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Reversal of Acute Renal Failure by Kidney Revascularisation

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Objectives: *To assess whether acute renal failure, due to total or subtotal renal artery occlusion, can be reversed by kidney revascularisation.*
Design: *A retrospective review of surgery for kidney salvage in anuric patients at a University Hospital.*
Methods: *From 1983 to 1993, eight patients were operated on for occlusive renal artery disease as a cause of acute renal failure, requiring preoperative haemodialysis. On admission the mean serum creatinine was 40 mg/l (354 μmol/dl). The oligoanuria lasted from 12 h to 3 weeks. Renal length of 8 cm or more and visualisation of a patent distal renal artery branches on aortography were arguments that return of renal function could be expected after revascularisation of these non-functioning kidneys.*
Results: *Revascularisation restored immediate urine flow in six cases, with no further need for dialysis in four. Two patients remained oliguric despite successful reperfusion. One of them could be weaned from dialysis after 1 month. Two patients died postoperatively. Five of the eight patients left the hospital with restored renal function.*
Conclusions: *Patients with acute renal function deterioration due to ischaemia of a single or both kidneys can benefit from prompt revascularisation, with significant recovery of renal function in most of them.*

Key Words: Acute renal failure; Renal artery thrombosis; Renal artery surgery; Renal revascularisation.

Introduction

Unexplained abrupt onset of oliguria in an elderly vascular, hypertensive patient should always raise suspicion of sudden progression of renal artery disease to critical stenosis or thrombosis. Once urinary tract obstruction has been excluded by echography, work-up should always include emergency angiography as a definite diagnostic procedure, even though there has been some reluctance to use nephrotoxic contrast medium in oliguric patients.

In a study by Schreiber *et al.*[1] who followed-up 85 renovascular patients, 16% of the stenotic lesions ultimately evolved towards total occlusion over a mean follow-up period of 52 months, as assessed on sequential angiograms. Gradual progression to renal artery thrombosis often remains subclinical, when the controlateral kidney ensures normal function.[2,3] Prompt revascularisation can reverse renal failure and offers the only alternative to permanent dialysis in these fragile patients. Morris *et al.*[4] was one of the first clinical investigators to obtain return of renal function by renovascular repair of totally occluded arteries supplying non-functioning kidneys. Many other short series or case reports[5–14] illustrate that even delayed revascularisation may re-establish renal function. We describe our experience with eight such cases.

Material and Methods

A retrospective review of our vascular surgery registry for last 10 years (1983–1993) identified revascularisation of 11 ischaemic kidneys in eight patients presenting with acute anuria. There were two women and six men, ranging in age from 55 to 86 years (mean 68.7 years). This represents 7.6% of all renal surgical revascularisations ($n = 105$) in our department during the same period. In four patients, the acute renal insufficiency was caused by total ($n = 3$) or subtotal ($> 90\%$ stenosis) ($n = 1$) occlusion of the main renal artery of a solitary functioning kidney (three previous nephrectomies (cases 2, 7 and 8) and one shrunken opposite kidney (case 3)). One of these renal artery thromboses was due to dissection after failed percutaneous angioplasty on a single kidney (case 2). Four other patients had bilateral subtotal ($> 90\%$ stenosis) (cases 5 and 6) or total (cases 1 and 4) occlusion of the

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renal arteries. Two of them were referred from elsewhere with acute anuria after aortoiliac surgery (cases 4 and 6). Angiography revealed bilateral occlusion in one and bilateral tight stenosis in the other. Another presented with a Leriche syndrome (aortic thrombosis) involving both renal arteries (case 1). In total, 12 kidneys were at risk. The use of angiotensine converting enzyme (ACE) inhibitors could have precipitated the acute renal failure in two of these eight patients (cases 5 and 8).

