The second case suffered from four spontaneous abortions, one threatened abortion and required forceps delivery for prolonged second stage in both her term pregnancies.

Although there is no cure for myotonic dystrophy the earlier recognition of the diagnosis would allow appropriate management particularly with a view to prognosis, anaesthesia and genetic counselling. These patients presented with cardiac rhythm disturbance before other clinical features of the disease were recognised by their medical attendants.

Experience from these two patients suggest that atrial flutter may appear at a stage before the clinical diagnosis becomes obvious. A combination of atrial flutter together with symptoms such as stiffness, 'rheumtics' weakness and lethargy or a difficult obstetric history should stimulate a search for other possible features of myotonic dystrophy.

REFERENCES

1 Wieringa B, Brunner H, Hulsebos T, Schonk D, et al. Genetic and physical demarcation of the locus for dystrophia myotonica. Adv Neurol 1988; 48: 47-69.
2 Harper PS. Myotonic dystrophy. Philadelphia: WB Saunders 1979.
3 Harper PS. The genetics of muscular dystrophies. Prog Med Gen 1985; 6: 53-90.
4 Olofsson BO, Forsberg H, Andersson S, Bjerle P, et al. Electrocardiographic findings in myotonic dystrophy. Artic Med Res 1988; 47 (Suppl): 420-42.
5 Walton JN. In Walton JN and Gardner D. Disorders of Voluntary Muscles, 4th Edition. Churchill Livingstone, Edinburgh 1981: pp 510-514.
6 Kuhn E, Lehmann-Horn F, Rudel R. Dystrophia myotonia a frequently misdiagnosed disease. Nerveenartz. 1990; 61(6): 323-331.
7 Olofsson BO, Forsberg H, Andersson S, Bjerle P et al. Electrcardiographic findings in myotonic dystrophy. Br Heart J 1988; 59: 47-52.
8 Chung HT, Tam AYC, Wong V, Li DFH et al. Dystrophia myotonica and pregnancy - an instructive case. Postgrad. Med J 1987; 63: 555-557.
9 Freeman RM. Placenta accreta and myotonic dystrophy. Two case reports. Br J Obstet Gynecol. 1991; 98: 594-595.

Scot Med J 1992; 37: 150-152

'ECSTASY' AND INTRACEREBRAL HAEMORRHAGE

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Abstract: *Four cases of intracerebral haemorrhage related to the use of amphetamine or 'Ecstasy' (methylene dioxy methyl amphetamine) are presented.*

Key words: Amphetamine, methylene dioxy methyl amphetamine, 'Ecstasy', intracranial haemorrhage, drug abuse.

Introduction

Intracranial haemorrhage related to drug abuse is poorly documented in Great Britain. Four young adult patients presented to our unit over a ten week period with intracerebral haemorrhage. They had all recently taken an amphetamine based drug. Three patients had normal angiograms and made a good functional recovery. One patient who had an arterio-venous malformation died.

Case reports

Case 1

A previously healthy thirty year old female sustained sudden onset headache, dysphasia and right hemiparesis. She was a smoker and was also taking the oral contraceptive pill. She volunteered that she had taken a mixture of 'ecstasy' and 'speed'(amphetamine) at a party just prior to the onset of her symptoms. On admission to the Institute of Neurological Sciences she opened her eyes spontaneously and was obeying commands. She was dysphasic with a right hemiparesis, her arm being flaccid. Her blood pressure was 100/60. A CT scan (Fig 1) revealed a large haematoma deep in the left fronto-parietal region, extending into the basal ganglia. It was decided to treat her conservatively. Cerebral angiography when performed six weeks later was normal.

