opposing sides, but there were some signs that such action might be necessary in similar circumstances.

Conclusions—As well as continuing to care for its normal patients an accident and emergency department will have to make special arrangements to prevent the department becoming overloaded with riot casualties. These arrangements should include (a) the use of a large area within the department, preferably next to the minor injury area, for receiving and sorting the riot casualties; (b) the provision of special teams for suturing, extra reception and portering staff, and facilities for rapid x-ray examinations; and (c) arrangements for detaining some patients for observation pending diagnosis because the emotional state of the casualties may make diagnosis difficult. There should also be plans for the hospital's security staff to be reinforced by police officers in case of violence among opposing factions within the department. Although it is accepted that when natural disasters produce large numbers of casualties the police should site a documentation team in the accident department,³ such action after riots, when criminal proceedings may be pending against some of the casualties, could be construed as a breach of confidentiality. Finally, our experience suggests that the specialised needs of a riot are not catered for by the major accident procedure.

We acknowledge the great help of the nursing staff in dealing with the casualties.

¹ Rutherford WH. Surgery of violence. *Br Med J* 1975;i:443-5.

² Moran Lord. *The anatomy of courage*. London: Constable, 1945:21-3.

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Dysentery-like syndrome associated with *Aeromonas hydrophila*

Aeromonas hydrophila has been isolated from stools of patients with diarrhoeal disease¹⁻³ and some of these isolates produce enterotoxin, but its enteropathogenic role is still to be established.⁴ The gastroenteritis associated with this organism is usually self-limiting and does not require treatment. We report here a case of prolonged "dysentery-like" illness in a healthy man associated with repeated recovery of *A hydrophila* in stool cultures and his cure after treatment with co-trimoxazole.

Case report

In early December 1979 a 35-year-old Indian doctor developed abdominal cramps and diarrhoea with blood and mucus while on an extensive tour in his own country, which included a visit to Calcutta. Seven days later he returned to the UK. Though the frequency of diarrhoea increased to 5-6 stools/day he remained afebrile and otherwise well. Examination of two stools, collected one week apart, showed no usual parasitic or bacterial cause except for a moderate growth of sucrose-fermenting, oxidase-positive colonies on thiosulphate bile sucrose agar from the first specimen. This organism, identified as *A hydrophila* using the API-20E system, was not considered significant. As the patient had not had any specific treatment for four weeks he was given a 10-day course of metronidazole on the presumptive diagnosis of amoebic dysentery. This treatment reduced the cramps but did not affect the diarrhoea.

At this stage examination did not show any abnormality except a gross haemorrhagic proctitis found at sigmoidoscopy. A rectal biopsy specimen showed non-specific chronic inflammatory (plasma cell) infiltration of the lamina propria. The appearances on barium enema were normal. The following laboratory tests were negative: routine haematology; liver function tests; serological tests for Epstein-Barr virus, cytomegalovirus, and *Entamoeba histolytica*; Widal test; and viral culture of stool. Rectal fluid obtained during sigmoidoscopy and a subsequent stool again yielded only a heavy growth of *A hydrophila*; identification was later confirmed by the Vibrio Reference Laboratory, Maidstone. On disc diffusion testing the isolate was

sensitive to all the antimicrobials commonly used against Gram-negative organisms except ampicillin, carbenicillin, and cephaloridine. A high-dose preparation of co-trimoxazole (trimethoprim 160 mg and sulphamethoxazole 800 mg) was prescribed twice daily for seven days. Within four days of starting treatment the bleeding had stopped but headaches, which the patient attributed to the drug, caused him to discontinue the treatment on the fifth day. Improvement continued and his stools became normal after a further two days. Twelve days after the end of treatment a repeat sigmoidoscopy showed an apparently normal rectum. The results of three stool cultures during the six-week follow-up period were negative and the patient did not have a recurrence.

Comment

Some workers are convinced that *A hydrophila* has an enteropathogenic role since it can produce an enterotoxin. Nevertheless, to our knowledge there is only one previous well-documented case of diarrhoeal disease associated with this organism requiring antimicrobial therapy.⁵ The cell-free filtrate of an overnight brain-heart infusion broth culture of our strain examined in the Food Hygiene Laboratory, Colindale, produced appreciable fluid accumulation in ligated rabbit intestines and increased vascular permeability in rabbit skin tests, which suggested that this organism was enterotoxigenic. The isolate also gave a positive lysine decarboxylase test and Voges-Proskauer reaction, properties which correlate with the production of cytotoxic enterotoxin.⁴ This case of prolonged diarrhoea associated with repeated isolation of *A hydrophila* from faeces in the absence of any other enteric pathogens, with recovery after appropriate antimicrobial therapy, has provided strong evidence in support of the enteropathogenic role of this species. More studies are needed to elucidate the epidemiology and pathogenesis of this diarrhoeal disease.

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³ Annapurna E, Sanyal SC. Enterotoxicity of *Aeromonas hydrophila*. *J Med Microbiol* 1977;10:317-23.

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⁵ Rosner R. *Aeromonas hydrophila* as the etiologic agent in a case of severe gastroenteritis. *Am J Clin Pathol* 1964;42:402-4.

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Lymphocyte sensitisation in nifedipine-induced hepatitis

Nifedipine (Adalat) is a new, highly effective agent in the treatment of angina, with beneficial haemodynamic responses.¹ Adverse side effects include gastrointestinal symptoms, sensation of heat, and facial flushing. We report on a patient who developed hepatitis after administration of nifedipine. The clinical course, immunological findings, and a positive challenge test result strongly indicated a hypersensitivity response.

