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Superior Vena Cava Rupture during Balloon Angioplasty and Stent Placement to Relieve Superior Vena Cava Syndrome: A Case Report

Louis E. Samuels,[1] Joseph B. Nyzio Jr,[1] John W. C. Entwistle III[2]

[1]Department of Cardiothoracic Surgery, Lankenau Hospital, Wynnewood, Pennsylvania, USA;
[2]Department of Cardiothoracic Surgery, Hahnemann University Hospital, Philadelphia, Pennsylvania, USA

ABSTRACT

Percutaneous stenting of the superior vena cava (SVC) has been an accepted therapy for SVC syndrome for more than a decade. Complications are uncommon and usually of minor consequence. Three previous reports have described ruptures of the SVC during venoplasty with death on one occasion. We report a fourth case of SVC rupture during angioplasty and stenting that required immediate pericardiocentesis followed by open surgical repair via sternotomy for direct control and repair. An algorithm for rapid recognition and prompt intervention is described.

INTRODUCTION

Percutaneous stenting of the superior vena cava (SVC) has been an accepted therapy for SVC syndrome for over a decade. Complications are uncommon and usually of minor consequence. The purpose of this report is to describe a fourth case of iatrogenic rupture of the SVC during balloon angioplasty and stenting that resulted in shock secondary to cardiac tamponade. Emergent pericardiocentesis followed by sternotomy/pericardiotomy and SVC repair was necessary.

CASE REPORT

A 21-year-old woman presented with a history of neurogenic syncope and a 3-year history of poorly controlled cardiac arrhythmias. She had been diagnosed with recurrent atrial tachycardia versus inappropriate sinus tachycardia. She had previously undergone multiple sinus nodal ablations and was on nadolol to suppress the tachycardia. Despite these therapies, she remained symptomatic.

Physical examination was unremarkable, with stable vital signs and a resting heart rate of 60 bpm. She underwent successful radiofrequency ablation of the sinus node followed by implantation of a DDD permanent pacemaker via the left subclavian vein. Over the next several days, she developed increasing shortness of breath and swelling of both upper extremities. A chest radiograph demonstrated bilateral pleural effusions. Ultrasound of the left arm and central venous system showed diminished flow. Venous angiography showed obstruction at the SVC/right atrial junction. A diagnosis of SVC syndrome was made; the patient was placed on intravenous heparin and underwent upper body elevation. Over several days, the patient's clinical condition showed no improvement. An angioplasty of a focal tight stenosis of the SVC at the right atrial junction was performed and resulted in some improvement. Placement of an SVC stent was considered for definitive therapy and long-term patency.

The patient was taken to the electrophysiology suite where, prior to stent placement, removal of the pacemaker leads was performed. The underlying rhythm at this point was a low atrial rhythm at 50 to 60 bpm. The SVC stent was placed (via transfemoral approach), and plans to replace the pacemaker leads were made. However, the patient developed acute hypotension and cyanosis. She was intubated for respiratory distress. An echocardiogram demonstrated a pericardial effusion. Angiography with fluoroscopy showed dye within the pericardial space. The patient was resuscitated with pericardiocentesis and brought rapidly to the operating room. A sternotomy was performed with relief of the cardiac tamponade and stabilization of the vital signs. Active bleeding was observed in the posterolateral region of the SVC/right atrial junction. Digital control of the area was obtained while the clot was evacuated from the pericardium. Surgical repair was completed with direct sutures to the perforated site. Prior to closure, permanent epicardial pacing leads were placed on the right atrium and ventricle. These were tunneled subcutaneously into the right subcostal region and were connected to a subcutaneous pacing generator. The patient's symptoms were significantly improved after the procedure, and she had an uneventful hospital recovery. She was discharged with no shortness of breath, arrhythmia, or upper extremity edema.

DISCUSSION

Treatment options for SVC syndrome depend on the underlying etiology and include external-beam radiation,

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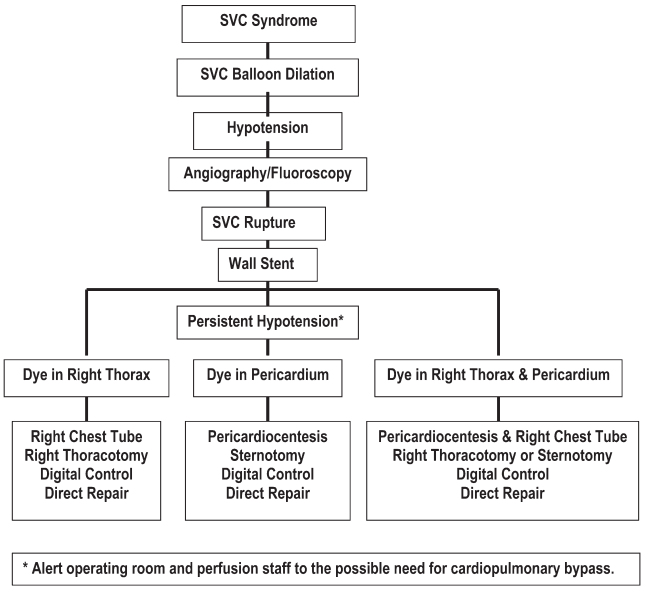
Correspondence: Louis E. Samuels, MD, Lankenau Hospital—MSB Suite #280, 100 Lancaster Avenue, Wynnewood, PA 19096, USA.



