tence of HB_sAg and HB_eAg, without evidence of infection with hepatitis A virus, hepatitis D (delta) virus, or hepatitis C virus. His symptoms abated, and the ALT level returned within 1 month (mid-April 1995) to that observed before he was treated with stavudine (80 U/L).

In early May 1995, the patient began receiving combination antiretroviral therapy with zidovudine (100 mg t.i.d.) and lamivudine (300 mg b.i.d.), which was well tolerated. His CD4 cell count increased to 538/mm^3 (23%) by week 6, while his ALT level decreased; after 15 weeks of therapy, all liver function parameters had returned to the normal range. At week 20 of therapy, serological test results indicated that the patient was HB_sAg negative and that he had developed antibodies to HB_sAg; hepatitis B virus DNA was not detected (as of this writing, these findings have not changed) (figure 1).

Hepatitis B virus is transmitted readily via parenteral and sexual routes, and as such, it is commonly found in individuals who are coinfected with HIV-1. For the immunocompromised host, such as the patient with HIV-1 infection, a frequent outcome of hepatitis B infection is the development of a chronic carrier state that does not spontaneously resolve over time. Chronic carriage of hepatitis B virus occurs after acute infection in ~20% of patients who are HIV infected prior to exposure to hepatitis B virus [1–2].

The complete clearance (either spontaneous or drug induced) of hepatitis B in a patient with chronic infection who is coinfected with HIV-1 is unusual [3]. Lamivudine ([−] enantiomer of 2′-deoxy-3′thiacytidine [3TC]) is a nucleoside analogue that has shown antiretroviral activity in vitro as well as in patients with HIV-1 infection; this drug has also shown activity against hepatitis B virus in vitro [4] and in vivo [5]. Whether the clearance of hepatitis B virus in our patient was due to the activity of lamivudine alone or was a result of enhancement of his immune system following combination antiretroviral therapy with zidovudine and lamivudine (in addition to lamivudine's activity against hepatitis B virus) is unknown. The experience with this patient argues for further investigation and clinical trials of agents such as lamivudine in patients who are coinfected with hepatitis B virus and HIV.

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Optic Disk Edema as the Presenting Sign of Lyme Disease

Lyme disease is a tick-borne infection caused by *Borrelia burgdorferi* [1]. Manifestations include a characteristic rash (erythema chronicum migrans), nonspecific flulike symptoms, oligoarticular arthritis, a chronic skin lesion (acrodermatitis chronica atrophicans), and neurological abnormalities [2–4]. A variety of ocular manifestations are associated with Lyme disease, including conjunctivitis, keratitis, optic neuritis, and edema [3, 4]. We describe a patient in whom optic disk edema was the presenting and primary feature of Lyme disease.

An 82-year-old man from New Jersey who had a 4-month history of malaise and myalgia and who reported weight loss was found to have bilateral optic disk edema during a routine eye examination. He did not recall any rash or exposure to ticks or animals. The patient's medical history was significant for well-controlled hypertension. No history of substance abuse was noted. Physical examination revealed a well-nourished man with stable vital signs. The only remarkable findings were ophthalmologic.

The patient's pupils were symmetrical and reactive to light and to accommodation. Extraocular movements, intraocular pressure, color vision, visual fields (as determined by the Goldmann perimeter), acuity, and visual evoked responses were normal. Bilateral optic disk edema (360°) and small splinter hemorrhages were noted on fundoscopic examination (figure 1A, B). No abnormalities were seen on an electrocardiogram or a chest roentgenogram, and the results of blood chemistry studies, a hemogram, thyroid function studies, and urinalysis were normal. Findings on a CT scan of the head were unremarkable. The opening pressure on lumbar puncture was 10 cm H_2O; examination of the CSF revealed 4 WBCs/mm^3 and 2 RBCs/mm^3.

A Venereal Disease Research Laboratory (VDRL) test of the CSF was nonreactive; cultures for bacteria, mycobacteria, and fungi were negative. ELISA of the CSF was positive for both IgG and IgM responses to *B. burgdorferi,* and an immunoblot assay of the serum was positive for IgG and IgM antibodies (13 and 7 bands, respectively), indicating the presence of Lyme borreliosis [5].

The patient was treated for 21 days with intravenous ceftriaxone (2 g/d). At follow-up several months later, his optic disk edema (figure 1C, D) and myalgia were completely resolved.

