Role of late renal revascularization in functional renal salvage

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ABSTRACT

The duration that renal parenchyma will tolerate ischemia has continued to be debated. We have reported the cases of three patients who had undergone revascularization procedures with successful return of baseline renal function after prolonged renal artery occlusion of 14 days to 3 months. These cases highlight that aggressive revascularization can lead to successful renal salvage in selected patients. We examined the characteristics of these patients and those of others in the literature and reviewed the factors favoring recovery. (J Vasc Surg Cases Innov Tech 2022;8:121-4.)

Keywords: Functional salvage; Late renal revascularization; Renal artery occlusion

Renal artery occlusion is a common, but treatable, cause of renal infarction. Renal artery occlusions are generally categorized as acute (presenting within 24 hours of occlusion), subacute (presenting with 24 hours to 2 weeks), or chronic (presenting after >2 weeks). The most common symptoms include abdominal pain, hematuria, fever, new or worsened hypertension, and signs of kidney impairment.[1-3] The current guidelines have recommended timely revascularization with percutaneous endovascular therapy (thrombolysis, thrombectomy with or without angioplasty, or stent placement) if the occlusion has <6 hours' duration.[4-6] Considerable controversy has continued regarding the ischemic time the renal parenchyma can tolerate. As such, no clinical guidelines have addressed the best modality of treatment for those presenting outside the typical treatment window. We have reported three cases of complete renal recovery after successful treatment of prolonged renal artery occlusion with ischemic times ranging from 14 days to 3 months. All three patients provided written informed consent for the report of their case details and imaging studies.

CASE REPORT

Patient A. Patient A was a 48-year-old woman with a medical history significant for Takayasu arteritis, prior thoracic aorta to infrarenal abdominal aortic bypass for midaortic syndrome, and a bypass to the solidary left kidney. She had presented with acute renal failure, pulmonary edema, and uncontrollable hypertension requiring urgent dialysis. Abdominal computed tomography angiography revealed left renal bypass occlusion (Fig 1, *A*). However, renal perfusion imaging showed uptake of radiotracer by the left kidney, suggesting persistent perfusion from collateral vessels recruited secondary to chronic arterial stenosis. After an unsuccessful endovascular attempt, she experienced flush pulmonary edema and uncontrollable hypertension that required hemodialysis. A redo aortorenal bypass graft was performed 20 days after her initial presentation. The patient experienced a complete return of renal function and resolution of cardiopulmonary instability. Her creatinine (Fig 2) and systolic blood pressure had normalized to baseline before discharge, and she was no longer dialysis dependent. At 3 years of follow-up, the patient remained normotensive with normal renal function and a creatinine of 0.8 mg/dL.

Patient B. Patient B was a 73-year-old woman with a history of chronic renal disease and a baseline creatinine of 2.3 mg/dL. The patient also had a solitary functioning right kidney and a right renal stent. She had presented with complete right renal stent occlusion, and severe hypertension and hyperkalemia requiring urgent dialysis. Angiography confirmed complete occlusion. After an unsuccessful attempt at recanalization of the occluded right renal stent, the patient was deemed to have end-stage renal disease and a routine hemodialysis schedule was started. However, she presented 13 days later with a contained rupture of an abdominal aortic aneurysm. Preoperative magnetic resonance angiography showed opacification of the distal right renal artery despite stent occlusion (Fig 1, *B*). The patient underwent emergent open aortic aneurysm repair and right aortorenal bypass. Postoperatively, she experienced progressive improvement of her blood pressure and a return of creatinine to baseline (Fig 2). She no longer required dialysis by postoperative day 20.

Patient C. Patient C was a 70-year-old man with a medical history significant for fenestrated endovascular aneurysm repair 10 months prior was found to have progressive hypertension for

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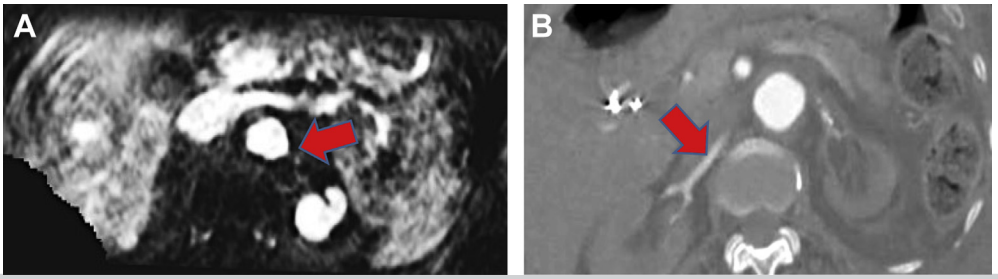

Fig 1. A, Abdominal computed tomography angiogram revealing origin of the occluded left aortorenal artery bypass (*red arrow*) of patient A. **B,** Magnetic resonance angiogram demonstrating a patent main right renal artery (*red arrow*) distal to an occluded stent of patient B.

