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Commentary: SVC syndrome: Venous stenting is the mainstay but may not stay open

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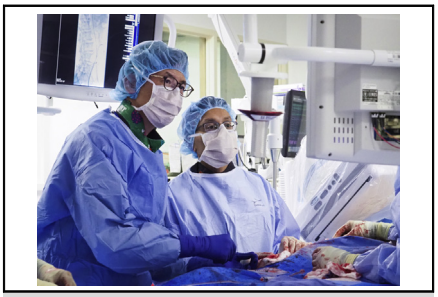

The authors in the vascular OR.

CENTRAL MESSAGE

Endovenous stenting is the main therapy for superior vena cava syndrome. Long-term patency is yet to improve, however, especially with benign pathologies on the rise.

Superior vena cava (SVC) syndrome related to device implantation is on the rise. While malignancy has historically been the primary etiology for SVC syndrome, recent estimates suggest that 40% of SVC syndrome cases are related to benign etiologies, with the presence of an indwelling central venous catheter or percutaneous pacemaker now the most common benign cause.[1] Open repair is not a fun operation for surgeons, and its long-term patency is poor. Currently, endovenous stenting is the first-line therapy for benign etiologies of SVC syndrome.[2]

In this issue of the *Journal*, Muller and colleagues[3] describe SVC and brachiocephalic vein stenting for tandem stenotic lesions related to a left upper extremity percutaneous pacemaker resulting in bilateral upper extremity venous occlusive symptoms of type II SVC syndrome. Originally, balloon-expandable stents were used for precise placement, but the more recently introduced uncovered self-expanding stents may be ideal because of their greater flexibility. Covered stents may offer benefits in the setting of hemorrhage, perforation, or prevention of tumor ingrowth.[4] Some have also proposed the use of drug-eluting technology to reduce the rates of intimal hyperplasia. Predilation and postdilation are often performed, requiring larger-diameter high-pressure balloons to treat particularly resistant stenoses. With the use of intravenous devices, the confluence of the right and left brachiocephalic veins inclusive of the SVC is commonly involved. This often requires parallel kissing stents for management.[5] There has been some thought given to purposefully undersizing venous stents to increase the velocity of blood at the same flow rate to prevent stasis and optimize patency. Unilateral repair may be used with adequate success, even in bilateral disease.[5] Postoperatively, oral anticoagulation is routinely administered for 6 months and then individualized thereafter based on the patient's underlying etiology of occlusion.

Unfortunately, the primary patency of both endovenous and open repair is poor, with reported rates of 70% at 1 year and <50% at 3 years.[2] Therefore, close surveillance and early reintervention are needed to maintain high rates of primary assisted and secondary patency. Duplex ultrasonography is limited for intrathoracic pathology, so computed tomography or magnetic resonance venography is recommended before discharge and at 3 to 6 months and 1 year postoperatively.[5] This may be particularly important in the setting of ongoing intravenous device utilization. Multiple endovenous attempts generally do not preclude subsequent open repair for longer durability, but careful attention to the costs of multiple devices and reintervention should be an area of future study.

Ideally, the best solution for iatrogenic SVC syndrome related to pacemaker placement is preventing this complication in the first place. The exact etiologic mechanism is not known but is likely related to mechanical irritation. Lead exchange or manipulation may be associated with an increased risk of venous injury and stenosis and thus should be minimized.[6]

In conclusion, the incidence of SVC syndrome related to indwelling intravascular devices is on the rise. Stenting has become a popular and enticing fix to this complex problem,

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but requires careful patient selection, proper technical execution, and close postprocedure follow-up for durable improvements for patients.

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