used. The case for oral systemic cortico-steroids is as yet unproven. Necrotic tissue should be removed. Pneumonia is com- mon in severe cases. 

In conclusion, TEN is a rare side-effect of indomethacin therapy. Awareness of this association is important and TEN is potentially lethal and indomethacin is widely used in the UK.

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Neuroleptic malignant syndrome with normal creatine kinase

Sir,

Neuroleptic malignant syndrome is an uncommon but life-threatening complication of neuroleptic drug therapy. It was first described in 1968 but a quarter of a century later, doubts still exist concerning the diag- nostic criteria. The syndrome has been char- acterised as the combination of autonomic dysregulation, muscular rigidity, hyperther- mia, confusion, agitation, elevated creatine kinase and leucocytosis. Serum creatine kinase is considered to be the single most important investigation in diagnosing this condition. We report a case of presumed neuroleptic malignant syndrome based on clinical features, but with a normal creatine kinase which responded to dantrolene.

Case report

A 54-year-old man with a four-year history of manic depression was transferred from a psychiatric hospital for emergency medical treatment. He was on lithium 700 mg daily, chlorpromazine 200 mg tid and had received depot haloperidol 200 mg twice weekly. He had been hospitalised for a relapse of his manic depressive illness and seemed to be making good progress. One week after admis- sion, however, he became aggressive and violent over a two-hour period and was noted to be pyrexial.

On examination he appeared dehydrated and was febrile with an axillary temperature of 38°C, there was no apparent focus of infection. He was promptly rehydrated by secre- tions from his mouth but his chest was clear. Muscle tone was increased in all limbs but there were no other neurological signs. Haemoglobin was 10.5 g/dl, white cell count 14.1 x 10⁹/l (80.4% neutrophils) platelets 390 x 10⁹/l. Arterial blood gases showed a PO₂ of 10.3 kPa PCO₂ 4.8 kPa, pH 7.35, O₂ saturation of 96%. Urea and electrolytes were normal and the creatine kinase was 35 IU/l (normal 30–250). In view of his restless and violent behaviour he was immediately sedated with intravenous fluids and intravenous diazepam. He remained pyrexial and there was no improvement in his aggres- sive behaviour. Daily creatine kinase measurements were normal and urine myo- globin was not detected.

By 48 hours he remained pyrexial and disturbed. Having excluded a septic focus neuroleptic malignant syndrome was postu- lated, with a normal creatine kinase. In- tentious sodium dantrolene was given at a dose of 1 mg/kg (six hourly for two days). There was a dramatic response in clinical state within two hours; within four hours his mental state improved and at 24 hours he was fully alert and orientated, and was apyrexial.

We believe this case illustrates that the neuroleptic malignant syndrome is a clinical diagnosis and the absence of an elevated creatine kinase does not exclude the condi- tion. In the management of the confused patient taking neuroleptic medication the clinician must have a high index of suspicion for making this diagnoses.

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Flavobacterium meningosepticum meningitis in an adult with acute leukaemia

Sir,

Flavobacterium meningosepticum is a Gram- negative bacillus, widely distributed in nature being found in water and moist areas. The hospital environment is often contaminated with this organism. It is an opportunistic, causing epidemics in postnatal intensive care units and occasional sporadic cases. It fre- quently causes purulent meningitis in non- hospitalised children. Cases of infection in adults are rare and meningitis remains excep- tional.

Case report

A 27-year-old man living in Niamey (Niger) was admitted to the intensive care unit with fever, pancytopenia, and localised cervical cellulitis. A diagnosis of acute myelogenous leukaemia (AML) of M1 subtype in the French/American/British classification was made. Septicaemia due to Pseudomonas aeruginosa was documented and the patient was treated with piperacillin, netilmicin and metronidazole, administered via an indwell- ing central venous catheter. When afebrile, induction chemotherapy was started with lomustine, doxorubicin and cytarabine. Two days after completion of chemotherapy, fever reappeared and he was empirically treated with vancomycin, amikacin and ceftazidim.

On day 13, meningitis developed and a lum- bar puncture was performed. Analysis showed abnormal cellularity with 160 white blood cells/ml, but normal glucose and protein levels. Three blood cultures and cerebro- spinal fluid grew F meningosepticum. In vitro bacteriological tests showed that rifampicin (1 g intravenously every 12 h), piperacillin (6 g intravenously every 4 h) and ciprofloxacin was the best association. On day 17, aplasia resolved with rapid recovery of neutrophils. On day 19, the patient’s neurological status was normalised. Fever rapidly resolved. Antibiotics were continued for 23 days. Repeated lumbar punctures remained without growth. Complete remis- sion of AML was documented on day 22 after completion of chemotherapy. Early intensification treatment for AML was administered two weeks after antibiotic cessa- tion.

In adults, F meningosepticum is rarely pathogenic and is mainly responsible for nosocomial infections in immunosuppressed patients. Ten cases of F meningosepticum adult meningitis have been described in the literature, in immunodeficient patients (polycythaemia vera, aplastic anaemia, disseminated tuberculosis, renal transplant, aplasia due to chemotherapy for acute lympho- blastoid leukaemia, post-partum), or post- surgical (craniofacial or neurosurgical). One patient had no apparent immune depression and died rapidly. F meningosepticum has a
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 immunocompromised 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 with post-streptococcal meningitis was cured with monotherapy poorly penetrable to cerebrospinal fluid (CSF) or poorly bacteriostatic (aminocillin for 13 days, chemotheravian erythromycin 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 *E. meningosepticum* septicemia and bacterial meningitis in a patient with AML. Our patient could have been an healthy carrier of *E. meningosepticum*, which became highly pathogenic because of persistent neutropenia. The literature is scarce for early introduction 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 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 phalanges of the left thumb and soft tissue swelling. The terminal phalanges 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 susceptible 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 left thumb 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.6,10 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 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.

**Pressor effect of metoclopramide in phaeochromocytoma**

**Sir,**

Metoclopramide is a widely used anti-emetic in hospital practice. Apart from its well known anti-emetic effect 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 were, 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 cafe 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 felt well, her blood pressure rose to 160/120 mmHg. A presumptive diagnosis of phaeochromocytoma was made and the patient was given an alpha-blocker, doxazosin, in addition to the b-blocker, atenolol. Nifedipine was discontinued on admission. Computed tomography (CT) scan of the abdomen revealed a 9 x 8 cm mass in the left adrenal gland. A 24-hour urine collection contained a 380 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 both before and after adrenalectomy in two patients with phaeochromocytoma. The mechanism may relate to a 3,4-dihydroxyphenylalanine inhibitory effect on the adrenomedulla which is blocked by both metoclopramide.