aminase, which are findings consistent with worsening hepatitis. We believe that these abnormal findings may be caused directly by protease inhibitor therapy, or, alternatively, by the acceleration of the effects of hepatitis C on iron metabolism.

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Clinical Infectious Diseases 2001; 33:413–4

Alendronate Treatment for Osteoporosis in Patients Infected with Human Immunodeficiency Virus

Sir—Drug-induced osteoporosis is not rare and is mainly caused by treatment with glucocorticoids. Highly active antiretroviral therapy (HAART) has also been shown to accelerate bone mineral loss in HIV-infected patients and has been proven to be a potent inducer of osteoporosis in this (usually young) population [1]. We have recently described pathologic fractures in patients who have osteopenia and osteoporosis induced by antiretroviral therapy [2]. Here we report the result of alendronate therapy in a 51-year-old man with AIDS and severe osteoporosis.

In February 2000, this patient suffered a trivial trauma while walking. A radiogram showed a fracture of the body of lumbar vertebra L1; soon thereafter, because of an anterior vertebral collapse, the patient developed severe disability and pain. At the time of the fracture, the patient's CD4 count was 522 cells/μL and virus load (as measured by branched-DNA level) was <50 copies/mL. He had been receiving a stable course of antiretroviral therapy with stavudine, lamivudine, and indinavir for 36 months. The patient was given 10 mg of alendronate daily with calcium (500 mg daily in the form of calcium carbonate) and vitamin D supplements (450 IU daily), for 6 months.

Before and at the end of treatment, dual-energy x-ray absorptiometry scanning (DEXA) was performed to determine the bone mineral density (BMD) of the whole body and of the lumbar spine (vertebrae L1 through L5). Before treatment, DEXA documented that the median BMD of the lumbar spine was 0.691 g/cm^2 (t score, −3.85; z score, −3.53). At the end of treatment, DEXA showed that the median BMD of the lumbar spine was 0.832 g/cm^2 (t score, −2.35; z score, −1.34). A 20.4% increase in lumbar spine BMD was obtained by the end of treatment, and the patient's pain was almost completely relieved. A radiograph did not reveal any new fractures.

After 6 months of treatment, the patient developed lactic acidosis and interrupted both antiretroviral therapy and alendronate therapy. Other authors have recently described the association of lactic acidemia with osteoporosis in HIV-infected men, which suggests that this is a real phenomenon and not a spurious association [3].

The effect of alendronate on BMD in HIV-infected patients with osteoporosis has not been evaluated yet. On the other hand, no approved treatment exists for osteoporosis that develops secondary to antiretroviral therapy. This case report encourages the development of clinical trials to study the effect of alendronate in HIV-infected patients with metabolic bone disease. Information regarding the safety of this drug in combination with HAART is urgently needed.

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Clinical Infectious Diseases 2001;33:414–5

Pseudomonas Pneumonia in Smokers

Sir—Hatchette et al. [1] described a previously healthy cigarette smoker with *Pseudomonas aeruginosa* community-acquired pneumonia who died 36 h after admission to the hospital. Their search of the medical literature published since 1960 revealed 10 other cases of *Pseudomonas* pneumonia in otherwise healthy subjects, 5 of whom were smokers. The authors concluded that *Pseudomonas* species should be considered an etiologic agent in anyone with a history of smoking who has rapidly progressive pneumonia.

Clinical investigators should be wary of making such recommendations, and editors should be wary of printing them. First, leaving aside the platitude that physicians should always consider every diagnosis, the recommendation is not supported by the data. The number of smokers and nonsmokers with *P. aeruginosa* community-acquired pneumonia who were reported in the literature was the same, and mention of 6 cigarette smokers with rapidly progressive pneumonia in 40 years of medical literature (0.15 patients per year) might as well lead physicians *not* to consider the diagnosis. Second, the recommendation encourages abuse of antibiotics (e.g., inclusion of an antibiotic to "cover" *Pseudomonas* species in cases of community-acquired pneumonia). Third, such recommendations have a way of serving as grist for the mill of malpractice attorneys when rare cases do occur subsequently.

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Clinical Infectious Diseases 2001;33:415

Bacteremia and Meningitis Caused by a Macrolide-Sensitive Strain of *Streptococcus pneumoniae* during Treatment with Azithromycin

Sir—Kelley et al. [1] describe 4 patients being treated with azithromycin or clarithromycin who presented with breakthrough bacteremia caused by pneumococci exhibiting low-level resistance to macrolide antibiotics. We have recently treated a patient who presented with breakthrough bacteremia and meningitis caused by a macrolide-sensitive strain of *Streptococcus pneumoniae* while she was receiving treatment with azithromycin.

A previously healthy woman aged 65 years consulted her general physician after 2 days of cough, fever, and malaise. During the previous year, she had experienced tenderness of the left ear region in association with episodes of the common cold. Her condition was diagnosed as tracheobronchitis, and treatment was initiated with azithromycin at a dosage of 500 mg once daily. During the following 36 h, the patient experienced a headache of increasing intensity and nausea, and she was admitted to the hospital with a diagnosis of meningitis.

At the time of admission, the patient was awake, febrile, and had nuchal rigidity, but there were no clinical signs of septicemia. The leukocyte level was 20.1 $\times 10^9$ cells/L (neutrophil count, 17.2 $\times$ 10^9 cells/L), and the C-reactive protein level was 364 mg/L (reference level, <10 mg/L). Analysis of CSF revealed a leukocyte count of 8.8×10^9 cells/L (97% neutrophils), a glucose concentration of 2.6 m*M* (serum glucose concentration, 6.7 m*M*), and an albumin concentration of 1.20 g/L. Microscopic analysis of a CSF sample revealed neutrophil pleocytosis and gram-positive diplococci. Findings of lung and heart stethoscopy were normal, and a thoracic radiograph also appeared normal; clinical examination by a specialist in otology the next day revealed no signs of mastoiditis or otitis media.

Immediately after the lumbar puncture was performed, treatment was initiated with iv penicillin G at a dosage of 3×10^6 IU 6 times per day combined with iv ceftriaxone at a dosage of 6 g once daily. Following the acute microscopic examination of a CSF sample, the treatment was changed to iv penicillin G at a dosage of 3×10^6 IU 6 times per day. Eighteen hours later, *S. pneumoniae* had grown on all culture media. Corresponding blood specimens that were obtained after the lumbar puncture but before initiation of iv therapy—and during treatment with azithromycin—yielded growth of *S. pneumoniae* in 2 of 2 culture flasks. The 2 isolates recovered from CSF fluid and blood had identical resistance patterns; both were fully susceptible to penicillin G (MIC, 0.016 mg/L), ceftriaxone (MIC, 0.012 mg/L), and erythromycin (MIC, 0.19 mg/L). Therefore, the pneumococci were able to grow both in the patient and in the laboratory despite being fully susceptible to azithromycin, the agent that the patient was receiving at the time when specimens were obtained for culture.

Aside from an asymmetric left-side hearing loss, the patient experienced complete clinical recovery and was discharged after completing 10 days of treatment with iv penicillin G. Azithromycin treatment of this patient failed to eliminate a macrolide-sensitive strain of *S. pneumoniae,* failed to prevent it from causing bacte-