Cardiology in the Young (2015), **25**, 797–799
doi:10.1017/S1047951114001115

Brief Report

Unusual cases of right-sided and left-sided May–Thurner syndrome

I. B. Vijayalakshmi, H. S. Natraj Setty, Chitra Narasimhan

Department of Pediatric Cardiology, Sri Jayadeva Institute of Cardiovascular Sciences and Research, Bengaluru, Karnataka, India

Abstract May–Thurner syndrome is a rare clinical entity involving venous obstruction of the left lower extremity. The May–Thurner syndrome is a phenomenon commonly described as an acquired stenosis of the left common iliac vein secondary to compression of the left common iliac vein between the right common iliac artery and the underlying vertebral body. We report one case of May–Thurner syndrome, and another rare case of reverse May–Thurner syndrome, incidently detected during intervention, in a case of aortic stenosis and mitral stenosis with dextrocardia and situs inversus.

Keywords: common iliac vein; common iliac artery; dextrocardia; situs inversus; intervention

Received: 22 April 2014; Accepted: 1 June 2014; First published online: 10 July 2014

Background

Iliac vein compression syndrome was described by May and Thurner.[1] Several surgical management strategies have been used to alleviate symptoms and correct the resulting obstruction,[2] mostly involving venovenous bypass, with autologous vein creation of a tissue sling to elevate the overriding right iliac artery.[3] More recently, endovascular techniques have been used to treat both the thrombosis and correct the anatomic abnormalities associated with the May–Thurner syndrome.[4] However, there are no reports of May–Thurner syndrome or reverse May–Thurner syndrome in a case of dextrocardia, with situs inversus in the literature, coming in the way of transcatheter intervention. We report two cases incidentally detected during intervention and diagnosis confirmed by CT angiogram.

Case profile

Case report (1) A 7-year-old female patient known case of congenital heart disease with situs solitus, large patent ductus arteriosus with perimembraneous ventricular septal defect (left → right shunt), pulmonary arterial hypertension, and normal left ventricular function, was taken up for device closure. The venous and arterial access was attempted simultaneously from both sides to save time. Although the Terumo guidewire passed through the left femoral vein, the catheter could not be negotiated. Hence, the hand injection of contrast was administered through the sheath in the left femoral vein that demonstrated obstruction at the origin of the left common iliac vein, with plenty of collaterals (Fig 1a).The compression of the left common iliac vein by the right common iliac artery was demonstrated by simultaneous injection of contrast in the iliac vein and aorta before bifurcation (Fig 1b). This clearly demonstrated the May–Thurner syndrome.

Case report (2) A 16-year-old male patient presented with a history of easy fatigability, choking sensation, and chest pain on walking for the past 2 months. Pulse was 84/minute, low volume, with pulsus parvus et tardus. Blood pressure was 100/80 mmHg. Cardiovascular system examination revealed heart sounds better heard on the right side, ejection systolic murmur in the pulmonary area, and mid-diastolic murmur in the apical region on the right side. The echocardiography revealed congenital heart

Correspondence to: Dr H. S. N. Setty, D.M, Post Graduate Trainee, Department of Cardiology, Sri Jayadeva Institute of Cardiovascular Sciences and Research, Bangalore-560069, Karnataka, India. Tel: +080 26580051; Fax: +080-22977261; E-mail: drnatrajsetty75@gmail.com

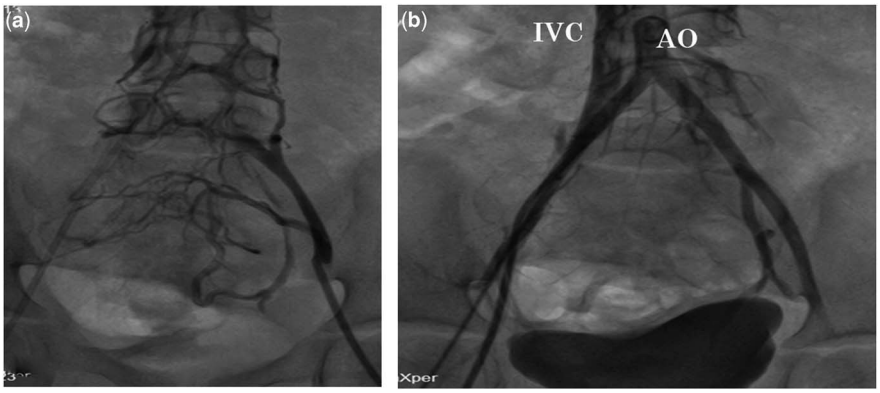

Figure 1.
*(**a**) Hand injection of contrast shows obstruction of the left common iliac vein. (**b**) Simultaneous injection of contrast in the aorta and right femoral vein showed obstruction of the left common iliac vein by the right common iliac artery.*

disease with situs inversus, dextrocardia, bicuspid aortic valve, severe aortic stenosis (gradient of 200/122 mmHg), and rheumatic heart disease, severe mitral stenosis (0.8 cm^2 mitral orifice area, gradient 63/34 mmHg), mild tricuspid regurgitation, pulmonary hypertension (pulmonary artery systolic pressure - 53 mmHg), concentric left ventricular hypertrophy, and normal left ventricular function. The patient was taken for dilatation of both the valves in the same sitting. As it is customary to dilate the distal lesion, we attempted to dilate the aortic stenosis first. As the balloon was slipping while inflating, we decided to pace the right ventricle. To our surprise the pacing lead could not be passed through the right femoral sheath. Hand injection of contrast showed the whirling of the dye and partial obstruction of right common iliac vein. Hence, CT angiogram was performed after the interventions. Anyway as it was a case of dextrocardia, the interatrial septal puncture was carried out by passing the Brockenborough needle through the left femoral vein in 7 o'clock position in 30° right anterior oblique view. After successful balloon valvuloplasty, by Accura balloon, a 0.025″ Terumo guidewire was passed into the left ventricle to cross the stenotic aortic valve into the aorta and to the descending aorta. A 4 F slip catheter (microcatheter) was passed over the wire and exchanged with a 0.035″ Terumo wire. The Terumo wire was snared through the right femoral artery, and arteriovenous loop was created from the left femoral vein to the right femoral artery. Over the extra support wire, a 12 × 40 Tyshak II balloon was introduced into the left ventricle by retrograde approach from the right femoral artery and placed across the aortic valve. As the waist had not disappeared, the dilatation was performed for the third time when the balloon burst. The left ventricle pressure had reduced from 332/14 to 168/8. The gradient reduced from 228 to mean 52 mmHg on pullback tracing.