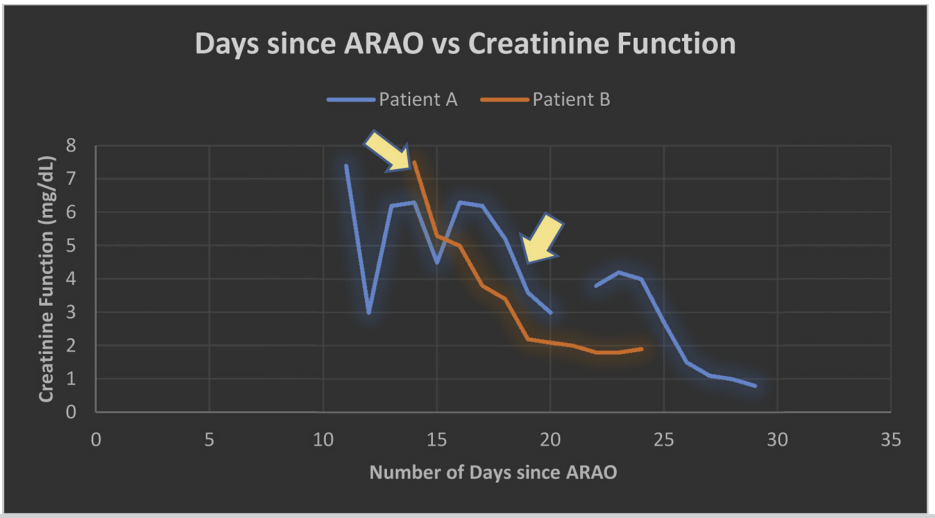

Fig 2. Changes in creatinine between patients A and B after acute renal artery occlusion (ARAO) and revascularization. Timing of revascularization indicated by *yellow arrows*.

3 months despite therapy with amlodipine, losartan, clonidine, and hydrochlorothiazide. His creatinine was 0.7 mg/dL; however, his plasma renin was elevated to 35 ng/mL/h. Computed tomography angiogram of the abdomen revealed complete occlusion of his left renal artery stent (Fig 3, *A*). He underwent successful endovascular recanalization of the left renal artery stent (Fig 3, *B*). Immediately after relining of the left renal stent, his blood pressure medication requirement decreased postoperatively, requiring only two antihypertensive agents, with significantly improved systolic blood pressure (Fig 3, *C*). At the 2-year clinical follow-up, the patient had stable blood pressure without requiring any antihypertensive medication.

DISCUSSION

No set guidelines are available for when to perform revascularization in patients with renal artery disease. One of the reasons is that these patients present in a variety of clinical settings, making it difficult to categorize the pathophysiology and duration of the occlusion from the clinical presentation and diagnostic features alone. We have reported the cases of three patients with acute symptoms of renal artery occlusion and an excellent response to delayed revascularization. We hoped to highlight the utility of pursuing salvage procedures in selected patients with a late presentation of acute symptomatic renal artery occlusion.

Although renal infarction will develop quickly after an acute occlusive event and revascularization has rarely been successful in salvaging kidney function, ischemic nephropathy can be a sequela of renal artery occlusion with preservation of full or partial renal function. Successful revascularization has been documented in patients with ischemic times of ≤48 hours.[7-10] Silverberg et al[8] reported better outcome for patients treated with catheter-directed thrombolysis for acute renal artery occlusion 48 hours after ischemia. Heidemann et al[9] reported function salvage 24 hours after acute occlusion of a renal stent after fenestrated endovascular aneurysm repair. Unlike previously reported series, our patients had undergone revascularization procedures at 20 days (patients A and B) and 3 months (patient C) after acute symptom onset and experienced complete functional rescue. These durations are well beyond the known reversible ischemic recovery time for acute renal artery

