Letters to the Editor

Recovery of adrenocortical function following treatment of tuberculous Addison's disease

Sir,

Penrice and Nussey report two cases of Addison's disease in which adrenocortical function recovered following treatment of tuberculosis.¹ Adrenal glands may be enlarged or atrophic in patients with Addison's disease due to tuberculosis. Large glands in tuberculosis mean a recent and probably active infection requiring treatment, whereas small calcified glands are in favour of remote and probably inactive infection.² Renner *et al.* reported two cases of adrenal tuberculosis in which adrenal glands had been enlarged and they concluded that computerized tomography (CT) is the method of choice for the noninvasive diagnosis of tuberculous adrenalopathy.³ In another report, the finding of enlarged adrenal glands on CT suggested the presence of early tuberculosis or in rare instances other potentially treatable diseases.⁴

Barnes *et al.* investigated adrenal function in 90 patients with active tuberculosis (30 pulmonary, 30 miliary, 30 extrapulmonary) before and after starting anti-tuberculous therapy. They found some degree of adrenal dysfunction in seven patients. After the treatment, the Synacthen response returned to normal in all but one patient, and they concluded that adrenal dysfunction is an uncommon problem in patients with active tuberculosis and anti-tuberculous therapy has a favourable effect on adrenal function.⁵

Recently we have reported a patient with active pulmonary tuberculosis, acute adrenal failure and an enlarged adrenal mass demonstrated by CT.⁶ The mass was removed surgically and histopathologic examination disclosed adrenal tuberculoma. We have also shown that adrenal glands are larger in acute pulmonary tuberculosis than that in chronic tuberculosis and also healthy subjects.⁷ CT scanning was not carried out in the patients reported by Penrice and Nussey. Since the symptoms and signs of Addison's disease appear after more than 90% of the glands have been destroyed by tuberculosis,⁸ we think that recovery of adrenal insufficiency is not possible in patients with Addison's disease due to remote tuberculosis in which adrenal glands are atrophic and calcified. In contrast, recovery of adrenocortical function after anti-tuberculous therapy in reported patients with tuberculosis is an expected outcome.^{2,9}

We suggest that CT of adrenal glands should be carried out in every patient presenting with Addison's disease thought to be caused by tuberculosis. If there is adrenal atrophy anti-tuberculous therapy may not be required. If adrenal glands are enlarged, anti-tuberculous therapy may be needed.

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Guillain–Barré syndrome in a patient with chronic lymphocytic leukaemia

Sir,

Guillain–Barré syndrome (GBS) has been frequently reported in association with malignant disorders, especially those of the lymphoid system, for example, Hodgkin's disease.¹ However, despite being the commonest lymphoid malignancy, chronic lymphocytic leukaemia has been associated with GBS in only three reported patients.^{2,3} I describe a fourth case.

A 77 year old woman with known stage III B-cell chronic lymphocytic leukaemia (CLL) presented with a 5-day history of progressive symmetrical limb weakness, distal limb paraesthesiae and breathlessness. There was no history of recent viral infection. CLL had been diagnosed 3 months before and treated with chlorambucil as well as a blood transfusion. Chlorambucil was stopped 8 weeks later because of myelosuppression. Examination revealed a frail, breathless, afebrile lady with moderate hepatosplenomegaly. The cranial nerves were normal. She had a flaccid quadriparesis with generally MRC grade 2 power, absent tendon reflexes and flexor plantar responses. There was impaired appreciation of vibration and proprioception below the ankles.

Investigation revealed a reduced forced vital capacity of 1.2 litres. Routine biochemistry was normal. Her haemoglobin was 11.5 g/dl and her lymphocyte count was $99.9 \times 10^9/l$. A direct Coombs' test was negative. Paired