coaptation depth of 8 mm with trivial AI (Fig 1B). The patient’s postoperative course was smooth, and he was discharged on postoperative day 14.

Comment

Our concept of BAV repair is based on previously reported principles [1–4] for transformation into a good bicommissural valve. In our patient, a prolapse of the conjoint cusp was observed by free margin elongation. The high TNI (0.63) of the reference cusp, which was reported to be most informative of the ability for it to be repaired [1], also supported our decision to repair. Complete detachment and closure of the raphe successfully created a good bicommissural valve, as planned. However, ventriculoaortic junction dilatation reduced the coaptation height to less than 2 mm. We then chose external annuloplasty to reduce the ventriculoaortic junction and increase the coaptation depth. Long-term recurrence of AI after BAV repair is a serious problem, especially in young patients. Our procedure theoretically reinforces coaptation of the valve by increasing its depth, which is expected to provide long-term durability of the repair.

Three techniques for aortic annuloplasty—subcommissural suture annuloplasty and internal and external ring annuloplasty—have been reported. In vitro comparison of the techniques [8] showed that the external and internal ring techniques had greater potential for reduction of the ventriculoaortic junction diameter. The external ring technique also provided paravalvular remodeling, in contrast with the internal ring technique. BAV is known to involve changes in the aortic root. Although the aortic root appeared normal in our patient, we chose external annuloplasty because of the expected paravalvular remodeling.

External ring annuloplasty combined with cusp repair provided excellent BAV repair. This approach is suitable for BAV patients whose ventriculoaortic junction is dilated without root dilatation.

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References


Open Surgery Repair for Superior Vena Cava Syndrome After Failed Endovascular Stenting

Huadong Li, MD, Xionggang Jiang, MD, and Tucheng Sun, MD

Department of Cardiovascular Surgery, Union Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China

Thrombosis is a rare cause of superior vena cava (SVC) syndrome. We report a 37-year-old man hospitalized because of swelling of the face and neck. A computed tomography angiography showed a thrombotic obstruction of SVC. The patient was treated by percutaneous transluminal balloon angioplasty of the SVC and placement of a stent. The symptoms disappeared, but the patient was hospitalized again after 3 months for the same complaints. Computed tomography angiography showed thrombosis in the stent in the SVC. The SVC was replaced with a prosthetic blood vessel. The patient’s postoperative recovery was uneventful, and SVC syndrome did not occur during 2 years of postoperative follow-up.


Superior vena cava (SVC) syndrome (SVCS) is the clinical manifestation of SVC obstruction, with severe reduction in venous backflow to the right atrium. William Hunter first reported SVCS in 1757. Most cases are caused by compression of the SVC due to malignancies such as lung cancer, lymphoma, or metastasis of solid tumors [1]. Other, less common causes include benign tumors, aortic aneurysm, thyroid enlargement, and fibrosis of the mediastinum [2]. Intravascular thrombosis is an extremely rare cause of SVCS. We present a patient with SVCS due to thrombosis caused by nonmalignant disease.

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Address correspondence to Dr Sun, Department of Cardiovascular Surgery, Union Hospital, Tongji Medical College, Huazhong University of Science and Technology, 1277 Jiefang Rd, Wuhan 430022, China; e-mail: suntucheng@126.com.
A 37-year-old man was first admitted to our hospital with complaints of swelling of the face and neck over 3 months. A computed tomography angiography (CTA) of the thoracic aorta demonstrated the obstruction of the SVC and vena azygos (Fig 1).

The decision was made to perform a percutaneous transluminal balloon angioplasty of the SVC and to insert a stent. Then, the patient was treated with aspirin (200 mg/d), and the symptoms disappeared for 3 months. At that time, the patient again experienced edematous facial swelling and anterior chest wall varicose veins, and was hospitalized immediately. There was no arthritis, lymphadenopathy, or diabetes, and there was no edema in the legs.

Blood pressure was 120/80 mm Hg, pulse was 80 beats/min, respiratory rate was 17 breaths/min, body temperature was 36.8°C, and there were no abnormalities in the chest auscultation. Blood test results indicated a hemoglobin level of 141 g/L, white blood cell count of 6.33 × 10^3/L, and platelets at 45 × 10^3/L. The activated partial thromboplastin time was 39.5 seconds and prothrombin time was 12.3 seconds. Concentrations of tumor markers were all within normal reference ranges. The functional levels of antithrombin III and protein S were within normal reference ranges. The coagulation parameters of protein C were 55.3%, which was lower than normal (reference range, 65% to 165%).

