

followed by administration of prednisolone (50 mg/day) and melphalan (6 mg for 10 days) each month with monthly intravenous injections of cyclophosphamide (300 mg). Thereafter, the patient showed gradual improvement in motor, respiratory, and renal functions (figure C; table). Four months after treatment, the tissue pressure of the quadriceps femoris in the supine position fell to 47 mm Hg. Serum IgA concentrations were consistently less than 200 mg/dl. There were no serious side effects of DMSO and an unpleasant breath odour was the patient's main concern. Nine months after treatment, we noted a levelling off or a slight decline in some variables. Sixteen months after treatment, the patient aspirated his secretions and died.

AL-amyloidosis results from conversion by proteolysis of monoclonal light chains into twisted β -pleated sheet fibrils,⁴ which can be recognised by Congo red staining. Light chain deposition disease is another pathological state associated with plasma cell dyscrasia.⁵ In our patient with IgA λ plasma cell dyscrasia and skeletal muscle pseudohypertrophy, simultaneous deposition of AL-amyloid and λ light chains⁵ were shown by amyloid staining, immunohistochemistry, and electron microscopy.

Precise mechanisms for physical disability in amyloid associated muscle pseudohypertrophy remain unclear. Most attention to date has been directed toward the weakness.¹ A possible factor causing motor impairment in our patient was a decreased range of motion, predominantly affecting proximal joints. Involvement of shoulder joints showing "shoulder pad sign" is pathognomonic of amyloidosis.⁴ Another factor hampering mobility is increased muscle tissue pressure reflected as wooden firmness. Increased muscle pressure is not produced by amyloid infiltration alone¹ but may be related to deposition of chondroitin-4-sulphuric acid and silicon in muscles,² tense muscle fascia secondary to amyloid deposition, or impaired tissue perfusion by amyloid angiopathy. The pressure is further increased by muscle activity to the point that it interferes with muscle blood flow.

The goal of treatment in amyloidosis is to prevent further deposition of amyloid and to promote its resorption. In our patient, plasmapheresis and DMSO treatment resulted in an appreciable level of improvement in motor, respiratory, and renal functions. The ability of DMSO to make amyloid fibrils soluble for digestion has been demonstrated.⁴ Amyloid/light chain-derived materials dislodged from various organs are likely to impair renal function. Therefore, to remove these breakdown products and the precursor monoclonal immunoglobulins, plasmapheresis was combined with DMSO. Because of the grave prognosis and disabling symptoms of amyloid associated muscle pseudohypertrophy, a trial of plasmapheresis and DMSO may be warranted even though the improvement may be moderate and of limited duration.

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Opsoclonus, a rare complication of cocaine misuse

Opsoclonus is a rare eye movement disorder, mostly seen in postviral encephalopathy or occult neuroblastoma in children, or as a paraneoplastic phenomenon in adults. It rarely occurs after giving drugs or toxins. A single report of the opsoclonus-myoclonus syndrome in association with cocaine use has been described in this *Journal*.¹ We present a patient with opsoclonus, myoclonus, and ataxia after taking cocaine.

A 29 year old man was admitted to hospital with vertigo, nausea, and vomiting. He was unable to stand and walk, because his legs were shaking. The first symptoms had occurred 18 days earlier, after taking cocaine, with paroxysmal vertigo which became continuous the next day, then progressive shaking of the legs, and finally of the whole body. The patient did not complain of headache. There was no weight loss, fever, or recent infectious disease. There was a medical history of migraine and hyperventilation. He admitted heroin misuse until eight years ago and incidental cocaine misuse in recent years. The patient took 10 mg diazepam daily because of nervousness but no other drugs. Since the appearance of nausea he used 6.5 mg thietilperazine a day.

General examination showed no abnormalities. Neurological examination showed normal consciousness and there was no evidence of nuchal rigidity. The optic fundi could not be examined because of intermittent involuntary eye movements. The pupil reactions were normal, as were the visual fields. There were continuously intermittent conjugated nystagmoid beats in all directions, often finishing with a circumduction movement. The abnormal eye movements increased under the influence of stress. The patient had a trembling voice and slight myoclonic jerks of his head and neck. He was unable to stand and walk because of vertigo and ataxia. When sitting he showed a disequilibrium. There was no other neurological deficit.

Blood and CSF examination were normal except for a slightly raised CSF protein (0.72 g/l). Viral serologies were negative. Electrocardiography, chest radiography, brain CT and MRI, EEG, and brainstem auditory evoked potentials were normal. Electro-oculography at fixation in different directions showed crescentiform eye movements with a short rotation at the end of the movement. With the eyes closed there were

coarse eye movements in all directions with a frequency of 8 Hz. The abnormal eye movements were seen in superposition of normal eye movements.

During his stay in hospital the disequilibrium gradually improved. The opsoclonus changed to flutter-like oscillations. After a few weeks oculomotor examination showed only sporadic horizontal ocular myoclonus in vertical movements. Follow up four months after his admission to the hospital yielded no oculomotor abnormalities or ataxia and the patient stated that he felt perfectly well.

In our patient opsoclonus was very likely associated with taking cocaine. After extensive diagnostic evaluation no other cause could be found. The disorder appeared after incidental misuse of cocaine and was self limiting. One other such patient was described by Scharf.¹

Various neurological complications of cocaine are known. Neurovascular disorders, either haemorrhagic or ischaemic, can occur after taking the drug.² Seizures and migraine are other neurological complications. Interestingly, increases in brain serotonin by inhibition of its uptake is an effect of cocaine. Maybe our patient, who had migraine, was more sensitive to this effect of cocaine, as serotonergic dysfunction has been reported in patients with migraine. The lack of any anatomical substrate supports this. On the other hand, a direct toxic effect of cocaine or one of its accompanying substances cannot be ruled out.

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Further evidence of increased risk of mortality from Parkinson's disease

It is often considered that since the introduction of levodopa treatment, there has been little difference in mortality from Parkinson's disease compared with the general population.¹ However, to date, only three studies have investigated the mortality in a group of patients with Parkinson's disease compared with a matched control group. Rajput *et al*, in their review of case notes, found a mortality for patients with Parkinson's disease 1.6 times that of controls,² and the community based prospective case-control survey of Ebmeier *et al* in Aberdeen found a 2.35-fold higher death rate.³ In a recent issue of this *Journal*, Ben-Shlomo and Marmot published the results of a long term community based prospective survey showing a 2.6-fold increased risk of mortality for Parkinson's disease.⁴ We report the results of a prospective population based survey of subjects aged 65 and over that provides further evidence of increased mortality due to Parkinson's disease.

The population studied was a representative, randomly selected sample of 2792 subjects, aged 65 and over, living at home in Gironde, France (PAQUID study), composed of 1122 men (40.2%) and 1670