Journal of Palliative Care & Medicine
ISSN: 2165-7386
Duvnjak, J Palliative Care Med 2012, 2:1
DOI: 10.4172/2165-7386.1000e106
Editorial Open Access

Malignant Superior Vena Cava Syndrome: Endovascular Stent Treatment- Current Status

Stevo Duvnjak

Odense University hospital-Radiological department, Sdr.Boulevard 29, DK-5000, Odense, Denmark

Introduction

Vena cava superior (VCS) is the largest vein in the body that collects returned blood from the head, upper extremities and upper chest to the heart. Superior vena cava syndrome (SVCS) occurs when obstruction of VCS restrain blood flow from the head, upper extremities and chest to the right atrium. The syndrome was first described by Wiliam Hunter in 1757 [1] and this case was due to syphilitic aortic aneurysm compression of VCS. Today malign diseases are responsible for syndrome in over 97% of cases, mostly due to extrinsic tumor compression following with compression by enlarged mediastinal lymph nodes [2]. However direct tumor vein invasion and intraluminal thrombosis can occurs. Lung cancer (small cell and non-small lung cell carcinoma) accounts for the majority of cases (75%) primary located on the right side which is four times frequent as those on the left because of vena cava superior anatomy [3]. Approximately 5 -10% of patients with lung malignancy will develop SVCS [4]. Other malignancy in thorax such a lymphoma accounting about 15% and metastasis (especially breast carcinoma about 7%) as well as other rarely mediastinal malignancy can be followed with SVCS also [5]. Benign conditions like, granulomatous mediastinal diseases, mediastinal fibrosis, struma, trauma, infections, aortitis, central venous lines, pacemaker which can cause SVCS which is beyond scope of this article. SVCS is usually diagnosed by clinical presentations with congestion and edema of the head and upper chest as well as vein distension across chest and neck. Dyspnoea is present in 50-80% of patients [6]. Pain, cough and hoarseness are reported relatively often also [6,7]. In severe cases of SVCS laryngeal and cerebral edema can occur following changing in mental status, syncopal attacks, lethargy, headache, stridor and coma [8]. Untreated tracheal obstruction/compression as well as brain herniation will result in death. The locations and time to onset of clinical manifest SVCS is in relation how severe symptoms are present. Collateral vessels that are usually described in SVCS are azygos and hemiazygos vein, intercostals, mediastinal, paravertebral, internal mammary, thoracico-epigastric, thoraco-acromioclavicular and anterior chest wall vein [9]. Usually it takes several weeks time to fully developing of collateral pathways. Vena azygos is very important vessel and symptoms are aggravated if the vena cava superior is compressed or occluded below origin of vein.

Diagnostic Methods

As mentioned earlier SVCV is diagnosed clinically verified by diagnostic methods. The majority of patients with SVCS has abnormal chest X ray with dominant sign of widening of mediastinum up to 60% of cases and pleural effusion up to 26% [10]. Computerized tomography (CT) of the chest with contrast is a preferable method today which allows precise detection of cause as well as extension of the blockade and collateral pathways. Magnetic resonance imaging can be used in patients with contraindication to contrast CT. Invasive venography is planned as a part of interventional procedure. Radionuclide venography is an option in detection of obstructive vein trombose but fails to detect cause and extent of obstruction.

Treatment of SVCS

The management of SVCS depends on the histological type of the tumor as well as tumor staging. It is mandatory to obtain histological diagnose before treatment. Principally there are two main strategies, one with primary focus for symptoms relief (endovascular stent) and other with focus to treat underlying cause of SVCV (radiotherapy and chemotherapy). The median estimated survival period for patients with SVCS is about 6 months, range from 1 to 9 months [11]. The treatment options include supportive steroid and diuretic therapy which is temporarily, radiotherapy (RT), and chemotherapy and stent placement. Due to terminal stadium of patients surgery is very rare a possibility.

Radiotherapy: Radiotherapy offer symptom relief in the 75% patients with small cell lung cancer and in 66% patients with non SCLC [12]. The majority of patients are symptom free about 2 weeks after course, but in some of the cases symptom relief is recorded after 72 hours [13]. There are to protocols, one with high initial fraction over 3 Gy and another with standard protocol. However there is no difference between the two protocols, quicker symptom relief can occur with high initial fractions protocol. The course long depends of tumor histology and nature of the treatment and varies from 1-2 up to 7-8 weeks [14]. There are some contraindications as well as complications of radiotherapy but that is beyond scope of article.

Chemotherapy: Chemotherapy is treatment of choice for SVCS caused by lymphoma, SCLS, germ cell tumor followed by symptom relief up to 80% patients with lymphoma as cause and up to 77% in patients with SCLC [5]. The response rate is similar as in the radiotherapy and ranges from one to two weeks. Radiotherapy can be used in these tumors groups, but chemotherapy is preferable due to better long term efficacy. Additionally radiotherapy plays a role in special group of the patients.

