Case Report

Superior Vena Cava Syndrome Following Central Venous Cannulation

M Abbasi, G Soltani, A Karamroudi, H Javan

Department of Cardiac Surgery, Imam Reza Hospital, Mashhad University of Medical Sciences ,Mashhad, Iran

Superior vena cava syndrome caused by blockage of the vein that carries blood from the head, neck, chest, and arms to the heart may occur due to various etiologies including thrombosis, occlusion and pressure on the superior vena cava. Foreign instruments in the vein, infections and also intimae injuries can lead to venacaval thrombosis . One of the most common causes of caval thrombosis is central venous catheterization for fluid administration and hemodialysis. This report presents an 8 years-old girl with chronic benign superior vena cava syndrome related to the long-term use of central venous catheters for hemodialysis. Treatment included resection of the obstructed segment and repair of the superior vena cava with an autologous pericardial patch. Reconstruction with an autologous pericardial patch without bypass of superior vena cava to right atrium is a safe and simple method but more importantly it is preferable and easier to prevent these events by simple nursing care in dialysis unit to secure the dialysis access.

Introduction

Superior vena cava syndrome (SVCS) is a collection of symptoms caused by blockage of the vein that carries blood from the head, neck, chest, and arms to the heart. Symptoms that may indicate this syndrome include dyspnea, coughing and swelling of the face, neck, upper body, and arms. In rare occasions, patients may complain of hoarseness, chest pain, dysphagia , and hemoptysis. Physical signs include swelling of neck or chest veins, collection of fluid in the face or arms, and rapid breathing. Rarely, cyanosis, Horner's syndrome (miosis, ptosis, and unilateral anhydrosis), and a paralyzed vocal cord may also be present.[1] SVC syndrome is known to occur secondary to many conditions such as malignancy, catheterization, granulomatous disease, retrosternal goiter and aortic aneurysm. Spontanous SVC syndrome is also to occur.[2] We report a case of SVC syndrome in a child with chronic renal failure on hemodialysis which was due to SVC stenosis secondary to catheter insertion.

Correspondence:
H Javan
General practitioner , Department of Cardiac Surgery, Imam Reza Hospital, Mashhad University of Medical Sciences, Mashhad, Iran
Tel : +98-511-8405559
E-mail: hadi61@gmail.com.

Case Report

In March 2006 an 8-year old girl was admitted to our hospital with SVC syndrome. She had a history of chronic renal failure secondary to reflux nephropathy. She had been hemodialysed since one year ago via A-V fistula on her wrist. Unfortunately, because of problems with arteriovenous fistula, hemodialysis was continued through a catheter which inserted into the right internal jugular vein. SVC syndrome appeared after 5 months. The catheter removed and peritoneal dialysis was begun. Upon admission to our department, the physical examination showed signs and symptoms of SVC syndrome. The diagnosis was confirmed with

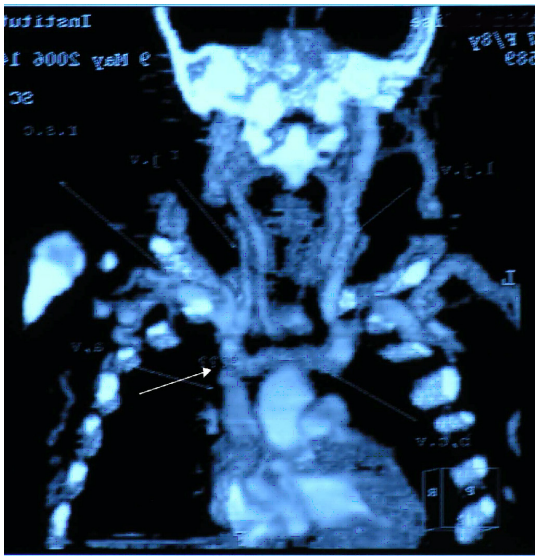

Figure 1. CT angiography showing occlusion of the SVC-inominate vein junction

CT angiography that showed total obstruction of junction of SVC and innominate vein, beyond the Azygus vein (fig 1). Due to extension of stenosis, percutaneous transluminal balloon angioplasty was unable to dilate the stenotic SVC, so she was scheduled for surgical treatment. Operation was done through a median sternotomy without CPB. Stenosis was at the level of junction of SVC and innominate vein with a length of 2 cm which extended below the Azygus vein. The anterior wall of SVC and innominate vein that was fibrotic, excised and repaired with a triangular piece of autologous pericardial patch .Operation was uneventful. Facial congestion and cyanosis resolved postoperatively. Edema of face and upper limb was resolved a few days after surgery. Patient discharged from hospital on the 7th day. Follow-up with vascular doppler ultrasonograply showed satisfactory graft patency with good blood flow.