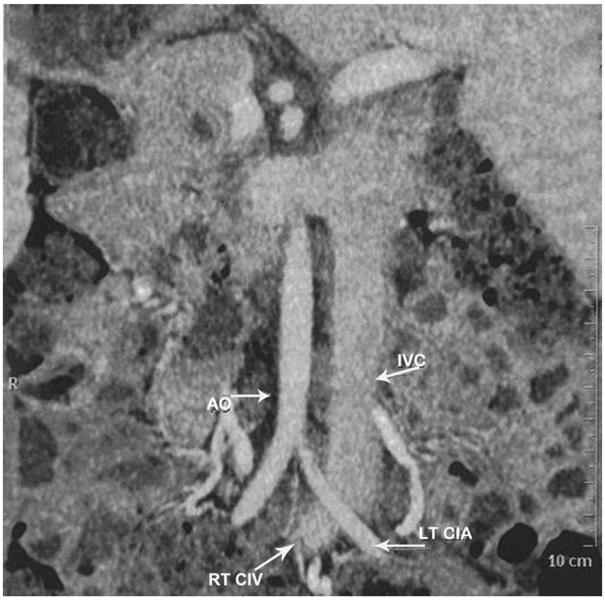

Figure 2.
Computed tomography (CT) angiogram showed the obstruction of the right common iliac vein by the left common iliac artery, (presentation of reverse May–Thurner syndrome). AO = aorta; IVC = inferior vena cava; LT CIA = left common iliac artery; RT CIV = right common iliac vein.

CT angiogram (Fig 2) showed compression of the right common iliac vein by the left common iliac artery (reverse of May–Thurner syndrome). Hence, we were unable to introduce the temporary pacing lead through right femoral vein.

Discussion

The prevalence of May–Thurner syndrome is unknown. With the advent of venography and non invasice CT angiogram the ante mortem detection of the syndrome can be done with more precision. In the past, many cases

https://doi.org/10.1017/S1047951114001115 Published online by Cambridge University Press

of the left iliofemoral venous thrombosis associated with May–Thurner syndrome were probably not recognised clinically.[5,6] Compression of the left common iliac vein against the pelvic rim by the overlying right common iliac artery was first described by Virchow.[7] In 1956, after examining 430 cadavers, May and Thurner described the development of intraluminal bands, or "spurs", within the left common iliac vein of 22% of the bodies. Considering an acquired anomaly, the stricture is felt to develop as a result of not only physical compression but also the repetitive trauma of the overlying arterial pulsation, leading to intimal hypertrophy and predisposing the patient to deep vein thrombosis.[1] One of our patients was incidentally detected during the interventional procedure where the left common iliac vein was compressed by the right common iliac artery. It clearly demonstrated May–Thurner syndrome. In our second case reverse of the May–Thurner syndrome was demonstrated showing compression of the right common iliac vein by the left common iliac artery, due to situs inversus associated with dextrocardia. The CT angiogram confirmed the findings.

The May–Thurner syndrome does not matter to the surgeons for intracardiac repair, but it matters a lot to the interventional cardiologist, as one may have to use the opposite vein or jugular approach for the intervention. The compression of artery can be clinically detected by a feeble pulse. However, there is no clinical way to detect venous compression. Hence, it is worthwhile to perform a venogram or CT angio if there is a difficulty in negotiating the catheter in the vein.

Conclusion

May–Thurner syndrome and rarely reverse May–Thurner syndrome may hamper the catheterisation and interventions. One should suspect May–Thurner syndrome if the guidewire can be passed and not the sheath or the catheter through the femoral vein.

Acknowledgements

We wish to acknowlege Dr Madhav Hegde, Consultant Radiologist for his help in performing the CT angiogram.

Financial Support

This research received no specific grant from any funding agency, commercial or not-for-profit sectors.

Conflicts of Interest

None.

Ethical Standards

Consent has been taken from the attendant of the patients.

References

1. May R, Thurner J. The cause of the predominantly sinistral occurrence of thrombosis of the pelvic veins. Angiology 1957; 8: 419–427.
2. Palma E, Esperon R. Vein transplants and grafts in the surgical treatment of post-phlebotic syndrome. J Cardiovasc Surg 1960; 1: 94–107.
3. Cockett FB, Thomas L. The iliac vein compression syndrome. Br J Surg 1965; 52: 816–821.
4. Berger A, Jaffe JW, York TN. Iliac compression syndrome treated with stent placement. J Vasc Surg 1995; 21: 510–514.
5. Taheri SA, Williams J, Powell S, et al. Iliocaval compression syndrome. Am J Surg 1987; 154: 169–172.
6. Giordano JM, Trout H. Anomalies of the inferior vena cava. J Vasc Surg 1986; 3: 924–928.
7. Virchow R. Uber die Erweiterung kleiner Gefasse. Arch Path Anat 1851; 3: 427.