Endovascular treatment: The placement of stent in vena cava superior can restore venous return following with prompt symptom relief. The procedure is performed under local anesthesia with punction of the femoral vein and/or jugular internal vein usually with ultrasound guided. Other vascular access such as subclavian vein can be used as alternative routes in a case of obstruction of jugular or femoral vein. In this situation ultrasound guided vein punction is recommended [15]. Superior vena cavography is performed to obtain information about distal and proximal landing zone, which should be at least 10 mm on the both side. After the crossing the stenosis with guide wire self-expandable stent is placed using bone markers for precise placement

Corresponding author: Stevo Duvnjak , Odense University hospital-Radiological department, Sdr.Boulevard 29, DK-5000, Odense, Denmark, E-mail: stevo.duvnjak@ouh.regionsyddanmark.dk

Received December 19, 2011; **Accepted** December 19, 2011; **Published** December 21, 2011

Citation: Duvnjak S (2012) Malignant Superior Vena Cava Syndrome: Endovascular Stent Treatment- Current. J Palliative Care Med 2:e106. doi:10.4172/2165-7386.1000e106

usually, however newer angiographic equipment has "road map" which is useful also. Generally self-expandable stent is used due to better adaptability to vessel wall as well as stent length and flexibility. There are several self-expandable stent on the marked and there is no significant difference between them [16] excluding one report about Gianturco stent with some difference. There is no evidence that bilateral stenting of both brachiocephalic veins is better compared with a stenting of only one brachiocephalic vein in a case of the involvement of both veins but complications could be higher in bilateral vein stenting [17]. There are still a few unanswered questions regarding interventional technique such as medications before and after stent placement. Some authors recommend routine pre-dilatation of the vein before stent placement and other performed direct stenting following with post dilatations if the residual stenosis is above 50% of lumen which is our technique also and predilatation is performed only if the stent cannot cross the stenossis /obstruction [18,20,22]. Especially, complications can occur if tromb is presented in VCS when pre-dilatation is performed even a case of vein rupture after pre-dilatation is described [21]. There is no consensus about pre-dilatations balloon size also however lower or the same balloon diameter as vein diameter is recommended. The anticoagulant therapy (AT) is another controversy and there is no general consensus about it due to bleeding risk. Some recently results suggest that is no differences between patient who received AT and / or aspirin and the patients who do not receive scheduled AT therapy in term of stent thrombosis occurrence [18,22]. Our policy is that patients received 5000 IU heparin during interventions and on the individual basis continuous with anticoagulant therapy if the pre-interventional was discovered thrombus in VCS. It is recorded high technically success after stent insertion [21-23]. The majority of patients record completely or significant disappearing of the head and upper extremity edema, following with reductions of headache, dyspnea and collateral venous circulation [21,23,24]. There are no prospective randomizer studies which compare RT or chemotherapy and stent placement. The systemic review from Rowell and Gleeson [25] is the biggest collect data which showed symptom relief in 95% of patients treated with stent placement and relapse rate op to 11% which is possible to treat endovascular in the majority of the cases with balloon angioplasty, new stent insertion and/ or with fibrinolysis. This is better result compared with chemotherapy, RT, and combination of chemotherapy and RT in patients with SCLC with symptom relief in 77%, 78% and 83% respectively. In NSCLC patients chemotherapy is followed by symptom relief in 59% and RT in 63%. Relapse rate range from 19 up to 50% of cases.

Currently patient with SCLC, lymphoma and other sensitive tumor is treated primary with chemotherapy. In patients with non SCLC, stent insertion seems to be reasonable option but RT is still primary method in the majority of center. Endovascular stent is mainly reserved after failure of RT or chemotherapy. Randomized study compared RT and primary stent insertion in non SCLC is necessary to shows primary role and quality of life after stent deployment in this group of patients, however primary stent insertion allows additionally RT and/or chemotherapy. Complications after stent insertion are a relatively rare and including stent fracture and migration (10%), pulmonary emboli, local hematoma, but several fatal cases are reported after intervention with pericardial tamponade [26]. Jean Baptiste et al described covered stent insertion to treat vein rupture successfully in to patients [27]. The interventional radiologist should be aware about those complications and treatment possibility.

Conclusion

SVCS treatment should be individualized based on tumor histology and symptoms severity as well as patient's life expectancy. Endovascular stent placement in SVCS is a safe interventions followed with rapid symptom relief. High clinical success and relative low relapse rate as well as possibility to treat relapse endovascular again are arguments for more liberally using of stent in these patients.