Discussion

Superior vena cava syndrome is frequently encountered by cardiovascular clinicians. In this condition, SVC obstruction increases the venous pressure in the head and neck, elevating the cerebral venous pressure to anywhere from 20 to 50 mmHg.[3] Most cases are due to malignant diseases, but 10% to 20% arise from benign disorders such as chronic fibrosing mediastinitis or compression caused by an expanding thoracic aneurysm[4] One potential benign cause, which is steadily increasing in frequency, is stenosis and obstruction of the SVC associated with the use of long-term central venous catheters or permanent pacing electrodes.

Initial management of any patient with SVC syndrome includes supportive measures such as providing supplemental oxygen and elevating the head of the bed. Mainstays of treatment also

Include diuretics to reduce intravascular volume and at least a short course of parenteral steroids (dexamethasone, 4 mg / 6 hours) to decrease edema and tumor burden in certain malignancies, although these therapies remain unproven. In sever or non-responding to supportive therapy cases we suggest performing imaging procedures like CT, MRI or MRV (Mag-

netic resonance venography) and Venogram. In presence of acute thrombus,thrombolytic therapy is indicated.[5]

Several treatments have been introduced for SVC syndrome depending on the underlying disease which led to superior venacava obstruction. Anticoagulation, thrombolytic and endovascular treatments should be considered in case of catheter induced SVC syndrome[2] In the patients with malignancy as a source of SVC syndrome radiation and chemotherapy may relief the symptoms[6] Surgery could help when medical or interventional treatments fail. Various surgical techniques are available for repairing, reconstructing, replacing, or bypassing the obstructed SVC. Ideally, the procedure should be tailored to suit each individual patient. Either method is used, the goals are to relief symptoms, minimize the risk of complications (infection, central nervous system sequel, and upper respiratory tract edema and stridor), and providing long-term patency of the SVC.[7] These goals are achieved by providing a high-flow (750-2000 ml/min) conduit for venous return from the upper body. Whenever possible, one should perform an anatomic reconstruction, thereby avoiding the need for postoperative anticoagulants.

Various materials have been proposed for SVC repair or reconstruction. Currently, two autologous materials are used.[1] 1) pericardium, which may be fashioned in the shape of a patch or tube; and 2) Saphenous vein grafts, which may be split longitudinally and sutured in spiral fashion around a 40F stent, so as to have a diameter of 12 mm.[5] The preferred synthetic material is poly tetra fluoro ethylene (PTFE), which can take the form of a thin membrane or patch for repairing the SVC; alternatively, PTFE can take the form of a tube graft (commonly with external reinforcing rings) for reconstructing or bypassing the vessel.[4] Most surgeons, including us, favor autologous pericardium because it is easy to procure and prepare. In contrast, spiral saphenous vein grafts require 60 to 90 minutes to harvest and prepare.[8] Unlike prosthetic materials, autologous pericardium involves no risk of infection and does not necessitate anticoagulation therapy.[9]

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References

1 Wan JF, Bezjak A. Superior vena cava syndrome. *Emerg Med Clin North Am* 2009;**27**:243-55. [19447309]

2 Bauset R. Pacemaker-induced superior vena cava syndrome: a case report and review of management strategy. *Can J Cardiol* 2002;**18**:1229-32. [12464987]

3 Moore WM Jr, Hollier LH, Pickett TK. Superior vena cava and central venous reconstruction. *Surgery* 1991;**110**:35–41. [1866692]

4 Abner A. Approach to the patient who presents with superior vena cava obstruction. *Chest* 1993;**103**:394S–397S. [8462332]

5 DeCamp MM Jr, Swanson SJ, Sugarbaker DJ. The mediastinum. In: Baue AE, Geha AS, Hammond GL, Laks H, Naunheim KS, eds. Glenn's Thoracic and Cardiovascular Surgery. 6th Edn. Connecticut. Appleton and Lange; 1996. p. 643-64.

6 Warren WH, Piccione WJ Jr, Faber LP. As originally published in 1990: Superior vena caval reconstruction using autologous pericardium. Updated in 1998. *Ann Thorac Surg* 1998;**66**:291–3. [9692495]

7 Piccione W Jr, Faber LP, Warren WH. Superior vena caval reconstruction using autologous pericardium. *Ann Thorac Surg* 1990;**50**:417–9. [2400262]

8 Cheng S. Superior vena cava syndrome: a contemporary review of a historic disease. *Cardiol Rev* 2009;**17**:16-23. [19092366]

9 Doty DB, Doty JR, Jones KW. Bypass of superior vena cava. Fifteen years experience with spiral vein graft for obstruction of superior vena cava caused by benign disease. *J Thorac Cardiovasc Surg* 1990;**99**:889–96. [2329828]