A 61-year-old man with ischemic cardiomyopathy with an ejection fraction of 10% to 15%, revascularized multivessel coronary artery disease, type 2 diabetes mellitus, and a history of smoking presented with recurrent episodes of heart failure exacerbations and pulmonary edema. He was on maximally tolerated medical therapy including aspirin, prasugrel, spironolactone, torsemide, and simvastatin. Prior attempts at initiation of β-blockers and angiotensin-converting enzyme inhibitors were limited by hypotension. On presentation, the patient had a heart rate of 96 bpm, respiratory rate of 22 breaths/min, blood pressure of 84/68 mm Hg, and jugular venous pulsations to 18 cm H2O. His cardiac exam showed evidence of a laterally displaced apical impulse with a parasternal heave and an S3 gallop on auscultation. There were bibasilar rales, and his lower extremities were cool with 3+ pitting edema. Admission labs demonstrated an elevated creatinine of 4.3 mg/dL (baseline 1.0 mg/dL). The patient was placed on inotropic support for cardiogenic shock and ultimately started on continuous venovenous hemofiltration (CVVH) for anuric renal failure. As workup for renal failure, a renal artery ultrasound was performed that showed isoechoic kidneys both approximately 11.0 cm in size without hydronephrosis. Duplex evaluation of the renal arteries was suggestive of bilateral renal artery stenosis >60% (Figure 33-1A). The patient underwent renal artery angiography showing >90% ostial stenosis for both renal arteries (Figure 33-1B). Given the clinical scenario, decision was made to perform renal artery stenting as salvage therapy for anuric renal failure and recurrent heart failure. Postintervention angiogram showed well-expanded bilateral stents with no residual stenosis (Figure 33-1C) and improved renal parenchymal blushing (Figure 33-1D). Within hours of the procedure, the patient started generating urine at a rate of >100 mL/h. His creatinine continued to improve even with cessation of renal replacement therapy (Figure 33-1E). At his 1-year follow-up, the patient continued to have stable New York Heart Association class II symptoms and an outpatient creatinine level of 1.28 mg/dL.
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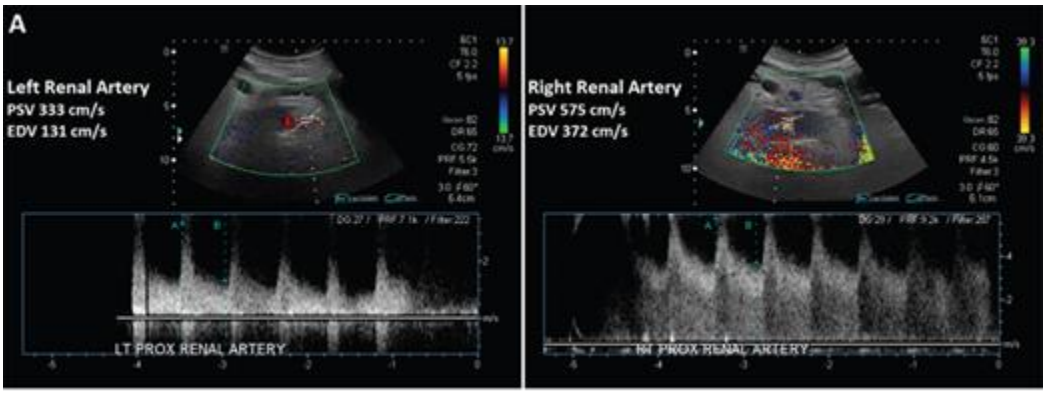
Figure 33-1

(A) Diagnostic renal artery Duplex ultrasound with elevated peak systolic and end-diastolic velocities, suggestive of bilateral renal artery stenosis. (B) Diagnostic renal angiogram with digital subtraction, illustrating bilateral severe proximal stenoses. (C) Poststent angiogram showing well-expanded stents bilaterally. (D) Following the placement of stents, there was a notable increase in arterial flow to both kidney parenchyma with improved “blushing.” (E) Trend of urine output (UOP) and creatinine, from before hospitalization and up to day of renal artery stenting (day 0, y-axis). The initial decrease in creatinine marks the onset of continuous venovenous dialysis.

Graphic Jump Location



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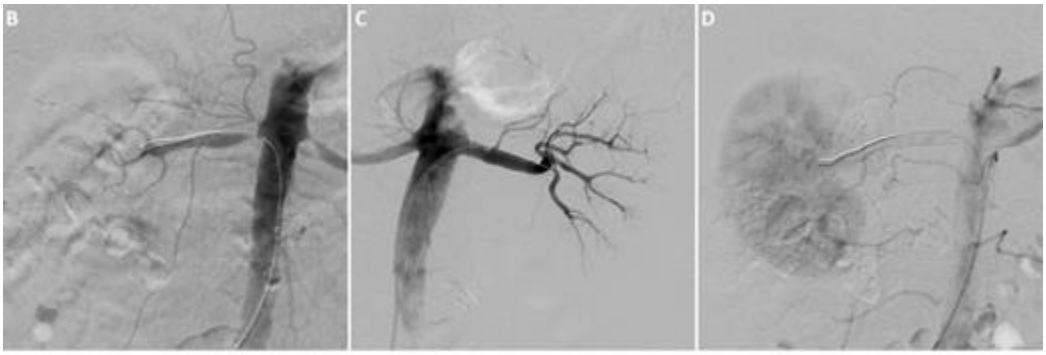