References

1. Hunter W (1757) History of aneurysm of the aorta with some remarks on aneurysm in general. Medical Observation and Inquiries 1- 323-357.
2. Wudel LJ Jr, Nesbitt JC (2001) Superior vena cava syndrome. Curr Treat Options Oncol 2: 77-91.
3. Fincher RE (1987) Superior vena cava syndrome: experience in a teaching hospital. South Med J 80: 1243-1245.
4. Escalante CP (1993) Causes and management of superior vena cava syndrome. Oncology 7: 61-68.
5. Ostler PJ, Clarke DP, Watkinson AF, Gaze MN (1997) Superior vena cava obstruction: A modern management strategy. Clinical Oncology 9: 83-89.
6. Parish JM, Marschke RF Jr, Dines DE, Lee RE (1981) Etiologic consideration in superior vena cava syndrome. Mayo Clin Proc 56: 407-413.
7. Markman M (1999) Diagnosis and management of superior vena cava syndrome. Cleve Clin J Med 66: 59-61.
8. Lewis MA, Hendrickson AW, Moynihan TJ (2011) Oncologic emergencies: Pathophysiology, presentation, diagnosis and treatment. CA Cancer J Clin 19
9. Stanford W, Jolles H, Elli S, Chiu LC (1987) Superior vena cava obstruction: A venographic classification. Am J Roentgenol 148: 259-262.
10. Brown G, Husband JE (1993) Mediastinal widening: A valuable radiographic sign of superior vena cava thrombosis. Clin Radiol 47:415-420.
11. Baker GL, Barnes JH (1992) Superior vena cava syndrome: etiology, diagnosis and treatment. Am J Crit Care 1: 54-64.
12. Armstrong B, Perez C, Simpson J, Hederman MA (1987) Role of irradiation in the management of superior vena cava syndrome. Int J Radiat Oncol Biol Phys 13: 531-539.
13. Davenport D, Ferree C, Blake D, Raben M (1978) Radiation therapy in the treatment of superior vena caval obstruction. Cancer 42: 2600-2603.
14. Sculier JP, Ewans WK, Feld R, DeBoer G, Payne DG, et al (1986) Superior vena caval obstruction syndrome in small lung cell cancer. Cancer 57: 847-851.
15. Uberoi R (2006) Quality assurance guidelines for superior vena cava stenting in malignant disease. Cardiovasc Intervent Radiol 29: 319-322
16. Miller JH, Mcbride K, Little F, Price A (2000) Malignant vena cava superior obstruction: stent placement via the subclavian route. Cardiovasc Intervent Radiol 123: 155-158.
17. Lanciego C, Chacon JL, Julian A, Andrade J, Lopez L, et al. (2001) Stenting as first option for endovascular treatment of malignant superior vena cava syndrome. AJR Am J Roentgenol 177: 585-593.
18. Nicholson AA, Ettles DF, Arnold A, Greenstone M, Dyet J (1997)Treatment of malignant superior vena cava obstruction: Metal stents or radiation therapy. J Vasc Intervent Radiol 8: 781-788.
19. Dinkel HP, Mettke B, Schmid F, Baumgrtner I, Triller J, et al. (2003) Endovascular treatment of malignant superior vena cava syndrome: is bilateral Wallstent placement superior to unilateral placement? J Endovasc Ther 10: 799-797.
20. Entwisle KG, Watkinson AF, Hibbert J, Adam A (1995) The use of Wallstent endovascular prosthesis in the treatment of malignant inferior vena cava obstruction. Clini Radiol 50: 310-313.
21. Brown KT, Getrajdman GI (2005) Balloon dilatation of the superior vena cava (SVC) resulting in SVC rupture and pericardial tamponade: a case report and brief review. Cardiovasc Intervent Radiol 28: 372-376.
22. Duvnjak S, Andersen PE (2011) Endovascular treatment of superior vena cava syndrome. Int Angiol 30: 458-461.

23. Fagedet D,Thony F, Timsit JF, Rodiere M, Monnin-Bares V, et al. (2011) Endovascular treatment of malignant superior vena cava syndrome: results and predictive factors of clinical efficacy. Cardiovasc Intervent Radiol 12.

24. Ariza MA, Gamboa P, Gimeno MJ, Alfonso E, Mainar A, et al. (2003) Percutaneous treatment of superior vena cava syndrome using metallic stent. Eur Radiol 13: 853-862.

25. Rowell NP, Gleeson FV (2002) Steroids, radiotherapy, chemotherapy and stents for superior vena caval obstruction in carcinoma of the bronchus: a systematic review. Clin Oncol 14: 338-351.

26. Ploegmakers MJ, Rutten MJ (2009) Fatal pericardial tamponade after superior vena cava stenting. Cardiovasc Intervnt Radiol 32: 585-589.

27. Jean BR, Williams DM, Gemmete JJ (2011) Successful treatment of superior vena cava rupture with placement of the covered stent: a report of the two cases. Cardiovasc Intervent Radiol 34: 667-671.