Case 2

A twenty-two year old woman awoke with the sudden onset of severe headache, urinary incontinence and agitation. She then had a grand mal seizure. On admission to the Institute of Neurological Sciences she opened her eyes to speech and could localise to painful stimuli. She had a mild right hemiparesis and an expressive dysphasia. Her blood pressure was 140/80. A CT scan (Fig 2) showed a small left frontal haematoma with blood in the left sylvian fissure. A left carotid angiogram was performed and was normal. She developed further seizures that were controlled with phenytoin. An anonymous phone caller stated that she had taken amphetamine sulphate just prior to the onset of her illness.

Case 3

A twenty year old man was taken to a public house by his friends at lunch time. He abstained from alcohol but his soft drink was 'spiked' with 'Ecstasy'. He "fell asleep" and was carried home. He "slept" all afternoon. The patient had a grand mal seizure later that afternoon and was taken to his local hospital where he was given intravenous diazepam and phenytoin. He was deeply unconscious and therefore ventilated for transfer. On arrival at the Institute of Neurological Sciences he was neither opening his eyes or making any verbal response to painful stimuli. He had a flexor response to pain in both upper limbs. Both pupils were fixed and dilated. His blood pressure was 170/90. A CT scan (Fig 3) revealed a large frontal haematoma with 1.5 cms of mid line shift. Contra-lateral ventricular dilatation was present. An emergency angiogram was performed and showed a left frontal arteriovenous malformation. The patient had an emergency craniotomy but a combination of severe brain swelling and torrential bleeding resulted in the operation being unsuccessful. The patient was declared brain dead the following day.

Case 4

A sixteen year old boy had been drinking cider with his friends. The patient's drink was 'spiked' with 'Ecstasy'. At 9pm he returned home

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when his parents found him to be "drowsy, slurring his speech and dragging his right leg". On admission to the Institute of Neurological Sciences he was opening both eyes and localising with both arms to painful stimuli. He had a mild right hemiparesis with an expressive dysphasia. His blood pressure was 130/70. A CT scan (Fig 4) demonstrated a deep left hemisphere haematoma. He was treated conservatively. Cerebral angiography when performed four weeks later was normal. He made a good functional recovery.

Discussion

Intracranial haemorrhage related to drug abuse is well recognised in the United States but remains uncommon. Drug abusers also present with non haemorrhagic stroke. Cocaine together with amphetamines and related compounds are the drugs most commonly involved in both intracranial haemorrhage and stroke.[1,2]

Drug abuse is a very rare cause of intracranial haemorrhage in Glasgow. We have however had four such cases present to us over a ten week period. In the following two months we have seen no further cases. Two mechanisms of haemorrhage are postulated in the literature:- hypertensive surges and cerebral angi-itis[3,4,5]

