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VASCULAR-INTERVENTIONAL

Percutaneous endovascular management of chronic superior vena cava syndrome of benign causes : long-term follow-up

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Abstract
Background The vast majority of superior vena cava (SVC) syndromes are of malignant causes, but with growing use of indwelling central catheters and implanted cardiac devices, benign SVC syndromes are becoming more frequent. The main objective of this study is to evaluate long term outcome in patients treated for benign SVC syndrome by endovascular techniques.
Methods Forty-four patients, 26 men and 18 women, mean age 56, treated for benign SVC syndrome using endovascular techniques between 2002 and 2015 were included. Type of obstruction was classified according to the site of disease and degree of occlusion. Complications and recurrence of symptoms were analyzed.
Results Technical and clinical success were achieved in all but one patient. Four patients (9 %) were treated by angioplasty alone and 40 (91 %) required stent implantation. Mean clinical follow-up was 1275 days. Nine patients had at least one episode of recurrence after a mean of 385 days. Four minor and two major complications were reported.
Conclusion Percutaneous endovascular techniques to treat benign SVC syndrome are safe with good long term patency. Recurrence of symptoms can easily be addressed by repeat procedure.
Key Points
- *Malposition of indwelling central catheter can cause superior vena cava obstruction.*
- *Image-guided catheter placement helps prevent superior vena cava obstruction.*
- *Imaging and superior vena cava obstruction classification allows adequate procedure planning.*
- *Endovascular techniques are safe and effective for superior vena cava syndrome treatment.*

Keywords Superior Vena Cava Syndrome · Endovascular Procedures · Angioplasty · Indwelling catheters · Oedema

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Abbreviation

AV	Arteriovenous
CNS	Central Nervous System
CT	Computed tomography
CTO	Chronic total occlusion
CVC	Central vein catheter
ICU	Intensive Care Unit
SVC	Superior Vena Cava

Introduction

Superior Vena Cava (SVC) syndrome is a group of pathologies that result in restriction or obstruction of venous blood flow from the upper part of the body back to the heart. It has a wide range of aetiologies including malignant and benign causes. SVC syndrome may present with neck or arm

swelling, but more serious presentations include laryngeal and bronchial oedema resulting in respiratory distress or coma from cerebral oedema [1]. Eighty to ninety percent of SVC syndromes are of malignant aetiologies [2–4], most frequently caused by extrinsic compression or invasion by bronchogenic carcinoma. However, with the increasing use of indwelling central catheters (implanted venous access devices, hemodialysis catheters) and transvenous cardiac devices (pace-maker or defibrillator), benign causes of SVC syndrome are becoming more frequent [5]. Since patients with benign SVC syndrome have a normal life expectancy as opposed to those with malignant disease, long-term relief of symptoms is a major factor in the choice of treatment modality. For a long time, surgical reconstruction of SVC or venous bypass were the only available treatments [2, 6, 7]. Superior vena cava stenting for palliative treatment of malignant SVC syndrome was introduced in 1986 [8] and has since been well described in the literature. A first series of 12 patients with endovascular treatment for benign SVC syndrome was published in 1999 [9].With the advances in the field, endovascular techniques have now become the first line treatment for patients with benign SVC syndrome [10]. Surgery is usually reserved for patients who are refractory to repeat angioplasty or stenting.

The aim of this study is to analyze the safety and efficiency of endovascular techniques for treatment of SVC syndrome of benign causes with specific regards to long-term results.

Material and methods

Patient selection

All patients referred to our institution's Radiology department for symptomatic SVC syndrome between October 2002 and March 2015 were analyzed. To be included, patients had to show signs or symptoms of SVC syndrome as described by Kishi et al. [11]. Patients had to have no active neoplasm and a sub-acute (2 weeks-2 months) or chronic (>2 months) clinical presentation. Treatment had to be done using endovascular techniques, i.e., balloon angioplasty and/or stenting. Acute (<2 weeks) cases were excluded because treatment of those patients relies mainly on thrombolysis and anticoagulation [12]. Asymptomatic patients and patients who were not treated using endovascular techniques were excluded. Patients who presented with SVC obstruction in the context of an arteriovenous fistula for hemodialysis were not excluded but analyzed separately because in these cases, arterialization of blood flow in central veins is in itself a precipitating factor for venous stenosis or obstruction [13, 14].

