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 $9{\cdot}2\times10^9$/l, neutrophils $5{\cdot}8\times10^9$/l, lymphocytes $2{\cdot}5\times10^9$/l (CD4 count $0{\cdot}86\times10^9$/l), platelet count 255×10^9/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 trimethroprim 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.[2,3] 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 bacteriaemia originated from the colon[1] 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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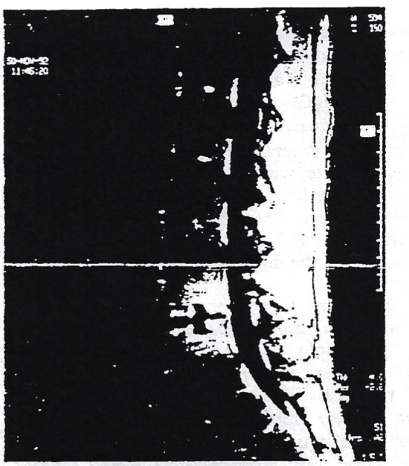

Magnetic resonance imaging of the lumbar spine showing erosions between L4 and L5 end plates and disc space narrowing.

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*,[3] and actively, through inactivated whole borrelia injection.[4] Immunisation with recombinant outer surface protein A (OspA) (strain N40),[5] and to a lesser degree, OspB[5] 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	92, 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

The second patient was a 9 year old girl who first presented with a history of recurrent left knee swelling (two episodes in six months). Her initial serological evaluation showed an ELISA titre of 1/640, with four IgG bands on the western blot in both serum and synovial fluid. She received a two week course of intravenous penicillin G consisting of 100 000 units per kg/day in four divided daily doses. Resolution of her symptoms was observed. At follow up, 13 weeks later, her ELISA was 1/1280 and the western blot showed seven IgG bands and no IgM reactivity. Four weeks later she developed a typical erythema migrans rash on her jaw after a deer tick bite, which resolved with oral penicillin, 250 mg four times a day for four weeks. Her ELISA titre three weeks after onset of this rash was 1/1280 and she had 12 reactive bands in the IgG western blot and one in the IgM.

These two cases suggest that reinfection with *B burgdorferi*, manifesting as erythema migrans, is possible even after a previous infection resulting in a well established immune response. The elapsed time and resolution of the initial symptoms after treatment make it unlikely that the second erythema chronicum migrans (following a documented tick bite) was a result of the primary infection with *B burgdorferi*. It is unclear if progressive disease would have occurred in the absence of treatment, but the presence of erythema migrans suggests that bacterial replication can occur, at least locally, despite prior naturally induced immune response to *B burgdorferi*.

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Health screening: the temporomandibular joint

Sir: Patients with temporomandibular joint (TMJ) disorders can be divided into two large groups: those with organic joint abnormalities, including ankylosis, neoplasia, trauma, and arthritis; and those with facial pain, noise in the temporomandibular joint, and restricted motion without organic joint disease.[1–8] Patients with TMJ disorders may present to ear, nose, and throat surgeons, neurologists, neurosurgeons, orthopaedic surgeons, rheumatologists, and primary care physicians, as well as those involved in dental care.

Management of TMJ disorders is covered comprehensively in the dental curriculum. Our impression is that the same is not true in the medical curriculum. We thus undertook, firstly, to determine the amount of instruction on management of TMJ disorders which is allocated in the medical curriculum and, secondly, to investigate the frequency with which the TMJ is assessed in the primary medical and dental health services.

A questionnaire (available from the authors) was devised and sent to the rheumatology departments of all 25 British medical schools. Its aims were to determine whether it is the policy of the local rheumatology department to instruct medical students routinely to: (*a*) ask the patients if they have had any pain/clicking or limitation of movements of their temporomandibular joint? (*b*) assess the temporomandibular joint during the physical examination?

Another questionnaire was sent to a random selection of 38 general medical practitioners in teaching practices in the London area inquiring: (*a*) During a general examination do you regularly ask your patients if they have had any pain/clicking or limitation of movements of their temporomandibular joint? (*b*) Do you assess the temporomandibular joint during the physical examination as a routine, only if symptomatic, or never?

A third questionnaire, asking essentially the same questions as those posed to the London general practitioners was sent to a random selection of 100 dental practitioners in London, Taunton, and Birmingham.

The medical and dental practitioners from London, Taunton, and Birmingham areas as listed in local telephone directories were chosen with the aid of statistical random number tables.[7]

Of the 25 medical schools contacted 23 (92%) replied. Ten of these (43%) confirmed that they taught their students to ask about TMJ symptoms. Thus 13 (57%) medical schools did not teach their students to ask about TMJ symptoms. Fourteen (61%) taught their students how to assess the TMJ; nine (39%) did not.

Thirty six of 38 (95%) general medical practitioners contacted replied to our London survey questionnaire. None of those answering the questionnaire either asked about the TMJ during a general medical examination or examined the joint as a routine. All did so, however, when it was directly symptomatic.

Of the 100 dental practices contacted 90 (90%) replied, comprising 157 dentists as the number in each practice was invariably more than one. Forty eight (31%) dentists asked about the TMJ during a routine inspection; 109 (69%) did not. Eighteen (11%) physically examined the TMJ as a routine; 137 (87%) assessed the joint only when symptomatic; two (1%) never assessed.

Thus the TMJ does not appear to be included in primary health screening programmes, either medical or dental, which in this era of preventive health management we find disappointing. Early recognition of, for example, rheumatoid arthritis in the TMJ might possibly prevent potentially disabling sequelae, such as limitation of condylar movement and marginal condylar irregularities,[2] anterior open bite,[3] micrognathia,[4] ankylosis,[5] and upper airway obstruction.[5]

Examination of the TMJ is not encouraged in rheumatological screening, but we suggest that a prospective study is required into the benefit of TMJ screening. This might enable early identification of TMJ disease, such as occurs in rheumatoid arthritis, resulting in a subsequent reduction in TMJ morbidity.

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