natural resistance to a number of antibiotics including those routinely bactericidal for Gram-negative bacilli, 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 immunodepression with post-surgical meningitis was cured with monotherapy poorly penetratable to cerebrospinal fluid (CSF) or poorly bacteriostatic (amikacin for 13 days, then intravenous erythromycin for 25 days and the CSF cultures remaining positive for 25 days).⁴

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 a healthy carrier of *F meningosepticum*, which became highly pathogenic because of persistent neutropenia. The favourable outcome underlines the need for early introduction of appropriate antibiotic therapy and the role of haematological remission in cure without sequelae.

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Eikenella corrodens thumb osteomyelitis

Sir,
Eikenella corrodens is a small, facultatively anaerobic and slowly growing Gram-negative rod,¹ which is a common inhabitant of the human oral cavity, upper respiratory, gastrointestinal and genitourinary tracts.^{2,3} It has been implicated in periodontitis, human wound infections and more serious diseases.³⁻⁸ We present a case of osteomyelitis of the terminal phalanx from the left thumb caused by *E. corrodens*.

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 bed 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. Radiographic examination revealed lytic bone defects on the terminal phalanx of the left thumb and soft-tissue swelling. The terminal phalanx was amputated. The operative material showed an acute suppurative response and necrosis of tissue. Culture of the bone yielded, three days later, heavy growth of *E. corrodens*. The pathogen was susceptible *in vitro* to penicillin, ampicillin, and tetracycline and resistant to clindamycin, cephalosporins, chloramphenicol and gentamicin. Amoxicillin, 500 mg tid, was prescribed for 14 days. The wound healed quickly and all symptoms promptly subsided.

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.^{9,10} Probably, in our patient, the organism was inoculated when he licked his finger. It is a matter for speculation why amoxicillin/clavulanic acid failed to cure the process or halt the development of osteomyelitis, in spite of the fact that the organism was susceptible *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.

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Pressor effect of metoclopramide in phaeochromocytoma

Sir,

Metoclopramide is a widely used anti-emetic in hospital practice. Apart from its well known extrapyramidal side-effects it is generally well tolerated. It has a little known pressor effect in normal individuals but can produce a massive rise of blood pressure in phaeochromocytoma. We describe a patient with 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 I, 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 the patient became profoundly unwell, clammy, pale, and vomited again. Her blood pressure rose to 280/160 mmHg. Intravenous labetalol (50 mg) was infused over several minutes, 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 x 7 cm² mass in the left adrenal gland. A 24-hour urine collection contained 80 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 1976.¹ Later this effect was confirmed by controlled administration of the drug before and after adrenalectomy in two patients with phaeochromocytoma.² The mechanism may relate to a D₂ dopaminergic inhibitory effect on the adrenal medulla which is blocked by both metoclopramide

Learning points

- many drugs may induce a severe pressor reaction in patients with a phaeochromocytoma
- these drugs should not be administered to patients with hypertension when phaeochromocytoma is a possible diagnosis

and sulpiride.³ Some common drugs such as corticosteroids, insulin, morphine, pethidine, monoamine oxidase inhibitors and tricyclic antidepressants, have been known to elevate the blood pressure in phaeochromocytoma. Most physicians are aware of the paradoxical rise of blood pressure with β -adrenergic blockers given alone in phaeochromocytoma without concomitant α -blockade.

With our patient it was fortunate that two of us (SF and MRL) were aware of the interaction and, in the presence of neurofibromatosis, the diagnosis was then straightforward. Nevertheless, we feel that this dangerous effect of a very commonly used anti-emetic should be emphasised more strongly.

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Fibrate monotherapy and profound hypoalphalipoprotein-aemia

Sir,

A proposed benefit of fibrate therapy in dyslipidaemia is to increase serum high density lipoprotein cholesterol (HDL) concentrations.¹ A recent report by Murphy *et al*² described two cases of profound reductions in serum HDL and apolipoprotein A1 concentrations following combination therapy with probucol and a fibrate, ie, bezafibrate and fenofibrate, respectively. The authors postulated an interaction between probucol and the fibrates as the underlying cause of this phenomenon. However, substantial paradoxical reductions in HDL have also been reported in association with fibrate monotherapy (ie, ciprofibrate, bezafibrate), although the pathogenesis is unknown.^{3–5} It is also unclear how soon after commencing monotherapy with the fibrate the fall in HDL occurs or whether the fall is transient or persistent. We report observations which address these questions in a patient who

Figure HDL-cholesterol pattern during treatment with ciprofibrate 100 mg daily

exhibited this unusual response to ciprofibrate.

Case report

A 41-year-old hypertensive woman presented with mixed hyperlipidaemia exacerbated by excessive alcohol intake. Her antihypertensive medication was nifedipine. She had been treated with diet and bezafibrate over a three-year period during which HDL ranged between 1.12 and 1.34 mmol/l. Apo E phenotype was confirmed as E3/3. Due to inadequate response to this therapy, her medication was changed to ciprofibrate 100 mg daily. Two weeks later, total cholesterol had fallen from 9.7 to 8.5 mmol/l but this was accompanied by a precipitate fall in HDL from 1.05 to 0.12 mmol/l. This very low HDL concentration was reflected in an equally low apolipoprotein A1 concentration of 0.12 g/l (reference range: 0.91–1.70). It was decided that ciprofibrate should be continued at the same dose but with fortnightly clinical and biochemical monitoring of the patient to assess whether this effect on HDL was sustained. This therapeutic decision was discussed fully with the patient. Subsequent HDL levels fluctuated between 0.12 and 0.80 mmol/l with four out of seven values being less than 0.20 mmol/l (figure). During this period, the median and range for total cholesterol and triglycerides were 8.5 (7.7–9.7) mmol/l and 4.8 (2.5–9.5) mol/l, respectively. Ciprofibrate was stopped at three months because of lack of improvement in her lipid profile and two weeks later HDL had increased from 0.13 to 0.90 mmol/l. Six weeks after discontinuing the medication, HDL was 0.88 mmol/l (figure). The patient did not report any adverse clinical effects during her treatment with ciprofibrate.

This case demonstrates that substantial anomalous reductions in HDL can occur in susceptible individuals in association with ciprofibrate monotherapy. This phenomenon may become evident as early as two weeks after initiating treatment which is much less than the four to six months reported in

previous cases.^{3,5} Our patient also demonstrated wide variability in HDL concentrations during treatment. Although most of the HDL values were very low, there was a near-normal value of 0.80 mmol/l obtained 10 weeks into treatment. If the HDL level had been assessed only at this time point, the very low values could have been missed. It is important that HDL should be included in the routine monitoring of patients on lipid-lowering medication.

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