essarily high. However, as demonstrated here, intraocular malignant neoplasms must also be considered and biopsy of the aqueous and/or vitreous is prudent.

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Highly Active Antiretroviral Therapy-Associated Ptosis in Patients With Human Immunodeficiency Virus

We describe 2 patients with human immunodeficiency virus (HIV)/AIDS receiving highly active antiretroviral therapy with bilateral ptosis. In both cases, the ptosis developed during the course of the disease and its treatment. Each patient underwent surgical ptosis repair. A biopsy of the advanced levator complex, including the levator muscle, aponeurosis, and orbicularis oculi muscle, was obtained intraoperatively. This was compared with a similar biopsy taken from an HIV-negative control subject.

Report of Cases. Case 1. A 57-year-old man with a medical history of HIV/AIDS was referred for treatment of vision-impairing upper eyelid ptosis. He was treated for cytomegalovirus retinitis in his left eye 15 years prior to his initial visit to us. His HIV medications included lamivudine (Epivir; GlaxoSmithKline, Philadelphia, Pennsylvania) and didanosine (Videx; Bristol-Myers Squibb Co, New York, New York), both of which are nucleoside analogues similar to zidovudine, as well as fosamprenavir calcium (Lexiva; GlaxoSmithKline), which is a protease inhibitor. The patient had a CD4 lymphocyte count of 180/µL.

External examination revealed lipoatrophic facies and bilateral ptosis. Eyelid fissures measured 5 mm OD and 4 mm OS. Levator function was 10 mm OU. The patient underwent a bilateral levator resection.

Case 2. A 53-year-old man had a 17-year history of well-controlled HIV/AIDS. He reported the development of droopy eyelids over the past several years. The patient underwent an uncomplicated levator advancement procedure 5 years prior. His condition remained undercorrected. His HIV medications included enteric-coated didanosine (Videx EC; Bristol-Myers Squibb Co), tenofovir disoproxil fumarate (Viread; Gilead Sciences, Inc, Foster City, California), and abacavir sulfate (Ziagen; GlaxoSmithKline), all of which are nucleoside analogues similar to zidovudine, as well as nevirapine (Viramune; Boehringer Ingelheim Pharmaceuticals, Inc, Ridgefield, Connecticut), which is a nonnucleoside reverse transcriptase inhibitor. The patient demonstrated a CD4 lymphocyte count of 343/µL.

External examination demonstrated lipoatrophic facies and bilateral ptosis. Eyelid fissures measured 5 mm OU. Levator function measured 10 mm OU. The patient underwent a bilateral levator resection.

Comment. Myopathy associated with HIV was first described in the 1980s.1,2 This myopathy may manifest as an inflammatory myopathy with numerous inflammatory cells in sarcolemmal complexes (ie, polymyositis) or less commonly as a type II muscle fiber atrophy or nemaline myopathy.2 Zidovudine and drugs of its class are indicated for the treatment of patients with AIDS. These drugs are dideoxynucleoside analogues that inhibit y-DNA polymerase, an enzyme found solely in the mitochondria. These drugs interfere with the replication of mitochondrial DNA and have been
implicated as the cause of a mitochondrial myopathy associated with atrophic ragged-red fibers and marked myofibrillar alterations.\(^4\)\(^5\)

The histologic samples in this small case series depict findings similar to those described in the first reported cases of zidovudine-associated myopathy.\(^4\)\(^5\) Dalakas and colleagues\(^3\)\(^4\) studied muscle specimens of 15 patients treated with zidovudine. These specimens displayed the morphologic changes in muscle architecture consistent with mitochondrial pathology, ie, abundant ragged-red fibers. None of these changes were seen in the untreated control subjects. Moreover, the patients who had HIV-associated myopathy did not have these findings. Similarly, our patients had a higher density of ragged-red fibers (Figure 1) in comparison with that in the control subject (Figure 2). Increased staining of reduced nicotinamide adenine dinucleotide and succinate dehydrogenase and in numerous muscle fibers as well as electron microscopy results showing mitochondrial hyperplasia and hypertrophy in our cases also support a mitochondrial abnormality.

We report 2 cases of ptosis likely secondary to HIV and highly active antiretroviral therapy. This association is based on histopathologic findings consistent with the mitochondrial myopathy associated with these drugs. Given the utility of highly active antiretroviral therapy, it is not feasible for these patients to discontinue this treatment. However, identification of the ptosis is important to the understanding of the disease and the consequences of its treatment.

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