Constipation and colonic perforation complicating calcium resonium therapy

Sir,

Constipation is a well recognized complication of treatment with many different medicines. Significant side effects of such constipation, however, are not common. Pseudo-obstruction induced by an amitriptyline overdose has been associated with intestinal perforation. We have treated a patient who developed absolute constipation and colonic perforation complicating the use of calcium resonium therapy.

A 62 year old male was admitted to hospital for treatment of chronic renal and cardiac failure. Drug therapy on admission was nifedipine, colchicine, sulphinpyrazone, probenecid, bumetanide daily, inhaled ipratropium bromide and fenoterol hydrobromide and codyramol for joint pain. He was commenced on captopril to improve his cardiac failure. Unfortunately, his serum potassium increased to 7.1 mmol/l. He was, therefore, commenced on calcium resonium 15 g (increased subsequently to 30 g) thrice daily. The serum potassium fell to 4.9 mmol/l and the patient was discharged from hospital continuing on calcium resonium 15 g thrice daily. Prior to discharge he had complained of constipation. Glycerol suppositories and lactulose 15 ml daily were prescribed as laxatives.

The patient was subsequently admitted with a 48 hour history of absolute constipation. Abdominal X-ray showed the descending colon to be loaded with faecal material. The constipation was presumed to be secondary to calcium resonium therapy. Conservative treatment was unsuccessful. At laparotomy he was found to have perforation of the sigmoid colon. A Hartmann’s procedure was performed with removal of 2 kg of inspissated faeces including calcium resonium. Despite further treatment the patient subsequently died.

Faecal impaction of calcium resonium has previously been reported in children following rectal administration of the drug and gastric concretions have been reported in neonates after oral administration. Constipation may also occur and normally responds to treatment with aperients. This case, however, reflects the possible severity of such constipation although to the knowledge of the manufacturer a similar case has not been reported.

There is no defined limit of calcium resonium dose that may be used. In this patient the hyperkalaemia was resistant to the starting dose necessitating a dose increase.

There are two possible contributing factors to this patient developing colonic obstruction. The patient had a normal bowel habit despite the use of drugs including codyramol which are associated with constipation. It is possible, however, that the addition of calcium resonium to these drugs increased the risk of developing large bowel obstruction. The patient had also been recommended to limit the intake of oral fluids to about 1.3 litres daily in view of his cardiac and chronic renal failure. Such a fluid restriction may have limited his ability to clear the resonium from the bowel.

It is suggested, therefore, that therapy with calcium resonium should be monitored so that possible interactions with other constipating agents can be avoided. In addition, adequate patient fluid intake and limitation of dosage should be ensured.

References


Rapid pulmonary cavitation due to Mycobacterium tuberculosis and infection with human immunodeficiency virus (HIV 1)

Sir,

Infection with Mycobacterium tuberculosis is increasingly recognized among patients infected with the human immunodeficiency virus (HIV). Cavitation of pulmonary lesions in patients with tuberculosis (TB) and HIV is considered to be rare but we have recently observed a rapidly expanding pulmonary cavity in a patient with HIV infection and pulmonary TB.

A 29 year old Ugandan businessman was admitted for investigation of a persistent cough, weight loss and diarrhoea. A diagnosis of pulmonary TB had been made 9 months previously in Uganda and he was treated for 3 months with streptomycin and thiacetazone. Six months later he arrived in England and was investigated at another hospital for cough, weight loss and a pruritic rash. His chest X-ray showed diffuse interstitial shadowing in both mid and upper zones. An antibody test for HIV 1 was positive. Bronchoalveolar lavage showed no organisms other than Haemophilus influenzae. He received oral cotrimoxazole for 2 weeks and because of possible recurrence of TB he received rifampicin, isoniazid and pyrazinamide. Culture of lavage fluid was subsequently sterile and his antituberculous therapy was discontinued after 3 months.