A CTA of the thoracic aorta showed thrombosis in the stent of the SVC (Fig 2). Positron emission tomography-CT imaging did not indicate malignancy. No abnormal observations were noted on a transthoracic echocardiogram. The patient had smoked for 17 years. There was no family history related to thrombophilia, and the patient had not undergone surgical operations or treatments with chemotherapy or radiotherapy. There were no mucocutaneous lesions or recurrent ulcers, indicating absence of signs for Behçet’s disease.

The patient underwent surgical intervention 3 days after the initiation of anticoagulation therapy with heparin. After a median sternotomy, cardiopulmonary bypass was initiated, and the SVC and the root of the innominate vein were replaced with a blood vessel prosthesis (Fig 3). During the operation, the wall of SVC proximal to the right atrium was observed to be much thicker than normal. The innominate vein proximal to the SVC was completely obstructed, and a small thrombus was found in the stent of the SVC (Fig 3). The excised tissue was confirmed to be a thrombus.

Outcome and Follow-Up
The patient’s postoperative recovery was uneventful. After discharge from the hospital, the patient received long-term oral anticoagulation with warfarin to an international normalized ratio of 2.5 to 3.0. However, the level of protein C was still less than normal. There was no recurrence of SVCS during 2 years of postoperative follow-up.

Comment
SVCS is the clinical condition of blood flow obstruction through the SVC resulting from intravascular obstruction or extrinsic compression. About 60% of SVCS cases result from malignancies such as lung cancer, lymphoma, or germ cell tumors. In benign cases, fibrosing mediastinitis, benign intrathoracic tumors, and inflammatory or infective processes typically cause SVCS [3].
Because of the increased use of pacemakers and central venous catheters for access or treatment purposes, thrombotic causes are increasing [4]. However, intravascular thrombosis leading to SVCS is still extremely rare.

We present a patient with SVCS due to unexplained thrombosis. The patient had no hypertension or diabetes and no obvious abnormal parameters in blood tests, other than a level of protein C that was lower than normal. It is entirely possible that the decreased level of protein C contributed to the pathology [5]. Another cause could have been that the patient was a smoker. Smoking is known to be a secondary risk factor for SVCS.

There are no exact guidelines for the clinical management of SVCS. The treatments include long-term anticoagulation, thrombolysis, percutaneous transluminal balloon angioplasty, stent implantation, and open surgical reconstruction. Surgical intervention has been the mainstay for treatment of benign SVCS [6]. However, Rizvi and colleagues [7] retrospectively reviewed a series of 70 patients with benign SVCS between 1983 and 2006. Of these patients, 93% were treated by open surgical reconstruction (OSR) or endovascular repair (EVR), which induced a significant relief from the symptoms [7]. These interventions are similarly effective for the short-term and midterm, whereas OSR is most effective for obtaining a long-term effect [7, 8].

To date, EVR is the first-line treatment for SVCS with a benign etiology because it is less invasive and is associated with lower morbidity by thrombosis [7]. Our patient was therefore treated by percutaneous transluminal balloon angioplasty and placement of an intravascular stent. Unfortunately, the patient presented after again 3 months later with thrombosis in the stent in the SVC. This recurrence was intervened on by surgical reconstruction. Postoperative anticoagulation with warfarin (international normalized ratio of 2.5 to 3.0) and aspirin (100 mg/d) [6] was effective for a period of 2 years of postoperative follow-up.

In conclusion, our report demonstrates that surgical management is an excellent choice for SVCS after failed EVR. EVR of benign SVCS was effective in the short-term and midterm and did not adversely affect subsequent OSR. After both EVR and OSR, long-term anticoagulation is suggested.

References

Pylorus-Preserving Pancreatectoduodenectomy After Coronary Artery Bypass Grafting Using Right Gastroepiploic Artery

Shinichi Fukuhara, MD, Marissa Montgomery, MD, Naruhiko Ikoma, MD, and Ryohei Miyata, MD, PhD

Department of Surgery, Beth Israel Medical Center, New York, New York; Department of Surgery, University of Texas Houston Health Science Center, Houston, Texas; and Department of Surgery, International Goodwill Hospital, Kanagawa, Japan

Coronary artery bypass grafting using right gastroepiploic artery and pylorus-preserving pancreatectoduodenectomy are both well known and commonly performed procedures independently. However, pylorus-preserving pancreatectoduodenectomy after coronary artery bypass grafting using right gastroepiploic artery has not been

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Address correspondence to Dr Fukuhara, Department of Surgery, Beth Israel Medical Center, First Ave at 16th Street, New York, NY 10003; e-mail: shin922@gmail.com.