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REVIEW ARTICLE

Aortosternal venous compression: the thoracic inlet syndrome — a scoping review of the literature

Andreia Pinelo, Daniel Mendes, Henrique Almeida, Miguel Queirós, João Cabral, Samuel Cardoso, Mónica Bandeira, Rui Machado

Department of Angiology and Vascular Surgery, Centro Hospitalar Universitário de Santo António

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ABSTRACT

OBJECTIVE: Aortosternal vein compression (AVC) is an underrecognized cause of central venous obstruction. This scoping review aimed to synthesize available case-based evidence on its clinical presentation, diagnostic work-up, and management strategies.

METHODS: A systematic search of the PubMed database was conducted, including case reports and case series reporting patient-level data on symptoms, imaging findings, and management.

RESULTS: Ten publications were identified. Clinical presentations ranged from incidental findings to upper extremity swelling, paresthesia, dysphagia, and visual disturbances. Diagnostic confirmation relied primarily on dynamic imaging modalities—contrast-enhanced CT, MR angiography, venography, and IVUS—often during respiratory maneuvers. Although no standardized diagnostic threshold exists, thoracic antero-posterior diameters below 6 cm during expiration were associated with compression. Management was predominantly conservative; however, endovascular interventions such as stenting were attempted in selected cases, with reported complications including stent deformation and collapse due to persistent external compression.

CONCLUSION: AVC is conceptually analogous to May–Thurner syndrome and should be considered in the differential diagnosis of unexplained central or upper limb venous thrombosis or venous hypertension. Recognition of AVC has direct implications for management, as if venous occlusion is due to dynamic extrinsic compression, endovascular interventions may be prone to failure, including risks of stent collapse or deformation. Accurate diagnosis is therefore essential not only for appropriate clinical assessment but also for guiding safe and effective treatment strategies.

Keywords: Aortosternal venous compression; central vein thrombosis; thoracic entrapment

INTRODUCTION

Aortosternal venous compression (AVC) is a rare form of central venous outflow obstruction in which the left brachiocephalic vein (LBCV) becomes entrapped between the posterior surface of the sternum and anterior thoracic outlet arteries – Figure 1 —most commonly the aortic arch or an aberrant right brachiocephalic artery. First described by Wurtz et al. in 1989, AVC arises from the LBCV's passage through a narrow mediastinal space in route to the superior vena cava, predisposing it to dynamic extrinsic compression, stenosis, and even occlusion.[1,2] Although most cases remain clinically silent—compensated by collateral drainage—symptomatic presentations can include left-sided head or neck discomfort, pulsatile tinnitus, headaches, and, more rarely, left upper-extremity edema or pain.[3,4] Recognition

Corresponding Author:
Andreia Pinelo | afbpinelo@gmail.com
Department of Angiology and Vascular Surgery, Centro Hospitalar Universitário de Santo António
Largo do Prof. Abel Salazar, 4099-001 Porto

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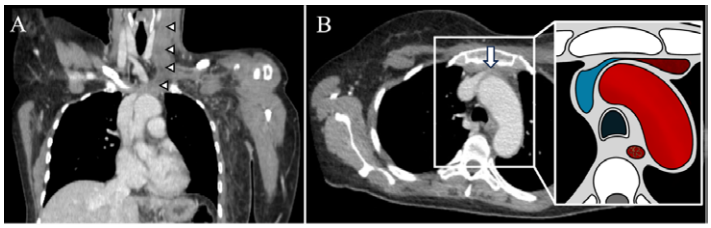
of this entity is critical, as it can precipitate upper-limb and central venous thromboses in individuals without classic thrombotic risk factors and may rise important management implications.

Figure 1. CT and Schematic representation of the Aortosternal Venous Compression.



A) Thrombosis of the left internal jugular vein and the subclavian-axillary-brachial venous axis; **B)** Complete collapse of the left brachiocephalic vein due to compression between the aortic arch and sternum

MATERIALS & METHODS

This study was conducted as a scoping review of the literature to characterize the clinical presentation, diagnostic approaches, and management strategies associated with aortosternal and innominate vein compression. A systematic search of the PubMed database was performed using the keywords “brachiocephalic”, “innominate”, and “aortosternal venous compression”. Reference lists of all eligible articles were manually cross-checked to identify additional relevant publications. Studies were selected based on abstract screening. Inclusion criteria were limited to case reports and case series that provided descriptive data on patient presentation, imaging findings, and therapeutic management.

RESULTS

A total of 10 studies, including 9 clinical case reports and 1 case series, were identified reporting on aortosternal compression of the brachiocephalic venous trunk – summarized in Table 1.