Algorithm for treatment of superior vena cava rupture/perforation.

chemotherapy, anticoagulation, thrombolysis, surgical bypass, and balloon venoplasty and/or stent placement. Percutaneous SVC stenting has been an accepted therapy for more than a decade and has resulted in satisfactory outcomes with minimal morbidity in the majority of cases [Hochrein 1998]. On 3 previous occasions, perforation of the SVC during venoplasty has been described [Burket 2003; Brown 2005; Oshima 2006]. In one case, the perforation was recognized promptly and successfully treated with a Wallgraft stent (Boston Scientific, Maple Grove, MN, USA) [Burket 2003]. In another case, the perforation resulted in cardiac tamponade—this complication was recognized late and the patient went on to expire despite closed-chest cardiopulmonary resuscitation [Brown 2005]. In a third case, the rupture was rapidly recognized and treated with pericardiocentesis (unsuccessfully) followed by transfer to the operating room for institution of percutaneous cardiopulmonary support and direct repair with an autologous pericardial patch [Oshima 2006]. In our case, the situation was recognized quickly and resuscitative manuevers were instituted to restore vital signs (ie, pericardiocentesis) while preparation for definitive repair (ie, sternotomy) was made in the operating room. Although percutaneous cardiopulmonary support was not necessary, a heart-lung machine was on standby.

The etiology of SVC syndrome in these 4 cases varied, although similarities between Oshima's report [2006] and ours is striking. In both cases, the patients were treated with multiple SA node ablations for inappropriate sinus tachycardia and had undergone placement of a pacemaker just prior to the development of the syndrome. Presumably, the SVC/right atrial junction was narrowed from the ablations and then occluded with the addition of pacemaker electrodes. This form of SVC syndrome has been described before [Leonelli 2000; Kai 2001]. In an effort to improve the caliber of the SVC, both patients underwent SVC balloon dilation. In our case, the expectation was that a stent would follow the balloon dilation, as described by Teo and others [2002]. Shortly after the procedure, however, both patients developed acute circulatory decompensation as a result of SVC rupture at the site of the stenosis. Both cases required formal operative repair since pericardiocentesis was inadequate.

Although complications are rare during SVC angioplasty and stenting, the consequences of rupture/perforation are life-threatening when they occur. Rapid recognition and prompt intervention are necessary for successful outcomes—as demonstrated in 3 of the 4 reported cases. In planning an SVC angioplasty with or without a stent, it is important to delineate a plan should a complication occur. An algorithm (Figure) for treating complications of SVC rupture/perforation must be established so that prompt treatment is instituted at the time of injury. First, rapid recognition is necessary with a high index of suspicion in a patient who becomes hypotensive during or following the procedure. There is very little reason for hypotension and shock, other than rupture/perforation, during this procedure—interruption of SVC flow into the heart during balloon inflation is not the cause of hemodynamic instability since there is little to no inflow due to the SVC syndrome in the first place. Anesthesia overdose and blood pressure monitoring malfunctions should be diagnoses of exclusion and not the primary reasons for an acute decompensation in vital signs. Thus, prompt recognition of a vascular complication should lead to a rapid response. Since angiography and fluoroscopy are available during the procedure, it may be useful to inject dye and demonstrate the injury. Fluoroscopy is excellent for showing the extravascular dye in the right chest, pericardium, or both. For those cases in which the injury is demonstrated in the absence of profound shock, application of a walled stent, as described by Burket [2003], is appropriate. In the event that cardiac tamponade is the case, a rapid pericardiocentesis can be instituted while the operating team is called and assembled. Bleeding into the right chest may require a chest tube, but this procedure will only serve to transfer blood from the thorax to an external container and not resolve the problem per se. Instead, an extra-pericardial injury with blood in the right chest associated with hemodynamic instability should prompt a thoracotomy and direct digital control and repair; for those cases in which dye is demonstrated in the right hemithorax without shock, a walled stent may alleviate the ongoing blood loss while a chest tube alone may be adequate. A trans-sternal approach may be considered for intrapericardial injuries—cardiac tamponade with shock refractory to pericardiocentesis should trigger the need for more aggressive access to the injury. In essence, SVC rupture/perforation differs from right ventricle perforation during pacemaker or defibrillator implants because the latter may seal itself with no further treatment whereas the former will likely go on bleeding. Last, in considering a stent for SVC dilatation, it makes more sense to use a walled/covered stent instead of a bare stent to minimize (if not eliminate) bleeding complications. Certainly if perforation/rupture occurred during dilatation prior to stent placement, then a walled/covered stent would be the device of choice.

In summary, SVC rupture/perforation during balloon angioplasty for SVC syndrome is rare, but potentially fatal when it occurs. Prompt recognition and rapid treatment are necessary to restore vital signs. Direct repair of the injury is necessary, either surgically or by the application of a walled stent.

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