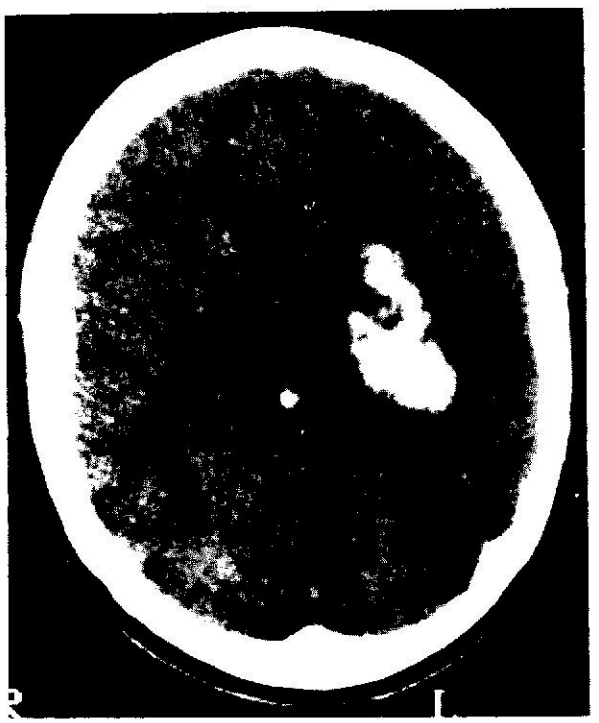
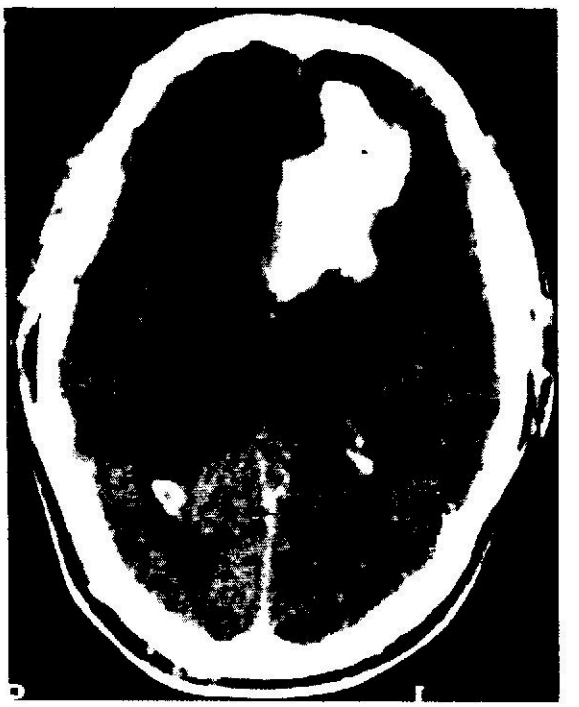


Fig 1 A large haematoma deep in the left fronto-parietal region, extending into the basal ganglia.



Flg.3 A large frontal haematoma with 1.5 cms of mid line shift. Contra-lateral ventricular dilatation is present.

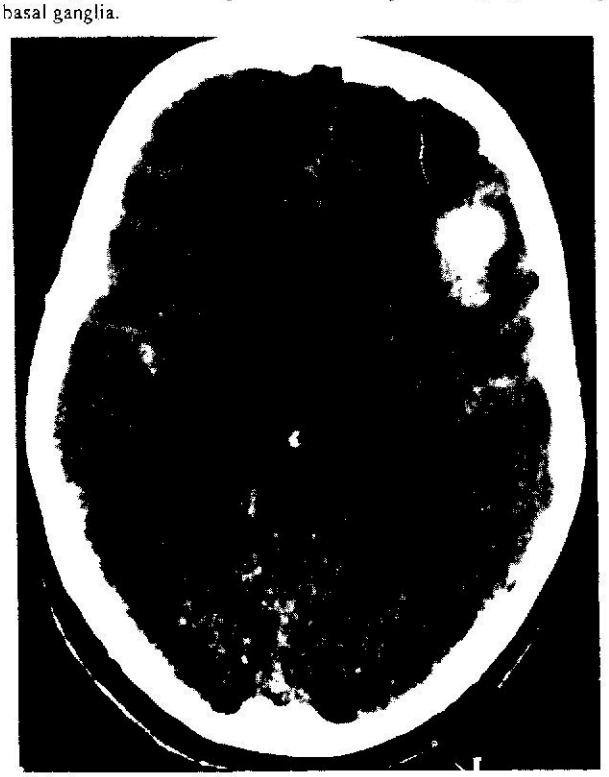


Fig.2 A small left frontal haematoma with blood in the left sylvian fissure.