All patients suffered ischaemia-induced acute renal failure, requiring preoperative haemodialysis to correct electrolyte balance or impending fluid overload (pulmonary oedema) (cases 4 and 5). They presented with sudden onset of oligoanuria, with an urine output of less than 100 cc per day. On admission, the mean serum creatinine attained 40 mg/l (354 μmol/l) (extremes 23–62 mg/l). All patients were known to be hypertensive. The hypertension was medically controlled in four and drug resistant (diastolic blood pressure above 95 mmHg despite optimised medical therapy) in the other four patients. Recent deterioration of hypertension occurred in half of the cases. Three patients presented acute pulmonary oedema due to fluid overload, that responded well to dialysis. Data on baseline renal function one month or more before the obstructive episode are available for seven of the eight patients. Five had impaired renal function (serum creatinine > 20 mg/l) (177 μmol/l), and two had normal serum creatinine values. For the patient with an acute aortic thrombosis extending to the renal arteries, no previous creatinine values were available.

All patients had preoperative angiography. As already stated, there were three single kidneys and one shrunken opposite kidney. In total there were eight occluded renal arteries (including the chronic arterial occlusion of the shrunken kidney) and five tight stenoses. On delayed films, intrahilar arterial branches of four occluded renal arteries were opacified by filling from perirenal collaterals. A parenchymal nephrogram was not seen in four kidneys (three patients). Isotopic renal flow scan revealed residual perfusion in two of these non-visualised kidneys. In the patient with Leriche syndrome involving both renal arteries, the kidneys could not be visualised by either method. The renal height was always more than 8 cm as estimated on angiography or by echography. Extrarenal atherosclerosis was present in all patients (aortoiliac 5, coronary 4 and cerebrovascular 3).

Renal artery repair was performed 12 h to 3 weeks after the onset of acute renal failure. The operations performed included venous aortorenal bypass to two single kidneys, bilateral aortorenal bypass in two patients (the two patients with acute anuria after previous aortoiliac surgery), aortorenal thrombendarterectomy in three patients (associated nephrectomy of a shrunken kidney in one), and bilateral thrombectomy in the patient with aortic thrombosis. Seven of the eight occluded renal arteries were repaired. Distal renal artery patency had been maintained through a network of collateral vessels as assessed by intraoperative Doppler before arterial repair. During the procedure, profuse back bleeding from the patent distal renal artery was evidenced in all cases.

Results

Renal revascularisation restored immediate urine flow during or soon after the operation in six patients with no need for postoperative dialysis in four (cases 2, 4, 5 and 7). Dialysis was necessary in the two others who unfortunately died postoperatively. One died from multiorgan failure on the tenth postoperative day while still on dialysis, the other had fatal myocardial infarction on the third postoperative day. Two patients remained oliguric. Anuria subsided progressively by day 5 in one patient who required dialysis for a further 4 weeks. The other patient remained dialysis dependent. In that patient, control angiography revealed early thrombosis of the renal artery repair. No reoperation was planned because of patient's critical status (postoperative myocardial infarction). Overall operative morbidity included pulmonary infection necessitating prolonged intubation in two patients and one non fatal myocardial infarction.

At discharge (six patients), one patient was dialysis-dependent (case 3) and five patients (cases 1, 2, 4, 5 and 7) recovered renal function. At first follow-up visit (6 weeks), the five patients with recovered renal function had a mean serum creatinine of 18 mg/l (160 μmol/l) (16–25 mg/l). Hypertension was cured in two of them (diastolic blood pressure < 90mmHg without medication) and improved in the three others (blood pressure control with less medication). The one patient who remained on dialysis continued to have drug-resistant hypertension.

Complete follow-up data are available for the six survivors. During the follow-up period (mean 39 ± 17 months), three patients died. One of them was the patient requiring long-term dialysis postoperatively. He died within the first year from myocardial infarction. Another patient evolved progressive renal failure in the fourth postoperative year, despite angiographic evidence of graft patency. He ultimately became

Table 1.