Optic disk edema is manifested by blurring of the disk margins, splinter hemorrhages, venous congestion of the retinal vessels, and retinal infarcts that are visible on funduscopic examination. The term *papilledema* pertains to optic disk swelling that results from increased intracranial pressure [6]. Conditions that may lead to optic disk swelling or edema include vascular and congenital abnormalities, optic neuritis, optic disk tumors, infiltrative processes, and thyroid-related

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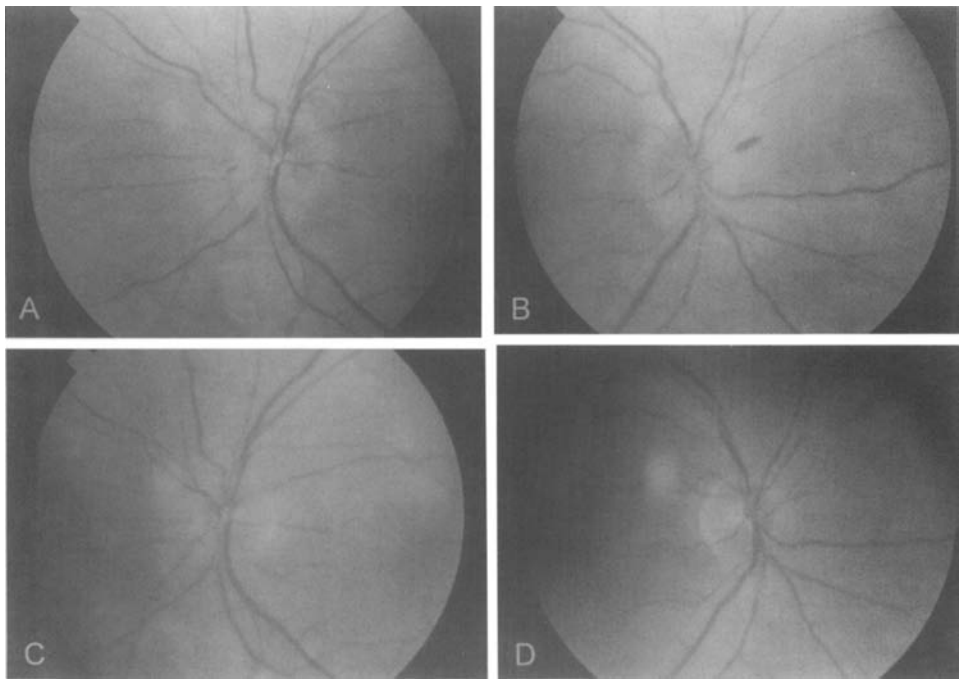

Figure 1. *A, B*: Bilateral optic disk edema and small splinter hemorrhages are evident on fundoscopic examination of a patient with Lyme disease. *C, D*: Bilateral resolution of the optic disk edema.

orbitopathy [6]. Normal visual evoked responses, thyroid function, and CSF pressure, and lack of evidence of vascular abnormalities allowed exclusion of alternative etiologies of our patient's optic disk swelling [6]. The results obtained on serological testing of his serum and CSF and the resolution of his ophthalmologic abnormalities with antibacterial therapy confirmed that Lyme borreliosis was the etiology of our patient's optic disk edema.

To our knowledge, optic disk edema has not been reported previously as the sole objective feature of Lyme disease [3, 4]. The varied ocular pathologies associated with Lyme disease usually occur within the context of a typical history of exposure and/or clinical syndrome [3, 4]. The pathogenesis of the neurophthalmologic findings is unknown [3]; however, it is possible that ocular structures are directly invaded by *B. burgdorferi* [7].

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Fatal Toxic Epidermolysis Induced by Zidovudine

Drug-related adverse cutaneous reactions occur more often in HIV-infected persons than in the general population. Toxic epidermolysis (Lyell's syndrome) is a serious adverse reaction; the mortality rate associated with this reaction in HIV-infected individuals is 21%–55% [1]. It has often been correlated with sulfonamide therapy [2], and its incidence increases as immune function deteriorates [3]. Despite the widespread use of zidovudine, to our knowledge, no case of toxic epidermolysis induced by zidovudine therapy has ever been reported. We describe herein a 42-year-old HIV-positive man in whom lethal toxic epidermolysis developed after retreatment with zidovudine.

The patient first presented to our service because of fever, gingival bleeding, pharyngodynia, and dysphagia. His medical history was notable only for oral thrush, herpes zoster, and chronic hepatitis B. His CD4 cell count was 27/μL. His medications included zidovudine (500 mg daily since 1 month before hospital admis-

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