Our institution's Ethical Committee approved the study. All patients gave informed consent before the procedure.

Inclusion criteria were met in 44 patients during the study period (Table 1). Twenty-six men (59 %) and 18 women (41 %) with a mean age of 56 years (range 5-88 years) were included. The cause of caval obstruction was noted for every patient. Twenty-seven patients (61 %) had a history of indwelling CVC, 25 of whom (93 %) had a previous history of catheter malposition. The others had either a transvenous cardiac device or another cause of benign SVC syndrome (e.g, . extrinsic compression by benign process, previously treated neoplasm; see Table 2). A total of eight patients had a previous history of cancer but were all treated and considered in remission. The most common clinical presentation was face and neck swelling in 82 % ($n=36$) of patients. Seven patients (16 %) had isolated upper limb oedema, eight (18 %) presented respiratory difficulties, and seven (16 %) had laryngotracheal symptoms (e.g., voice change, cough). Six patients (14 %) showed symptoms of the central nervous system (CNS) including headaches and blurry vision. Almost all patients ($n=40$; 91 %) had apparent or dilated veins on their upper body. At the time of the procedure 35 (80 %) patients were under anticoagulation therapy.

Table 1 Patients' characteristics

		n	%
Gender	Male	26	59 %
	Female	18	41 %
Age (y)	Mean	56	
	Range	5–88	
Occlusion type	I	10	23 %
	II	11	25 %
	III	5	11 %
	IV	18	41 %

Patient classification and management

Patients were first assessed by CT phlebography [15] to evaluate central vein and azygos system patency (Fig. 1). SVC obstruction was classified according to the site and degree of obstruction using a modified Stanford classification [16]: type

Table 2 Causes of SVC obstruction

	n	%
Indwelling Central Catheter	27	61 %
History of Catheter Malposition	25	93 %
Tranvenous Cardiac Device	6	14 %
Previously Treated Neoplasm	3	7 %
Extrinsic Compression by Benign Process	3	7 %
Previous Mediastinal Surgery	3	7 %
Mediastinal Fibrosis	1	2 %
Other	2	5 %

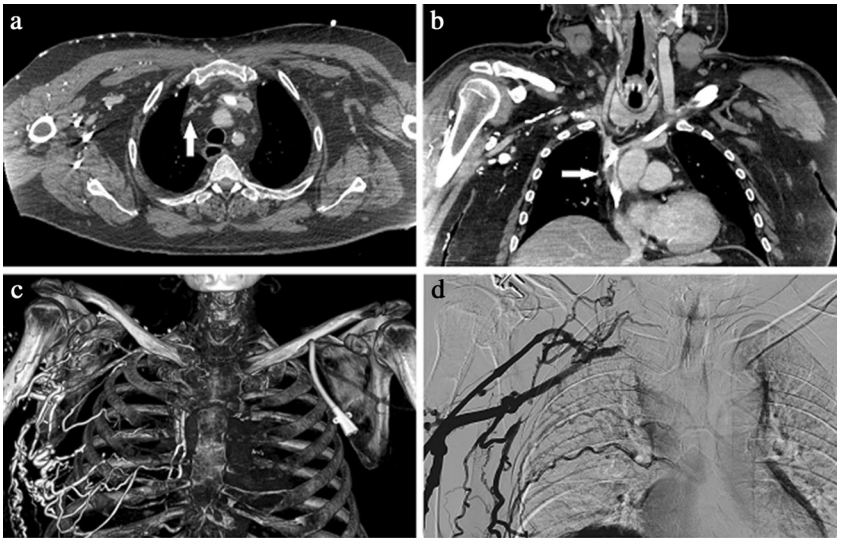

Fig. 1 CT-phlebography, patient with SVC obstruction from chronic dialysis catheter. **a**) Axial CT images showing total occlusion of right brachiocephalic vein (white arrow). **b**) Coronal CT images show extension to the SVC (white arrow). **c**) 3-D surface rendering in same patient shows the abundance of collateral circulation developed over time. These dilated veins were visible on patient's skin. **d**) Subtracted venography correlation showing total occlusion of SVC and right brachiocephalic vein. Contrast flows through parietal collateral circulation and into the intercostals veins

I corresponds to an isolated stenosis of the superior vena cava; type II, stenosis of central veins (subclavian/brachio-cephalic) with or without extension to SVC; type III, chronic total occlusion (CTO) of SVC and type IV, CTO of one or more central veins with or without extension to SVC (Fig. 2). Ten patients (23 %) had a type I SVC obstruction, 11 (25 %) had a type II, five (11 %) had a type II,I and 18 (41 %) had a type IV.

