used. The case for oral systemic corticosteroids is as yet unproven. Necrotic tissue should be removed. Pneumonia is common in severe cases.[7]

In conclusion, TEN is a rare side-effect of indomethacin therapy. Awareness of this association is important for two reasons: 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 treatment.[1] It was first described in 1968 but a quarter of a century later, doubts still exist concerning the diagnostic criteria. The syndrome has been characterised as the combination of autonomic dysregulation, muscular rigidity, hyperthermia, confusion, agitation, elevated creatine kinase and leucocytosis.[2] Serum creatine kinase is considered to be the single most important investigation in diagnosing this condition.[3,4] We report a case of presumed neuroleptic malignant syndrome based on clinical features, but with a normal creatine kinase which responded to dantrolene.

Neuroleptic malignant syndrome

Clinical features
- confusion and agitation
- hyperthermia(> 38°C)
- muscular rigidity
- tachycardia
- tachypnea
- autonomic dysregulation

Laboratory findings
- elevated serum creatine kinase
- leucocytosis
- hypoxia
- metabolic acidosis
- myoglobinuria

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, chloropromazine 200 mg tid and had received depot haloperidol 200 mg two weeks earlier. He had been hospitalised for a relapse of his manic depressive illness and seemed to be making good progress. One week after admission, 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 producing copious secretions 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 14.1×10^9/l (80.4% neutrophils) platelets 399×10^9/l. Arterial blood gases showed a PO_2 of 10.3 KPa PCO_2 4.8 KPa, pH 7.35, O_2 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 midazolam. He remained pyrexial and there was no improvement in his aggressive behaviour. Daily creatine kinase measurements were normal and urine myoglobin was not detected.

By 48 hours he remained pyrexial and disturbed. Having excluded a septic focus neuroleptic malignant syndrome was postulated, with a normal creatine kinase. Intravenous 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 maligant syndrome is a clinical diagnosis and the abscence of an elevated creatine kinase does not exclude the condition. 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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Learning points

- neuroleptic malignant syndrome is underdiagnosed and carries a high mortality
- a high index of suspicion is essential
- exclude a septic focus
- stop all neuroleptics
- treat early with rehydration and intravenous dantrolene
- the dopamine agonists, amantadine bromocryptine, carbidopa/levadopa may also be considered, benzodiazepines may help with muscle rigidity

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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 opportunist, causing epidemics in postnatal intensive care units and occasional sporadic cases.[1] It frequently causes purulent meningitis in non-hospitalised children.[2] Cases of infection in adults are rare and meningitis remains exceptional.

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 indwelling 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 lumbar puncture was performed. Analysis showed abnormal cellularity with 160 white blood cells/ml, but normal glucose and protein levels. Three blood cultures and cerebrospinal fluid grew *F meningosepticum*. *In vitro* bactericidal tests showed that rifampicin (1 g intravenously every 12 h), piperacillin (6 g intravenously every 6 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 remission of AML was documented on day 22 after completion of chemotherapy. Early intensification treatment for AML was administered two weeks after antibiotic cessation.

In adults, *F meningosepticum* is rarely pathogenic and is mainly responsible for nosocomial infections in immunosuppressed patients.[1] 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 lymphoblastic leukaemia, post-partum),[3] or post-surgical (craniofacial or neurosurgical).[4] One patient had no apparent immunodepression and died rapidly.[5] *F meningosepticum* has a