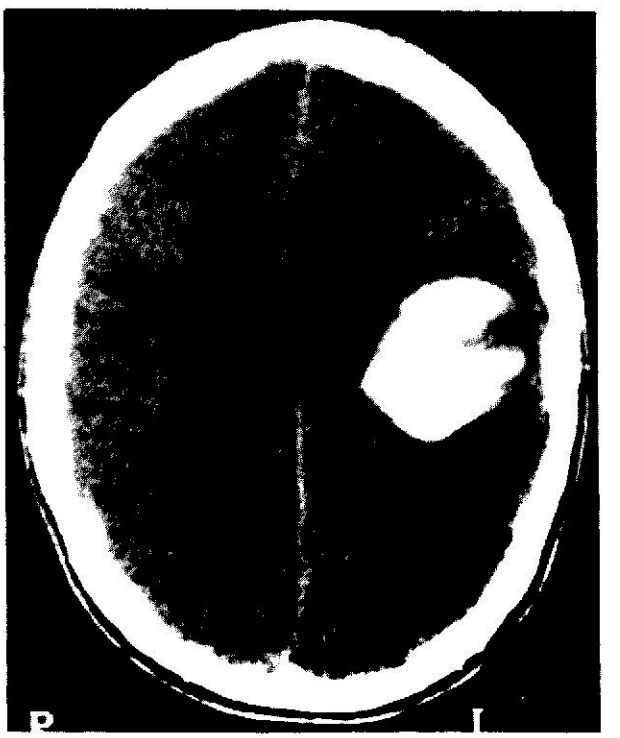


Fig.4 A large dominant hemisphere haematoma is shown.

These mechanisms do not appear to be dose related and both may play a role in patients with and without vascular malformations. Amphetamine and methamphetamine cause cardiac stimulation, peripheral vasoconstriction and hypertension. In Delaney's review[3] only five of fifteen patients were found to be hypertensive on admission, however a short lived hypertensive surge may have occurred in some of these patients. The only patient in our study who was hypertensive on admission to the Institute had already coned and this would alone explain his elevated blood pressure. Cerebral angi-itis is a well documented complication of methamphetamine. Pathologically the lesion is indistinguishable from that of polyarteritis nodosa and disappears with steroid treatment.[3] Drug impurities may play a role via either hypertensive surges or cerebral angi-itis.[3] The close timing of our four cases makes us suspicious that impurities in a batch of drugs may have been a major factor in the concentration of cases in Glasgow over such a short period.

'Ecstasy' (methylene dioxy methyl amphetamine) is one of the more recent arrivals on the Scottish illicit drug scene. Very little is known of its long term effects. At present it does not appear to be addictive. 'Ecstasy' has unfortunately become very popular in Glasgow, it is widely available in public houses and discotheques. 'Ecstasy' is usually taken so as to "better appreciate" some kinds of music. A single dose costs £15-20. 'Ecstasy' and amphetamine sulphate are generally taken orally, parenteral use does not appear to take place in Strathclyde. 'Ecstasy' is relatively easy to manufacture and it appears in many forms around Glasgow. Multiple routes of supply are suspected although no laboratories have been discovered in Scotland as yet.

Some instances of the stronger methylene dioxy amphetamine (MDA), or compressed amphetamine powder mised with LSD, being sold as 'Ecstasy' are known to the police.

Drug abuse should be remembered as a potential cause of intracranial haemorrhage in young patients. Urinary screening for amphetamines and related compounds should be considered in young patients presenting with unexplained stroke.

ACKNOWLEDGEMENTS: We would like to thank Mr R A Johnston consultant neurosurgeon and Strathclyde Police drug squad for their assistance with this report.

REFERENCES

1 Kakdu DA, Lowenstein DH. Emergence of drug abuse as a major risk factor for stroke in young adults. Ann Intern Med 1990; 113(11): 821-827.
2 Vinken PJ, Bruyn GW, Klawans HL. In: Handbook of Clinical Neurology (Revised series 2) Vol 55: 519-521.
3 Delaney P, Estes M. Intracranial hemorrhage with amphetamine abuse. Neurology 1980; 30: 1125-1128.
4 Yu YJ et al. Cerebral angi-itis and intracerebral hemorrhage associated with methamphetamine abuse. J Neurosurg 1983; 30: 1125-1128.
5 Lukes SA. Intracerebral hemorrhage from an arteriovenous malformation after amphetamine injection. Arch Neurol 1983; 40:60-61.