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Atypical May-Thurner syndrome caused by endovascular aortic aneurysm repair

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ABSTRACT

May-Thurner syndrome is characterized by unilateral lower extremity venous hypertension and stasis due to compression of an iliac vein between an iliac artery and the lumbar spine. In almost all cases, the left common iliac vein is compressed by the right common iliac artery; however, other patterns have been described. Rarely, May-Thurner syndrome may be created iatrogenically as a result of iliac artery stenting. We present an unusual case of new left common iliac vein thrombosis caused by ipsilateral left iliac artery compression after aortobi-iliac endovascular aneurysm repair. (J Vasc Surg Cases and Innovative Techniques 2020;6:397-400.)

Keywords: Iliac vein compression; Aortic repair; Iatrogenic injury

CASE REPORT

A 71-year-old woman was evaluated for an extent III thoracoabdominal aortic aneurysm with maximal diameter of 5 cm. Her vascular history was also notable for bilateral lower extremity claudication due to iliac and infrainguinal arterial occlusive disease. Given her small stature, medical comorbidities, and cardiovascular risk, repair through a predominantly endovascular strategy was recommended. She first underwent bilateral iliofemoral bypasses to enable safe aortic endograft delivery and to preserve bilateral hypogastric perfusion. She later underwent staged thoracoabdominal aortic coverage, with a four-vessel physician-modified endograft bridging the visceral segment.

Each iliofemoral bypass was performed with 10-mm Dacron, sewn end to end to the transected distal common iliac artery after circumferential dissection and end to side to the common femoral artery. The transected end of the distal common iliac artery was oversewn, and the hypogastric artery was perfused in a retrograde fashion; no venous injuries were incurred. Infrarenal aortobi-iliac endografting was performed with a Cook Zenith Flex 28-82 bifurcated device (Cook Medical, Bloomington, Ind) extended bilaterally with 16-mm Cook Spiral-Z limbs crossing the narrow, calcified common iliac arteries and sealing distally in the Dacron bypasses. After deployment, the iliac limbs were expanded to nominal diameter using high-pressure 12-mm noncompliant balloons.

At 2 weeks postoperatively, the patient complained of new moderate left leg discomfort and swelling, and her physical examination was notable for mild left leg pitting edema. Preoperatively, the patient reported no history of leg swelling. Computed tomography angiography with venous-phase imaging demonstrated widely patent aortic and iliac endografts and Dacron bypass grafts, new-onset left common iliac vein thrombosis, and new-onset high-grade compression of the proximal left common iliac vein between the ipsilateral stented left common iliac artery and the L5 vertebral body, consistent with iatrogenic May-Thurner syndrome (MTS; Fig 1). The left external iliac and hypogastric veins remained patent. Prompt venography and intravascular ultrasound were performed, confirming extrinsic compression and complete occlusion of the left common iliac vein. After crossing of the occlusion with a wire, a 16- × 60-mm Wallstent endoprosthesis (Boston Scientific, Marlborough, Mass) was placed from the distal vena cava into the left external iliac vein (Fig 2). No residual thrombus was identified after stent placement, and thus thrombectomy or thrombolysis was deemed unnecessary. After this procedure, leg swelling and pain improved rapidly. On postoperative day 2, repeated computed tomography angiography imaging demonstrated successful treatment of iatrogenic MTS with venous stent patency and luminal opacification without extrinsic compression of the left common iliac vein (Fig 3). The patient was maintained on a 3-month course of oral anticoagulation in addition to compression therapy. Ultimately, the patient completed planned endovascular thoracoabdominal aneurysm repair once she was able to cease anticoagulation. At 22 months postoperatively, computed tomography imaging demonstrated a widely patent venous stent, and the patient exhibited no clinical sequelae of post-thrombotic syndrome.

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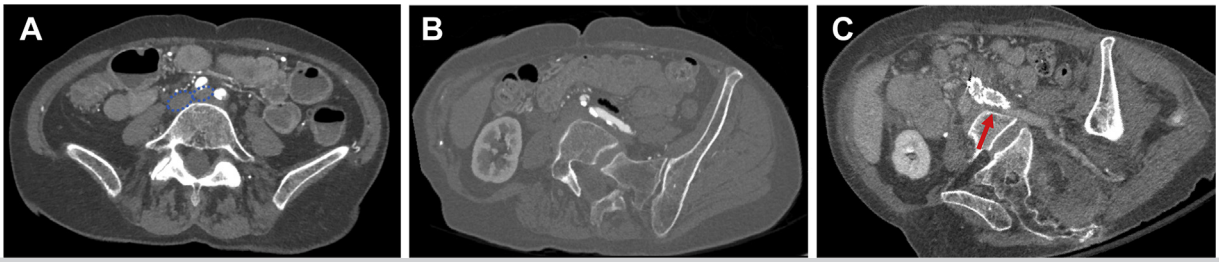

Fig 1. Computed tomography angiography demonstrating **(A)** the native iliac veins (*outlined*) in relation to the aortic bifurcation and L5 vertebral body in the axial plane. Single multiplanar reconstructions **(B)** of the native left common iliac vein and **(C)** after left common iliac artery stenting with compression of the left common iliac vein against L5 (*arrow*).