Classification allowed for better treatment planning prior to the intervention. Treatment strategy was established according to the type of disease. For type I and II, treatment was limited to the site of stenosis. For type III and IV, the main goal was to reestablish direct venous flow between the heart and the brain through the dominant jugular axis. When this could not be achieved (sometimes in type IV), the non-dominant side was

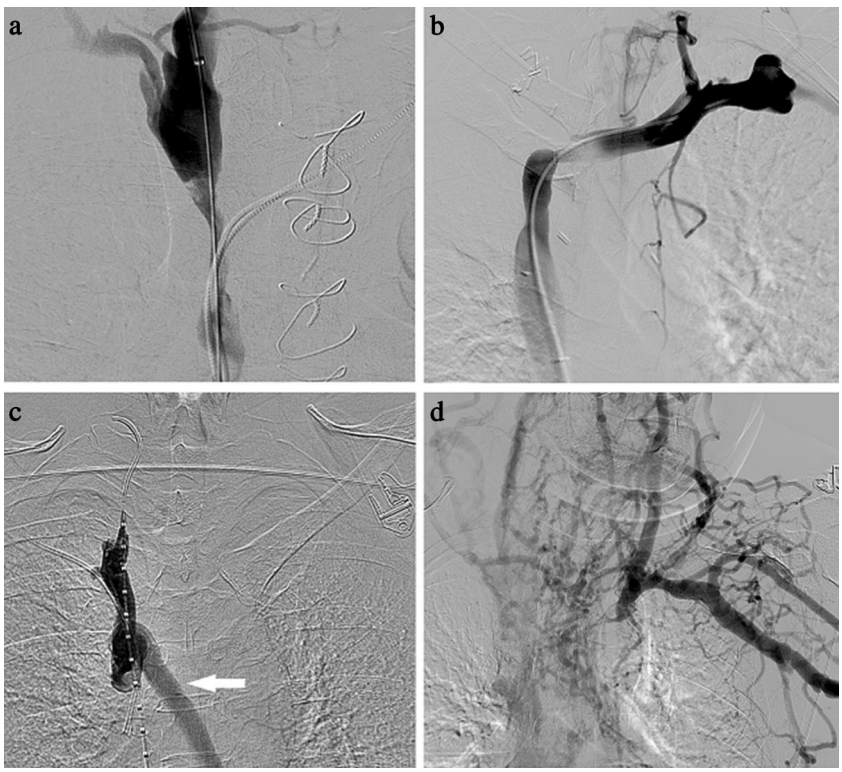

Fig. 2 Digitally subtracted venography showing different types of SVC obstruction. **a**) Type I: Patient with isolated SVC stenosis due to pace maker leads. **b**) Type II: Stenosis of the innominate vein just before its junction with the SVC. **c**) Type III: Complete occlusion of SVC. Note that the right brachiocephalic vein is patent and blood flows through the azygos system (white arrow). **d**) Type IV: Total occlusion of SVC and innominate vein. Note the important collateral circulation that developed over time

treated. Ultimately, if none of the jugular axis could be treated, flow was reestablished between the superior vena cava and a subclavian vein.

Procedure description

In patients presenting with complex SVC obstruction (typically type III and IV), especially if multiple venous access was needed for treatment, procedures were performed under general anaesthesia. Patients with severe comorbidities, such as heart disease, were referred to the intensive care unit after treatment for risks of pulmonary oedema after restoration of venous blood flow. Local anaesthesia was used for patients with less complex cases.