The earliest report by Wurtz et al. described a 65-year-old patient presenting with neck and left chest wall edema. Imaging revealed dynamic compression of the left brachiocephalic vein by the origins of the aortic arch vessels, with expiratory phlebography demonstrating extrinsic venous compression.[1]

Subsequent reports confirmed the recurring anatomical theme of left brachiocephalic vein compression between the sternum anteriorly and aortic arch vessels posteriorly. Moes et al. described a pediatric case of venous narrowing discovered during the investigation of ventricular fibrillation.[5] Hammer et al reported two dialysis patients with left arm swelling and ipsilateral arteriovenous fistulas.[6] Venography demonstrated significant stenosis of the brachiocephalic trunk in both cases. One patient was treated with a Palmaz stent (PS 564, Cordis, Miami, FL) deployed using a 12-mm balloon, while the other received a 12 × 40-mm self-expandable nitinol stent (Sinus-Stent, Optimed, Ettlingen, Germany). Both patients experienced symptom recurrence at 2 and 6 months, respectively. Follow-up CT imaging revealed stent compression and collapse between the manubrium and the aortic arch in both cases. Conservative management was adopted with progressive resolution of symptoms in both patients.

The largest dataset was provided by Itkin et al., who reviewed 106 venograms from 48 hemodialysis patients.[7] Extrinsic compression of the left innominate vein was observed in 44% of cases, with three patients requiring stent (Wallstents, Boston Scientific, Natick, MA) placement following symptom onset and failure of angioplasty.

A report by Hernandez et al. detailed two female patients with intermittent upper extremity swelling. CT and venography in both cases demonstrated dynamic extrinsic compression of the left brachiocephalic vein, aggravated during expiration.[8] Similarly, Lo et al. described a 64-year-old man with dizziness and reversed jugular flow on MR angiography due to compression of the left innominate vein against the sternum and major arteries.[9]

Later, Lee et al. documented a 19-year-old woman with arm swelling and paresthesia, in whom MR angiography and provocative venography identified compression of the innominate vein by the innominate artery.[10] Esteban et al. reported on a young man with longstanding superficial venous dilation and non-disabling symptoms; dynamic imaging revealed compression of the left brachiocephalic vein with hypertrophic collateral drainage.[11]

A 2019 report by Horita et al. described a dialysis patient with access limb swelling. Multimodal imaging, including IVUS, confirmed left brachiocephalic vein compression, and successful treatment was achieved via 7mm balloon-expandable stent (Express, Boston Scientific, Marlborough, Massachusetts, EUA) deployment using a "dog-bone" technique to prevent migration.[2]

The most recent contribution by Giglio et al reviewed two female patients, aged 72 and 34, who presented with non-specific neurologic and visual symptoms. Dynamic CT and angiographic imaging demonstrated expiratory compression of the left brachiocephalic vein by an aberrant right subclavian artery, consistent with aortosternal venous compression. Both patients were managed conservatively with nocturnal arm elevation.[12]

DISCUSSION

The LBCV traverses a narrow anteroposterior (AP) mediastinal space bordered anteriorly by the manubrium and posteriorly by the aortic arch or its major branches. This anatomical configuration predisposes the LBCV to extrinsic compression, particularly during expiration when the AP diameter decreases. This phenomenon, referred to as AVC, is physiologically relieved by deep inspiration, which expands the thoracic inlet and restores venous patency.[1,7,12,13]

AVC is estimated to occur in 0.2–0.4% of the general population based on postmortem and radionuclide studies, with a modestly higher incidence in the elderly, likely due to age-related arterial ectasia and decreased thoracic compliance.[4,14,15] Although typically asymptomatic, AVC may manifest with upper extremity or supraclavicular edema, neck pulsation, or neurologic symptoms such as paresthesia

Table 1. Summary of the five articles concerning CDT in chronic aortic occlusions, included in this literature review.

Title, Authors, Date	Study Type	N	Clinical Presentation	Imaging Methods	Treatment/Approach
Wurtz et al., 1989	Clinical report	1	Chest wall + neck edema	CT-scan, phlebography	Conservative
Moes et al., 1995	Clinical report	1	Not reported	MRI, phlebography	Conservative
Hammer et al., 2000	Clinical report	2	Left arm edema	Duplex scan, phlebography, CT	Stent placement [Palmaz stent, PS 564, Cordis, Miami, FL; n=1] and [Sinus-Stent, Optimed, Ettlingen, Germany); n=1] occluded at 2 at 6 months
Itkin et al., 2004	Case series	21(out of 48)	Arm edema, high venous resistance during dialysis	Phlebography	Stent placement [Wallstents, Boston Scientific, Natick, MA; n=3]
Hernandez et al., 2005	Clinical report	2	Left arm edema, supraclavicular pain	CT, phlebography	Not reported
Lo et al., 2007	Clinical report	1	Dizziness	MRI, CT	Conservative
Lee et al., 2011	Clinical report	1	Left arm edema, headache	Phlebography, MRI	Conservative with prophylactic anticoagulation
Esteban et al., 2011	Clinical report	1	Forearm vein engorgement, pain	Ultrasound, dynamic phlebography, angioMRI	Conservative
Horita, 2019	Clinical report	1	Hemodialysis patient with edema of access limb	CT, phlebography, IVUS	Stent placement [Express, Boston Scientific, Marlborough, Massachusetts, EUA; n=1]
Giglio et al., 2022	Clinical report	2	Paresthesia, pulsations, dysphagia, visual aura	CT, ultrasound, phlebography	Conservative

