

Percutaneous stent placement in malignant cases of superior vena cava syndrome

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Summary

Purpose: Superior vena cava (SVC) syndrome is caused by SVC stenosis or occlusion, frequently as a consequence of lung cancer or a mediastinal tumor. SVC syndrome is characterized by unpleasant symptoms and the condition usually leads to death if untreated. Treatment with radiation therapy and chemotherapy may produce an initial relief, whereas operations with bypass are associated with high mortality and morbidity. The purpose of our study was to show the efficiency of percutaneous stenting in the SVC for relieving SVC syndrome secondary to malignant diseases.

Patients and methods: From January 1999 to March 2003, 17 patients with malignant SVC syndrome were evaluated at the "Metaxa" Cancer Hospital. Their caval stenoses were confirmed by means of computed tomography and venography. There were 15 males and 2 females with a median age of 62 years (range 47-79).

The SVC syndrome was caused by malignant disease

in all patients: bronchogenic carcinoma in 14 and lymphoma in 3.

All patients underwent placement of a self-expandable (wallstent) endovascular (vena cava) prosthesis.

Results: All procedures were successfully carried out without complications. The average time for wallstent placement was 37 min. There was no sign of bleeding and the wallstent was well positioned on chest roentgenograms.

All patients, without exception, noticed an immediate improvement, with relief of dyspnea and rapid resolution of headache. Cyanosis disappeared over the first hour and swelling resolved gradually over the first 24 hours.

Conclusion: Percutaneous venous wallstent placement in the SVC is a simple, safe and effective technique to rapidly relieve SVC syndrome caused by malignant diseases.

Key words: lung cancer, percutaneous stent placement, superior vena cava syndrome, thoracic malignancies

Introduction

Obstruction of blood flow in the SVC results in symptoms and signs of SVC syndrome. SVC obstruction can be caused either by invasion or external compression of the SVC by contiguous pathologic pro-

cesses involving the right lung, lymph nodes, and other mediastinal structures, or by thrombosis of blood within the SVC. Occasionally, both mechanisms coexist.

SVC syndrome was first described by William Hunter in 1757 [1]. This syndrome is caused by malignant diseases in 85-95% of the reported cases [2,3]. Bronchogenic carcinoma is the etiology in about 80% of the cases, with lymphoma and metastatic disease comprising 15% and 5%, respectively. Mediastinal inflammation as a result of fibrosing mediastinitis and granulomatous disease caused by histoplasmosis predominates the benign causes of SVC syndrome [4].

With the advent of long-term central venous catheterization for diagnostic or therapeutic interventions, catheter-related thrombosis causing SVC syndrome is characterized by swelling of the face, neck and arms, with dyspnea, cough, and collateral thoracic circulation [5].

Received 17-02-2005; Accepted 07-04-2005

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Treatment with radiation therapy and chemotherapy may produce an initial relief, whereas operations with bypass are associated with high mortality and morbidity [6,7].

The purpose of our study was to show the efficiency of percutaneous stenting in the SVC for relieving SVC syndrome secondary to malignant diseases.

Patients and methods

From January 1999 to March 2002, 17 patients with malignant SVC syndrome were evaluated at the "Metaxa" Cancer Hospital. Their caval stenoses were confirmed by computed tomography and venography (Figure 1). The procedure was performed under local anesthesia. Patients were monitored for oxygen saturation, blood pressure and cardiac function (electrocardiogram). The stents were introduced into the right common femoral vein through a 9- French sheath. We used self- expanding wallstents in all patients.

A SVC cavogram was systematically carried out in all patients by femoral injection of 50 ml Ultravist-300 with opacification of the bronchiocephalic confluence (Figure 1).

A final SVC cavogram confirmed the potency of the stent, showing that the stent placement was correct in all patients with sufficient caliber of the stenosed SVC (Figures 2-4). Intravenous heparin was adminis-