Procedures were all done by the same team; one interventional radiologist with 25 years of experience did 36 procedures (82 %). Using a single or combined venous approach (femoral, basilic and/or internal jugular), a 6 to 8 French, 45-60 cm long introducer sheath was inserted (Destination, Terumo Medical Corporation, Elkton, MD, USA). First, digitally-subtracted venography was performed (Fig. 2). Then, hydrophilic guidewire-based recanalization was performed. Prior to angioplasty and/or stenting, 5000 to 10000 UI of heparin was administered. Measurements were obtained in the adjacent patent venous segments, using transcatheter venography and/or CT-phlebography. High-pressure balloon angioplasty was then performed starting with a small-size balloon and moving to progressively larger balloons using techniques described by Qanadli et al. [9]. Self-expandable metallic stents (stainless steel alloy or nitinol; details in Table 3) were used to complete treatment if residual stenosis was noted or if blood flow through collateral circulation was still present at control venography (Fig. 3). Stent diameter was adapted to patent vein caliber, oversizing by 10 to 20 %. Stent length was adapted to stenosis extent and multiple overlapping stents (of same brand and model) were used if necessary. Stent remodelling was obtained by in-stent (shorter) balloon angioplasty. Caution was used to avoid stent displacement when manipulating balloons. In the SVC, the midportion of the stent was underdilated whereas the distal and proximal ends were correctly apposed to the venous wall. This manoeuvre is expected to avoid caudal migration of the stent into the right atrium.

Technical success was defined as reestablishment of central vein patency and restoration of venous blood flow to a physiological pattern. Clinical success was defined as regression (Kishi score < 4) or resolution of symptoms 48 hours to one week after treatment or at discharge. Complications were classified as minor or major according to the Society of Interventional Radiology standards [17]. After the procedure, anticoagulation was continued in patients who were already under such regimen before SVC syndrome treatment, and for a minimum of 6 months. Six patients received anticoagulation and aspirin and three received aspirin and clopidogrel.

All patients were systematically re-examined by the performing radiologist 3 months after treatment. Clinical evaluation and physical examination was obtained. Imaging was done only if recurring symptoms were noted (Kishi score > 4). Follow-up was performed by the referring physician after that period. If clinically significant symptoms occurred during the follow-up period, patients were treated again using endovascular techniques. At the end of the study period, patient files were analyzed to look for recurrence of symptoms during follow-up.

Table 3 Details of initial procedure

		n	%
Type of anesthesia	General	17	39 %
	Local	27	61 %
Venous Access	Femoral	24	55 %
	Basilic	9	20 %
	Internal Jugular	1	2 %
	Combined Access	9	20 %
	Other	1	2 %
Stents used	Wallstent™	20	50 %
	SinusXL™	8	20 %
	Luminexx™	6	15 %
	S.M.A.R.T™	4	10 %
	Philon™	1	3 %
	Maris™	1	3 %

Wallstent™ (Boston Scientific Corporation, Marlborough, Massachusetts, USA); SinusXL™ (Optimed Medizinische Instrumente, Ettlingen, Germany); Luminexx™ (Bard, Tempe, Arizona, USA); S.M.A.R.T.™ (Cordis Corporation, Hialeah, Florida, USA); Philon™ (Biotronik, Berlin, Germany); Maris™ (Medtronik, Dublin, Ireland)

Results

Twenty-seven procedures were done under local anaesthesia and general anaesthesia was required for the remaining 17 procedures (Table 3). Treatment was done by a femoral ($n = 24$; 55 %), basilic ($n = 9$; 20 %) or combined ($n = 9$; 20 %) approach. A left internal jugular approach was used in one (2 %) patient and another had a left sub-clavian tunnelled dialysis catheter which was exchanged for the introducer sheath over a guidewire. Only a minority of patients were treated with balloon angioplasty alone ($n = 4$; 9 %) and most required stent implantation ($n = 40$; 91 %). Stent diameter varied between 10 and 24 mm and stent length ranged from 30 to 100 mm. Most of the stents that were used were either Wallstent™ (Boston Scientific Corporation, Marlborough, Massachusetts, USA) ($n = 17$), SinusXL™ (Optimed, Ettlingen, Germany) ($n = 9$) and Luminexx™ (Bard Peripheral Vascular Inc., Tempe, AZ, USA) ($n = 5$). Technical and clinical success was achieved in all but one

