rupture of the spleen has been reported, but it was not possible to determine the stage of HIV infection in that case [1]. There are many hypotheses regarding the mechanism of the splenic rupture, including lymphoid infiltration of trabecular veins, as has been described in cases of Epstein-Barr virus infection, as well as endothelial cell dysfunction and acute vasculitis [2]. In our patient’s case, lymphoid infiltration of trabecular veins was the main feature of his condition, and no acute vasculitis was noted. His thrombocytopenia likely played an important but insufficient role in the development of the hematoma.

We conclude that in all cases of spontaneous rupture of the spleen in which no other etiology can be determined, it seems appropriate to screen for acute HIV infection by means of ELISA and measurement of p24 antigenemia.

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References

Recurrent Iritis After Intravenous Administration of Cidofovir

Although iritis is a relatively common side effect with intravitreal injections of cidofovir [1, 2], this adverse drug reaction rarely occurs with administration of the iv form, and its occurrence has not yet been clearly linked to administration of this drug [3]. We describe a case of recurrent unilateral iritis following iv administration of cidofovir (Vistide, Gilead Sciences, Foster City, CA) to a patient with AIDS and cytomegalovirus (CMV) retinitis.

A 41-year-old HIV-infected homosexual male (CD4 cell count, 15/mm³) with a history of cryptococcal meningitis in 1993 and unilateral CMV retinitis (right eye) presented to the Ochsner Clinic in New Orleans on 26 November 1996 with a 4-day history of right red eye and photophobia. He denied ocular pain, visual blurring, or any constitutional symptoms. His temperature was 37.0°C, his blood pressure was 110/70 mm Hg, and his heart rate was 85. His right conjunctiva was hyperemic and inflamed, but the rest of the physical examination was unremarkable. Except for a WBC count of 3.0 × 10³, his laboratory values were within normal limits.

CMV retinitis of the right eye had been diagnosed in January 1995. At that time, therapy with iv ganciclovir was started; however, the patient developed a skin rash and his therapy was switched to foscamet. Although his CMV retinitis remained stable for 22 months, therapy was changed to iv cidofovir because of its convenient administration (every other week rather than daily).

On 23 October 1996, he received the first dose of cidofovir (5 mg/kg or 340 mg) along with iv saline and 4 g of oral probenecid. The patient did not report any complications or side effects. On 6 November 1996, he received the second dose of cidofovir and probenecid. Two days later he called to report the onset of a right eye inflammation and photophobia. He refused to be examined, but 6 days later he called again to report that the symptoms had spontaneously disappeared. On 20 November 1996 he received the third dose of cidofovir and probenecid, and 2 days later he noticed the recurrence of right eye inflammation and photophobia. This time he decided to come to the clinic.

The patient’s other medications were ritonavir, zalcitabine, lamivudine, fluconazole, and aerosolized pentamidine (given monthly). Ophthalmologic examination revealed a visual acuity of 20/20 normal vision in both eyes. The right eye had 1 + bulbar conjunctival injection and 1–2 + cell and flare in the anterior chamber. The left cornea was clear, but inferior keratic precipitates were seen on the right inferior corneal endothelium. His CMV retinitis was quiescent. The vitreous had no cells or posterior vitreous detachment. Numerous examinations performed before cidofovir therapy was begun showed no signs of anterior uveitis.

The patient received treatment with topical 1% prednisolone acetate eight times daily until the inflammation resolved. As of 18 May 1997, he had had three consecutive episodes of iritis after the administration of cidofovir without visual loss.

Cidofovir is an acyclic cytosine nucleoside phosphonate analog that is highly active against CMV. Its iv formulation was approved by the Food and Drug Administration in June 1996 for the treatment of CMV retinitis in patients with AIDS. Significant adverse reactions to cidofovir include elevation of the creatinine level, proteinuria, neutropenia, and metabolic acidosis. In clinical trials, a decrease in intraocular pressure (hypotony) was the most common ocular adverse reaction observed, and this reaction occurred in 12% of the patients. Other ocular adverse effects encountered and listed in the prescribing information for cidofovir as “regardless of causal relationship” include amblyopia, conjunctivitis, iritis, retinal detachment, uveitis, and abnormal vision.

In contrast, the intravitreal form of cidofovir was shown to cause a variety of ocular complications, including iritis, uveitis, vitreitis, and hypotony [1, 2]. In one study with low-dose (20-mg) intravitreal cidofovir for CMV retinitis, iritis occurred in 14% of the patients who received prophylaxis with oral probenecid and in 41% of the patients who did not receive probenecid [1]. The presumed role of probenecid is to lower the uptake of cidofovir into the ciliary body. This appears to have an effect on decreasing the incidence of ocular hypotony and iritis [1]. Probenecid helps prevent uptake of cidofovir into epithelial tissues by competitive inhibition. Damage to this ciliary body epithelium results in hypotony as well as in inflammation secondary to tissue damage or irritation.

According to a report received by the manufacturer (Gilead Sciences; J. Buchanan, personal communication), 10 cases of iritis had been associated with the administration of iv cidofovir as of
December 1996. None of those cases has been published in the literature; the clinical characteristics of the patients, the possible interaction of cidofovir with other medications, and the patient outcomes are unknown. Clinicians who treat CMV retinitis with cidofovir should be aware of this rare complication.

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