Teaching Point

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Charcoal haemoperfusion in a child with amitriptyline poisoning

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Tricyclic antidepressant (TCA) overdose is a relatively common cause of poisoning. According to the American Association of Poison Control Centers, 7961 patients with amitriptyline poisoning were reported in 2000. Of these, 958 were under 6 years old, 6388 needed treatment in a health care facility and 68 patients died [1]. The current therapy of TCA overdose includes preventive measures such as gastric lavage, activated charcoal, anticonvulsants, vasopressors for hypotension, and sodium bicarbonate infusion for dysrhythmias [2,3]. Although charcoal haemoperfusion (CHP) has been used in the management of amitriptyline overdose, the data regarding CHP in the management of amitriptyline overdose is controversial [3–5]. To our knowledge, there is no report related to the use of CHP in children with amitriptyline poisoning in the English literature. Here, we report a child with amitriptyline poisoning who showed dramatic improvement after CHP treatment.

Case report

A 2.5-year-old, 14 kg male was admitted to our paediatric emergency department 4 h after ingesting 20 tablets of amitriptyline (25 mg each, totally 500 mg). The parents stated that he ingested a total of 20 tablets of amitriptyline at 19.00 h, was sleepy, and minutes later became unresponsive. The parents observed that he developed tonic–clonic seizures on his way to hospital. At the local hospital, diazepam was given and he was referred to our hospital. At presentation in our paediatric emergency room, he was comatose, and blood pressure was 90/50 mmHg, pulse rate was 110/min and irregular. Respiration rate was 30 with shallow breathing. He was unconscious with mild flexion withdrawal to painful stimuli. The Glasgow coma score was 4 (E1, M2, V1). Pupils were mid-range and non-reactive to light. Babinski reflexes were bilaterally unresponsive. Deep tendon reflexes were negative. Laboratory investigation revealed Hb 6.5 g/dl, WBC 7300/mm³, platelet 162 000/mm³, BUN 10 mg/dl, serum creatinine 0.3 mg/dl, Na 126 mEq/l, K 3.7 mEq/l, Cl 109 mEq/l, Ca 9 mg/dl, ALT 37 IU/l and AST 20 IU/l. Urinalysis showed pH 6, specific gravity 1010 without protein or blood. Predialysis ECG showed diffuse ventricular extrasystoles, QRS prolongation (0.12 s), QT prolongation, intraventricular branch blocking, and diffuse ventricular arrhythmia (Figure 1). A nasogastric tube was inserted and gastric lavage was started with normal saline and activated charcoal (1 g/kg). A Foley catheter was inserted and an 8-Fr double lumen catheter was placed for dialysis access. Normal saline was used to treat the hypotension and intravenous sodium bicarbonate to prevent cardiac toxicity. Blood samples for amitriptyline level were obtained just before CHP, at midtime and at the end of the session. Blood amitriptyline levels were measured using HPLC (Hewlett-Packard Model 1050, USA). CHP was initiated 7 h after ingestion. A 140 ml CHP cartridge (Gambro Adsorba 150c; Gambro Dialysatoren, Germany) was used. The CHP session lasted 3 h. No adverse effect related to CHP treatment was seen.

After the CHP session, the child began to respond, showing extremity flexion withdrawal and eye movement (opening) to painful stimuli. The Glasgow coma
score was 7 (E2, M3, V2) and the ECG became normal 3 h after the first CHP session (Figure 1). He was still unconscious with mild flexion withdrawal to painful stimuli 24 h after admission. The second CHP session was done on the second day. The serum amitriptyline levels are given in Table 1. In the follow-up, the patient improved completely and was discharged after 5 days of admission. On control examinations, the patient was healthy.

Discussion

Amitriptyline poisoning was the cause of coma in our patient. Although the beneficial effects of haemoperfusion in the management of amitriptyline overdose have been reported, the experience with the dialysis modality in the treatment of amitriptyline overdose is limited and contradictory. Neither textbooks nor reference books recommend haemoperfusion for the treatment of amitriptyline overdose [2,4–7]. In our patient, the response to CHP treatment was excellent and this case is the first reporting beneficial effects of CHP in the management of amitriptyline overdose in a young child. The administration of other preventive measures does not eliminate the beneficial effect of these measures in addition to CHP. Some authors state that haemoperfusion is unnecessary in the management of amitriptyline overdose [2,4,5]. However, the recommendation of haemoperfusion in the management of amitriptyline overdose is based on (i) its effectiveness for shortening the coma, (ii) its stability to severity of complications due to prolonged therapy and to the cost of hospital admission, (iii) the prevention of the broad QRS complexes in the ECG during treatment [5–7]. It has been reported that the QRS interval and the clinical manifestations of overdose were reliable and more readily available indicators of toxicity than serum concentrations of TCA [2,3]. Our indications for CHP treatment in our patient were deep coma, generalized seizures, shallow breathing and prolonged QRS complex. The Glasgow coma scores increased from 4 to 7 after CHP treatment, the coma disappeared gradually over 1–3 days [2,3] and our patient was fully awake and conscious after the second CHP session.

In conclusion, our experience showed a dramatic improvement of an amitriptyline overdose with CHP treatment in a very young child but controlled studies are needed to clarify the indication of CHP treatment in the management of this intoxication.

Conflict of interest statement. None declared.

References