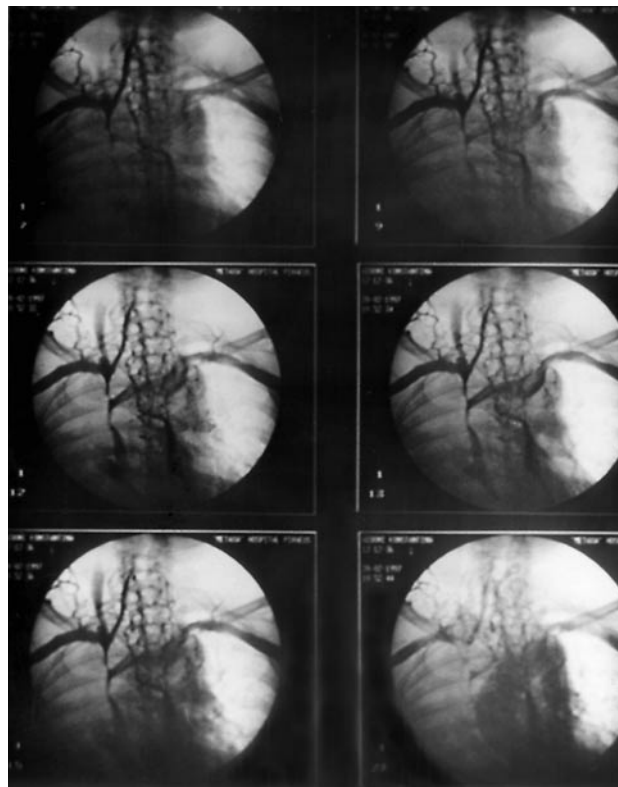


Figure 1. Venography in patients with superior vena cava syndrome.

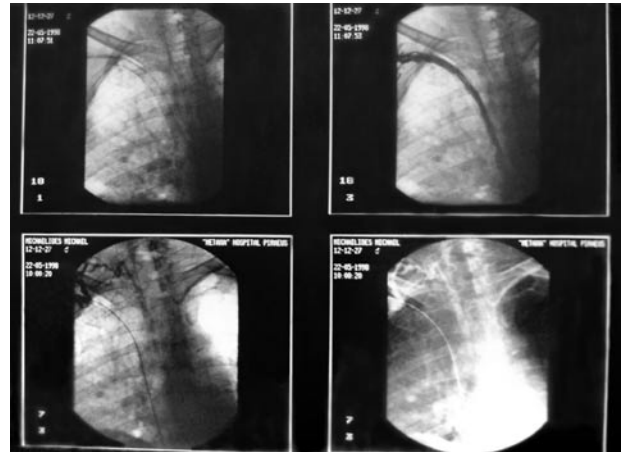


Figure 2. Venography after placement of the stent.

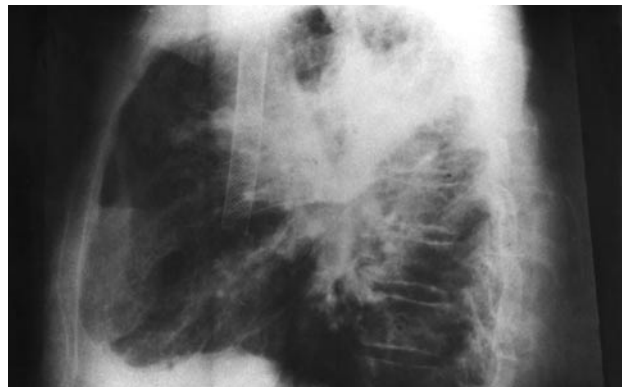


Figure 3. Thoracic radiograph after positioning of the stent.

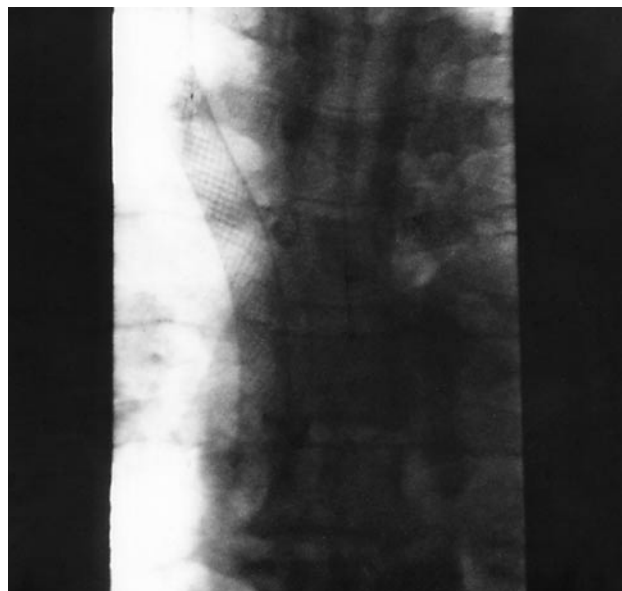


Figure 4. Positive control of the stent placement in the superior vena cava.

tered to all patients (5000 IU bolus) at the beginning of the procedure, followed by infusion of 1000 IU per hour for the first 24 hours to maintain the heparin plasma level at twice the normal thromboplastin level. On day 2 after the procedure, low molecular weight heparin was administered and continued for at least 1 month and then oral anticoagulation therapy (1 mg acenocoumarol and aspirin 100 mg daily) was given.

