

Intracerebral haemorrhage and vasculitis secondary to amphetamine use

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Summary

We report a case of amphetamine-related intracranial haemorrhage and vasculitis, responding to immunosuppressants. Angiograms obtained before and after therapy are shown; the importance of immunosuppressive therapy is discussed.

KEY WORDS: hemiplegia, cyclophosphamide, prednisolone.

Introduction

Intracerebral haematomas are a rare complication of amphetamine abuse, and may or may not be associated with arteriographic evidence of a vasculitis. We report a further case to draw attention to the important clinical implications of the serious prognosis associated with this disease.

Case report

An 18-year-old male developed a severe left frontal headache and subsequent progressive right hemiparesis several hours after taking three tablets of amphetamine orally. His past medical history was unremarkable aside from a long history of drug abuse with numerous agents, largely amphetamines and marihuana.

The patient was afebrile with a blood pressure of 106/60 mmHg and a pulse rate of 72 per min. He was oriented to person, time and place. Speech, recent and remote memory were normal. He had a dense right hemiplegia and a right hemisensory loss. There was no evidence of trauma or signs of systemic vasculitis.

Investigation revealed a normal blood count, biochemistry, sedimentation rate, platelet count, coagulation studies, antinuclear factor, rheumatoid factor, serum complement levels and blood cultures. The electrocardiogram and chest X-ray were also normal.

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A computed tomographic (CT) head scan (Fig. 1) demonstrated a 2.3 × 2.7 cm left intracerebral haematoma, involving the internal capsule and basal ganglia. A 4 mm left to right shift was noted. Cerebral angiography revealed diffuse bilateral multi-focal areas of ectasia and narrowing in all intracranial arteries, consistent with a vasculitis (Fig. 2). The patient was treated with dexamethasone 24 mg per day intravenously, for 5 days, followed by 50 mg of prednisone per day. Cyclophosphamide 2 mg per kg per day was added. He improved steadily walking with assistance 1 week after admission. A repeat angiogram 18 days after the first showed marked improvement (Fig. 3). Complications during the angiography prevented renal and hepatic angiograms from being performed. The prednisone was tapered after 1 month and the patient was discharged 6 weeks after admission with a mild residual right hemiparesis.

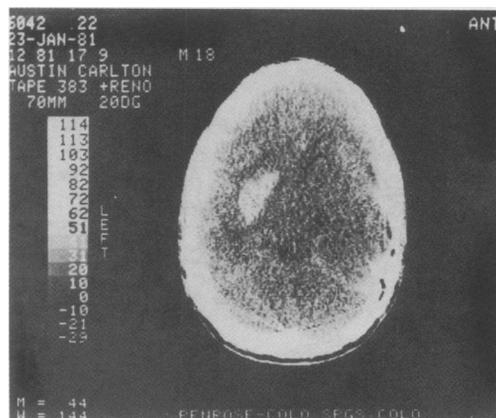


FIG. 1. CT head scan on admission showing a left intracerebral haematoma involving the internal capsule and basal ganglia.

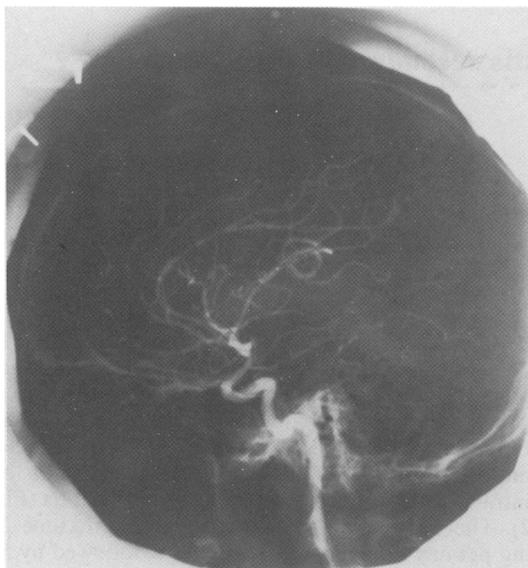


FIG. 2. Cerebral angiogram (lateral view). Showing diffuse multifocal areas of ectasia and narrowing of the anterior and middle cerebral arteries.

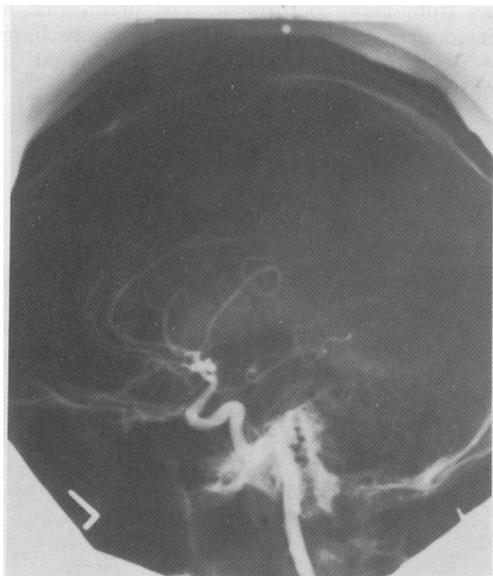


FIG. 3. Cerebral angiogram (lateral view) repeated 18 days after the first one, showing marked improvement.

Discussion

A small number of patients with intracerebral haemorrhage associated with amphetamines have been reported. Delaney and Estes (1980) reported one case and reviewed 14 others reported in the literature. Amphetamines were taken intravenously in eight patients and orally in seven. Symptoms consisting of headaches, decrease in the level of consciousness and hemiparesis began minutes after consuming the drug. Five patients were found to have an elevated blood pressure, and in two the diastolic exceeded 120 mmHg. Arteriograms revealed beading of the intracranial arteries in four of the cases studied. In eight cases (five at autopsy) intracranial pathological studies were obtained, and in two of these cases an intracranial vasculitis was demonstrated. The findings suggest that the intracerebral haemorrhage is due to either a vasculitis or amphetamine related hypertensive crisis or both.

Amphetamine-related vasculitis has been well defined in the literature, and the pathology of this necrotizing vasculitis angiitis was delineated by Citron *et al.* (1970). The early vascular changes include fibrinoid angiitis, necrosis of the media and intima and a leukocytic infiltrate; marked intimal proliferation is seen. In the late stages, muscular and elastic tissue is replaced by collagen. Medium- and small-sized arteries and arterioles are involved. The vessel lumen is usually narrowed considerably in the involved segments and occasionally a nodular aneurysmal dilatation is found. Yu *et al.* (1983) reported a patient with amphetamine-related vasculitis and intracerebral haemorrhage, responding to prednisone. Our case similarly responded briskly to immunosuppressive agents, and the angiograms demonstrated the improvement. In view of the five deaths in the 15 patients reviewed by Delaney and Estes (1980), this response certainly underlines the need for urgent immunosuppressive therapy in patients with amphetamine-related vasculitis.

References

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