natural resistance to a number of antibiotics including those routinely bactericidal for Gram-negative bacteria, such as ampicillin, third-generation cephalosporins and aminoglycosides. It is usually susceptible to trimethoprim–sulfamethoxazole, imipenem, and rifampicin. Among cases of adult meningitis, five immunosuppressed patients died and the four others were cured without ill effects. The seriousness of the disease is more from the terrain on which it occurs rather than the virulence of the germ. One patient without immunosuppression but with post-traumatic meningitis was cured with monotherapy poorly penetratable to cerebrospinal fluid (CSF) or poorly bacteriostatic (amikacin for 13 days, ceftriaxone, cefepime, and cefotaxime for 25 days and the CSF cultures remaining positive for 25 days).

To our best knowledge, this is the first case report of an association between *F. meningosepticum* septicaemia and bacterial meningitis in a patient with AML. Our patient could have been an healthy carrier of *F. meningosepticum*, which became highly pathogenic because of persistent neutropenia. The serious condition of our patient is an early indication that molecular tests for early identification of appropriate antibiotic therapy and the role of haematological remission in cure without sequelae.

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**Case report**

A 55-year-old man was working in his garden when he injured his left thumb with a stone. He licked his finger to relieve pain. Five days later, he presented with left thumb nail haematoma, finger pain and fever. On examination he was pyrexial (37.8°C) and had a tender, swelling, left thumb with subcuticular abscess of the nail folds. X-Ray examination was normal. The nail was removed and amoxicillin/clavulanic acid (500/125 mg tid) was prescribed for 10 days. Two weeks later he remained febrile (37.5°C), and had left thumb pain and swelling. Radiologic examination revealed lytic bone defects on the terminal phalans of the left thumb and soft tissue swelling. The terminal phalanx was amputated. The operative material showed an acute suppurrative response and necrosis of tissue. Culture of the bone yielded, three days later, heavy growth of *E. corrodens*. The pathogen was not
cultured to *in vitro* to penicillin, ampicillin, and tetracycline and resistant to clindamycin, cephalosporins, chloramphenicol and gentamicin. Amoxicillin, 300 mg tid, was prescribed for 14 days. The infection healed quickly and all symptoms promptly subdued.

As *E. corrodens* is an endogenous oral bacterium it is not surprising that the most common clinical sources of this organisms are human bite wounds, head and neck infections, and respiratory tract infections. Several clinical manifestations including soft-tissue infections or osteomyelitis caused by this organism, have also been described. Thus, most infections caused by *E. corrodens* involve areas contaminated by oral secretions. Probable, in our patient, the organism was inoculated when he licked his finger. It is a matter for speculation why amoxicillin/clavulanic acid failed. The process which halted the development of osteomyelitis, in spite of the fact that the organism was susceptible to *in vitro* to ampicillin. Presumably the daily dose and length of treatment were not sufficient to cure the infection, especially if osteomyelitis were already present when the patient was seen for the first time.

**Pressor effect of metoclopramide in phaeochromocytoma**

Sir,

Metoclopramide is a widely used anti-emetic in hospital practice. Apart from its well established indications it is generally well tolerated. It has a little known pressor effect in normal individuals but can produce a massive rise in blood pressure in phaeochromocytoma. We describe a patient with a phaeochromocytoma given metoclopramide where, fortunately, the outcome was good.

**Case report**

A 34-year-old woman, with recently diagnosed hypertension and a past history of neurofibromatosis Type 1, was referred with episodic dizziness, sweating, recurrent headaches and nausea. Her blood pressure was initially 208/134 mmHg. Control of blood pressure had been poor over the past few weeks, despite nifedipine and atenolol prescribed by her general practitioner. On admission to hospital her blood pressure was 150/90 mmHg. Multiple café au lait spots and axillary freckling were noted. She had had a right below-knee amputation some years previously for neurofibromatosis. Fundoscopy revealed grade III hypertensive changes. Physical examination was otherwise normal. Shortly after admission she complained of severe nausea and vomited once. She was given intramuscular metoclopramide (10 mg). Within minutes she became profoundly unwell, clammy, pale, and vomited again. Her blood pressure rose to 280/160 mmHg. Intravenous labetalol (50 mg over 3 minutes) was infused over 1 minute, which relieved her symptoms and lowered the blood pressure to 160/120 mmHg. A presumptive diagnosis of phaeochromocytoma was made and the patient was given an alpha-blocker, doxazosin, in addition to the beta-blocker, atenolol. Nifedipine was discontinued on admission. Computed tomography (CT) scan of the abdomen revealed a 9 cm² mass in the left adrenal gland. A 24-hour urine collection contained a 880 μmol of normetadrenaline (normal < 4 μmol) and more than 70 μmol of metadrenaline (normal < 4 μmol). The patient underwent left adrenalectomy and histological examination of the tumour confirmed the diagnosis of phaeochromocytoma.

Metoclopramide-induced hypertensive crisis was first described in 1976. Later this effect was confirmed by controlled administration of the drug before and after adrenalectomy in two patients with phaeochromocytoma. The mechanism may relate to a D₂ dopamineergic inhibitory effect on the adrenergic medulla which is blocked by both metoclopramide

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**References**

Eikenella corrodens thumb osteomyelitis.

M. Cuenca-Estrella, J. M. Ramos, J. Esteban, F. Soriano and J. V. Vallejo

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