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DISCONTINUATION OF CHRONIC HAEMODIALYSIS AFTER CONTROL OF ARTERIAL HYPERTENSION; LONG TERM FOLLOW-UP

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Summary

Five patients with varying chronic renal diseases had to be started on haemodialysis with malignant hypertension. After several months, dialysis treatment could be interrupted when long term blood pressure control had been attained. In three of them no long term complications were observed and renal function has continued to improve. In the two other patients, uncontrolled hypertension or acute infection made a return to haemodialysis imperative. In the case of malignant hypertension, the arteriolar necrosis has been shown to be reversible.

Introduction

The initiation of treatment of arterial hypertension generally induces a transient decline of renal function through a reduction in renal perfusion. This has led to the generalised concept that in patients with renal failure hypertension should not be treated aggressively or not be treated at all [1]. Bilateral nephrectomy has been proposed for most of those patients. Recently, a few cases have been described where an improvement of renal function became evident after blood pressure had been brought under control [2–6]. In most of those cases, renal failure was only moderate.

This report concerns the long term follow-up of five patients started on chronic haemodialysis after developing malignant hypertension and where improvement in renal function allowed the discontinuation of haemodialysis several months later. The short term outcome of four of these patients has been reported previously [7].

Patients

Relevant clinical, biochemical, and therapeutic details concerning those patients are given in Table I.

TABLE I

	Case 1	Case 2	Case 3	Case 4	Case 5
Age	52	46	31	64	28
Diagnosis	Scleroderma	Nephro-angiosclerosis	Chronic glomerulonephritis	Nephro-angiosclerosis	Interstitial nephritis
Hospital admission:					
BP (mmHg)	250/130	265/150	190/140	290/180	230/160
Diuresis (ml/d)	500	1100	50	470	290
Creatinine (μmol/L)	1150	1500	1115	1150	1330
Antihypertensive therapy (mg/d)	Sodium nitroprusside Propranolol 320 Frusemide 500	Sodium nitroprusside Propranolol 80 Frusemide 500	Hydralazine Clonidine Atenolol 100 Frusemide 500	Sodium nitroprusside Propranolol 240 Frusemide 250	Sodium nitroprusside Clonidine 0.450 Propranolol 320 Frusemide 250
Duration of haemodialysis	20 months	2 months	4 months	1 month	30 months
Withdrawal dialysis:					
BP (mmHg)	120/90	165/105	120/90	120/75	140/90
Diuresis (ml/d)	1200	5060	1500	1100	1200
Creatinine (μmol/L)	590	335	250	815	510
Return to dialysis:					
After	–	–	–	31 months	8 months
BP (mmHg)	–	–	–	210/120	140/80
Creatinine (μmol/L)	–	–	–	1500	1400
August 1982:					
Duration from withdrawal of haemodialysis	54 months	67 months	45 months	–	–
BP (mmHg)	165–90	135/95	135/90	140/85	140/100
Diuresis (ml/d)	2000	4200	1500	400	300
Creatinine (μmol/L)	225	210	130	–	–
Antihypertensive therapy (mg/d)	Atenolol 100 Frusemide 20	Atenolol 100 Frusemide 250 Dihydralazine 75	Atenolol 100 Frusemide 20	Captopril 50	Captopril 100 Atenolol 100

Case 1

This woman, aged 52, suffered from cutaneous manifestations of scleroderma for four years. Renal function and blood pressure were normal 10 months before admission. In 1976 acute pulmonary oedema related to malignant hypertension developed. Blood pressure at 250/150mmHg, stage IV retinopathy and plasma renin activity (PRA) of 201ng/ml/h for natriuresis of 62mmol/24h were found. Hypertension could be brought under control with sodium nitroprusside and propranolol. She had to be started on haemodialysis for uraemic pericarditis. While blood pressure was continuously controlled, pre-dialysis serum creatinine values progressively declined despite reduction of dialysis treatment times. After 20 months of haemodialysis renal function had recovered to such a degree that we decided to stop haemodialysis treatment. Serum creatinine decreased to the present level of 225μmol/L, more than four years after the interruption of dialysis. Blood pressure is still controlled by beta blocking agents and frusemide.

Meanwhile cutaneous manifestations of scleroderma have regressed and Raynaud's phenomenon have disappeared.

Case 2

This patient, aged 46, was suffering from dizziness and vomiting for many months before admission. In December 1977 he was hospitalised with a blood pressure of 260/150mmHg and stage IV retinopathy. Serum creatinine was 1500 μmol/L, Hb 64g/L, and PRA 15ng/ml/h for a natriuresis of 76mmol/24h. A diagnosis of nephroangiosclerosis was made. Blood pressure could be controlled with sodium nitroprusside, propranolol and frusemide. After a few days haemodialysis had to be started for gastrointestinal problems (serum creatinine 1600 μmol/L). With prolonged blood pressure control, pre-dialysis serum creatinine progressively declined and haemodialysis could be discontinued two months later. The addition of dihydralazine to the previous therapy was necessary a few months ago due to a sudden increase of arterial hypertension. At this time, five and a half years since we stopped haemodialysis, serum creatinine is 220 μmol/L.

Case 3

This patient, aged 31, was suffering in August 1977 from oedema and hypertension of 170/130 which was left untreated. One year later acute pulmonary oedema and hypertension at 190/140 with stage IV retinopathy were noted and clonidine was given. The diagnosis of glomerulonephritis was confirmed by renal biopsy. Renal failure (serum creatinine at 1115μmol/L) necessitated the initiation of dialysis. Progressively pre-dialysis serum creatinine values decreased despite the reduction in time and frequency of the dialysis sessions. After 4 months haemodialysis could be interrupted. Serum creatinine further decreased to 200μmol/L; at that time the patient was found to be pregnant despite medical advice. During this pregnancy, serum creatinine surprisingly decreased and blood pressure remained under control by the continuation of previous treatment, frusemide

and atenolol. After 34 weeks of pregnancy complications and increasing blood pressure made caesarian section imperative and a healthy child was delivered. Two years later the mother and child are doing well. Blood pressure is still controlled with frusemide and atenolol.