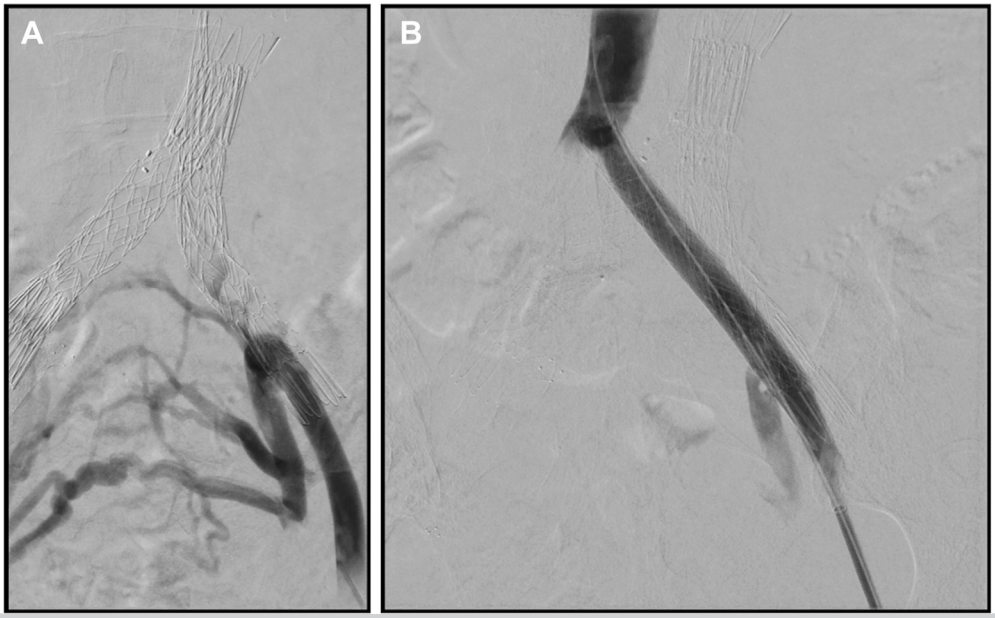

Fig 2. Venogram demonstrating **(A)** compression of the left common iliac vein with preferential filling of cross-pelvic collateral veins to empty into the inferior vena cava and **(B)** subsequent stenting of the left common iliac vein with emptying into the inferior vena cava without opacification of the pelvic collateral veins.

The patient provided consent for this case report, and it was exempt from Institutional Review Board approval at our institution.

DISCUSSION

MTS, or iliac vein compression syndrome, is characterized by unilateral lower extremity venous hypertension and stasis secondary to extrinsic compression of the iliac vein.[1] Virchow[2] first noted a left-sided predominance of iliofemoral deep venous thromboses in 1851. However, the eponym of this syndrome originates from a cadaveric study in 1957 by May and Thurner describing a "vascular spur" arising in 22% of the examined individuals from chronic irritation of the left common iliac vein by anterior crossing of the right common iliac artery.[3] In almost all cases, the left common iliac vein is compressed between the right common iliac artery and L5 vertebral body, although other anatomic patterns have been described.[1,4,5]

Some degree of left iliac vein compression may be physiologic and harmless; in fact, one study of left iliac vein anatomy in asymptomatic patients found that 66% of patients had >25% compression and 24% of patients had >50% compression of the left iliac vein on axial imaging despite no clinical symptoms.[6] In symptomatic patients, however, MTS may lead to both acute and chronic consequences, including lower extremity swelling, venous claudication, deep venous thrombosis, superficial venous thrombophlebitis, chronic venous insufficiency, and post-thrombotic syndrome.[4,5]

The incidence of MTS is largely unknown, although it is most commonly manifested in women between the second and fourth decades of life.[4,5] Currently, it is estimated to affect 2% to 5% of patients with a lower