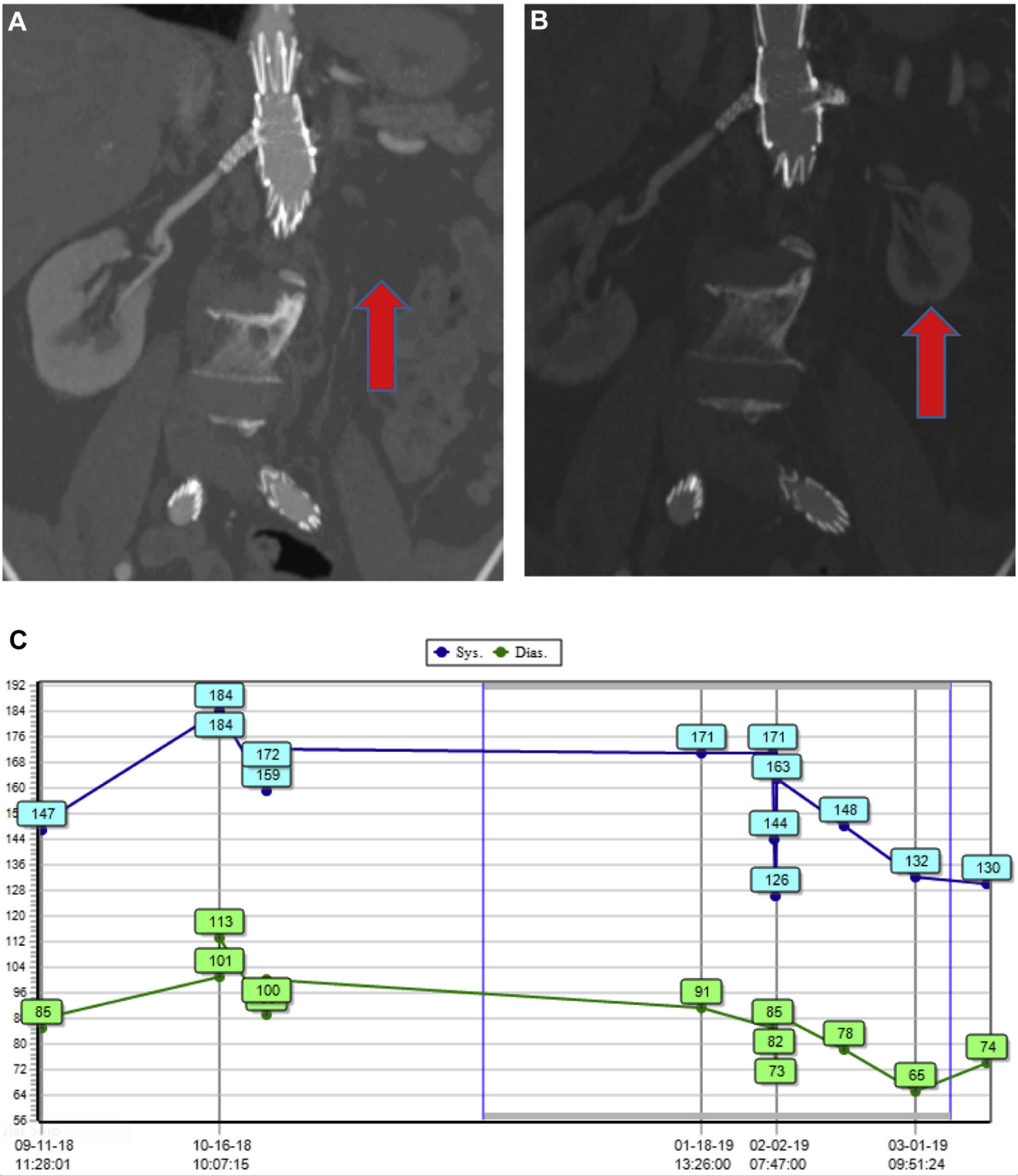

Fig 3. **A,** Coronal abdominal computed tomography scans demonstrating complete occlusion of the left renal artery stent and absence of perfusion of the left kidney (*red arrow*). **B,** Coronal abdominal computed tomography scan demonstrating opacification of the left kidney after endovascular recanalization (*red arrow*). **C,** Changes in systolic and diastolic blood pressure of patient C after acute renal artery stent occlusion and recanalization. *Dias.,* Diastolic; *Sys.,* systolic.

occlusion. We believe this might be related to the insidious nature of the renal artery occlusion despite the acute onset of symptoms.

Revascularization in cases of chronic ischemic nephropathy has had mixed outcomes of meaningful functional recovery, with no clearcut guidelines for when to perform revascularization. Ouriel et al[10] evaluated 16 cases of acute renal occlusion and found that none of the patients with embolic or traumatic occlusion had regained function, even if revascularization had

been performed within 6 hours. However, revascularization in the thrombosis group was successful, irrespective of ischemia times ≤14 days after occlusion. They believed this had resulted from the persistent collateral flow present in the stenotic renal artery vessels that had led to thrombosis compared with the normal renal artery vessels in embolic and traumatic occlusions.[10] Although our patients experienced sudden renal function deterioration indicative of acute renal artery occlusion, it is possible that these patients had had slow progression of renal artery stenosis that had ultimately resulted in occlusion. Such a situation would allow for recruitment and development of collateral vessels. Supporting this is their history of intervention, either bypass or stenting.

Several historical studies that examined the predictors of renal rescue after revascularization have remained very relevant today. Dean et al[11,12] and Oskin et al[13] reported the predictive variables were the status of the vessel beyond the occlusion, bilaterality of reconstructable disease, and the amount of residual renal mass.[11-13] The return of renal function several days or weeks after an occlusive event has also been observed.[13,14] All three patients had patent renal arteries distal to the occlusion and severe hypertension, suggesting retained renal tubular function and, subsequently, experienced complete recovery of renal function after delayed revascularization.

Although far from conclusive, our findings have highlighted a dilemma in managing acute renal artery occlusion and have added valuable information to the scant literature on late renal revascularization for functional rescue. This is especially important with the rapid adoption of fenestrated and branched endovascular approaches for aortic aneurysm repair.

CONCLUSIONS

Our findings support aggressive renal revascularization in selected patients who have evidence of persistent renal perfusion and residual renal tubular function despite renal artery occlusion. We advise larger studies to validate these observations.

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