Case 4

This patient, aged 64, was suffering from arterial hypertension for many years with a systolic blood pressure of 240mmHg which was left untreated. In February 1978, with a blood pressure of 290/180 acute pulmonary oedema appeared. The diagnosis of nephroangiosclerosis was made. Blood pressure could be controlled with sodium nitroprusside, then propranolol and frusemide. Haemodialysis had to be started for uraemic pericarditis and severe gastrointestinal symptoms. After one month, while blood pressure remained controlled, haemodialysis could be interrupted. Following this the serum creatinine continued to drop. During subsequent months a high blood pressure was noted each time the patient came to the clinic but he claimed his blood pressure was normal at home and that he did not tolerate the increase in the dosage of frusemide. He finally accepted hospitalisation in order to start captopril treatment when it became apparent that his renal failure had increased to such a degree that haemodialysis had to be resumed. The patient has now been on haemodialysis for the past 21 months.

Case 5

This patient, aged 28, with chronic interstitial nephritis due to vesico-ureteral reflux, had to be started on haemodialysis in 1977 with malignant hypertension and severe uraemia. Blood pressure was difficult to control but after 18 months haemodialysis could be interrupted. Seven months later this non-compliant patient developed acute pyelonephritis which was left untreated for several days. Haemodialysis had to be resumed a few weeks later. She has now been on haemodialysis for the past 18 months.

Discussion

Withdrawal of haemodialysis due to recovery of renal function after control of malignant hypertension has been possible in five of our patients. They had varying renal diseases and direct or indirect evidence of having had renal failure for more than six months prior to admission. They recovered sufficient renal function to stop haemodialysis after 1–30 months of treatment.

All those patients had malignant hypertension in whom the late recovery seems to be greater than in patients with severe renal failure due to benign nephroangiosclerosis [6]. It has indeed been shown that the acute process of arteriolar necrosis, characteristic of malignant hypertension, can be reversed once blood pressure has been brought under control [7].

After withdrawal of dialysis, renal function in four of the five patients continued to improve. This supports the hypothesis that reducing the blood pressure to normal interrupts the vicious circle between aggravation of renal function and

acceleration of hypertension. In three of them no long term complications were observed and renal function is now stable at serum creatinine values between 130 and 225μmol/L four to six years after the withdrawal of dialysis. One of those patients has even had a normal pregnancy (nearly to full term) and a healthy child was delivered.

However dialysis may have to be resumed as in our two other patients when complications occur: untreated arterial hypertension or acute renal infection, represented triggering mechanisms leading to irreversible renal damage.

Our experience combined with the other reported cases [2, 5, 6] shows that in patients with malignant hypertension, bilateral nephrectomy is definitely obsolete. Blood pressure can be brought under control with the modern drugs available. The chance to recover renal function afterwards is not negligible since five out of 16 patients with malignant hypertension have stopped dialysis in the last six years in our group and similarly four out of nine in the series of Mitchell [6].

In *conclusion* those five observations seem to indicate that:

1. Strict blood pressure control in patients with end-stage renal disease may have a prolonged beneficial affect on renal function.
2. Improvement in renal function may occur even after several months of haemodialysis in patients with malignant hypertension.
3. Renal function may continue to improve after interruption of haemodialysis if blood pressure is kept under control and other renal complications are avoided.
4. Bilateral nephrectomy should not be performed for malignant hypertension.

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Open Discussion

RITZ (Heidelberg) Of course, Dr Wauters, the observation is not new that patients in the malignant phase of hypertension can regain renal function sufficient that they may be taken off dialysis, and I can confirm this from my own clinical experience. There is however one aspect of your observation which deserves comment. You are certainly aware of the concept of Brenner that glomerular hyperperfusion causes progressive renal damage. One observation often quoted in this context is Jean-Pierre Grunfeld's observations from Hôpital Necker, Paris, that after a limited period of time such patients described above return to dialysis again after several months, your observation extending to five years, of no progression of renal failure is, therefore, notable. As in the well-known detective story it is perhaps the dog which did not bark who provides the most important clues.

WAUTERS The protein restriction of our patients was not very severe. They were advised to take a diet consisting of 1g protein/Kg/day but I must say that one or two of these patients were so happy they could stop dialysis that they continued a very severe restricted diet, perhaps this could be one of the reasons for the persisting amelioration.

DRUEKE (Paris) I am concerned about the possibility that in some patients, at least one of your patients, blood pressure might not be perfectly controlled. Would you retrospectively regret that you had interrupted the dialysis treatment in this patient in whom hypertension was not well controlled, since in this patient, the cardiovascular risk is particularly high? She might be at risk for myocardial infarction later?

WAUTERS Yes, exactly. That was the reason why we kept her on haemodialysis for such a long time, in fact her creatinine values were not that high but we continued haemodialysis for many months to obtain a better control of hypertension. Once we had the impression, after starting Captopril treatment, that her hypertension was better controlled then we decided to interrupt. Now this patient continued to have a steady creatinine value until she developed an acute infection which I think was the factor which made dialysis necessary again.

LA GRECA (Vicenza) What was the residual renal function in your patients starting dialysis?

WAUTERS They were all anuric or oliguric, the greatest urine output in one patient was 400ml/24 hours. All the others were less. When we stopped dialysis the mean urinary output was around 1L/24 hours.