Self-induced myocardial infarction

PETER CARR
B.M., M.R.C.P.

Department of Medicine, Manchester Royal Infirmary, Oxford Road, Manchester M13 9WL

Summary

A case of acute myocardial infarction and peripheral vascular insufficiency following overdose with ergotamine is described.

Introduction

Despite the prevalence of migraine and the widespread use of ergotamine preparations in its treatment, there are few reports of the effect of a massive overdose of these compounds. This case illustrates the consequences of an overdose of Migril, a compound containing 2 mg ergotamine tartrate B.P., 50 mg cyclizine hydrochloride B.P. and 100 mg caffeine hydrate B.P. per tablet.

Case report

A 29-year-old man was admitted as an emergency after taking 60 Migril tablets. A migraine sufferer for several years, he had a past history of attempted suicide, but otherwise had been well. There was no family history of heart disease.

On physical examination he was obese and had old scars on the wrists from a previous attempted suicide. The apex rate was 160 beats/min, BP 130/70 mmHg, heart sounds, peripheral pulses and skin circulation were normal. There was no abnormality in the chest, abdomen or nervous system.

On admission, the serum urea and electrolytes were normal. An electrocardiogram showed atrial fibrillation which reverted spontaneously to sinus rhythm with a normal record.

Gastric lavage was not performed because the patient had been vomiting. He remained well until 16 hr after admission when he had a series of cardiac arrests (ECG showed both ventricular fibrillation and asystole), and aspirated vomit. He was resuscitated but remained hypotensive for several hours and developed transient oliguria with azotaemia (serum urea 24 mmol/l). He also developed pneumonia.

During the second day of the admission the peripheral pulses became impalpable, the toes of the left foot becoming gangrenous and anaesthetic. Left

femoral angiography confirmed arterial spasm in the popliteal artery. Serial electrocardiographs showed the changes of an acute infero-lateral myocardial infarction.

Treatment with i.v. sodium nitroprusside 200 µg/min, and hydroxocobalamin was started on the third day and the peripheral pulses became palpable again within 30 min. The infusion was continued for 5 days until withdrawal was not followed by signs of vasoconstriction. He made good progress and the pneumonia and renal failure resolved completely. There was sloughing of the skin at the tip of the toes of the left foot only. Three months later the changes in the electrocardiogram were still present.

Discussion

The sequelae of this patient's overdose are thought to be related directly to ergotamine, although it is possible that the transient atrial fibrillation was related to the caffeine ingestion (Goodman and Gilman, 1980). The patient ingested 120 mg of ergotamine tartrate; the maximum recommended dose is 4 mg/day, and 12 mg in one week.

Myocardial ischaemia has been described after the administration of conventional doses of ergot derivatives in migrainous subjects with and without heart disease (Snell, Russell-Smith and Coys, 1978), but myocardial infarction has been reported rarely (Goldfischer, 1960; Fuchs and Blumenthal, 1950). Ischaemia of the extremities due to ergot alkaloids is well recognized and historically is known as St Antony's Fire.

Sodium nitroprusside is the treatment of choice for ergotamine poisoning, and should be given with hydroxocobalamin. Its use is influenced by impaired renal function (Leading Article, 1978). In this case it was commenced after the development of gangrenous change, and when renal function was improving. In retrospect it may be argued that its prompt use after the patient's admission may have avoided any untoward complications. It is certain that it prevented more extensive peripheral ischaemia and possibly further myocardial damage.

References

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