MATTERS ARISING

Enterobacter cloacae vertebral infection in a heroin addict with HIV infection

Sir; We read with interest the recent case reported by Solans *et al* concerning an infectious discitis caused by *Enterobacter cloacae*.¹ We would like to report a similar case in an intravenous drug abuser with human immunodeficiency virus (HIV) infection.

A 28 year old heterosexual man was admitted to hospital in October 1992 with a two month history of severe low back pain, fever, night sweats, and weight loss. He had been using heroin intravenously for several years, and the last dose had been given two weeks before onset of symptoms. He was known to have been seropositive for HIV-I since September 1985, but he remained asymptomatic (CDC III). He had also had asymptomatic hepatitis C since 1990.

On admission his temperature was 38.8°C. The lumbar spine was markedly tender with bilateral paravertebral muscle spasm. His chest was clear, and there were no heart murmurs. Lymphadenopathy was also noted. Laboratory data showed haemoglobin 132 g/l, white blood cell count $9.2 \times 10^9/l$, neutrophils $5.8 \times 10^9/l$, lymphocytes $2.5 \times 10^9/l$ (CD4 count $0.86 \times 10^9/l$), platelet count $255 \times 10^9/l$, erythrocyte sedimentation rate (ESR) 50 mm/h, and normal serum protein electrophoresis results. Two urine and three blood cultures were sterile. A chest radiograph was normal. Radiographs and magnetic resonance imaging of the lumbar spine (figure) suggested an infectious process affecting the fourth lumbar interspace. Bone scan showed increased uptake at L4 and L5. Puncture biopsy yielded a purulent fluid from which *E cloacae* were isolated. The organisms were resistant to ampicillin and sensitive to amikacin, pefloxacin, and trimethoprim with sulphamethoxazole. Treatment was started with intramuscular amikacin (1 g/day) and intravenous pefloxacin (800 mg/day). The latter was given orally after the third week of treatment. At the sixth week the patient became asymptomatic, and the ESR was normal (5 mm/h). He received an additional eight week course of oral pefloxacin and cefixime (800 mg/day).

To our knowledge only two cases of vertebral osteomyelitis due to *E cloacae* have been reported.^{1,2} Elderly patients were affected in both cases. The present case concerns a young male drug addict with HIV infection. Whether the HIV seropositive status has had a role is unclear.³ Septic bone and joint lesions are uncommon in such patients and pyogenic infection of the spine has been rarely reported.⁴⁻⁶ In previous reports enterobacter bacteraemia originated from the colon¹ or the urinary tract.² In our case there was no overt site of infection. Possibly, the disease resulted from intravenous self inoculation as haematogenous septic arthritis and osteomyelitis are common complications in drug abusers, with 53% occurring in vertebral bodies.⁷ Most infections are due to aerobic Gram negative

organisms with a predominance of pseudomonas species.^{7,8} Our case report indicates that heroin addicts may also be infected by *Enterobacter cloacae* spondylodiscitis.

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Magnetic resonance imaging of the lumbar spine showing erosions between L4 and L5 end plates and disc space narrowing.

LETTERS TO THE EDITOR

Reinfection in paediatric Lyme borreliosis

Sir: The nature of the humoral immune response to *Borrelia burgdorferi* in patients with Lyme disease is not completely understood. A variety of antibody patterns have been observed in the serum of infected patients as the immune response 'matures'.¹ A protective effect of those antibodies in humans has not yet been shown.² Animal studies have shown that 'immunity' can be conferred to hamsters, both passively, by antiserum to *B burgdorferi*,² and actively, through inactivated whole borrelia injection.³ Immunisation with recombinant outer surface protein A (OspA) (strain N40),⁴ and to a lesser degree, OspB⁵ prevented disease in mice subsequently infected with *B burgdorferi*. The degree of immunity induced by the natural infection is not known, however, and few studies on the protective effect of prior Lyme infection on human 'immunity' to the disease have been published.

Recently, we observed erythema migrans following tick bites in two children after appropriate treatment for previously well established Lyme arthritis. Both patients reside in the Delaware Valley region of the eastern United States, which is endemic for *Ixodes dammini* and *B burgdorferi*. Their prior diagnosis was supported by typical clinical findings and positive serological results (table).^{6,7}

The first patient was a 12 year old girl with an eight month history of fatigue followed by unilateral knee arthritis. Antibodies to borrelia measured by enzyme linked immunosorbent assay (ELISA) were present at a 1/640 dilution; 10 IgG bands and one IgM band were noted by western blot technique (ATCC strain B31).⁷ The child received a four week course of oral tetracycline (250 mg four times a day). Total compliance was reported and her symptoms resolved. Eleven weeks later her ELISA titre was 1/160 and her western blot reactivity had decreased to two IgG bands and no IgM reactivity. Twenty three weeks later we noted a typical erythema migrans rash in her groin 24 hours after a tick bite. Her serology 11 days after the onset of the rash was ELISA titre 1/80, five IgG bands and one IgM band in the western blot. The patient received a similar course of oral tetracycline with resolution of the rash and no subsequent symptoms.

Molecular weights of bands observed on western blots of patients reinfected with *B burgdorferi*

	Patient 1		Patient 2	
	IgG bands (kilodaltons)	IgM (kilodaltons)	IgG (kilodaltons)	IgM (kilodaltons)
Primary infection	60, 42, 41, 40, 37, 25, 18, 17, 14, 10	41	62, 41, 37, 35	None
After treatment	41, 40	None	62, 41, 37, 35, 24, 21, 16	None
Reinfection	64, 41, 37, 25, 17	41	83, 74, 67, 64, 60, 48, 41, 28, 25, 19, 18, 17	41