Intracranial section of the glossopharyngeal and upper roots of the vagus nerves has been recommended, the cardiac dysrhythmia being controlled with atropine, isoprenaline, or transvenous pacemaker. Carbamazepine is known to relieve the pain of uncomplicated glossopharyngeal neuralgia.^{2,3} In our first patient the combination of carbamazepine and demand pacemaker abolished both pain and syncope within hours. The pacemaker was later withdrawn. In the second patient both neuralgia and syncope were suppressed by carbamazepine for one year.

By its inhibitory effect on brain stem reflex activity carbamazepine appears not only to be an effective treatment for the pain of glossopharyngeal neuralgia but also to control the more dangerous bradycardia and asystole.

We thank Dr R Bradley for permission to report case 1.

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Gonococcal ophthalmia neonatorum caused by beta-lactamase-producing *Neisseria gonorrhoeae*

Certain strains of *Neisseria gonorrhoeae* produce the enzyme beta-lactamase that destroys the penicillin nucleus, rendering the drug ineffective. In the West these mutant strains probably originated from conjugation between *N. gonorrhoeae* and strains of *Escherichia coli* which produce beta-lactamase. There is some evidence that resistant strains in the Far East arose from conjugation between the gonococci and *Haemophilus influenzae*.¹ Genital gonococcal infection by these penicillin resistant strains were first reported in England and the USA² in 1976. Reports followed from Belgium, Sweden, Norway, Ghana, Singapore, Thailand, Philippines, and Korea.³ In Singapore in 1977 22 resistant strains were reported out of a total of 7668 infections (0.29%). In the first six months of 1978 188 were reported out of 4195 infections (4.48%). The number of gonococcal ophthalmia neonatorum cases reported in 1977 and the first six months of 1978 was 20 and 15 respectively. We report a case caused by a beta-lactamase-producing strain of gonococci. It is the first reported in Singapore and, we think, in the world.

Case report

An 8-day-old boy attended outpatients for a bilateral purulent eye discharge for five days. Three days before he had been treated by a general practitioner with parenteral and oral penicillin and sulphacetamide eye drops with no apparent improvement. The infant's mother had had a yellowish vaginal discharge for one week before parturition, otherwise she was asymptomatic. Her last sexual intercourse with her husband had been about one month before delivery. She denied other sexual contacts. Her husband admitted having a urethral discharge at about the same time but he had been treated by a general practitioner with full recovery. The patient was unaware of this and thus remained untreated.

Smears from the infant's eyes and the mother's endocervix and urethra showed Gram-negative intracellular diplococci. Specimens were sent to the laboratory for culture for *N. gonorrhoeae*. Gonorrhoea was provisionally diagnosed and both mother and child were admitted. The mother was treated with intramuscular procaine penicillin 4.5 mega units and probenecid 1 g by mouth. The child was given intramuscular crystalline penicillin 100 000 units six-hourly for four doses and crystalline penicillin drops (10 000 units/ml) were instilled into both eyes, after swabbing with normal saline, every 10 minutes in the first hour, every 30 minutes in the second, third, and fourth hours, and thereafter hourly for 60 hours. Despite this, smears from the child's eyes and mother's endocervix still showed Gram-negative intracellular diplococci after three days' treatment. By then the results of culture of the initial specimens had confirmed the presence of penicillin-resistant strains of gonococci. The mother was then treated with intramuscular kanamycin 2 g and the child intramuscular kanamycin 1 g

with intensive eye toilet, as described above, using 1% kanamycin eye drops. Follow-up smear cultures on days 3, 7, and 14 of kanamycin treatment were negative for *N. gonorrhoeae*. Both mother and child have since been well.

Comment

Crede's method of instilling silver nitrate eye drops to newborns has not been practised in Singapore for many years. The low incidence of gonococcal ophthalmia neonatorum, about 0.07% of total live births a year, reflects satisfactory control and antenatal screening. But with the increase in cases of genital infections caused by penicillin-resistant gonococci in the East we expect this incidence to rise. We have treated 356 cases of beta-lactamase-producing gonorrhoea with single intramuscular doses of 2 g kanamycin, with an overall cure rate of 98.2%. We therefore think that kanamycin is very effective.

In a recent study we found that treatment of gonococcal ophthalmia neonatorum with intensive eye toilet alone is unsatisfactory. The failure rate was 18.75%. But combined local and parenteral therapy with penicillin cleared infections in 100% of cases.⁴ We therefore emphasise the importance of combined treatment.

