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Teaching Point
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Malignant hypertension with protracted but not definitive oligoanuric acute kidney failure

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Introduction

Malignant hypertension (MHTN) is diagnosed when a patient presents with accelerated hypertension and multi-organ compromise including severe retinopathy. The renal manifestations may be oliguric renal failure or rapidly progressive renal failure. Oliguria in MHTN occurs as a consequence of malignant nephrosclerosis, microangiopathic haemolytic anemia, polyarteritis nodosa, lupus, acute cortical necrosis and scleroderma. However, acute kidney insufficiency (AKI) may be seen in the absence of systemic disease or of nephropathy.

The clinical presentation of anuria with MHTN is rare, and in such situations renal recovery is unlikely. There are few case studies reporting reversible renal failure in MHTN [1–16].

A literature search supports the view that the entity of reversible anuric renal failure in MHTN is recognized by early clinical presentation (with anuria/oliguria), the presence of normal sized kidneys, normal main renal arteries and kidney biopsy without significant vascular changes.

Herein, we report two such cases with anuria due to MHTN who recovered after remaining on haemodialysis for an average 2 months.

Case reports

A 35-year-old man was admitted for management of MHTN and anuria. The blood pressure (BP) was 220/130 mm Hg. At admission, the blood urea nitrogen (BUN) was 120 µmol/L, serum creatinine 1229 µmol/L and haemoglobin 67 g/L. An ultrasound examination showed normal sized kidneys. Serology for anti-nuclear antibodies (ANA), anti-dsDNA, C3, C4 and anti-scl-70 was normal. There was no evidence of haemolysis on a peripheral blood smear. A renal angiogram did not show stenosis at the main renal vessels or branch segments. There was no cortical perfusion. A kidney biopsy showed hypertensive changes in the vessels. There was no evidence of fibrinoid necrosis or proliferative endarteritis. The patient's BP could be controlled with five antihypertensive drugs which included clonidine (0.8 mg/day), minoxidil (10 mg/day), torsemide (40 mg/day), long-acting nifedepin (90 mg/day) and prazosin (20 mg/day).

The BP was maintained at 130/80 mm Hg. He remained anuric for 17 days. After 7 weeks of dialysis, the urine output amounted to 4 L/day and haemodialysis could be stopped. At the end of 3 months off dialysis, his serum creatinine level was 274 µmol/L. Supportive treatment was continued with amlodepin (2.5 mg/day).

Case 2: A 30-year-old man was admitted with MHTN and anuria. The BP was 200/120 mm Hg. The BUN was 96 µmol/L and serum creatinine was 742 µmol/L. An ultrasound showed normal sized kidneys. Serology for ANA, anti-ds DNA, anti-scl-70, C3 and C4 was negative. There was no evidence of haemolysis on a peripheral blood smear. The renal angiogram was normal. The main renal arteries showed normal perfusion. But the cortical perfusion was absent. The renal biopsy disclosed only features of hyperplastic arteriosclerosis. There was no evidence of fibrinoid necrosis. Immunofluorescence was negative. The patient's BP could be controlled with four antihypertensive drugs which clonidine (0.8 mg/day), minoxidil (7.5 mg/day), torsemide (40 mg/day), long-acting nifedepin (60 mg/day). With these drugs, the BP was normalized at 120/80 mm Hg. After 8 weeks of haemodialysis, improvement in urine output and renal function was noted and haemodialysis was stopped. At discharge, his urine output was 3.5 L/day and serum creatinine was 318 µmol/L. Supportive treatment was continued with amlodipin (2.5 mg/day).

Both patients had a similar presentation of acute renal failure with oligoanuria. There was no evidence of bilateral renal vascular occlusion. The clinical parameters did not give clues for an aetiology of MHTN. The angiogram of the renal vessels ruled out renal vascular hypertension. On histopathology vascular changes suggestive of MHTN

were not present. At the end of 6–8 weeks, both patients showed improvement and remained dialysis independent at a 6-month follow-up after discharge.

Discussion

MHTN is a clinical syndrome characterized by very high diastolic BP, along with hypertensive retinopathy and multiorgan compromise. MHTN may present as rapidly progressive renal failure and rarely as acute oligoanuric renal failure. Renal recovery is thought to be unlikely and patients are prepared for definitive renal replacement therapy (RRT).

