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. 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. 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 tuberculosis. 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.

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.

Recently we have reported a patient with active pulmonary tuberculosis, acute adrenal failure and an enlarged adrenal mass demonstrated by CT. The mass was removed surgically and histopathologic examination disclosed adrenal tuberculosis. We have also shown that adrenal glands are larger in acute pulmonary tuberculosis than that in chronic tuberculosis and also healthy subjects. 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, 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.

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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References


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. However, despite being the commonest lymphoid malignancy, chronic lymphocytic leukaemia has been associated with GBS in only three reported patients. 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 paresthesiae 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 x 10^9/l. A direct Coombs’ test was negative. Paired
serology showed no evidence of recent viral or mycoplasma
tissue. Serum IgA was slightly reduced at 1.17 g/l (normal 1.25–4.25 g/l) and the IgM was 0.47 g/l (normal 0.5–1.75 g/l), with a normal IgG. A chest X-ray was normal. Spinal fluid examination showed a protein of 0.7 g/l (normal up to 0.45 g/l), glucose 4.8 mmol/l, with no white cells, and no growth on culture. Nerve conduction studies showed motor nerve conduction of the median nerve was 30 m/second with a distal motor latency of 4.7 milliseconds (normal, up to 4.2 milliseconds). F responses were unobtainable. Distal sensory nerve conduction velocity of median nerve was 29 m/second, the sensory action potential being reduced at 1.5 μV. The extensor digitorum muscle was denervated and motor conduction along the peroneal nerve could not be obtained. Motor latency from the capitulum fibulae to the tibialis anterior was slightly prolonged, but the M wave was markedly decreased (0.1 mV).

She received a 5-day course of intravenous immunoglobulin (Venoglobulin, Alpha Pharmaceuticals) at 0.4 g/kg/day, and steadily improved. After 3 weeks she was able to walk unaided.

Our patient fulfills the well-established diagnostic criteria for GBS. GBS is an acute inflammatory demyelinating polyradiculoneuropathy in which both protein and lipid antigens in peripheral nerve are the target of immune attack. Neither the precise antigens nor the relative roles of humoral or cell-mediated immune responses are known. A prominent role of the humoral immune system in the pathogenesis of GBS is supported both by the presence during the acute phase of the illness of complement-fixing anti-peripheral nerve myelin IgM antibodies whose kinetics correlate with the clinical course of the disease, and by the efficacy of treatment with high-dose intravenous immunoglobulin.

Under certain circumstances partial or transient immunosuppression could serve as a contributing factor in precipitating GBS. Impaired cell-mediated immunity is the proposed mechanism for the increased incidence of GBS in Hodgkin's disease. Indeed, Hughes et al. reported reduced suppressor T-cell function in about a quarter of patients during the early phase of GBS.

In CLL, in contrast with Hodgkin's disease, almost all patients eventually develop hypogammaglobulinaemia while the cell-mediated immune system remains largely intact. This may explain why the association between CLL and GBS is rare. In our patient it is possible that the transient myelosuppression induced by the chlorambucil may have predisposed to the development of GBS, whilst the almost normal humoral response may have mediated its subsequent development.

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References


Anti-epileptic therapy and fatal chickenpox

Sir,
A 15 year old epileptic boy on carbamazepine, primidone, sodium valproate, acetazolamide and clobazam presented with severe backache following a grand mal seizure. An X-ray revealed no abnormality and he was sent home on ibuprofen. Two days later he re-attended with fever, anorexia, vomiting and a rash which had appeared the previous night. He had recently been exposed to chickenpox. On examination his temperature was 37.8°C and he had a florid papular, vesicular and purpuric rash involving his face, trunk and proximal limbs. Ecchymoses were present at a venepuncture site. His pulse was 120/minute, blood pressure 165/110 mmHg, the abdomen was distended and tender, and the urine and vomitus contained blood. Investigations showed: haemoglobin 16.3 g/dl, white cell count 17.0 x 10^9/L, platelets 49 x 10^9/L, international normalized ratio > 8 (normal 0.8 – 1.2), activated partial thromboplastin time ratio 3.4 (normal 0.8 – 1.2), fibrin degradation products (D-dimer) 32.0 mg/l (normal <0.5 mg/l) and glucose 6.7 mmol/l. Disseminated intravascular coagulation (DIC) precipitated by a drug, meningococcal septicaemia or chickenpox was considered. Despite infusions of fresh frozen plasma, platelets and cryoprecipitate together with intravenous acyclovir, benzylpenicillin and chloramphenicol, he died within 24 hours. Varicella zoster virus was isolated from vesicular fluid.

An explanation for the fulminating course was not apparent. He was, however, on a number of drugs and the possibility that the DIC was due in part to a drug effect cannot be excluded. In this regard it is relevant that carbamazepine and acetazolamide can induce autoimmune thrombocytopenia, and sodium valproate can cause various disorders including abnormal bleeding times, impaired clotting, platelet antibodies, and low fibrinogen levels. Fatal DIC has been reported in one neonatal patient on sodium valproate, but it was not clear whether causes other than the drug could have been responsible for the DIC (personal communication from Sanofi Winthrop).
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