Results

Fifteen males and 2 females patients aged from 47-79 years (median 62), were subjected to stent placement. All of them had swelling of the neck and face with venous distention (Figure 5a and b). One patient presented cerebral edema and another one hoarseness. The SVC syndrome was caused by malignant disease in all patients: bronchogenic carcinoma in 14 and lymphoma in 3. The mechanism of SVC syndrome was caused by either lymph node compression of the SVC in 9 patients or direct compression of the SVC by lung cancer invading the mediastinum in 8 patients.



Figure 5a and b. A patient with neck and face edema, and vein distention of the anterior thoracic wall.

All procedures were successfully carried out without any complication. The average time for wallstent placement was 37 min (range 23-49). All patients, without exception, noticed an immediate improvement with relief of dyspnea and rapid resolution of headache. Cyanosis disappeared over the first hour and swelling resolved over the first 24 hours. SVC syndrome was completely relieved in all patients within 2 weeks. No clinical complications occurred; in particular, no patient experienced clinical symptoms of pulmonary embolism or pulmonary edema during or after the procedure.

The patients' follow-up ranged from 0.5-7 months (median 5.5). Re-obstruction of the stent and recurrence of the SVC syndrome occurred in 2 (11.4%) patients, due disease progression. The median survival time of the 14 lung cancer patients was 3.2 months (range 1-7) from the date of the stent placement, while the 3 patients with lymphoma were lost to follow-up 7 months after stent placement, being alive and well at that time.

Discussion

Because SVC syndrome is mainly caused by advanced lung and mediastinal cancers, it is generally associated with poor prognosis [8]. As shown in our study, the median survival time was only 3.2 months. Therefore, good palliation is the main objective in such cases. When radiation therapy and chemotherapy are inefficient to improve SVC syndrome, other techniques must be discussed. With recent advances in percutaneous interventional vascular technology, thrombolytic agents, angioplasty and intravascular stents have been used in the management of SVC obstruction [9].

The intraluminal self-expanding percutaneous stent placement was performed under local anesthesia with radiological control. The stent can be compressed and introduced through a Teflon polytetrafluoroethylene catheter sized 8F-12F, depending on the caliber of the wire and the diameters of the stent.

When the stent is released from the catheter, it expands to its original diameters [10]. The wallstent device maintains the advantage of the metal stent and its narrow woven mesh greater flexibility and longer length make it superior to the Gianturco stent [10].

Pulmonary edema, due to the sudden increase in venous return after SVC stent placement, has been reported [11]. However, patients with chronic complete vessel occlusion or severe coagulopathy and those with cardiac failure are candidates for intravascular stent placement [12].

Before placing a SVC stent, the extent and degree of stenosis or obstruction must be meticulously studied [13,14]. A venogram in two different projections is essential. The extent of stenosis may be poorly visualized and may actually be misleading if only one projection is obtained. The venogram should allow for evaluation of the length, severity and location of the obstruction. Careful evaluation of the extent of collateral circulation and the presence of thrombus or tumor ingrowth should be well documented before intervention [15,16]. Not infrequently, patients with SVC syndrome present with acute or subacute thrombosis of the SVC. The initial treatment of choice in those circumstances is thrombolytic therapy [17]. The thrombolytic agent is delivered directly into the thrombus by infusion catheters that are centered in the thrombus [18]. When the thrombus has been dissolved, the underlying obstruction can be identified and treated with stent placement. The thrombolytic agents most commonly used are urokinase (2000 IU/kg/h) and tissue plasminogen activator (0.02 mg/kg/h) [19].

In conclusion, percutaneous venous stent placement in the SVC is a safe and simple palliative procedure for treating SVC syndrome caused by advanced lung cancer and lymphoma [20-22]. Palliation of symptoms is achieved immediately in the vast majority of patients. Complications are rare and usually minor.