No	Age (years)	Sex	Left kidney	Right kidney	Preoperative creatinine (mg/l) (x 8.84 μmol/l)	Operative technique	Preoperative dialysis (days)	Postoperative creatinine (mg/l) (x 8.84 μmol/l)	Postoperative dialysis	Operative outcome	Follow-up
1	64	Male	100% (Leriche syndrome)	100% (Leriche syndrome)	37	Thrombectomy x 2 + Ao Bif bypass	3	35	+ (4 weeks)	Postoperative pneumonia	Evolved to permanent dialysis 4 years later, † fatal stroke (52 m)
2	55	Male	100% (failed PTA)	Previous nephrectomy	39	TEA**	2	17	–	Uneventful	Well and alive
3	72	Female	100% (shrunken kidney)	99%	62	TEA + nephrectomy	21	55	+ (permanent)	Postoperative myocard infarction - postoperative ren art thrombosis	† 11 months later (infarction)
4	60	Female	100%	100% (ARI* after Ao aneur surg.)	23	Ao Ren bypass x 2	1	16	–	Uneventful	Well and alive
5	73	Male	95%	100%	29	TEA x 2	3	19	–	Postoperative pneumonia	† 2 years later (infarction)
6	57	Male	99%	99% (ARI after aortic surg.)	46	Ao Ren bypass x 2	1	60	+	†Multiorgan failure (10th postop day)	–
7	82	Male	100%	Previous nephrectomy	45	Ao Ren bypass	1	18	–	Uneventful	Reintervention 3 years later (acute graft thrombosis), well and alive
8	86	Male	Previous nephrectomy	99%	38	Ao Ren bypass	8	40	+	†Myocard infarction (3rd postop day)	–

*ARI= acute renal insufficiency.
**TEA= thrombendarterectomy.
†= death

dialysis dependent, and died 8 months later from fatal stroke. The third death was due to fatal myocardial infarction 2 years after successful kidney revascularisation. The survival ranges from 11 months to 6 years (mean 3.5 years). One of the patients still alive became oliguric again in the third postoperative year, due to an acute thrombosis of the aortorenal bypass graft to his single kidney. He was successfully reoperated. For three other patients the recovered renal function remained stable throughout follow-up.

Discussion

Acute thrombosis of atherosclerotic renal arteries causing oliguric renal insufficiency, does not mean an irreversible loss of renal excretory function. Some preoperative markers are useful in predicting cases in which retrieval of renal function is achievable : kidney size of at least 9 cm length,[2,5–7,14,16–18] residual isotopic glomerular filtration,[7] disease-free distal renal artery[3,7,16,17] on late angiogram or evidenced by echo-Duplex, or renal biopsy.[2,3,5,13] Kidney size and a patent renal artery beyond the main stem occlusion are the most reliable predictors. Isotopic function tests lose much sensitivity in poorly perfused kidneys and intraoperative biopsy is questionable since it offers focal information and will be normal if other criteria are met. The acute occlusion may be secondary to thrombus apposition on an irregular plaque, intraplaque haemorrhage (sudden progression of the stenosis)[12] or iatrogenic trauma after balloon angioplasty (plaque dissection).[19] Other possible causes are surgically induced hypotension[12,14] that creates low flow across the stenosis and favours *in situ* thrombosis, typically in an immediate postoperative setting characterised by a thrombogenic state or acute intrarenal hypotension with decreased renal blood flow due to angiotensin converting-enzyme inhibitors.[20,21] Extensive aortic thrombosis is another rare cause of acute bilateral renal artery occlusion.[6] In our series, a precipitating event preceded the development of anuria in four of the eight patients (one failed PTA, two aortic replacement procedures, and one Leriche syndrome). In most cases, the pre-existing renal artery stenosis had caused renovascular hypertension and progressive impairment of renal function during the years preceding the sudden onset of oligoanuria. There is typically an exacerbation of the hypertension, with escape from previous pharmacological control. Preoperative control of metabolic derangements or congestive heart failure is of utmost importance and may necessitate one or more sessions of haemodialysis prior to surgery.[8]

The extent of renal ischaemia is variable and depends on the magnitude of available collateral supply from ureteric, adrenal or lumbar arteries. The presence of collateral blood flow, stimulated by preexisting renal artery stenosis, improves the tolerance to persistent ischaemia of these non-functioning kidneys. The critical hypoperfusion at subfiltration pressure by way of capsular collaterals is sufficient to preserve viability of the glomeruli, even for prolonged periods, but is inadequate to ensure renal excretory function.[5,8–10,22] This has been experimentally demonstrated by Morris *et al.*[23] Perfusion pressures as low as 20 mmHg offer protection from parenchymatal anoxic injury but are suboptimal for urine production. The renal tubuli are more prone to ischaemic lesions, but also have a marked regenerative potential once normal kidney perfusion is restored. Some authors report functional recovery after delayed restoration of renal blood flow, even after 1 or more months of anuria.[2,5,7–9,11,15,22,24] The delay for recovery of renal function varies from some minutes to some months.[7,8,11,14,24] Early operation tends to limit the severity of postoperative tubular dysfunction. In six of our patients, urine flow resumed immediately after successful revascularisation, followed by progressive reversal of azotaemia. This illustrates the dramatic increase in glomerular filtration that can be obtained with reperfusion.