However, some cases of unexpected renal recovery in anuric MHTN have been reported since 1971 [11], particularly in black patients of African descent. Following a period of maintenance haemodialysis that lasted from 2 months to more than 2 years, renal function resumed allowing cessation of dialysis. This unexpected recovery, although often partial but sufficient to stop RRT, was ascribed to intensive antihypertensive treatment with vasodilators and particularly minoxidil. These reports indicate that anuria in MHTN is not the consequence of cortical necrosis but of a protracted period of renal vasoconstriction. This is reminiscent of oligoanuric renal failure occurring in the hepato-renal syndrome where angiography discloses intense renal vasoconstriction and a renal blood flow that does not reach the cortex [17]. That this vasoconstriction can be reversible has been shown in a few observations of kidneys harvested in cirrhotics who died of final complications and kidneys that were transplanted in a non-cirrhotic environment and resumed diuresis. In a few cases, such as the one reported by Meyrier *et al*. in 1990, angiography carried out at the time of anuria showed that, similarly to the hepatorenal syndrome, the renal circulation was almost abolished and did not reach the cortex. (Figure 1a). The radiologist's diagnosis was that of 'complete cortical necrosis'. This visual interpretation was refuted by a kidney biopsy that did not show significant arterial lesions, following a period of haemodialysis of 15 months and drastic control of hypertension, by progressive improvement in renal function allowing to stop dialysis treatment. At that time, a new renal angiography still showed a very abnormal appearance of the renal vasculature but the circulation reached the cortex (Figure 1b). This led to an interpretation proposing that this type of AKI is more functional than organic despite its unusual duration.

The matter of its pathophysiology remained and is still speculative. An increased sympathetic activity did not seem likely. Neither was the role of intense production of angiotensin 2 a satisfactory explanation as converting enzyme inhibitors played no role in improving renal function, despite the fact that initially plasma renin activity was extremely high. Endothelin appeared to be a possible suspect but could not be measured.

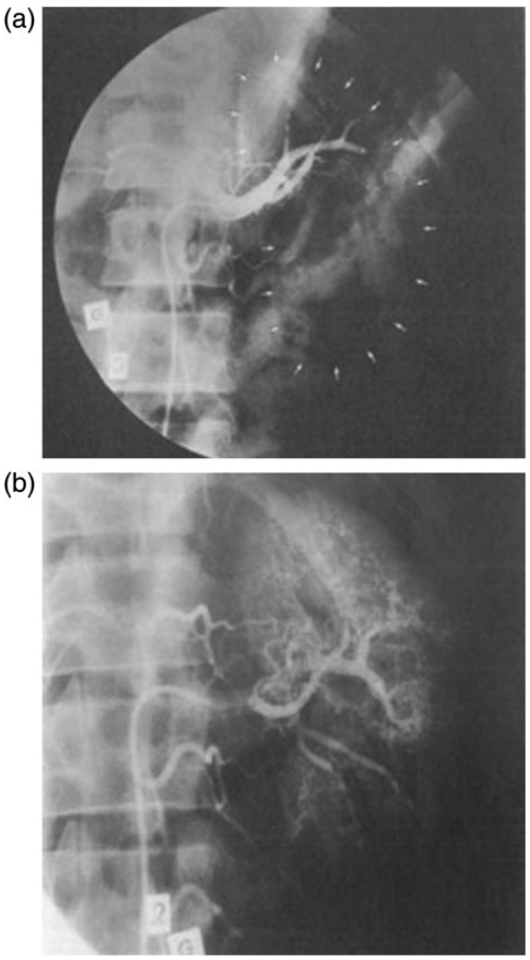

Fig. 1. (**a**) Renal angiogram showing intense renal vasoconstriction in a 27-year-old black male with MHTN and anuria. The contrast medium opacified only the main renal artery and its initial branches. (**b**) Renal angiogram of the same patient after the onset of diuresis. There was reappearance of renal circulation to the cortex, although with very abnormal arteries (Reproduced with permission from the author and Oxford University Press, NDT (5); 174–178:1990) (License agreement n°: 2863161255119).

Teaching points

(i) MHTN can be complicated by AKI despite absence of irreversible lesions of the renal tissue.
(ii) This unusual form of functional renal insufficiency is due to an intense renal vasoconstriction with an arrest of the circulation to the cortex.
(iii) This form of AKI may require months of dialysis before the time to recovering renal blood flow allows a return to supportive treatment.
(iv) Therefore, a decision of kidney transplantation should be postponed.
(v) Treatment of hypertension seems to be based more on vasodilators than on angiotensin antagonists.

Conflict of interest statement. None declared.

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