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Reactive arthritis associated with *Campylobacter jejuni* enteritis

Reactive arthritis, defined as an acute arthritis associated with an infection elsewhere in the body and without the infecting micro-organism being present in the joints, may follow infections of the intestinal and urinary tract. The intestinal pathogens *Shigella*, *Salmonella*, and *Yersinia enterocolitica* have been associated with reactive arthritis. We describe a patient who developed an arthritis after enteritis caused by *Campylobacter jejuni*, a bacterium which has not been described as an aetiological agent in reactive arthritis.

Case report

A 20-year-old man was seen with arthritis of both ankle joints. Five weeks before he had become suddenly ill with fever (39.5°C), watery diarrhoea contaminated with blood, anorexia, and weight loss of 6 kg. These symptoms lasted for five days. *C. jejuni* was isolated from the faeces. Two weeks after the onset of the enteritis an acute arthritis of both ankle joints developed. He had no dysuria, conjunctivitis, skin abnormalities, or lesions of the mucous membranes and no history of bowel or joint complaints. When seen three weeks after the onset of the arthritis the ankle joints were warm, tender, red, and swollen. The other peripheral joints and the spine, skin, nails, mucous membranes, penis, and eyes were normal. Laboratory examination initially showed an erythrocyte sedimentation rate of 40 mm in 1 hour, which later became normal. Haemoglobin, leucocyte and platelet counts, serum creatinine, urate, cholesterol, liver function tests, and complement factors C3, factor B, and C4 were within normal limits. A PPD skin test was negative. Aspiration of the right ankle joint produced clear yellow synovial fluid. No micro-organisms or polymorphonuclear leucocytes were seen in the Gram smear. Culture of the aspirate failed to grow *C. jejuni*. Bacteriological examination of the stool at this time was also negative for *C. jejuni* as well as for salmonella, *Yersinia enterocolitica*, and shigella. Serological tests were negative for rheumatoid factors, antinuclear and antiperinuclear antibodies, complement fixing antibodies against *Neisseria gonorrhoeae*, and agglutinating antibodies against *Y. enterocolitica* types 3 and 9 and *Y. pseudotuberculosis*. When *C. jejuni* were isolated from the stools no

agglutinating antibodies could be detected against the isolated strain, but by the time the enteritis was over the titre had risen to 1/1280. When we saw the patient in our outpatient clinic the titre had declined to 1/160. After the arthritis had disappeared no antibodies could be detected. Radiological examination of the sacroiliac and ankle joints showed no abnormalities. Tissue typing showed HLA-A 2,28; B 12,27. Eight weeks after the onset of the arthritis all signs and symptoms had disappeared.

Comment

The findings in this case suggest that the oligoarthritis was reactive to the *C jejuni* enteritis. The arthritis started two weeks after the onset of the enteritis, and the antibody titre against the isolated campylobacter significantly increased. Other known causes for acute arthritis could be excluded. Moreover, reactive arthritis after certain infections occurs especially in HLA-B 27-positive patients.¹ This antigen was present in our patient. We do not know of other reports of reactive arthritis associated with *C jejuni* infections. One report² attributes exacerbations of a Reiter syndrome to *Campylobacter fetus* infections. This species, however, is distinct from *C jejuni*. *C jejuni* is now identified more often as a cause of enteritis.³ Possibly, therefore, reactive arthritis after *C jejuni* infection is not rare and will be diagnosed more often in the future.

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Renal carcinoma with thrombocytopenia, clubbing, and hypertrophic osteoarthropathy

Renal carcinoma has not been reported in association with thrombocytopenia, while clubbing and hypertrophic osteoarthropathy in the absence of pulmonary metastases has also not been described.

Case report

A 71-year-old man was admitted in December 1974 with aching in his left loin, weight loss of 18 kg in two months, and clubbing of fingers and toes. His erythrocyte sedimentation rate (ESR) was 120 mm in the first hour (Westergren). Skeletal x-ray survey and bone scan showed hypertrophic osteoarthropathy of the tibiae, fibulae, and femora. Investigations with normal results included the following: urine microscopy, intravenous urography, blood and platelet counts, chest radiograph, sputum cytology, bronchoscopy with washings for cytology, barium enema, barium meal, liver biopsy, faecal fat excretion, urinary and serum protein electrophoresis, and bone marrow aspiration. His left loin pain and his raised ESR persisted until his death.