Case report

A 69-year-old man with a history of myocardial infarction was treated with nifedipine, 40 mg daily, because of severe, stable angina. Ten days after beginning the drug he experienced anorexia, nausea, and shaking chills with a temperature of 38.8°C and was admitted to hospital with painless jaundice. His blood pressure was 140/80 mm Hg and pulse 100 beats/min. There was no

ascites or abdominal organ enlargement. The laboratory results included: erythrocyte sedimentation rate 95 mm in first hour, conjugated bilirubin concentration 51.3 $\mu\text{mol/l}$ (3 mg/100 ml), serum alkaline phosphatase activity 420 IU/dl, serum aspartate aminotransferase activity 56 U/l, and serum alanine aminotransferase activity 68 U/l. Titres to hepatitis B surface antigen were negative. Antibody titres to hepatitis B surface and core antigens and hepatitis A associated antigen were positive on admission. Immunoglobulin G (IgG) concentration was initially raised (22.2 g/l), while concentrations of complement components were normal. Antibodies to smooth muscle and mitochondria were not detected.

Withdrawal of the drug resulted in a gradual decline of the temperature and return to normal of all laboratory values. An oral cholecystogram was normal. A liver biopsy was not performed.

The assessment of the feasibility of readministration of nifedipine seemed important because of the patient's continuing severe angina. Therefore, four months later and with the patient's informed consent we rechallenge him with a single dose of 10 mg nifedipine. Twelve hours later his temperature had risen to 37.5°C with a concomitant rise in serum alkaline phosphatase activity and IgG concentration but no changes in the activities of aminotransferases. Results of liver function tests at follow-up at 10 months were normal.

A macrophage migration inhibition factor assay² for nifedipine was performed two months after the acute icteric episode and gave positive results (figure). After rechallenge with nifedipine the migration indices abruptly returned to normal. In five control patients the migration inhibition factor test gave negative results (figure).

There was no lymphocyte stimulation³ with nifedipine, while lymphocyte response to regular mitogens was normal. An indirect mast-cell degranulation test⁴ gave negative results in the presence of nifedipine.

Migration index (migration of macrophages with donor lymphocytes in presence of antigen: migration of macrophages in control chamber) in patient and five control subjects. Arrow indicates time of challenge with nifedipine. Broken lines show limits of normal values.

Comment

The clinical features of this patient indicated that the liver damage may be attributed to hypersensitivity, since discontinuing the drug resulted in rapid and spontaneous improvement in his clinical condition and the biochemical findings. The positive challenge test with a small dose of nifedipine confirmed its causative role. The clinical course, normal cholecystogram, and results of viral hepatitis-associated tests could reasonably exclude other causes.

The raised alkaline phosphatase activity and IgG concentrations during the acute hepatic injury, their parallel decrease after withdrawal of nifedipine, and their concomitant rise on rechallenge are also important.

Migration inhibition factor production against nifedipine indicated that the patient's lymphocytes had become sensitised to that drug. The abrupt change of the positive result of the migration inhibition factor test to a negative one during challenge with nifedipine may reflect a shift of the sensitised lymphocytes from the peripheral blood pool to the site of injury.

Thus the immunological processes in the hepatic damage may include humoral and cellular mechanisms. The serial migration inhibition factor assays confirmed the clinical evidence when differentiating between allergic drug-induced and viral hepatitis. Nifedipine may be added to the list of drugs causing allergic hepatitis in susceptible people.

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Failure of rubella immunisation after blood transfusion: birth of congenitally infected infant

The dangers of maternal rubella in early pregnancy and the promotion of rubella immunisation programmes have been much publicised recently. This case of congenital rubella syndrome in the offspring of a mother who had received postpartum immunisation on two previous occasions illustrates the potential hazard of failure of immunisation when rubella vaccine is given shortly after blood transfusion.

Case report

The mother's obstetric and immunisation history is summarised in the table. Immunisation was with Almevax (Wellcome). In her fourth pregnancy (last menstrual period 4 November 1978) she was in contact with rubella between 6 and 8 weeks. All three of her children had a febrile illness with a rash diagnosed clinically as rubella by her family doctor. She remained asymptomatic. On 20 December her blood was reported to have "rubella antibodies present"—that is, an HAI rubella screening test titre of 1:80 or greater. The report on a specimen sent from the hospital antenatal clinic on 26 February 1979 was similar. Her baby was born on 22 August 1979 by normal delivery at term + 11 days weighing 3280 g. The infant was pale and had a generalised petechial rash. The platelet count was $34 \times 10^9/\text{l}$ ($34\,000/\text{mm}^3$). The right cornea was opaque and the left hazy. Tonometry showed bilateral glaucoma. The liver was palpable 3 cm below the costal margin and the spleen 1.5 cm. There was a systolic murmur at the 2nd-4th left intercostal spaces. Subsequent cardiological investigation (Dr G Watson) revealed a large patent ductus arteriosus, moderate ventricular septal defect, an atrial shunt through an incompetent foramen ovale, and slight pulmonary stenosis. Audiometry has since shown severe bilateral nerve deafness. The rubella HAI titre was greater than 1:960 and rubella-specific IgM was present. Retrospective testing of the mother's antenatal serum of 20 December 1978 showed a rubella titre of 1:1280 with rubella IgM present.

Obstetric and rubella immunisation history of mother who gave birth to an infected infant in her fourth pregnancy

Pregnancy	Antenatal screening (HAI titre)	Delivery date	Blood transfusion (date and amount)	Indication for transfusion	Immunisation date
First	Not done	6 April 1973	6 April 1973 2 units	PPH	Not given
Second	<1:20	21 June 1974	20 June 1974 2 units	Anaemia	21 June 1974
Third	1:20	31 May 1977	31 May 1977 4 units	PPH	3 June 1977

PPH = postpartum haemorrhage.