

probably accelerated the fatal course in one of them.¹ The same cause might explain the outcome in other cases of fatal side effects of valproate.¹ The families of such children need to be investigated because (a) a common cause of fatal valproate toxicity might be found, (b) a procedure to detect people at risk might be advised—for example, measuring plasma ammonia concentrations for initial monitoring of treatment—and (c) confidence about using the drug for children and adults without a metabolic abnormality might be restored.

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(Accepted 18 September 1985)

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Apheresis in the management of loiasis with high microfilariaemia and renal disease

Membranous glomerulopathy is sometimes associated with loiasis,¹⁻³ a common disease in western and central Africa. We describe a case in which renal symptoms improved after treatment of loiasis with apheresis followed by diethylcarbamazine.

Case report

A 28 year old man, who had been born in Cameroon but had lived in France from 1976, presented with recurring episodes of itching. Loiasis was diagnosed with microfilariaemia of $35 \times 10^6/l$. The eosinophil count was $1.6 \times 10^9/l$. Stools and urine were negative for parasites; results of serological tests were negative for syphilis, hepatitis B surface antigen, and schistosomes and positive for filarias and malaria. Serum creatinine concentration was $122 \mu\text{mol/l}$ ($1.4 \text{ mg}/100 \text{ ml}$). Urine

Effects of three successive sessions of apheresis on microfilariaemia

	Microfilaria count ($\times 10^6/l$)	
	Before apheresis	After apheresis
Day 1	30.1	22.3
Day 2	26.1	15.5
Day 3	16.8	7.1

contained albumin (2.8 g/24 h) and red blood cells ($25 \times 10^3/\text{min}$) but no leucocytes. Serum complement concentrations (C3, C4) were in the normal range. Renal biopsy showed membranous glomerulitis with microfilarias in the glomerules and the vessels as described in other cases^{1,2}; immunofluorescence microscopy showed IgG, IgM, and C3 in a diffusely granular pattern without IgA. To avoid massive lysis of microfilarias the microfilariaemia was reduced by apheresis (Haemonetics V50 blood processor, Hemonetics, Massachusetts, USA) before treatment with diethylcarbamazine. The standard procedure for collection of platelets was followed by centrifugation at 4800 rpm. About 3.2 litres of blood were processed in seven cycles during each session of apheresis. Three sessions were performed on consecutive days at midday, when the microfilariaemia was highest. The table shows effect of the three sessions on the number of

microfilarias in the blood. About 2.8×10^8 microfilarias were extracted during the three sessions. This procedure was well tolerated with the platelet count falling from 383 to 140×10^9 platelets/l.

He was then treated with diethylcarbamazine, starting with low doses (5 mg/day) that were progressively increased; prednisolone (0.5 mg/kg/day) was added for the first few days. Transient moderate itching occurred. After 15 days of treatment (diethylcarbamazine 400 mg/day) no microfilarias were detectable. After nine months (diethylcarbamazine 100 mg/day) microfilarias remained undetectable, the haematuria had cleared, and proteinuria ranged between 0.4 and 1.0 g/24 h. Serum creatinine concentration was $114 \mu\text{mol/l}$ ($1.3 \text{ mg}/100 \text{ ml}$).

Comment

In loiasis with high microfilariaemia diethylcarbamazine may be responsible for serious side effects such as encephalitis^{4,5} due to the lysis of microfilarias, and the risks of treatment can be more important than the risks associated with the disease. Loiasis with visceral complications such as renal or cardiac conditions, however, should be treated. In our patient the loiasis was treated because it was considered to be the cause of the membranous glomerulopathy and we hoped to relieve the renal symptoms. Glomerulopathy associated with loiasis is believed to be immunologically mediated¹⁻³; massive lysis of microfilarias at the start of treatment with diethylcarbamazine induces an antigen overload that could worsen renal symptoms, and so the microfilariaemia should be reduced before specific chemotherapy. In 1983 Muylle *et al* first reported the success of apheresis in reducing microfilariaemia in two cases,⁵ showing that microfilarias are concentrated in the buffy coat by discontinuous flow centrifugation and can be extracted with a blood cell separator. To our knowledge this technique has never been used in patients with loiasis and renal disease. In this case, with a follow up of nine months, we observed an improvement in urinary sediment and stabilisation of renal function. Further observations are warranted to confirm the benefit of specific treatment for renal symptoms in the management of such patients.

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(Accepted 21 August 1985)

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Renal artery occlusion in patients with renovascular hypertension treated with captopril

In patients with renal artery stenosis reversible renal failure of the kidney on the affected side may occur during treatment with captopril. We describe three patients with atherosclerotic renal artery stenosis who developed occlusion of the affected renal artery during treatment with captopril while awaiting percutaneous transluminal angioplasty. This was preceded by long term treatment with captopril (13-21 weeks), which is said to guide prediction of curability.

Case reports

Case 1—A 62 year old man after 10 years of severe hypertension (235/135 mm Hg) seemed to have 75-90% stenosis of the left and minimal stenosis of the right renal artery. Renal function was unimpaired (plasma creatinine concentration $89 \mu\text{mol/l}$ ($1.01 \text{ mg}/100 \text{ ml}$), creatinine clearance $100 \text{ ml}/\text{min}$) (table). Treatment