Hypotensive convulsion associated with desmopressin and imipramine treatment

DTS M. HAMED, H MITCHELL, and D J CLOW (Dumfries and Galloway Royal Infirmary, Dumfries DG1 4AP) write: In September 1992 a 10 year old boy was admitted unconscious (Glascow coma scale 3) and hypotensive (temperature 33°C). He had previously had primary nocturnal enuresis, for which he was initially given intranasal desmopresin 5 μg at night for two months; later the dose was increased to 40 μg for five months, after which imipramine 25 mg at night was added. Two weeks before admission he had had a minor head injury. On the day of admission he had fine twitching movements of both hands and clenched teeth, which progressed to a generalised tonic seizure lasting 10 minutes and which was terminated by 5 mg of rectal diazepam. The cause of his deep coma was not clear. His weight was 31.8 kg and his height 138.8 cm, both measurements being above the 50th centile. His haemoglobin level was 125 g/l, white blood cell count 19 x 10⁹/l, neutrophils 16 x 10⁹/l. Although blood urea, calcium, and glucose concentrations were normal, his serum sodium was 113 mmol/l, serum potassium 3.3 mmol/l, plasma osmolality 244 mmol/l, and urine osmolality 160 mOsm. Hyponaemia was diagnosed and treated.

There was progressive clinical and biochemical improvement over 36 hours, confirming the diagnosis of hypotensive convulsion secondary to desmopressin or imipramine, or both. He was discharged after four days without medication and reviewed 10 days after discharge, when he appeared in good health. A drug overdose was unlikely because of parental supervision. The other possibility was hyponaemia induced by imipramine or desmopressin. Although the manufacturer of desmopressin recommends reassessment after three months’ treatment, this is often ignored. We would recommend that imipramine is not prescribed in combination with desmopressin, that parents should be warned of the dangers of excessive fluid intake when desmopressin is used, and that the initial trial period before reassessment should be only four weeks.

Hemiplegia after measles, mumps, and rubella vaccination

DTS A H SACKEY and R L BROADHEAD (Royal Liverpool Children’s Hospital, Liverpool) write: A previously healthy 16 month old girl was admitted with an acute prolonged right sided tonic clonic convulsion associated with a fever of 39.4°C, having received measles, mumps, and rubella vaccine (Immervax) six days earlier. She was subsequently found to have a flaccid left hemiparesis, together with complex partial seizures, which were subsequently controlled with sodium valproate. Consciousness returned to normal within two weeks, but she was then noted to have a left homonymous hemianopia, which persisted for three months. On review after six months there was some mild weakness of the right leg, but the hand appeared normal.

On admission blood glucose and electrolyte concentrations were normal, and bacterial, viral, and serological studies showed no evidence of infection. Oral, oral, computed tomography showed initial mild generalised oedema and a persistent subarachnoid cyst. An electroencephalogram one week after admission was grossly abnormal with diffuse slow activity consistent with encephalopathy. Six months later it looked normal.

Systemic symptoms attributable to measles, mumps, and rubella vaccination usually occur 5–12 days after immunisation. There have been reports of severe neurological reactions after immunisation with serious sequelae, but no consistent association has been shown.2–4 Fever convulsions are well recognised after vaccination against measles, mumps, and rubella, but we are not aware of a previous report of prolonged hemiparesis.

This reaction was probably non-specific in that the high fever induced by the vaccine, in association with the added risk factors of susceptible age and maternal history of epilepsy, precipitated the prolonged convulsion and resulting encephalopathy.

Psychomotor retardation and semistuporous state with paroxetine

DTS J LEWIS, J BRAGANZA, and T WILLIAMS (St Tydfil’s Hospital, Merthyr Tydfil) write: Specific serotonin reuptake inhibitors have an advantage over tricyclic antidepressants in producing less sedation and psychomotor retardation.1 We present a patient who developed severe psychomotor retardation, becoming semistuporous, when taking the serotonin reuptake inhibitor paroxetine.

A 67 year old mentally handicapped woman was admitted with an agitated depressed illness. Her depression had endogenous components, with occasional persecutory beliefs. She was started on paroxetine 20 mg daily, increased to 40 mg, with thioridazine 10 mg three daily. Trifluoperazine 10 mg was added after a few days because she remained deluded. After three weeks her agitation subsided, and she appeared sedated; consequently the thioridazine and trifluoperazine were stopped. She deteriorated, however, becoming progressively more withdrawn, immobile, and unreactive, and rarely speaking. Her muscle tone remained normal, with no peripheral movements, but she could not feed or care for herself, and was occasionally incontinent. Physical examination and investigations, including computed tomography and electroencephalography, failed to show any cause for her deterioration. It was not until paroxetine was stopped (two weeks after withdrawal of the neuroleptics) that she began to improve, and after five days had completely recovered.

Two months later her depression recurred, and she restarted paroxetine 20 mg daily, with 3 mg of haloperidol. After two weeks she again developed psychomotor retardation, lapsing into a semistuporous state. As before, this did not resolve until paroxetine (but not the haloperidol) was withdrawn.

The patient did not have any renal or hepatic impairment that might have increased her sensitivity to paroxetine. However, it is possible that paroxetine was solely responsible for the adverse reaction, or whether the neuroleptics also played a part. Furthermore, mentally handicapped people are known to react to idiosyncratic or paradoxical ways to medication. The Committee on Safety of Medicines has reports of four further cases of snupor associated with paroxetine treatment. None of the patients was reported as concurrently receiving neuroleptics, nor were they elderly.


Muscle cramps related to corticosteroids

DTS J LEAR and R G DANIELS (Northampton General Hospital NN1 5BD) write: Muscle cramps have been reported with β₃ agonists with muscle pain with nebulisation. Some diopropionate reported to the Committee on Safety of Medicines, but there have been no reports of muscle cramps with prednisolone. A 48 year old non-smoking woman with mild asthma had been started four years earlier on a twice daily regimen of fenoterol hydrobromide 90 μg and ipratropium bromide 75 μg by nebulisation. During that time each administration of prednisolone (40 mg orally once daily) or beclomethasone dipropionate (200 μg twice daily by inhaler) precipitated severe muscle cramps. Starting one day after the dose, they mainly affected her left calf. They occurred mostly at night with three to four attacks lasting five minutes and subsiding spontaneously. Direct pressure and stretching did not help. They stopped within three days of her finishing the corticosteroids, and did not recur. While taking the corticosteroids she did not alter her use of inhaled β₂ agonists. Serum potassium concentration at this time was normal.

In a summary her hay fever was occasionally severe enough to warrant topical beclomethasone dipropionate (200 μg twice daily each nostril) for one week and oral corticosteroids (prednisolone, 20 mg once daily) for three days. The muscle cramps were again precipitated, although she was not using inhaled β₂ agonists at the time as her asthma was not a problem. She did not complain of weakness in her legs, and there was no muscle wasting or diminished power. She had had severe pneumonia secondary to measles at the age of 6 years and was diagnosed as asthmatic at the age of 21. She had left sided varicose veins. There was no family history of atopy. She had occasional cramps while taking salbutamol (100 μg per metered inhalation) but these were less severe and less frequent than the steroid related ones. Since starting fenoterol she had had no cramps unless she was also taking corticosteroids.