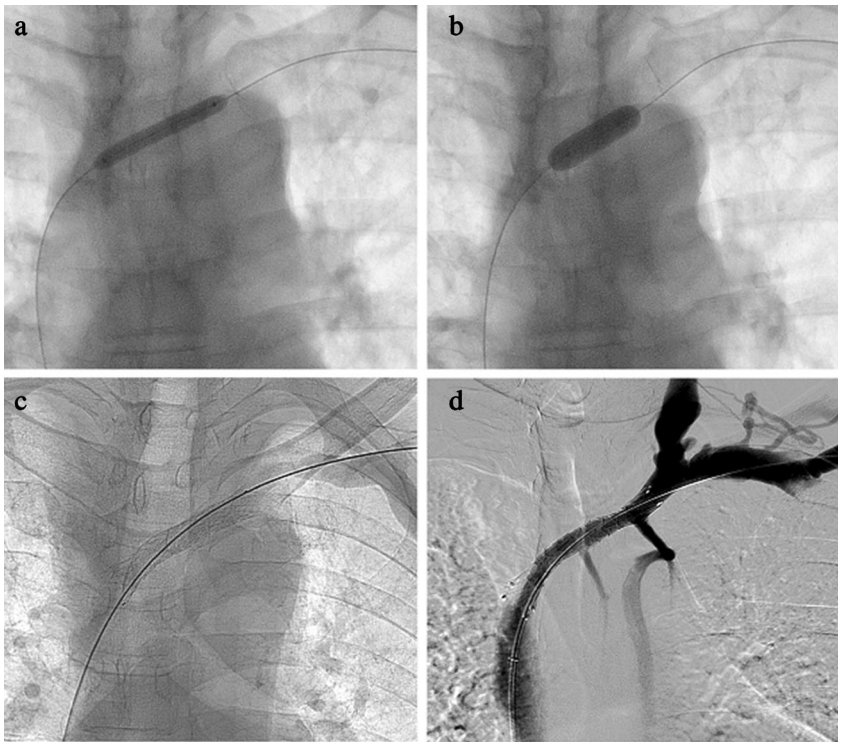

Fig. 3 Progressive balloon angioplasty in patient with type III obstruction. **a**) - **b**) To avoid vein rupture, progressive balloon angioplasty is indicated. Prolonged inflation of smaller balloons is first performed before moving on to larger sizes. This patient required stent implantation because of residual stenosis (**c**). **d**) Final result after angioplasty and stent placement

patient. Puncture site hematoma was noted in three patients. Two major complications occurred. One patient had acute pulmonary oedema after the procedure and was admitted to the intensive care unit. The second major complication was SVC rupture during angioplasty. The procedure was interrupted and the patient died of cardiac tamponade shortly after. No other complication was reported.

Clinical follow-up was available for 31 patients and ranged from 30 to 5764 days (mean 1275 days). Nine patients (19.6 %) showed recurrences. Five patients had one episode, one had a total of three episodes, two patients had three episodes and one patient had four episodes of recurrence during the study period. All episodes were successfully treated again using endovascular techniques after a mean of 385 days (range 60-1269 days). Intimal hyperplasia causing intrastent restenosis was the main cause for recurrence and most were treated using angioplasty alone ($n = 13$; 72 %), the others had new stent implantation. One patient had acute cruoric intra-stent thrombosis after 394 days and was successfully treated by IV and oral anticoagulation. Intra-stent stenosis was found in one asymptomatic patient on a thoracic CT-scan 614 days after initial treatment. He was treated by balloon angioplasty to prevent stent occlusion. Four recurrences were attributed to stent retraction and malposition. One patient had SVC stenosis caused by pacemaker leads and presented with severe face, neck and upper limb oedema, a changed voice, and some difficulty breathing. Angioplasty was performed and a stent was placed with good technical and clinical success. Three and a half years later, his pacemaker was changed and the leads were removed. The stent was displaced and damaged during this procedure and the patient presented with recurring symptoms. Intra-stent stenosis was found at phlebography and angioplasty was successfully performed before the new pacemaker leads could be placed.

