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CEREBRAL INFARCTION ASSOCIATED WITH ORAL VERAPAMIL OVERDOSE

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ABSTRACT

Verapamil is a widely used calcium channel blocker. Side effects with oral use are usually mild, however, major hemodynamic complications and death can occur if the drug is given in overdose. We describe a case of cerebral infarction associated with attempted suicide with verapamil overdose, the first reported to our knowledge.

INTRODUCTION

Verapamil is a synthetic drug related to papaverin, which has coronary dilator, antiarrhythmic and antihypertensive properties (1). The drug acts on cellular membrane permeability by blocking calcium ion influx and to some extent, sodium ion influx (2).

Serious side effects have been reported after intravenous use (3). Several cases of oral overdose have been described in the English literature (4-18). Only two cases of neurological side effects due to verapamil overdose are reported (5,11).

We present a case of permanent focal brain damage after attempted suicide with verapamil.

Case Report

A 39-year-old woman was admitted to the emergency room after a suicide attempt. She had ingested 2280 mg of verapamil, 120 mg of propranolol and 400 mg of opipramol.

On admission, the patient was in a deep stupor, peripheral pulses were not palpable and blood pressure was undetectable. The heart rate was 55-60 per minute on monitor and electrocardiogram showed AV dissociation and nodal rhythm with retrograde P wave.

Intravenous atropin and normal saline were started and the blood pressure raised to 80/50 mmHg. The patient was transferred to the Intensive Coronary Care Unit. On physical examination: the lungs were clear, heart sounds were normal, without murmurs. The patient was conscious, responding to her name, and no neurological deficits were seen. Several hours later, blood pressure and heart rate fell; intravenous isoproterenol was immediately started and vital signs returned to normal. On the next day, routine examination identified left hemiparesis and 48 hours later, brain CT scan showed right temporo-parietal infarction. The patient refused carotid artery angiography. Echocardiogram showed normal heart contraction without any sign of thrombus.

Laboratory findings were as follows: Hemoglobin 17 g%, white blood cell count = 13800 cm³m; glucose - 171 mg%; urea - 3.6 mg%; creatinine - 1.2 mg%; Na - 138 mEq/L; K - 47 mEq/L; pH - 7.36; PaO₂ - 75 mm Hg; PaCO₂ - 38 mmHg; H₂CO₂ - 22 mEq/L.

Three months later, on routine followup, mild right hemiparesis was still found.

DISCUSSION

Verapamil has been used successfully for over 25 years as an anti-anginal, antiarrhythmic and antihypertensive agent. Oral use may cause occasionally moderate side effects such as headache, dizziness, constipation, flushing rash, peritibial edema, first and second degree AV block and hypotension (3). Intravenous use can be associated with transient severe hypotension, exaggerated bradycardia or even asystole (3). Seventeen cases of oral verapamil overdose were reported in the English literature. Doses ranged from 0.8-9.6 g. The observed effects of these intoxications were extensions of the drug's pharmacologic activity including severe hypotension, bradycardia, AV dissociation and complete AV-block, sinus arrest and asystole (4,6-18). Altered mental status and metabolic acidosis due to decreased peripheral perfusions were also noted (5,6,9-11). Three cases were fatal in spite of treatment with calcium and isoproterenol (8,9,12). One patient was left with severe diffuse anoxic brain damage due to prolonged hypoperfusion and metabolic acidosis before institution of therapy (11).

Our patient ingested 2880 mg of verapamil and showed hemodynamic changes similar to those reported in the literature in such cases.

In addition, she developed cerebral infarction. To our knowledge there are no reported cases of such complication as a result of verapamil overdose. Two cases of brain damage were described, the first had diffused cerebral anoxic damage, secondary to severe prolonged hypoperfusion (11). The second is a child with transient seizures (5), but no focal brain damage was described.

In our patient, the cerebral infarction was probably secondary to severe hypotension caused by verapamil overdose. Propranolol ingested in a low dose (120 mg) could not have such exaggerated effect seen in the case under discussion. Opipramol is a tricyclic antidepressant. It is given in doses of 150-300 mg daily in divided doses or with the major part given at night. Of 18 reported instances of overdosage with opipramol, six had been fatal and four of these attributed to sudden cardiac arrest. Hypotension was not described as a side effect (19,20). Our patient ingested 400 mg which is very close to the therapeutic regimen. We suppose that this did not play any role in the hypotension the patient suffered. As mentioned above, the patient refused carotid angiography. We cannot be sure that any preexistent pathology did not contribute to the focal damage although the patient's young age makes this unlikely.

In conclusion, this patient exhibited a serious irreversible complication of oral verapamil overdose.

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