Reiter's syndrome and reactive arthritis in health care workers after vaccination

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Health care workers who are in direct contact with blood are at increased risk of acquiring hepatitis B infection. The Department of Health recommends vaccination against hepatitis B for people at special risk.¹ We report a case of Reiter's syndrome after the second dose of Engerix B vaccine and a case of reactive arthritis after the first, both occurring in health care workers.

Case reports

CASE 1

A 29 year old health care worker developed polyarthrits four weeks after the second injection in a course of hepatitis B immunisation. Initially, he had developed pain, stiffness, and swelling in his left wrist and his knees and had been unable to walk. He had also developed bilateral conjunctivitis and dysuria. There had been no prodromal illness.

On examination he was not feverish, and he had bilateral conjunctivitis with scanty purulent discharge. His right knee was hot, swollen, and tender but no effusion was detectable. He had a normal full blood count with a raised plasma viscosity (1.90, normal range 1.5-1.72). Liver enzyme activities were normal, and screening for rheumatoid factor and autoantibodies gave negative results. Urine cultures were sterile; a urethral swab was not taken. He improved with bed rest and splinting and was discharged from hospital taking indomethacin. Over the next six months he had intermittent dysuria but urine cultures remained sterile. He subsequently developed persistent swelling in his wrists and knees. Enteric coated prednisolone 5 mg daily was added, and his symptoms improved gradually over the next four months, when all drugs were withdrawn. He was observed for a further four months with no recurrence in his symptoms and remained well during the following year.

CASE 2

A 41 year old female nurse developed migratory polyarthrits two weeks after receiving a single dose of hepatitis B vaccine. On examination her right wrist and her knees were tender and swollen and she had only restricted, painful movement in her left shoulder. She also had asymmetrical swelling in the small joints of her hands and feet. She had no history of recent infections and no relevant medical or family history. She had a

normal full blood count with a raised plasma viscosity (2.12). Liver enzyme activities were normal and urine cultures were sterile. Tests for rheumatoid factor and autoantibodies gave negative results.

Her symptoms were initially controlled with naproxen 500 mg twice daily, but after two months the joint pain and stiffness increased and she started taking sulphasalazine 500 mg a day, which was increased to 1 g twice daily. Hand radiographs never showed erosive changes. Her symptoms settled after five months, and drug treatment was discontinued. She was observed for a further six months with no recurrence.

Comment

Although reactive arthritis after hepatitis B vaccination has been reported,^{2,4} to our knowledge Reiter's syndrome has not been described. Engerix B vaccine was first marketed in 1987, and over 3.2 million doses have been distributed across the United Kingdom and Republic of Ireland. The Committee on Safety of Medicines in the United Kingdom has received many reports of arthralgia and a small number of cases of reactive arthritis or exacerbation of pre-existing arthritis after vaccination (personal communication). Joint symptoms usually develop within a few weeks of the first or subsequent vaccination and commonly resolve in a further few weeks. In both our patients, however, disabling joint symptoms lasted for almost six months. The pathogenesis of reactive arthritis is not known, but the presence of HLA-B27 predicts a prolonged and more severe disease.⁵ Circulating immune complexes are also thought to have a role.³ We did not test our patients for circulating immune complexes, complement activation, or HLA-B27 antigen.

SmithKline Beecham Pharmaceuticals, the manufacturer of Engerix B vaccine, has received 11 reports of rechallenge vaccination in people who developed joint symptoms after receiving Engerix B; six of them developed polyarthralgia again, and in most of them rechallenge led to more severe symptoms (personal communication). We did not revaccinate our patients because of the duration of their disabling symptoms. The long term prognosis in people developing disabling arthritis after hepatitis B vaccination is not documented.

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3 Hachulla E, Houvenagel E, Mingui A, Vincent G, Laine A. Reactive arthritis after hepatitis B vaccination. *J Rheumatol* 1990;17:1250-1.

4 Biasi D, De Sandre G, Banbara LM, Carletto A, Caramaschi P, Zanoni G, et al. A new case of reactive arthritis after hepatitis B vaccination. *Clin Exp Rheumatol* 1993;11:215.

5 Svenungsson B. Reactive arthritis. *BMJ* 1994;308:671-2.

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Immunity to hepatitis B among health care workers performing exposure prone procedures

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All health care staff who have contact with blood or bloodstained body fluids should be vaccinated against hepatitis B.¹ More recent recommendations are that all surgeons should be immunised against hepatitis B by mid-1994 and all staff involved in exposure prone

procedures by mid-1995,² exposure prone procedures being those in which injury to the employee could result in exposure of a patient's open tissues to the employee's blood. We examined the immunity of staff of two health authorities and estimated the risk of a health care worker who performs exposure prone procedures being an infectious carrier of hepatitis B.

Subjects, methods, and results

Dudley Health Authority and Hammersmith and Queen Charlotte's Special Health Authority separately introduced policies before and in keeping with the government's guidelines² in 1993 and 1991 respec-

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See editorial by Delamothé

tively. All employees performing exposure prone procedures were asked to provide evidence of immunity to hepatitis B. Those without such evidence were asked to undergo testing for hepatitis B surface antigen and, if positive, for e markers.