Recurrence-free survival was evaluated at 1 and 3 years and analyzed separately for patients who had AV fistula for hemodialysis access. For patients without fistula, recurrence-free survival was 91 % at one year and 73 % at three years. In patients with AV fistula, recurrence-free survival was 44 % at one year and 60 % at 3 years. Assisted patency of the SVC system was 100 % at one and three years. Clinical follow-up was unavailable for seven patients, all of them because they were referred from another hospital and their external files could not be consulted. Four patients died during follow-up period after a mean of 1118 days (range 42-3511 days) after procedure.

Discussion

Superior Vena Cava syndrome was first described in 1795 and has since then been well documented in the literature. More than 80 % of cases are caused by extrinsic compression or invasion of superior vena cava by bronchogenic carcinoma and other tumours. Non-malignant obstruction is much less frequent and accounts for only 10-20 % of cases [2–4]. It has not been described as much in the literature. Indwelling central catheters such as dialysis catheters or implanted venous

access devices have been proposed as a main cause for central vein thrombosis, stenosis or occlusion of benign aetiologies. Some studies have shown that this risk is even higher if the catheter is not in optimal position (i.e., the cavo-atrial junction), especially if catheter is too short [18–21]. This was the case in the vast majority of our patients. Transvenous pacemaker leads have also been shown to cause vein stenosis and occlusion [22–24]. Other less frequent benign causes include mediastinal fibrosis, extrinsic compression by a nonmalignant process (mediastinal hematoma, thyroid goiter, benign tumour), and cardiovascular causes such as aortic aneurysm or dissection [25]. We believe simple measures can be taken to minimize the risks of catheter-related complications in a central venous system, such as fluoroscopy-guided CVC implantation. Furthermore, as suggested by Haller et al. [26], efforts should be made to recanalize occluded central veins when placing a new device, rather than potentially sacrificing the only patent side. We believe these measures could diminish the incidence of benign SVC obstruction.

As mentioned earlier, patients with benign SVC obstruction have a normal life expectancy as opposed to patients with malignant causes who show a mean survival of 6 months after stenting [27]. Therefore, long-term relief of symptoms is an important factor when choosing a modality of treatment in these patients. Some studies have reported a good short-, mid- and long-term patency rate in patients treated for benign SVC syndrome with endovascular techniques, similar to those achieved with surgery [10, 28, 29]. Some case reports showing absence of symptoms 2 to 5 years after endovascular treatment of benign SVC syndrome have also been published [30–32]. In our study, nine patients presented a total of 18 episodes of recurring symptoms during the study period, which can seem like a great many. However, five of them had an upper-limb arteriovenous fistula for dialysis treatment, which in itself is a precipitating factor for central vein complications. They accounted for 11 of the 18 episodes. Recurrence-free survival at one and three years was still very good for patients without AV fistulas. The majority of recurrences were attributed to intra-stent restenosis due to intimal hyperplasia (Fig. 4). Drug-coated balloons (DCB) are thought to reduce intimal hyperplasia, and their use is becoming more recognized for de novo treatment and reintervention in patients with coronary and peripheral artery disease [33–36]. A recent study showed good results in patients with hemodialytic AV fistulae treated for central vein stenosis [37]. The use of DCBs in SVC obstruction of benign causes could potentially improve primary patency and reduce the incidence of restenosis. More dedicated studies are needed to evaluate the use of DCBs in this setting.

Also, most of the patients presented in this study were implanted with Wallstents™, an older device that is known to get shorter when deployed (stent retraction), sometimes months after implantation [38, 39]. This was identified as the cause for recurrences in four of our patients. This, however, is not an issue with latest generation nitinol-based stents, and we believe the use of these newer devices will help diminish the number of recurrences.

