Possible cause of recurrent renal artery stenosis

To the Editor: We read with interest the recent renal consult presented by Kiernan et al. As the case presented, some cases of renal artery stenosis (RAS) are resistant to percutaneous transluminal renal angioplasty (PTRA) and necessitate surgical revascularization. Some of these ‘PTRA-resistant’ cases might be caused by retroperitoneal fibrosis, including chronic periaortitis, and steroid therapy would be effective for them.

Retroperitoneal fibrosis, a rare inflammatory disease, presents symptoms caused by the entrapment of retroperitoneal structures. It can cause renovascular hypertension by entrapment of the renal artery, and frequently causes urinary tract obstruction by entrapment of the ureter. Most cases are idiopathic, but the remainder occur as an inflammatory process secondary to other factors such as trauma and infections; previous surgery is a possible primary cause and smoking is a risk factor for retroperitoneal fibrosis.

The image after stent implantation indicated that the RAS of the presented case was rigid, which would represent fibrosis surrounding it. As the patient had previous aortal-renal artery reimplantation and was an active smoker, this case could be one of retroperitoneal fibrosis. With a patent renal artery, the atrophic left kidney could have been caused by a former ureteral obstruction. Sonography and magnetic resonance imaging could have revealed periarterial thickening or presence of a soft tissue mass in the retroperitoneum. The image after stenting described as ‘rigid’ by Tanemoto et al. probably reflects the non-elastic composition of the synthetic conduit material and not extrinsic fibrotic compression.

Surgical exploration and revascularization performed at our institution did not reveal any evidence of periarterial thickening or presence of a soft tissue mass in the retroperitoneum. The image after stenting described as ‘rigid’ by Tanemoto et al. probably reflects the non-elastic composition of the synthetic conduit material and not extrinsic fibrotic compression.

We thank Tanemoto et al. for their letter and agree on the importance of including other diagnoses of ‘percutaneous transluminal renal angioplasty-resistant’ renal artery stenosis, such as retroperitoneal fibrosis (RPF) and chronic aortitis, that we did not mention in our renal case report.

However, our case does not represent a case of renal artery stenosis caused by RPF for a number of reasons.

The 39-year-old patient underwent surgical revascularization for presumed congenital renal artery stenosis at an outside hospital at a young age. Unfortunately, limited medical and surgical data were available from that hospital, but at open repair we found that a synthetic conduit was used for the initial renal artery revascularization as a child.

Magnetic resonance imaging and sonography performed at our institution did not reveal any evidence of periarterial thickening or presence of a soft tissue mass in the retroperitoneum. The image after stenting described as ‘rigid’ by Tanemoto et al. probably reflects the non-elastic composition of the synthetic conduit material and not extrinsic fibrotic compression.

Surgical exploration and revascularization performed at our institution did not reveal any particular macroscopic evidence of RPF but revealed instead a synthetic graft that was now size mismatched with respect to the mature right kidney.

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Thomas J. Kiernan1, Bryan P. Yan1 and Joseph M. Garasic1
1Section of Vascular Medicine and Interventional Cardiology, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, USA
Correspondence: Thomas J. Kiernan, Section of Vascular Medicine and Interventional Cardiology, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, USA. E-mail: tom_kiernan@hotmail.com