Those with a titre of hepatitis B surface antibody > 100 mIU/ml were recorded as being immune. Those with a titre of 10-100 mIU/ml were given a booster dose (20 µg) of hepatitis B vaccine (Engerix B), and their antibody response was measured two months later. Those with a titre < 10 mIU/ml were given another course (at 0, 1, and 6 months) of vaccine, or the alternative recombinant hepatitis B vaccine (H-B-Vax II) if they had already had two courses.

The table shows immunity to hepatitis B in staff in the two health authorities. In Dudley two consultant surgeons refused to provide evidence of immunity or to undergo testing for hepatitis B surface antigen or core antibodies. The mean number of doses of vaccine given was 3.7, with most people developing adequate immunity after three; four surgeons, however, required either six or nine doses of vaccine. In London no employee refused to provide evidence of immunity or to undergo testing for hepatitis B surface antigen when appropriate. Four out of 230 employees (1.7%) who performed exposure prone procedures were positive for hepatitis B surface antigen; one of them, a surgeon, was also positive for the e antigen. Of these four, one was a doctor and three were midwives; three were born outside the United Kingdom.

Comment

An immunity to hepatitis B of 94% among consultant surgeons in Dudley is encouraging. This figure is greater than published seroconversion rates of about 85% in normal adults because some of the staff had had up to three courses (nine injections) of vaccine. In fact, no doctor who was negative for hepatitis B surface antigen or naturally immune failed to respond to vaccine. This observation challenges the notion that 1-4% of those vaccinated will fail to respond to one or more courses of vaccine³ and supports the concept of slow responders.

Some people will not mount an immune response on vaccination because they are long term carriers of the virus; this applied to 1.7% of health care workers who performed exposure prone procedures in the London

Percentages (proportions) of employees who were immune to hepatitis B in Dudley Health Authority and Hammersmith and Queen Charlotte's Special Health Authority

	Dudley	Hammersmith and Queen Charlotte's
<i>Staff performing exposure prone procedures</i>		
Consultant surgeons	94 (34/36)	65 (35/54)
Junior doctors	64 (48/75)	68 (26/38)
Dentists and surgery assistants	90 (28/31)	81 (13/16)
Midwives	47 (81/171)	90 (110/122)
<i>Others</i>		
Staff in renal dialysis units	74 (23/31)	93 (14/15)
Nurses (grades C to I)	21 (362/1753)	78 (855/1096)
Porters	4 (4/110)	49 (22/45)
Domestic staff	0 (2/550)	27 (70/260)

authority, but it applies to up to 11% of health care workers in parts of the world where hepatitis B is endemic.⁴ A prevalence of 1.7% is higher than that found in blood donors and at antenatal screening but in keeping with a mixed ethnic group of employees in a high risk occupation.⁵ For personal reasons a few employees will refuse to be vaccinated or provide evidence of immunity, but the government recommends that these staff are restricted in the same way as staff positive for hepatitis B e antigen.¹ This number can be limited by counselling about the different types of hepatitis B serological markers and by reassuring staff that testing for the surface antigen will be done only with informed consent.

Few domestic staff and porters were immune, which is disappointing because needlestick injuries are regularly reported by them. The provision of occupational health services to NHS staff is, however, patchy, and the amount of budget allocated to the purchase of vaccines is controlled. The ability of occupational health departments to achieve the targets set by the NHS Executive will largely depend on their resources.

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- 3 Boxall EH. Risks to surgeons and patients from HIV and hepatitis. *BMJ* 1993;306:652-3.
- 4 Gupta HL, Sebastian M, Sinha VP, Gupta SK, Gupta VK. Hepatitis B positivity among medical professionals. *J Assoc Physicians India* 1989;37: 618-9.
- 5 Alexander G, Williams R. Antiviral treatment in chronic infection with hepatitis B virus. *BMJ* 1986;293:956.

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Outcome of childhood asthma in mid-adult life

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See editorial by Sears

A prospective 28 year follow up of 7 year old children with wheezing and asthma began in 1963 in Melbourne, Australia.^{1,2} We aimed to describe the outcome of childhood asthma at 35 and the changes since the last review, at 28.³

Subjects, methods, and results

The children who were selected at 7, on the basis of parents' responses to a questionnaire, had been classified in terms of wheeze as those who had never wheezed (controls) (n = 106); those with fewer than five episodes associated with apparent respiratory infection (mild wheezy bronchitis) (n = 75); those with five or more

episodes associated with an apparent respiratory infection (wheezy bronchitis) (n = 107); and those with wheezing not associated with respiratory infection (asthma) (n = 113). A fifth group of children, with severe asthma (n = 79), was selected at age 10 from the same age cohort.

During 1992 we followed up 401 of the 480 subjects; 101 answered a telephone questionnaire, and 300 were examined physically. Eleven subjects had died since the study began. The follow up rate was therefore 86%. The subjects were classified as those who had been controls at age 7 (controls) (n = 85); those who had not wheezed in the previous three years (no recent asthma) (n = 132); those who had wheezed in the previous three years but not in the previous three months (infrequent wheeze) (n = 132); those who had not wheezed in the previous three months but less than once a week (frequent asthma) (n = 45); and those who wheezed at least once a week in the previous three months (persistent asthma) (n = 91).

The table shows the pattern of asthma at age 35 in relation to the initial classification. The presence of atopy at age 7 in the children with mild wheezy