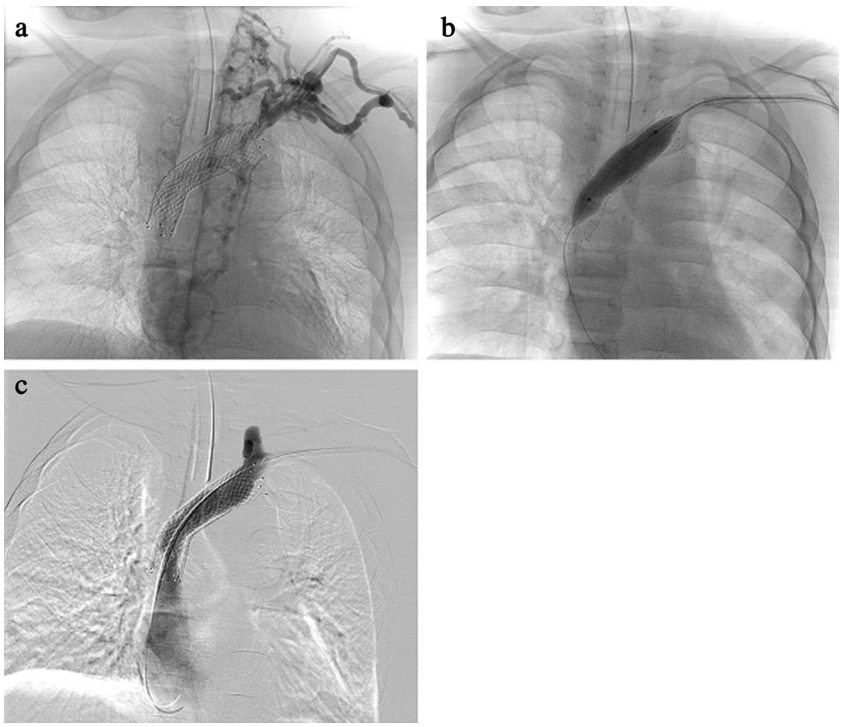

Fig. 4 Patient presenting with recurring symptoms of SVC syndrome 2 years after initial procedure. **a**) Subtracted venography shows intra-stent occlusion and development of multiple collateral veins. **b**) Recanalization was achieved with a hydrophilic guidewire and intra-stent angioplasty was performed. **c**) Control venography shows a good result with the absence of residual stenosis and complete regression of collateral circulation. Symptoms regressed in 48 hours

Endovascular procedures for management of benign SVC obstruction are quite safe. When comparing different treatment options for SVC obstruction of benign causes, Rizvi et al. showed that patient treated with open surgery were exposed to more complications and had longer hospital stays after surgery than those treated by endovascular reconstruction [10]. They showed a low periprocedural morbidity rate of 4 % in patients treated with endovascular repair as opposed to 19 % in the open surgical repair group. In our study, we reported very few complications directly linked to the procedure. Most of them were minor complications. One patient suffered severe pulmonary oedema after treatment and was transferred to the ICU for management. This happened at the beginning of our experience. Afterwards, patients with greater risks of developing pulmonary oedema (e.g., heart disease) were preventively admitted to the ICU immediately after the procedure. The only other major complication that occurred was iatrogenic superior vena cava rupture and patient death by cardiac tamponade shortly after the procedure. To avoid vein perforation or rupture, angioplasty should be done in a progressive manner, starting with longer inflation of smaller diameter balloons and slowly increasing in size. Immediate angiographic control should be obtained after angioplasty or if vein rupture is suspected to allow early detection. Prolonged (two 5-minutes cycles) low-pressure balloon inflation over the site of the rupture is indicated to stop bleeding [40]. A covered stent should be placed if haemorrhage persists.

The modified Stanford classification that was described earlier was especially useful for therapy planning. It is a purely morphologic classification and does not take into account the severity of the symptoms. It was merely used as a tool to plan the procedure and predict which patients would need a combined femoral and jugular/basilic approach. We did not evaluate the impact on patient outcome.

Our study has some limitations. First and foremost, non-malignant SVC obstruction is much less common than malignant disease. It is, therefore, almost impossible to have a prospective or randomized trial to compare different treatment strategies. The second limitation is the long-term follow-up that was hard to obtain for all patients as some of them were referred from other hospitals and were lost after the first systematic follow-up visit.

To our knowledge, this is the biggest published series of non-malignant superior vena cava syndrome. Mid- to long-term follow up was available for a majority of patients, and only a few of them presented with recurrence. Those results are consistent with the literature and quite similar to what can be achieved by surgery. Furthermore, endovascular treatment does not preclude surgery for refractory cases. Endovascular techniques being less invasive and requiring a shorter hospitalization and recovery than surgery, should, as other authors have suggested, be used as first line therapy in patients with non-malignant SVC syndrome. Furthermore, thorough understanding of pathogenesis of benign SVC syndrome can be integrated and simple measures should be taken to reduce the occurrence of this potentially debilitating disease.

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