In April 1976 a radiograph of the renal areas was normal, as was his platelet count and findings on urine microscopy. In July 1977 he was found to have developed asymptomatic thrombocytopenia (platelet count below $40 \times 10^9/l$). At that time he was receiving regular paracetamol and dextropropoxyphene (Distalgic) tablets for his loin pain, but the thrombocytopenia was unaffected by the withdrawal of the drug.

He received no other medication during the illness. The cause of the thrombocytopenia was not identified, the results of the following being normal: bone marrow aspiration and trephine, diffuse intravascular coagulation screen, serum and urinary protein electrophoresis, estimation of immunoglobulins and antinuclear factor, liver function tests, urine microscopy, platelet surface IgG estimation, and tests for platelet antibodies with and without paracetamol and dextropropoxyphene. In December 1977 the spleen was just palpable. In February 1978 the patient was found to have haematuria. Urine microscopy had been normal on six previous occasions. An abdominal radiograph showed calcification within the upper pole of the left kidney; intravenous urography and renal arteriography showed that the calcification was within a renal carcinoma. The previous x-ray films were reviewed and again passed as normal. An inoperable locally invasive renal

carcinoma was found at laparotomy. The patient recovered sufficiently to go home for a month, but was subsequently readmitted with a terminal bilateral bronchopneumonia. He had never had macroscopic haematuria.

Necropsy—A large, poorly differentiated renal cell carcinoma with pseudo-sarcomatous change in some areas was found. Tiny foci with the more typical pattern of renal papillary carcinoma still remained and a thin rim of normal kidney surrounded parts of the tumour. The tail of the pancreas, the left adrenal, and the spleen were affected by a mass of necrotic tissue, and multiple, small, wedge-shaped infarcts with associated thrombosed vessels were seen in the spleen. No definite invasion of the spleen by malignant cells was seen, and no viable tumour was identified in the necrotic material. There were no secondary deposits in any other organ and the left renal vein was not invaded. His terminal bronchopneumonia was confirmed.

Comment

The variable clinical presentation and course of renal carcinomas are well known.^{1,2} This patient presented with loin pain three years before he had other symptoms or radiological evidence of renal carcinoma. Hypertrophic osteoarthropathy and clubbing have been described only in association with pulmonary metastases and have disappeared after resection of the metastases.³ In this case only the legs were affected by the hypertrophic osteoarthropathy, although the fingers were clubbed, and no metastases were seen. Haematuria occurred only when the thrombocytopenia was well established. The thrombocytopenia had no clear cause and contrasts with the more usual thrombocythaemia seen with carcinomas. The thrombocytopenia may be related to splenic infarction.

We thank Dr R W E Watts for permission to report this case.

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Acute renal failure after TAB and cholera vaccination

Reactions to TAB and cholera vaccination ranging from local erythema to constitutional disturbance are commonplace.¹ We record here an episode of acute renal failure and hepatitis after vaccination, which apparently has never been reported.

Case report

A fit 21-year-old man who several years earlier had undergone vaccination with TAB and cholera was revaccinated with typhoid A and B and cholera 0.5 ml subcutaneously. Within one hour he vomited and complained of headache and abdominal pain. He vomited several times during the following night and one episode of diarrhoea occurred; fluids were encouraged throughout this period. He developed myalgia. The next day he had neck stiffness and his temperature rose to 37.4°C. On the third day of the illness he was admitted to hospital because he had been anuric since the injection.

Examination showed jaundice, but he was not clinically dehydrated. There was obvious tenderness of the neck, limb, and abdominal wall muscles. The blood pressure was 90/70 mm Hg and the pulse rate was 80 beats/min, regular and of good volume. The heart sounds were normal and the chest clear. The results of examination was otherwise normal.

Investigations showed: haemoglobin 15.2 g/dl, white cell count $23.6 \times 10^9/l$; blood concentrations of urea 23.0 mmol/l (138 mg/100 ml), potassium 3.8 mmol(mEq)/l, sodium 129 mmol(mEq)/l, total CO₂ 20 mmol(mEq)/l, and creatinine 0.38 mmol/l (4.4 mg/100 ml). Bilirubin concentration was raised at 75 µmol/l (4.5 mg/100 ml), alanine transaminase at 160 IU/l, and aspartate transaminase at 85 IU/l; alkaline phosphatase concentration was normal.

There was no history of exposure to nephrotoxins, contact with infectious disease, or excessive exercise. The following blood tests all gave normal or negative results: blood culture, viral studies, Paul-Bunnell test, Leptospira complement fixation test, immunoglobulin and complement estimations,