SHORT REPORTS

Adult polycystic kidney disease and intracranial aneurysms

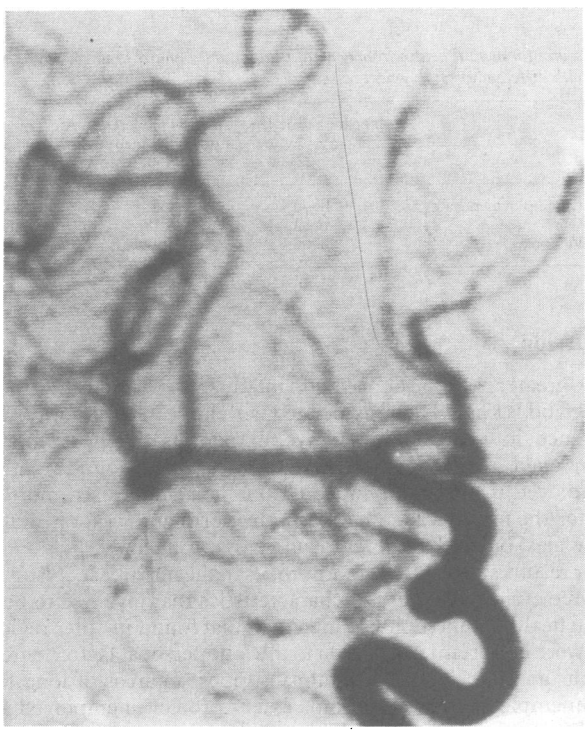
We describe for the first time a family whose three members all had adult polycystic kidney disease and intracranial aneurysms.

Case reports

Case 1—A 34 year old man presented in 1981 with accelerated hypertension. Renal ultrasonography confirmed that he had polycystic kidneys. In 1985 he was admitted with a subarachnoid haemorrhage. Neurosurgery showed a right cerebral haematoma, probably from an intracranial aneurysm. This was evacuated, but he died soon afterwards.

Case 2—A 39 year old woman, the sister of the patient in case 1, presented in 1978 with a subarachnoid haemorrhage. Cerebral arteriography showed bilateral intracranial aneurysms, but she was managed conservatively. In 1983 intravenous pyelography showed that she had polycystic kidneys. In 1986 she had a prolonged grand mal fit, and subsequently her renal function deteriorated. She was referred for continuous ambulatory peritoneal dialysis and remained well.

Case 3—A 36 year old man, the brother of the patients in cases 1 and 2, presented in 1978 with haematuria. Intravenous urography showed that he had polycystic kidneys. In 1986 his family history became known and he underwent elective cerebral digital subtraction angiography, which showed an aneurysm 6 mm in diameter of the right middle cerebral artery (figure). This was clipped, and he remained well.



Cerebral digital subtraction angiography showing aneurysm at trifurcation of right middle cerebral artery.

Comment

The association between intracranial aneurysms and adult polycystic kidney disease is well recognised. Recently, it has been suggested that patients with polycystic kidneys and a family history of intracranial aneurysms or subarachnoid haemorrhage have a greater risk of having aneurysms themselves than those without such a family history,[1] and our cases support this.

The risk of rupture of intracranial aneurysms has been estimated at 2% a year,[2] and once rupture has occurred the prognosis, short term and long term, is poor.[3] The outcome of surgery for unruptured aneurysms, however, is excellent.[4] For these reasons the need to screen for intracranial aneurysms in patients with polycystic kidneys has been emphasised.[5] Two such studies have been performed in Japan. Wakabayashi *et al* found seven patients with aneurysms among 17 with polycystic disease from 10 different families.[5] Five of these underwent surgery without complications. Similarly, Matsumara *et al* performed cerebral angiography on five patients with polycystic kidneys and found that three had aneurysms.[4] Two of these underwent surgery successfully.

Routine screening for intracranial aneurysms in people with polycystic kidneys, however, poses some problems. Firstly, there is no simple, non-invasive screening method. At present, the only sensitive method for detecting small aneurysms is cerebral arteriography,[2] and although complications are rare, they are potentially devastating. The advent of digital subtraction angiography has improved the situation only marginally since arterial injections of contrast are still necessary to achieve the resolution required to detect small aneurysms. Secondly, the optimum age at which to screen has not been defined, and it is by no means certain that a patient who has undergone angiography and been found not to have aneurysms will not develop aneurysms later.

For these reasons screening all patients with polycystic kidneys for intracranial aneurysms may not be worth while unless a subgroup at higher risk of having aneurysms can be identified. The only possible subgroup at present seems to be patients with a family history of intracranial aneurysms or subarachnoid haemorrhage, and we therefore suggest that these patients should be investigated.

1 Kaehny W, Bell P, Earnest M, Stears J, Gabov P. Familial clustering of intracranial aneurysms in autosomal dominant polycystic kidney disease. Abstract from American Society of Nephrology. *Kidney Int* 1987;31:204.
2 Levey AS, Pauker SG, Kassirer JP. Occult intracranial aneurysms in polycystic kidney disease. When is cerebral arteriography indicated? *N Engl J Med* 1983;308:986-94.
3 Nishioka H, Torner JC, Graf CJ, Kassell NF, Sahs AL, Goettler LC. Cooperative study of intracranial aneurysms and subarachnoid haemorrhage; a long term prognostic study. II. Ruptured intracranial aneurysms managed conservatively. *Arch Neurol* 1984;41:1142-6.
4 Matsumara M, Wada H, Ohwada A, Shinoda T. Unruptured intracranial aneurysms and polycystic kidney disease. *Acta Neurochir (Wien)* 1986;79:94-9.
5 Wakabayashi T, Fujita S, Ohbora Y, Suyama T, Tamaki N, Matsumoto S. Polycystic kidney disease and intracranial aneurysms. Early angiography diagnosis and early operation for the unruptured aneurysm. *J Neurosurg* 1983;58:488-91.

(Accepted 3 June 1987)

Royal South Hampshire Hospital, Southampton SO9 4PE
A SAIFUDDIN, BSC, MB, senior house officer in general medicine
J R E DATHAN, MD, FRCP, consultant physician

Correspondence to: Dr Dathan.

Drug abuse: a new problem

There have been many reports relating to aspects of drug abuse. Over the past two years we have witnessed a new hazard—namely, children presenting to hospital with needlestick injuries from discarded, used needles found in public places. We report one incident, review our total caseload, and recommend a policy for treating this problem.

Case reports

In May 1985 three children (aged 3-4) found a used 2 ml syringe and needle in a local dump known to be frequented by drug addicts. All three children stabbed themselves with the needle. They presented within 12 hours to hospital and received local toilet to their wounds. Blood eluted from the syringe and needle was tested for hepatitis B virus and antibody to human immunodeficiency virus (HIV). While awaiting these results the children were given hepatitis B immunoglobulin. The blood from the syringe and needle proved positive for hepatitis B surface antigen (HBsAg) but negative for antibody to HIV. The children were given a course of hepatitis B vaccine. Subsequently, they developed antibodies to HBsAg, proving uptake of the vaccine; none became clinically infected.

From June 1985 to April 1987, 15 further children (13 incidents) presented to our hospital. Most of these occurred after January 1987. In all cases the syringes and needles were available for analysis but none proved positive for either HBsAg or HIV antibodies.

Comment

These cases illustrate an ever increasing problem. From previous reports of the infectivity, diagnosis, and treatment of infections with hepatitis B