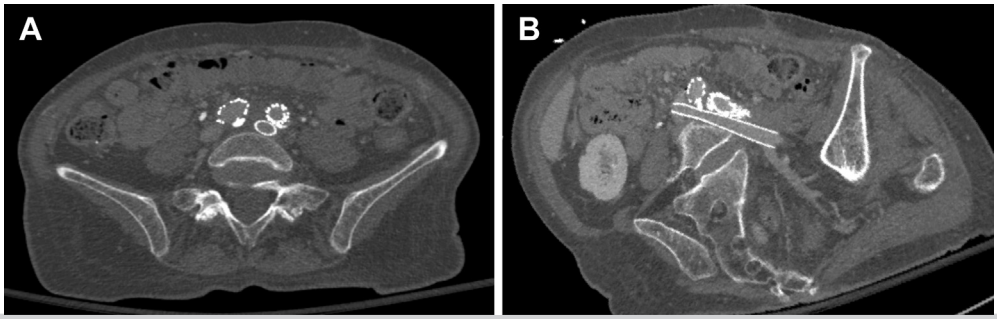

Fig 3. Computed tomography angiography after intervention demonstrating patent left common iliac vein stent passing underneath the left iliac limb of the bifurcated endovascular aortic aneurysm repair device **(A)** in the axial plane and **(B)** with single multiplanar reconstruction.

extremity venous disorder[4] but nearly 15% of patients with isolated left leg chronic venous disease.[7] Diagnosis can be challenging; ultrasound is a common initial study because of its noninvasive and inexpensive nature. It can demonstrate vessel patency or presence of deep venous thrombosis. However, several factors can limit the technical success of imaging of the inferior vena cava and iliac veins with ultrasound.[8] Axial imaging such as computed tomography or magnetic resonance imaging can be used to demonstrate narrowing of the iliac vein and to identify other potential causes of clinical symptoms. However, timing of these studies for optimal opacification of the venous system can be challenging. Furthermore, magnetic resonance venography in particular has a high false-positive rate for venous obstruction. Venography and intravascular ultrasound are the most accurate for diagnosis, but these are invasive modalities and should be reserved for clinical scenarios with a high likelihood for subsequent intervention.[9] Endovascular treatment with iliac vein stenting is highly successful; adjunctive treatment with thrombectomy or thrombolysis may also be needed in the presence of acute or subacute thrombus.[5,7]

Unusual causes of MTS have been previously described in the literature, including iliac artery aneurysm, distended bladder, and endometriosis.[6] Less frequently seen are iatrogenic cases of MTS; reports in the literature include impingement on the vein due to intra-abdominal adhesions, orthopedic hardware, and a penile prosthetic reservoir.[10-13] Endovascular stenting of an iliac artery causing MTS is a known but rare phenomenon. Two cases in the literature describe stenting of the right iliac artery for occlusive disease resulting in iliac vein compression, one causing the classic left iliac vein compression, the other causing ipsilateral right iliac vein compression.[14,15] Pandit et al[16] published a case of MTS after endovascular aneurysm repair in which the stented left iliac artery compressed the left iliac vein, but this involved treatment of a 2.2-cm left common iliac aneurysm that may have predisposed to venous compression. In our case, the iliac arteries were narrow and heavily calcified, with no evidence of pre-existing venous impingement. Notably, aneurysmal degeneration of the aorta had resulted in migration of the aortic bifurcation from its normal position across the midline to the right side of the spine. As a result, the left iliac artery, rather than the right, was positioned to cause venous compression against the spinal column.

Given the rarity of this occurrence, it is difficult to identify clear risk factors. Whereas the presence of pre-existing mild or moderate venous compression by either iliac artery might raise concern, no such compression existed in our case. As such, the absence of pre-existing compression cannot be considered exclusionary of the diagnosis. One unique factor in our case involved migration of the aortic bifurcation to an atypical location as a result of aortic aneurysm formation. Conceptually, a malpositioned aortic bifurcation might increase risk if it changes the natural relationship between iliac arteries and veins, although this case alone is insufficient to make any firm claims in this regard. We sought luminal expansion of the native iliac artery system to facilitate passage of large-bore sheaths for eventual complex endovascular thoracoabdominal aortic aneurysm repair. This intentional alteration to native anatomy may also have contributed to the development of MTS.

As the boundaries of complex endovascular and hybrid reconstruction of patients expand, unusual complications due to novel anatomic configurations may occur with increasing frequency. Regardless of preoperative imaging, providers should have a high index of suspicion with close attention to detail on postoperative imaging if patients have new-onset leg pain or swelling after endovascular aneurysm repair. Given the natural history of iliac vein thrombosis, early recognition and prompt treatment of iatrogenic MTS may confer a better therapeutic response to intervention.

CONCLUSIONS

Iliac vein compression after iliac artery stenting, including endovascular aortic aneurysm repair, is a known but rare complication that requires a high index of suspicion for the provider to recognize, diagnose, and treat this entity. Predisposing factors are often difficult to identify. As in our patient, iliac vein stenting may be used successfully to durably restore venous patency in such cases.

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