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Clinical effects of occupational clonidine poisoning and efficacy of extracorporeal blood purification therapies

Dear Editor,

Although limited data on adults with clonidine poisoning exist [1], occupational exposure is uncommon. Also, the role of extracorporeal blood purification techniques in patients with clonidine toxicity is unclear.

A healthy 32-year-old male involved in manufacturing clonidine was brought to the emergency department unresponsive. His face and clothes were covered with powder, and examination of the oral cavity, ear, nose, and throat revealed cream-colored deposits. Forty minutes prior to hospitalization, he was rescued from a poorly ventilated workshop by his coworkers. Approximately 10 min before his rescue there was an unintentional leakage of clonidine from a transporting pipeline. After rescue he complained of severe cough, wheeze, tinnitus, dyspnoea and dizziness and after a few minutes lapsed into unconsciousness.

On examination, pupils were symmetrical with an average size of 6 mm, and slowly responsive to light. His Glasgow Coma Scale score was 5 and he responded to painful stimuli. Vital signs were: blood pressure, 180/112 mmHg; respiratory rate, 10 beats/min and regular; body temperature 35.1 °C; and oxygen saturation was 92% on 7 litres nasal cannula. Laboratory evaluation was unremarkable. Electrocardiography showed sinus bradycardia at 43 beats/min. A chest radiograph and computed tomography scan of the brain were normal. The patient was decontaminated using soap and water, then gastric lavage was performed followed by administration of 80 g of activated charcoal. Supportive management included fluid resuscitation and atropine. After administration of two intravenous boluses of urapidil (an α_1 -adrenergic antagonist) 25 mg and a total of naloxone 10 mg, he had minimal improvement of hypertension and mental status.

The patient was then transferred to a tertiary hospital, where an elevated serum concentration of clonidine (164 $\mu\text{g/L}$) (therapeutic range 0.5–4.5 $\mu\text{g/L}$) was confirmed by liquid chromatography-tandem mass spectroscopy 6 hours after the leakage occurred. A 3-hour session of simultaneous, in-series haemodialysis and haemoperfusion (HA130 cartridge, Jafron Biomedical Co.) was performed and serial serum clonidine concentrations were performed before, during and following the session (Figure 1). The combined haemodialysis and haemoperfusion was performed according to the Standard Operating Procedures for Blood Purification by the National Health

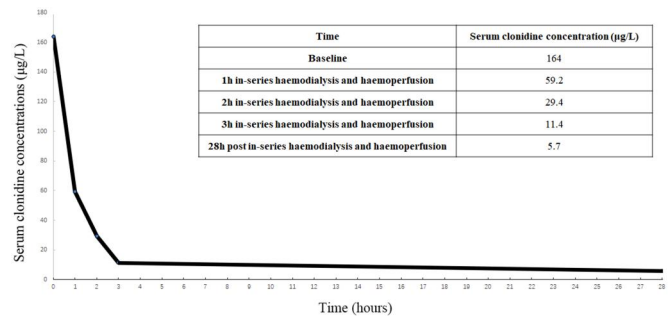


Figure 1. Serum clonidine concentrations before, during and after the in-series haemodialysis and haemoperfusion session.

Commission of the People's Republic of China as described in detail elsewhere [2]. The patient's consciousness, bradycardia and hypertension resolved within one hour of his extracorporeal treatment. He was discharged on the second day after admission with a full recovery and no complications.

Clonidine, an α_2 -adrenergic agonist that crosses the blood-brain barrier, has both central and peripheral α -adrenergic agonist effects. By stimulating α_2 -adrenergic receptors in the brain, clonidine can modulate sympathetic outflow, resulting in decreased peripheral vascular resistance and cardiac output. With massive overdose, the peripheral α -adrenergic agonist properties may predominate, resulting in vasoconstriction and marked hypertension as shown by our case [3, 4]. Clonidine has a molecular weight of 230 Da, is fat-soluble with a volume of distribution of 1.7–2.5 L/kg, is 20% protein bound and has an elimination half-life of 6–23 hours [5]. These characteristics suggest that in-series haemodialysis and haemoperfusion provides the possibility of removing clonidine which could rapidly reduce the serum concentrations with few side effects.

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