On admission to this hospital he was complaining of a productive cough, diarrhoea and malaise. He was a thin man with no lymphadenopathy and a rash due to chronic tinea corporis. He was pyrexial with scattered expiratory wheezes throughout the chest. Investigations showed a haemoglobin of 9.8 g/dl, total white cell count 9.46 × 10⁹/l, CD4 count 800 × 10⁹/l, and platelets 113/ml. Urea, electrolytes and liver function tests were normal apart from an albumin of 28 g/dl. Blood cultures, thick films for malarial parasites and stool samples for ova, cysts, parasites and other pathogens were negative. Chest X-ray
showed dense consolidation in the left upper lobe. Sputum samples showed numerous acid-fast rods on smear. He was started on rifampicin, isoniazid, pyrazinamide and streptomycin. Seven days later his chest X-ray showed a left upper lobe cavity 3 cm in diameter containing a dense central opacity. This enlarged to a maximum size of 4 cm at 10 days and subsequently resolved over 3 months. Because of the rapid appearance of cavitation, a bronchoscopy was performed which showed purulent secretions draining from the left upper lobe with underlying erythematous bronchial mucosa. Bronchoalveolar lavage showed numerous acid-fast bacilli, no *P. carinii*, fungi or neoplastic cells. Cultures grew no pathogens other than fully sensitive *M. tuberculosis*.

The radiological pattern of pulmonary tuberculosis is often atypical in AIDS patients. Hilar or mediastinal lymphadenopathy and localized pulmonary infiltrates in the middle or lower lung fields are the most common features, while pulmonary cavitation is infrequent. Cavitation may occur due to *P. carinii* and can mimic TB with the rapid appearance of single or multiple cavities. Although Klebsiella sp., Nocardia, Cryptococcus neoformans, and *Mycobacterium avium-intracellulare* can also cause pulmonary cavitation in HIV patients, *M. tuberculosis* alone can cause very rapidly progressive pulmonary cavitation in patients with HIV infection.

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References

Pseudogout associated with thyrotoxicosis

Sir,

Pseudogout is a frequent cause of arthritis in the elderly and is characterized by calcium pyrophosphate dihydrate (CPPD) crystals in the joint fluid and chondrocalcinosis. We describe a case of acute pseudogout associated with thyrotoxicosis.

An 89 year old woman was admitted with abdominal pain and vomiting of 4 days duration. She had lost 7 kg in weight over 1 year and was in atrial fibrillation. There were no other clinical features of thyrotoxicosis. The abdomen was distended and the rectum loaded with faeces. A diagnosis of faecal impaction was made. She responded to treatment with enemas and laxatives, but 3 days later she developed a swollen tender hot right knee and a temperature of 37.8°C. A total of 60 ml of cloudy fluid was aspirated and she responded to treatment with non-steroidal drugs. The synovial fluid aspirate contained large numbers of calcium pyrophosphate crystals. An X-ray of the right knee showed chondrocalcinosis. The serum calcium level was normal. Thyrotoxicosis was confirmed biochemically and she was then referred on for radioactive iodine treatment.

The association of pseudogout with hypothyroidism is well recognized. Alexander et al. found 10% of their CPPD study patients to be hypothyroid. However, there is no recorded case of pseudogout associated with thyrotoxicosis. Hypothyroid patients with previously asymptomatic CPPD crystal deposition often have a flare-up of arthritis when thyroid medication is started. Thyroxine thus probably has some role in precipitating acute crystal deposition, the mechanism being unknown. Hence the association of thyrotoxicosis in our patient with pseudogout may be more than casual.

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References

Morphine and severe dryness of the lips

Sir,

Dryness of the mouth is not generally recognized as a side effect of morphine, but clinical experience suggests that it is a common complaint when used in patients with cancer. The mechanism for this effect is unclear. We report a case of dryness of the lips accompanied by severe pain after intravenous administration of morphine, and suggest possible pathophysiological mechanisms.

A 60 year old man presented with acute myocardial infarction with no complications. The patient received 5 mg morphine intravenously for the chest pain. No other drugs were given. A few minutes later, gradual relief of his chest pain began accompanied by a complaint of progressive dryness of the lips which shortly became very dry and