fection syndrome has a poor prognosis, with a 50% mortality rate. The nervous system is seldom involved in the syndrome. If such involvement appears, it usually does so towards the end of the course of the disease. A patient with sensorimotor neuropathy related to anoxapony and occasional demyelination has been recently reported, but in the context of a fulminant illness.1

The distinctive feature of our finding is the occurrence of transient cranial nerve involvement as the probable first sign of macrophage activation syndrome. It could be claimed that the symptomatology is related to the lymphoma. However, very little is known about neurological complications in T cell lymphoma, and their occurrence is probably rare.2 Kaufman et al3 have reported an involvement of the nervous system in 14 patients out of 104 cases, eight being related to direct complications. In only one patient, palsy of the sixth cranial nerve was the first sign. Neurological signs occurred between 10 and 102 weeks after diagnosis of lymphoma. If polyneuropathy occurs in T cell lymphoma it is due to infiltration and the clinical evolution is usually stereotyped with slowly evolving sensorimotor signs.4 Because the process of infiltration of peripheral nerves cannot be eliminated; but it is unlikely, considering the improvement in neurological signs. Meningoradiculitis could be evoked, but if that were so, there would have been a worsening of the initial signs.4 Moreover, CSF examination and cerebral MRI were normal. All these indications lead us to suggest that the neurological signs in our patient could be related to a remitting/relapsing neuropathy due to non-curable T cell lymphoma infiltrating peripheral nerves, to vasculitis or, more likely, to the neurotoxic effects of cytokines. Cytokines, especially TNF, are secreted in large amounts in macrophage activation syndrome, and TNF can induce general side effects and cerebral damage.5

We are grateful to L Rose for her help with the English version of this paper.

**Figure 2.** Lymphoid cells with azurophil granules. Myelogram (originally × 100).

**Paraneoplastic opsonus associated with cancer of the gall bladder**

Opsonus is an ocular dyskinesia consisting in simple, conjugated, arrhythmic, multidirectional ocular movements which persist even with the eyes closed.1 This syndrome has been described during the course of different cancers.2 In infancy, neuroblastoma is the cancer most often associated with opsonus (in 2% to 7% of cases).3 In adults, opsonus is less common. Nevertheless, it is associated with a tumour in 20% of cases.4

Here, we report a case of opsonus associated with a cancer of the gall bladder.

A 72 year old, treated hypertensive woman, experienced the sudden onset of vertigo followed by impaired consciousness. At initial examination her Glasgow score was 13. She showed opsonus associated with a bilateral kinetic cerebellar syndrome. The cranial nerves were intact and there was no sensory or motor deficit. Complete physical examination only showed conjunctival icterus.

Brain MRI showed a left frontal angiomata measuring 7 mm in diameter without any impingement on cerebral parenchyma; the brain stem was normal. Two spinal taps were normal. A chest radiograph was normal. Laboratory studies showed an increase in alanine aminotransferase (43 IU/L), y-glutamyl transpeptidase (160 IU/L) and CA 19-9 (111 kU/L). Abdominal ultrasound showed a heterogeneous, polypoid tumoral structure within the gall bladder associated with hyperchogenic lesions in the liver and a thrombosis of the portal vein. Abdominal CT disclosed thickening of the left lateral wall of the gall bladder, liver metastases, and hilar adenopathy. Liver biopsy showed a fibroelastic, necrotic, disorganised medullated adenosarcoma most suggestive of a paraneoplastic origin.6

Tests for anti-Hu, anti-Ri, and anti-Yo antibodies were negative. Immunoglobulin IV (0.4 g/kg/day) and cortisone (Solumedrol, 0.5 g/day for five days) was ineffective. The patient died five weeks later. No necropsy was performed.

The diagnosis of opsonus remains clinical. It usually has an abrupt onset.7 It is probably the result of a diencephalic or mesencephalic lesion with production of the abnormal movement by removal of normal saccadic generator inhibition.8 Dysfunction of the pause neurons, which play a part in inhibiting the phasic neurons responsible for the appearance of jerks, is likely. The frontal angiomata does not explain the opsonus. The opsonus was considered paraneoplastic because it was not associated with an infectious or tumoral cerebral syndrome. Other possible causes (toxic, metabolic, degenerative,1 and vascular)9 were excluded. The normal MRI, lumbar punctures, and the absence of anti-Ri antibodies, which have been associated with paraneoplastic opsonus occurring with carcinomas of the breast,10 did not cast doubt on the diagnosis. Breast cancer and small cell cancer of the lung represent 70% of reported cases associated with opsonus in adults11 and are sometimes discovered during necropsy.4 In the present case, the histological differentiation seen during liver biopsy was strongly suggestive of a primary lesion in the gall bladder. Therefore, it is not likely that the lesions discovered were metastases from one of the above cited cancer localisations. The possibility of this association means that the gall bladder should be included in the investigation of a paraneoplastic opsonus.

**Lethal hyperoral behaviour from the Klüver-Bucy syndrome**

Clinicians have not sufficiently appreciated the danger of hyperoral behaviour in neurological disorders. A major clinical syndrome is the Klüver-Bucy syndrome.1 Originally described in monkeys after anterior bitemporal lobectomies, this syndrome includes indiscriminate dietary behaviour and a tendency to examine objects by mouth.2 The complete syndrome also results in placidity, hypersexuality, hypermorphemor Johns and therapeutic options.3

Paraneoplastic opsoclonus associated with cancer of the gall bladder.

P Corcia, B De Toffol, C Hommet, D Saudeau and A Autret

*J Neurol Neurosurg Psychiatry* 1997 62: 293
doi: 10.1136/jnnp.62.3.293