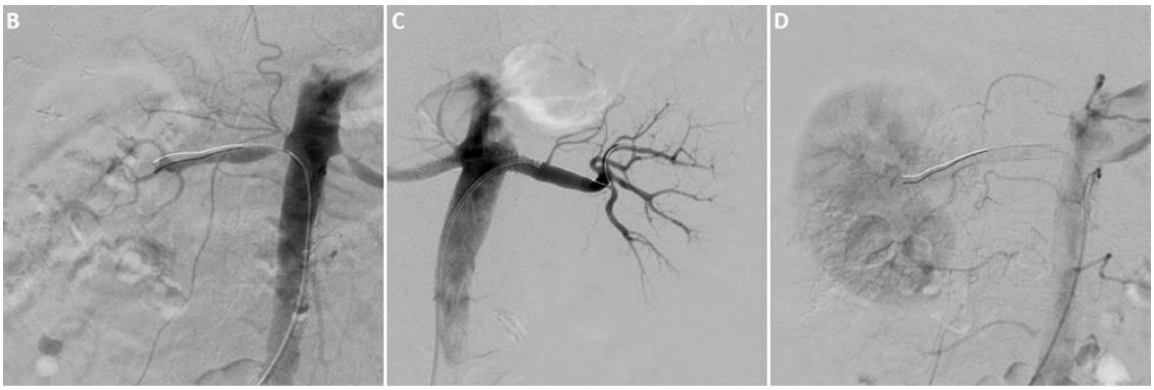

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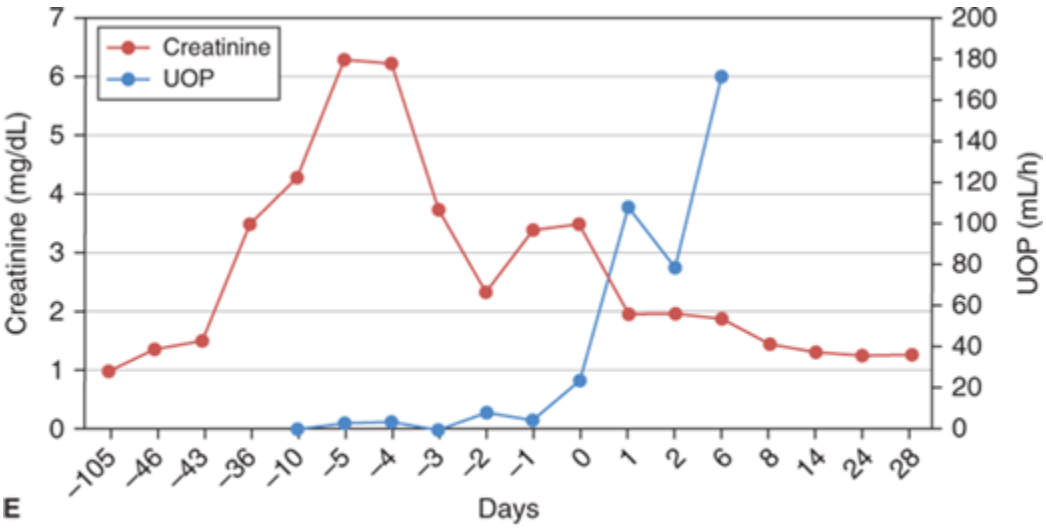

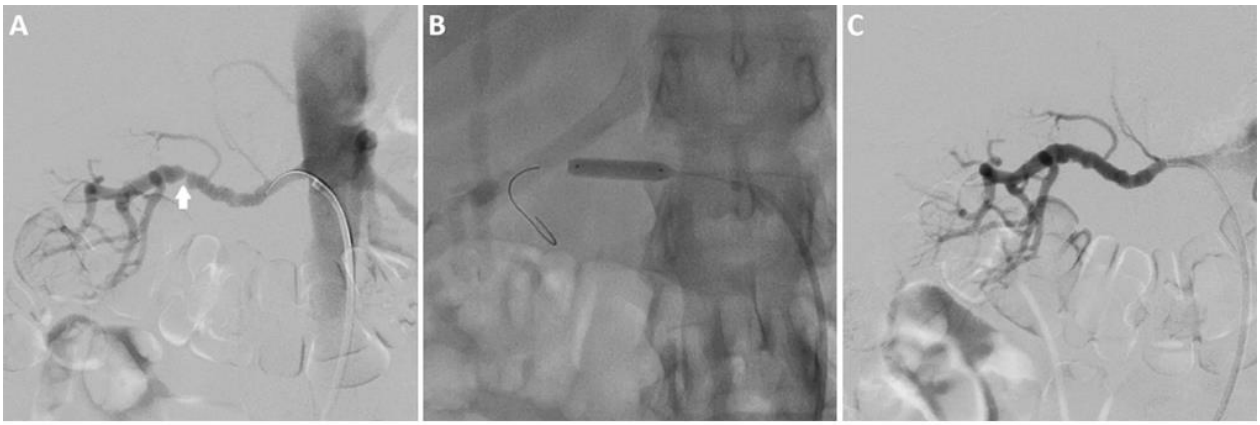

Table 33-1Classification of Fibromuscular Dysplasia

Classification	Frequency	Histopathology	Angiography
Medial dysplasia			
Medial fibroplasia	80%	Thinned media and thickened fibromuscular ridges that alternate and contain collagen	String of beads Beads larger than artery diameter
Perimedial fibroplasia	10%-15%	Significant collagen in the outer half of the media	Beading Artery diameter larger than beads
Medial hyperplasia	1%-2%	Smooth muscle hyperplasia No fibrosis	Concentric smooth stenoses
Intimal fibroplasia	<10%	Circumferential intimal collagen	Focal, concentric band Long, smooth narrowing
Periarterial (adventitial) fibroplasia	<1%	Collagen within the adventitia; can extend to surrounding structures	Unknown

Adapted from Begelman SM, Olin JW. Fibromuscular dysplasia. Curr Opin Rheumatol. 2000;12:41-47.