIV infection — for example, as a possibly rheumatic manifestation,1 as a complication of virustatic therapy with zidovudine,2 or as pyomyositis due to a secondary infection with bacteria3 or protozoa. Moreover, some cases of polymyositis in the early stages of HIV infection have been described in which HIV-1 itself was found in CD4-positive inflammatory cells surrounding or invading damaged muscle fibres. Here we report on a patient with ocular myositis as the presenting symptom of an HIV-1 infection and its therapeutic response to high-dose cortisone therapy.

Case report
The patient, a 30-year-old male, had already complained for a few weeks of lassitude, headache, eye related pain, swelling of the eyelids and conjunctiva, reddening of the eyes, and double vision, until he consulted us in March 1989. Treatment with antibiotic-steroid eyedrops administered up to that time had not resulted in any improvement.

On examination the visual acuity was 20/20 in both eyes. Both upper and lower lids were swollen. Apart from a tarsal conjunctivitis a slight bulbar conjunctivitis, an episcleritis, and slight conjunctival oedema were noted. The deeper eye segments were normal. In primary position the eyes were parallel. Motility testing revealed restriction of elevation and abduction of the right eye and restriction of abduction of the left eye. In the corresponding gaze directions the patient had double vision. The provisional diagnosis of an ocular myositis was confirmed by an ultrasonogram of the orbit. The patient was admitted to hospital for a general check up by electrocardiogram, chest x-ray, cerebral computed tomogram, and a physical examination. All results were negative. Lymphadenopathy and oral candidiasis were absent. An abdominal sonogram showed no splenomegaly or enlarged abdominal lymph nodes.

Laboratory tests revealed a raised erythrocyte sedimentation rate (Westergren) of 27 mm after one hour. The differential blood count, platelets, and serum enzymes, in particular creatinine kinase, were within the normal range. Electrophoresis showed a slightly raised gammaglobulin fraction. The level of IgG was within the normal range. Tests for anti-smooth-muscle antibodies (ASMA) and antinuclear antibodies (ANA) gave positive results, whereas other autoantibodies could not be detected. A reduced CD4/CD8 ratio of 0·62, with an absolute CD4-positive cell count of 526·106/l resulted in testing for HIV infection. An enzyme linked immunosorbert assay (ELISA) test for HIV-1 antibodies showed positive results and was confirmed by Western blot. IgG antibodies in low titres were also demonstrated for Epstein-Barr virus, cytomegalovirus, herpes simplex virus, and toxoplasmosis. The Treponema pallidum haemaggulitin test was negative. Other infections in stool, sputum, and blood samples were excluded.

In addition to steroid eyedrops, oral steroid treatment was started with a daily dose of 1·5 mg/kg body weight fluocortolone for five days. The initial daily dose was tapered to 0·3 mg/kg/day over the next 20 days. The clinical symptoms resolved only slowly while changing their pattern (the left superior rectus muscle became temporarily paralytic, and the episcleritis affected different locations of the bulbus before it resolved), so that the patient was maintained on this daily dose for another six weeks. After a further five weeks on a daily dose of 0·12 mg/kg body weight fluocortolone the systemic treatment was stopped, when complete resolution of the clinical symptoms was achieved and the sonographic controls of the eye muscles were normal (Fig 1).

Since then, after a follow-up of 15 months, the patient has remained free of symptoms without any therapy. He had received a cumulative dose of 2780 mg of fluocortolone over a period of 15 weeks without suffering any infectious complications by the potential immunosuppressive effect of cortisone and the underlying HIV-1 infection.

Discussion
The pathogenesis of HIV associated myositis is not entirely clear. Apart from an invasion of the musculature by HIV infected inflammatory cells' our case of ocular myositis perhaps sug-
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