

Case No	Age (years)	Blood pressure (mm Hg)	Serum creatinine ($\mu\text{mol/l}$)	Comment
1	20	120/80	80	
	34	110/75	118	
2	22	120/80	72	Regular haemodialysis started at age 35
	32	140/100	260	
3	29	200/?	130	Regular haemodialysis started at age 35; kidney transplantation done at age 36
	36	130/80	145*	

*After kidney transplantation.

had the disease but did not have symptoms of intracranial aneurysms; his sister developed end stage renal failure at the age of 28. Four vessel angiography showed an aneurysm of the right middle cerebral artery, which was clipped. Cerebral angiography four months postoperatively yielded normal findings. At the age of 35 he required maintenance dialysis for renal failure, and his blood pressure was subsequently normal. Renal transplantation was performed one year later. Two months later he was readmitted in status epilepticus. Cerebral computed tomography showed a left frontal haematoma with intraventricular haemorrhage, and angiography showed an aneurysm of the anterior communicating artery. The haematoma was evacuated and the aneurysm clipped. He was left with a severe neurological deficit.

Comment

These cases indicate that patients with autosomal dominant polycystic kidney disease may develop intracranial aneurysm over time. Rupture may first occur early in the third decade before hypertension or renal failure have developed. In our three cases rupture

of aneurysms occurred at intervals of from seven to 14 years, and multiple aneurysms developed over 10 years in one patient (case 2).

It has recently been shown that patients with autosomal dominant polycystic kidney disease from families with a history of subarachnoid haemorrhage are at higher risk of intracranial aneurysm than those without a family history, and routine screening in such patients is advocated.^{2,3} In addition, regular screening should be offered to patients after an intracranial aneurysm has ruptured as they are probably at high risk of developing further aneurysms.

Interestingly, two of our three patients progressed rapidly to end stage renal failure, as did some of their relations, requiring regular haemodialysis before the age of 40. This occurs in only about 5% of patients with autosomal dominant polycystic kidney disease and end stage renal failure.⁴ Intracranial aneurysm may be associated with rapid progression to end stage renal failure in a subset of patients with the disease. Further study is required to test this hypothesis and to define the most appropriate imaging procedure for screening for aneurysms, the optimal age at which screening should be started, and the best programme for repeated investigation in high risk patients with autosomal dominant polycystic kidney disease.⁵

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- 2 Kaehny W, Bell P, Earnest M, Stears J, Gabow P. Family clustering of intracranial aneurysms in autosomal dominant polycystic kidney disease [Abstract]. *Kidney Int* 1987;31:204.
- 3 Kaehny WD, Chapman AB, Stears JE, Gabow PA, Earnest MP. Prospective imaging study of asymptomatic intracranial aneurysms in polycystic kidney disease [Abstract]. *Kidney Int* 1990;37:249.
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Prevalence of Dupuytren's contracture in patients infected with HIV

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An increased prevalence of Dupuytren's contracture has been reported among patients infected with HIV.¹ We had not been aware of a high prevalence among our patients and therefore determined the prevalence among the patients attending our clinic.

Patients, methods, and results

Fifty consecutive patients attending the genitourinary medicine clinic at this hospital who fulfilled the Centers for Disease Control's diagnostic criteria for AIDS² were assessed for Dupuytren's contractures. Contractures were diagnosed only if both examiners agreed that they were present. Factors associated with the development of Dupuytren's contractures were sought, including a history of diabetes, alcohol misuse, heavy mechanical labour, and taking hepatotoxic drugs.

The patients were all men aged 23 to 56. Thirty two patients had opportunistic infections, six Kaposi's sarcoma alone, and 12 Kaposi's sarcoma and an opportunistic infection. Only three patients had Dupuytren's contractures. The contracture was unilateral in each case with no fixed flexion deformity of the finger. One of these three patients had dementia related to HIV infection and gave a history of severe

alcohol misuse (about 100 units of alcohol a week for over four years). The second patient had cutaneous Kaposi's sarcoma as well as an eight year history of chronic active hepatitis. The third patient gave a history of retinitis caused by cytomegalovirus and pneumocystis pneumonia but had no underlying recognised cause of Dupuytren's contractures.

Comment

The prevalence of Dupuytren's contractures in our patients, who were positive for antibody to HIV-I, was 6%. This is similar to the prevalence in the population at large (4.6-5.0%).³ It is appreciably different from the 36% reported by Bower *et al* in their group of patients who were positive for antibody to HIV-I.¹ It may be argued that the patients attending our clinic had less advanced disease than those in the previous survey, which was of inpatients. All our patients, however, had AIDS, and all except four had been admitted to hospital at least once with conditions related to HIV infection.

Clearly, our results are appreciably different from those of Bower *et al* and further studies are needed to determine the true prevalence and importance of Dupuytren's contracture in patients with HIV infection.

- 1 Bower M, Nelson M, Gazzard BG. Dupuytren's contractures in patients infected with HIV. *Br Med J* 1990;300:164-5.
- 2 Revision of the CDC surveillance definition of the acquired immunodeficiency syndrome. *MMWR* 1987;36 (suppl):1-15.
- 3 Evans RA. The aetiology of Dupuytren's disease. *Br J Hosp Med* 1986;36:198-9.

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