Comment

There is no doubt that this patient had acute hepatitis. Diclofenac or non-A non-B hepatitis are the most likely causes. In the absence of specific tests non-A non-B hepatitis can be diagnosed only by exclusion. The presence of eosinophils in the inflammatory infiltrate seen on liver biopsy favours a drug aetiology, but this feature may also be seen in relapsing or prolonged acute viral hepatitis, which may be indistinguishable histologically.³ This patient had not, however, been exposed to any of the known risk factors for non-A non-B hepatitis. Furthermore, relapsing non-A non-B hepatitis usually follows a more chronic course,⁴ whereas our patient was completely well with normal serum liver function tests between the two episodes of hepatitis, which resolved rapidly and totally after diclofenac was stopped.

The gap between starting diclofenac and the onset of hepatitis is incompatible with a drug-induced liver injury of the "hypersensitivity" type but is in keeping with an idiosyncratic drug-induced hepatitis of the "metabolic aberration" type.⁵ Diclofenac has been reported to cause abnormalities in serum liver function tests,² and it was the only drug our patient was taking. This fact and the temporal relation of the two episodes of hepatitis to ingestion of diclofenac strongly suggest that the drug was responsible for the liver injury.

This case emphasises the difficulties encountered in separating viral hepatitis from that caused by drugs.

We thank Dr K Greenlaw of Stock, Essex, who referred this patient.

- ¹ Ciccolunghi SN, Chaudri HA, Schubiger BI. The value and results of long term studies with diclofenac sodium (Voltarol). *Rheumatol Rehabil* 1979;2:100-15.
- ² Ciccolunghi SN, Chaudri HA, Schubiger BI, Reddrop R. Report on a long term tolerability study of up to 2 years with diclofenac sodium (Voltarol). *Scand J Rheumatol* 1978;22:86-96.
- ³ Bianchi L, DeGroot J, Desmet VJ, et al. Guidelines for the diagnosis of therapeutic drug-induced liver injury in liver biopsies. *Lancet* 1974;ii: 854-7.
- ⁴ Thomas HC, Bamber M. Clinical aspects of non-A non-B hepatitis. In: Jewell DP ed. *Advanced medicine* 17. London: Pitman Medical, 1981: 159-67.
- ⁵ Zimmerman HJ. Drug hepatotoxicity: spectrum of clinical lesions. In: Davis M, Tredger JM, Williams R, eds. *Drug reactions and the liver*. London: Pitman Medical, 1981:35-53.

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Psittacosis presenting with Reiter's syndrome

Polyarthritides is a common feature of chlamydial respiratory infection in many animal species,¹ but this association, while recognised, is rarely reported in man.^{2,3} We report such a case, which presented as Reiter's syndrome.

Case report

A 35-year-old woman was admitted in March 1981. Three weeks before admission she had developed a fever, general weakness, sore throat, cough, and purulent sputum. Ten days later she had bilateral conjunctivitis, back pain, and painful swollen knees. There was no dysuria or vaginal discharge. She visited a pet shop regularly but had no direct contact with psittacine birds. On examination gingivitis and purulent conjunctivitis were present. There were no clinical abnormalities of the respiratory, cardiovascular, or alimentary systems. Rotation of the cervical spine was limited with tenderness over C4/C5. The right first metacarpophalangeal joint, both knees, and the left ankle had tense effusions with pronounced tenderness and limitation of movement.

The initial results of investigations were as follows: full blood count normal; erythrocyte sedimentation rate 114 mm/first hour; Paul Bunnell test negative; antinuclear factor, DNA antibodies, and rheumatoid factor absent; antistreptolysin O titre less than 50 units; serum albumin 31 g/l

(3.1 g/100 ml) (normal 35-55 g/l) (normal 3.5-5.5 g/100 ml); serum alanine aminotransferase 52 IU/l (normal 5-35 IU/l). A midstream specimen of urine showed no abnormality. Throat and conjunctival swabs and sputum grew commensal organisms only. Synovial fluid from the knee contained total protein 65 g/l (6.5 g/100 ml) and albumin 27 g/l (2.7 g/100 ml); 60% of the cells were lymphocytes and 40% neutrophils. The glucose content was 5.5 mmol/l (100 mg/100 ml). Chest x-ray films showed a raised right hemidiaphragm with increased basal markings. X-ray films of the sacro-iliac joints were normal. Complement fixation test for the psittacosis/lymphogranuloma venereum organism was positive at a titre of 1/256, which fell to 1/64 three weeks later. HLA-B27 antigen was present. The patient was treated with tetracycline and then prednisolone and recovered fully over the following eight weeks. To date no other cause for her polyarthritides has been discovered.

Comment

This patient had a characteristic psittacosis pneumonia, which was diagnosed retrospectively by serology. The lack of a history of a contact is characteristic of this disease, which is being increasingly reported.⁴ Asymmetrical polyarthritides and conjunctivitis with the presence of the HLA-B27 antigen satisfied the criteria for the diagnosis of Reiter's syndrome.⁵ This is, we believe, the first report of such an association.

¹ Storz J. *Chlamydiae and chlamydial induced diseases*. Springfield Illinois: Charles Thomas, 1971.

² Favour CB. Ornithosis (psittacosis). *Am J Med Sci* 1943;205:162-87.

³ Simpson R, Huang C, Grahame-Smith DG. Psittacosis masquerading as rheumatic fever. *Br Med J* 1978;ii:694-5.

⁴ DHLS Communicable Diseases Surveillance Centre Report. Trends in human ornithosis/psittacosis 1975-80. *Br Med J* 1981;281:1411.

⁵ Arnett FC, McCluskey OE, Schottor B, et al. Incomplete Reiter's syndrome: discriminating features and HLA W27 in diagnosis. *Ann Intern Med* 1976;84:8-12.

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Spontaneous persistent pseudomembranous colitis related to Clostridium difficile in ischaemic bowel disease

Clostridium difficile is now considered to be a major factor in the pathogenesis of antibiotic-associated pseudomembranous colitis,¹ though in the past bowel ischaemia was considered to be important.^{2,3} I describe a patient with chronic ischaemic bowel disease who developed spontaneous pseudomembranous colitis associated with *C difficile* infection.

Case report

A 69-year-old woman with a history of stroke affecting the right hemisphere, angina, and hypertension presented in December 1980 after three weeks of non-bloody diarrhoea. She had not received antibiotics in the preceding six months. Sigmoidoscopy and rectal biopsy showed the florid changes of pseudomembranous colitis. Barium enema showed a pancolitis with diffuse thumb-printing pattern. Vancomycin 500 mg six-hourly was given by mouth for eight days. Stools were not examined for *C difficile* before this course of vancomycin. Her diarrhoea responded dramatically.

Within three days of her stopping vancomycin the diarrhoea returned; faecal *C difficile* and its cytotoxin were isolated. Vancomycin was given for a further three weeks at the same dose and was stopped when repeated stool testing shown no *C difficile* and the diarrhoea had resolved. One week later *C difficile*, sensitive to vancomycin, was again isolated, though there was no detectable cytotoxin. Vancomycin was initially withheld. Two weeks later she developed rectal bleeding and diarrhoea. Both the cytotoxin and *C difficile* were then present in the stools.

A third two-week course of vancomycin was begun, again with improvement. Angiography was carried out because of the rectal bleeding and the appearance seen on the initial barium enema. Complete occlusion of the origin of the inferior mesenteric and severe stenosis of the superior and coeliac arteries were shown. Her symptoms again resolved with vancomycin.