or visual disturbances due to increased venous pressure; dysphagia may also occur and should raise suspicion of an aberrant right subclavian artery.[4,6,8,10,13]

Multiple reports, including dynamic imaging studies, highlight the role of respiratory cycles in modulating the severity of venous compression. Tanaka et al. demonstrated that MR signal asymmetry of the left sigmoid sinus and internal jugular vein (IJV) was attributable to expiratory LBCV occlusion, which resolved with full inspiration.[4] Similarly, Chung and Hu reported a higher prevalence of jugular venous reflux (JVR) on the left side, implicating mechanical narrowing of the LBCV as a contributing factor, which was associated with neurologic syndromes such as transient global amnesia.[16] Kudo et al. corroborated these findings, showing that physiologic venous stenosis at the thoracic inlet was relieved during inspiratory breath holding, further supporting the dynamic and positional nature of this condition.[3]

Diagnosis relies on high clinical suspicion, particularly in patients presenting with unexplained upper limb swelling, JVR, or neurologic complaints without alternative causes. Duplex ultrasonography is a valuable first-line tool for detecting sluggish or retrograde venous flow, but it lacks specificity for extrinsic compression. Contrast-enhanced computed tomography (CT), particularly when performed during both inspiratory and expiratory phases, is a key diagnostic tool for evaluating the thoracic inlet in suspected cases of aortosternal venous compression. It enables direct measurement of the AP diameter at the level of the LBCV, visualization of respiratory variation, and exclusion of alternative causes such as masses, aneurysms, or vascular anomalies. Although no standardized AP diameter cutoff has been established to predict clinically significant compression, two reports described expiratory-phase AP diameters below 6 cm associated with radiologically confirmed LBCV compression. [1,12] Digital subtraction venography remains the gold standard for confirming both the anatomical severity and the respiratory variability of the stenosis.[3,7,12]

Management is guided by symptom severity and associated complications. In asymptomatic or mildly symptomatic cases, conservative treatment—including postural strategies and activity modification—remains the mainstay. In the presence of thrombosis, full-dose anticoagulation should be instituted per current guidelines, often resulting in symptom resolution without invasive intervention.[17]

Endovascular therapy has proven useful in hemodialysis-related central venous stenosis, including AVC.[2,7] However, stent placement in this setting is controversial due to mechanical limitations. Hammer et al. reported two patients with dialysis-related symptomatic AVC who developed stent deformation and recurrent symptoms due to continued extrinsic forces, with CT confirming stent collapse between the manubrium and aortic arch.[6] Surgical decompression—such as manubrioplasty or division of compressive vascular structures — has been employed in select cases, but experience remains limited and outcomes are not well characterized.[18]

Conceptually, AVC bears pathophysiologic resemblance to May–Thurner syndrome, functioning as an upper-body analog where in a major artery compresses a central vein, predisposing to chronic venous outflow obstruction and thrombosis. This parallel reinforces the need for systematic

evaluation of thoracic inlet dynamics in patients with idiopathic upper extremity or central venous occlusion.

CONCLUSION

AVC is an uncommon but likely underdiagnosed cause of central venous obstruction and thrombosis. Diagnosis relies on high clinical suspicion and dynamic imaging—particularly contrast-enhanced CT, MR angiography, venography, or IVUS—performed during respiratory maneuvers to detect positional venous stenosis. Management should be individualized based on symptom severity. Conservative approaches remain first-line. Endovascular or surgical interventions may be considered in refractory cases; however, the risk of stent deformation due to persistent external mechanical forces at the thoracic inlet must be carefully weighed. AVC should therefore be considered not only during diagnostic workup but also in treatment planning for central venous occlusion, as lesions at this level may carry a high risk of stent collapse, warranting careful device selection and procedural strategy.

Further research is needed to define standardized diagnostic thresholds and to elucidate the long-term efficacy and durability of interventional therapies in this unique anatomical context.

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