References

1. Parish JM, Marschke RF, Dines DE, Lee RE. Etiologic considerations in SVC syndrome. *Mayo Clin Proc* 1981;56: 407-413.
2. Perez CA, Presant CA, van Ambury AP. Management of superior vena cava syndrome. *Semin Oncol* 1978;5: 123-134.
3. Lochridge SK, Patrick K, Poty DB. Obstruction of superior vena cava. *Surgery* 1979;85:14-24.
4. Dotty DB, Doty JR, Jones KW. Bypass of superior vena cava: Fifteen years' experience with spiral vein grafts for obstruction of superior vena cava caused by benign disease. *J Thorac Cardiovasc Surg* 1990;99:889-896.
5. Capek P, Cope C. Percutaneous treatment of superior vena cava syndrome. *AJR* 1989;152: 183-184.
6. Davenport D, Fueue C, Blake D, Raken M. Radiation therapy in the treatment of superior vena cava obstruction. *Cancer* 1978; 42: 2600-2603.
7. Chu CJ, Terzis J, McRace ML. Replacement of superior vena cava with spiral composite vein graft. *Ann Thorac Surg* 1974; 17: 555-560.
8. Courtheoux B, Alkoter B, Refai M, Gervais R, Le Rochais JP, Icard P. Stent placement in SVCS. *Ann Thorac Surg* 2000; 75: 158-161.
9. Dyet JF, Nicholson LA, Cook AM. The use of the Wallstent endovascular prosthesis in the treatment of malignant obstruction of the superior vena cava. *Clin Radiology* 1993;48: 381-385.
10. Shah R, Sabanathay S, Lowe RA, Mearus AJ. Stenting in malignant obstruction of superior vena cava. *J Thorac and Cardiovasc Surg* 1996;112:335-340.
11. Kishi K, Sonolnura T, Mitsuzane K et al. Self-expandable metallic stent therapy for superior vena cava syndrome: clinical observations. *Radiology* 1993;89:531-535.
12. Oudkerk M, Heystraten FMJ, Stater G. Stenting in malignant vena cava obstruction. *Cancer* 1993;71:142-146.
13. Yim CD, Sane SS, Bjarnason H. Superior vena cava stenting. *Intern Chest Radiol* 2000;38:409-424.
14. Garcia Monaco R, Bertoni H, Pallota G et al. Use of self-expanding vascular endoprosthesis in superior vena cava syndrome. *Eur J Cardiovasc Surg* 2003; 24: 208-211.
15. Greillier L, Barlesi F, Doddoli C et al. Vascular stenting for palliation of superior vena cava obstruction in non-small-cell lung cancer patients: A future 'standard' procedure. *Respiration* 2004; 71: 178-183.
16. Urruticoechea A, Mesia R, Dominquez J et al. Treatment of malignant superior vena cava syndrome by endovascular stent insertion. Experience on 52 patients with lung cancer. *Lung Cancer* 2004;43:209-214.
17. Lanciego C, Chacon JL, Julian A et al. Stenting as first option for endovascular treatment of malignant superior vena cava syndrome. *Am J Rontgenol* 2001; 177: 585-593.
18. Slonim SM, Semba CP, Sze DY, Dake MD. Placement of SVC stents over pacemaker wires for the treatment of SVC syndrome. *J Vasc Interv Radiol* 2000;11:215-219.
19. Kee S, Kinoshita L, Ravzi M et al. Superior vena cava syndrome: treatment with catheter-directed thrombolysis and endovascular stent placement. *Radiology* 1998;206:187-193.
20. Marcy PY, Magne N, Bentolila F, Drouillard J, Bruneton JN, Descamps B. Superior vena cava obstruction: is stenting necessary? *Support Care Cancer* 2001 9:103-107.
21. Rowell NP, Gleeson FV. Steroids, radiotherapy, chemotherapy and stents for superior vena cava obstruction in carcinoma of the bronchus: a systematic review. *Clin Oncol* 2002; 14: 338-351.
22. Bierdrager E, Lampmann LE, Lohle PN et al. Endovascular stenting in neoplastic superior vena cava syndrome prior to chemotherapy or radiotherapy. *Neth J Med* 2005; 63: 20-23.