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Pseudotumour cerebri with amiodarone

Sir: Amiodarone is a relatively safe anti-arrhythmic agent, but is also has some extracardiac side effects, which may involve the cornea, the skin, the thyroid, the lungs, the gut and the nervous system. The neurological side effects include tremor, sleep disturbances, headaches¹ and, less commonly, peripheral neuropathy,² proximal weakness and cerebellar dysfunction.³ We report a case of pseudotumour cerebri (PTC) as another possible side effect of amiodarone.

A 58-year-old man had been treated for 6 months with amiodarone (400 mg/day) for supraventricular arrhythmias on exercise. He had also received pindolol (5 mg/day), allopurinol (100 mg/day) and clofibrate (500 mg/day) for several months prior to

amiodarone. He was referred to us after he developed acute blurring of vision in the right eye. The general examination was normal except for a moderate obesity (81 kg, height 162 cm). The blood pressure was 180/90 mm Hg and the ECG was normal. The neurological examination showed bilateral papilloedema, which predominated in the right eye, with normal visual acuity (1.0 in both eyes). A partial inferior field defect was present in the right eye. The CSF pressure was 300 mm H₂O with normal protein (305 mg/l), leucocytes (2.8.10⁶/l) and glucose (5.0 mmol/l). A brain CT scan, including a survey of the pituitary and orbital regions, was entirely normal. Standard blood and urine tests were normal. A diagnosis of pseudotumour cerebri was made. Acetazolamide (3 × 250 mg/day) was administered for 5 months and prednisone (60 mg/day) for 1 month, but both were discontinued in the absence of improvement. The CSF pressure also remained elevated (range 270 to 300 mm H₂O) on follow up 2, 5 and 12 months later. At 12 months, neurological examination still showed bilateral papilloedema with developing disc atrophy. Visual acuity was 1.25 (OD) and 0.8 (OS). Corneal deposits typical of amiodarone were present. Serum levels of amiodarone were determined by HPLC and were within the optimal range,⁴ that is 1.5 mg/l for the parent drug (n = 1.93 ± 0.80) and 0.9 mg/l for its major metabolite, desethylamiodarone. Because of the possible side effects, amiodarone as well as any other medication were withdrawn. Sequential spinal taps after 4 and 7 days showed a decrease in CSF pressure (180 and 80 mm H₂O). During the following months the visual acuity improved (1.5 (OD) and 1.0 (OS)) with disappearance of the papilloedema. Some degree of optic disc atrophy persisted bilaterally. A partial field defect also remained in the inferior nasal quadrant of the right eye.

This observation suggests that pseudotumour cerebri was induced by amiodarone because it developed shortly after amiodarone was administered and resolved after the drug was discontinued. The other drugs had been administered previously for a much longer time. The role of amiodarone in the pathogenesis of pseudotumour cerebri is also suggested by the fact that the toxicity of this drug is similar to that of perhexiline, which indeed may produce pseudotumour cerebri.^{5,6} The same types of keratopathy^{1,5} and peripheral neuropathy with lysosomal inclusions^{2,7} have also been reported as side effects of both drugs. This is possibly due to the fact that these drugs are amphiphilic.⁸ A

rise in venous pressure or an impairment in CSF outflow have been proposed to explain the elevation of intracranial pressure in pseudotumour cerebri.⁹ In the cases of pseudotumour cerebri associated with perhexiline and in our patient the exact mechanism remains unsettled.

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Adult onset spinal muscular atrophy with atrophic testes: report of two cases

Sir: Adult onset progressive spinal muscular atrophy is often considered to represent a variant of amyotrophic lateral sclerosis¹ or, less commonly, a hereditary entity.²⁻⁵ Here we report two sporadic cases of severe adult onset progressive spinal muscular atrophy associated with testicular atrophy and normal hormone levels.

Patient 1, a 31-year-old male native of the Ivory Coast, presented with a 2 year history