an abnormal TFT that has not been acted upon within a specified time.

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Cytomegalovirus colitis in an elderly patient

Sir,

We report a patient with severe cytomegalovirus (CMV) colitis, in association with *Clostridium difficile* infection.

An 83 year old previously fit woman was admitted with collapse. She was confused and chest X-ray confirmed left basal pneumonia. She was treated with intravenous ampicillin but next day she became pyrexial, with vague abdominal pain and was noted to have diarrhoea. Sigmoidoscopy revealed inflammatory changes up to 15 cm. Stool culture showed a significant growth of *Clostridium difficile*. Oral vancomycin rendered her stool culture negative but the diarrhoea continued sometimes mixed with blood. Barium enema showed some irregularity in the sigmoid colon confirmed to be gross sigmoid colitis by colonoscopy. She was treated with prednisolone and olsalazine which were stopped when the biopsies reported a large number of cytomegalovirus-infected cells with inclusion bodies. Treatment with intravenous ganciclovir was commenced. Human immunodeficiency virus (HIV) test was negative, serum electrophoresis excluded gammopathy and CMV IgM titres were significantly raised. She failed to improve and subsequently died. Postmortem examination revealed no evidence of ulcerative or ischaemic colitis, no CMV inclusion bodies were seen in the colonic mucosa which was denuded and inflamed and her lungs were severely bronchopneumonic.

Although detecting CMV inclusions by cytologic or histologic methods is diagnostic. It is not considered a sensitive means of diagnosis.¹ It reflects activity of infection and could be a useful marker to guide antiviral therapy.²

There is a high prevalence of CMV IgM antibodies among elderly patients despite the lack of obvious evidence of clinical sign or viral shedding. This might be attributed to the progressive decline of the immune system in old age; however the exact reasons are still unknown.³

CMV colitis is a recognised complication in severe immunodeficiency states but it is also reported to occur as an isolated phenomenon. The associations with ulcerative colitis and colonic adenocarcinoma have been reported, but it remains unclear as to how significant these associations are.⁴ In ulcerative colitis the susceptibility to CMV infection may be attributed to either steroid treatment or the growth of readily infected granulation tissue as a result of tissue damage.⁴ In our case CMV colonic biopsies were taken before starting steroid therapy. It is possible that the tissue damage associated with the *Clostridium difficile* colitis increased the patient's susceptibility to CMV colitis as ulcerative colitis may do. This hypothesis requires further studies for confirmation.

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Monozygotic twins concordant for both open-angle glaucoma and bronchospasm induced by beta-blockers

Sir,

A female monozygotic twin pair, born in 1914, is reported which demonstrates concordance for both open-angle glaucoma and bronchospasm precipitated by beta-blockers. The monozygosity of the twin pair was inferred by their morphological identity and homologous blood group antigens. There was no personal or family history of bronchospasm, atopy, or glaucoma; neither twin was a tobacco smoker.

Primary open-angle glaucoma was diagnosed in one twin at the age of 67 years and treated with pilocarpine eyedrops. Propranolol was subsequently prescribed for angina pectoris and bronchospasm developed which resolved upon withdrawal of the propranolol. Ten years later, an incidental diagnosis of advanced open-angle glaucoma was made in her asymptomatic co-twin. Treatment was commenced with betaxolol eyedrops but after 6 weeks, she developed bronchospasm. The betaxolol eye-

drops were replaced and her bronchospasm remained quiescent, with no bronchodilators being required.

There are few reports of adult twin pairs with glaucoma and most of these are incomplete with regard to either zygosity or the type of glaucoma. However, a Finnish population-based twin study has found three out of 27 monozygotic and three out of 79 dizygotic twin pairs to be concordant for open-angle glaucoma,¹ indicating the role of genetic factors in open-angle glaucoma. This is the rationale for screening first-degree relatives of patients with open-angle glaucoma² but the probability of a monozygotic co-twin developing glaucoma will be even greater. Unfortunately the co-twin in this report was not screened and presented independently. She would have been detected much earlier with less advanced disease had screening been instigated.

Bronchospasm precipitated by beta-blockers is attributed to pharmacological antagonism at the beta-2 adrenoreceptors in the bronchial smooth muscle. Both non-selective and cardio-selective beta-blockers can precipitate bronchospasm which can occur both in patients with and without a personal or family history of asthma.³ Bronchospasm can occur both following the administration of systemic beta-blockers and secondary to topical ocular beta-blockers, the small amounts of which (0.5 mg) are absorbed into the systemic circulation through the conjunctiva and nasolacrimal mucosa thereby avoiding first-pass metabolism in the liver to produce profound systemic effects.⁴ Thus bronchospasm can be produced by any beta-blocker in any subject.

This is the first known report of bronchospasm being precipitated by beta-blockers in both members of a monozygotic twin pair who also had glaucoma. The clinician should be aware of the possibility of similar diseases and adverse drug reactions occurring in monozygotic twins.

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Cauda equina syndrome associated with ankylosing spondylitis in a female

Sir,

A 61 year old woman had ankylosing spondylitis (AS) since the age of 23. At 43 years she had a recurrent effusion of the right knee and this was treated with synovectomy and patellectomy. At 57 years she had a left

total hip replacement and at that time the right knee was noted to be stiff in extension.

She was admitted for investigation with a two month history of urinary incontinence, loss of sensation in the perianal area, some faecal incontinence and altered sensation on defaecation. On examination there was evidence of severe AS. On neurological examination, power and tone were normal. Reflexes were absent in the right lower limb but plantars were bilaterally flexor. Saddle anaesthesia was found and anal tone was diminished. A diagnosis of a cauda equina lesion was made. A myelogram was unsuccessful. A computed tomographic (CT) scan of the lumbosacral region showed no evidence of a space occupying lesion or disc prolapse. No further treatment could be offered.

Cauda equina syndrome associated with AS is rare and was first described by Bowie and Glasgow¹ who documented three patients who developed cauda equina lesions many years after the active disease had settled. In a review by Bartleson *et al.*,² most patients' AS was asymptomatic for years before neurological complaints began. Sphincter disturbance and cutaneous sensory loss were the most frequent manifestations. All patients were able to maintain ambulation much as they had before the onset of their cauda equina syndrome, and sensory and sphincter function was disturbed more than motor function. Painless neurotrophic skin ulcers secondary to the sensory loss have been described.^{1,3} The progress of the syndrome is slow and the neurological defects are symmetrical. Post-irradiation sarcoma may mimic this syndrome, in those patients previously treated for their AS by radiotherapy.

A variety of treatments have been tried, including high dose steroids and laminectomy, but none has been shown to be effective. Surgical exploration should be avoided, as it usually results in transient worsening of symptoms with no proven benefit.^{4,5} In the review by Bartleson *et al.*,² seven of the 12 men had a notable history of prior prostate surgery with little or no benefit from the procedure and often resulting in increased urinary incontinence which may be the first presentation of this syndrome. The presentation is slowly progressive with symmetrical neurological defects developing long after the rheumatological symptoms have diminished. CT scanning or magnetic resonance imaging is the preferred method of investigation and fails to demonstrate a space occupying lesion.

What is particularly interesting about this case is that the patient was female. As far as we are aware, only two previous reported cases have occurred in females.² There is no effective treatment but awareness of this complication will prevent needless invasive investigation and the earlier institution of supportive care.

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