The conviction that non-functioning kidneys are salvageable brought Dean *et al.*[16,17,22] and others[3,14,18,21,22] to prefer revascularisation of chronically occluded renal arteries in hypertensive patients rather than removal of the non-functioning kidney of 9 cm length and without intrarenal disease. Dean *et al.*[22] obtained a 75% success rate, with six of eight dialysis-dependent anuric patients rendered dialysis-independent by kidney revascularisation. In the series of Scoble *et al.*[21] three of nine dialysis-dependent patients came off dialysis after the intervention. These results (70% success rate) are reasons to state that a potentially functional kidney may be found distal to a totally occluded artery and that revascularisation should be attempted. From these small series (less than 10 cases each), no difference appears for the response rate to revascularisation of spontaneous thrombotic occlusions or of traumatic or postoperative acute occlusions of preexisting stenotic renovascular lesions. However small series should be interpreted with caution.

This situation of reduced but not completely interrupted renal perfusion is totally different from acute total ischaemia after traumatic or embolic occlusion of

a previous normal renal artery, where renal dysfunction becomes irreversible after 90 min due to lack of collaterals.

The recovery of renal excretory function after repair of totally or subtotally occluded renal arteries is more spectacular than the effect of revascularisation of poor functioning kidneys with moderate stenosis.[2,7] In such cases there is a contributary irreversible nephrosclerotic parenchymal damage secondary to long-standing hypertension or to microembolic disease.

Percutaneous transluminal angioplasty has little role in the salvage of ischaemic kidneys, because of its bad results in cases of total occlusion or severe ostial stenosis[2,15,19] and its inherent risk of contrast-induced nephropathy. However Pattison *et al.*[10] obtained remarkable results (10 of 13 patients with acute renal failure could be weaned off dialysis after angioplasty).

Extrarenal atherosclerosis is common in elderly renovascular patients and accounts for their increased operative risk. The mortality for kidney revascularisation in anuric patients is about 15%.[21,22] However any effort to salvage an ischaemic poor functioning kidney is worthwhile. Failure to restore renal excretory function and ultimate dialysis dependency predisposes to early death during follow-up. Novick *et al.*[24] identified a group of 25 dialysed patients with end-stage renal failure due to advanced atherosclerotic renovascular disease. Eight of these patients underwent surgical revascularisation on the basis of criteria suggesting salvagable renal function. The operation was successful in all of them and their mean survival was 65 months compared to 32 months for non-operated patients who remained dialysis-dependent. This poor outlook for dialysed elderly patients is mainly due to accelerated generalised atherosclerotic vascular disease. Held *et al.*[25] observed a 34% annual death rate for dialysed patients of 65 years or older. In a series of Mailloux *et al.*[26] the 5 year survival for azotaemic elderly patients with uncorrected renal artery stenosis, who progressed to dialysis, was only 12%.

In conclusion, acute obstruction of an atherosclerotic renal artery is an uncommon but potentially correctable cause of renal failure in the elderly. A network of capsular collaterals may maintain a subcritical blood flow in the disease-free hilar branches assuring kidney survival despite arrest of renal excretory function. On the basis of criteria suggesting salvagable renal function, surgical revascularisation should be undertaken for non-functioning kidneys larger than 7 cm, with signs of residual perfusion on angiogram or scintigraphy. The patient's condition must be optimised preoperatively by one or more sessions of haemodialysis. Widespread atherosclerosis in these elderly patients accounts for an increased postoperative morbidity and mortality. However, surgery offers the only chance of kidney salvage, with a success rate of about 60%, and probably enhances late patient survival.

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