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CIRSE GUIDELINE

CIRSE Quality Assurance Guidelines for Superior Vena Cava Stenting in Malignant Disease

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Introduction

Superior vena cava obstruction (SVCO) is a clinically important condition manifesting as progressive plethora and oedema of the upper limbs, head and neck due to venous hypertension [1]. SVCO was first described by Scottish anatomist William Hunter in 1757 following the post-mortem of a 39 year old with a syphilitic aortic aneurysm [2, 3]. The classical presenting features of progressive upper limb and torso oedema with plethora of the head and neck are present in ~80% of cases [1]. Other features of SVCO may include glottal oedema, dyspnoea, chest wall collateral venous distention, headache, and rarely cerebral venous hypertension. Clinical manoeuvres to augment upper limb venous return, such as arm raising, can be demonstrated to exacerbate the symptoms of SVCO. Collateral venous distension is demonstrated in up to 80% of cases of SVCO, with venous return to the IVC via oesophageal, hemi-azygous, lateral thoracic, and vertebral venous plexi [4]. However despite this rich collateral network, vascular calibre is often insufficient to decompress the raised venous pressure [1].

The underlying causes of SVCO have evolved over time from infectious causes such as TB and syphilis which accounted for the majority of SVCO cases in 1949 [6], to the most

common modern day cause; cancer. Malignancy is the cause for SVCO in more than 90% of cases, with bronchogenic carcinoma accounting for at least 50% [5]. Extra-luminal SVC compression is either directly mediated by malignant growth from a central tumour, usually right bronchial, or by associated mediastinal lymphadenopathy. SVCO is reported in up to 4% of all diagnosed bronchogenic cancers, with squamous cell carcinoma the histological type most frequently associated with SVCO [36]. Other malignant causes include lymphoma, metastatic disease, germ cell tumours, thymoma and mesothelioma [7].

Benign causes of SVCO are now also considered to be increasing due to the rising use of central venous catheters and indwelling cardiac devices [8, 9]. Up to 75% of patients with benign SVCO have an indwelling venous device [8, 10]. Mediastinal lymphadenopathy, fibrosis and substernal goitre are the other main causes of benign SVCO [8].

Whilst the spectrum of clinical presentation of SVCO may vary widely, the most common clinical course is one of subacute progressive upper limb venous insufficiency in benign diseases, or a more fulminant course, over days to weeks, with underlying malignancy [11]. The severity and duration of symptoms of SVCO are an important guide to the timing of intervention, with evidence that treating SVCO as a medical emergency may not translate to an improved outcome in all patients [12,13]. However, clinical features of CNS depression, stridor and glottis or bronchial oedema remain strong indications for emergency treatment.

Until 30 years ago the mainstay of treatment for SVCO was non-invasive therapy for the treatment of malignancy; primarily radiotherapy or chemo-therapy [14, 15]. In 1986 an endovascular approach for the treatment of SVCO was described by Charnsangavej et al[16]. SVC stenting has now become the treatment of choice for SVCO to provide rapid relief of severe venous congestion and its associated morbidity. Furthermore SVC stenting has demonstrated good longer term patency, and alleviation of symptoms in this cohort [17].

Definitions

SVC: Superior vena cava.

IVC: Inferior vena cava

SVCO: Superior vena cava obstruction

MDCT: Multi-detector computed tomography

Pre-treatment imaging

Venography, usually performed as a prelude to stenting, remains important in the confirmation and assessment of SVCO, accurately depicting venous anatomy and the extent of thrombus formation [18]. However, non-invasive imaging prior to intervention is now clinical routine. Contrast-enhanced multi-detector computed tomography (MDCT) is usually the modality of choice due to its widespread availability and ability to determine the location and severity of SVCO with a very high degree of sensitivity [19]. MDCT will also demonstrate the underlying disease burden, presence of thrombus and involvement of other structures relevant to the technical success of intervention [19,25]. The presence of venous collateral vessels on MDCT is highly suggestive of SVCO, with a sensitivity of 96% and a specificity of 92% [22]. MRI is increasingly being used to diagnose SVCO with a sensitivity and specificity approaching 100% [20, 21]. MRI and CT are both sensitive enough to diagnose early and impending SVCO even before the development of clinical symptoms [19].

Indications for treatment

The indications for SVC stenting are symptomatic malignant SVCO, either at initial presentation or following failed chemotherapy or radiotherapy, and symptomatic benign SVCO. There is insufficient evidence to support primary SVC stenting in asymptomatic individuals.

Contraindications

There are no absolute contraindications to SVC stenting. The relative contraindications are patients with underlying malignancies with a very good chance of early cure or remission, patients who cannot lie flat or semi-supine and patients with systemic sepsis or non-correctable coagulopathy.

Patient preparation

Thorough clinical assessment is mandatory before any procedure is undertaken. Infection at vascular access sites, as well as systemic infection should be excluded. Pre-procedural blood testing including platelets, coagulation screening, and renal function is mandatory.

Standard pre-procedural advice for all patients undergoing conscious sedation should be provided and adhered to. Informed consent should be obtained prior to any intervention or

sedation. Intravenous access for potential fluids and medications should be established in all patients prior to the procedure as well as supplemental oxygen via an appropriate face-mask if needed.

Equipment specifics

Vascular access:

A colour-Doppler ultrasound with appropriate ultrasound probes (3–9 MHz) should be available for ultrasound-guided puncture of the access vessels.

Standard materials include:

1) 4–5 Fr catheters, typically with Multipurpose, Cobra or Sidewinder configuration.

2) 0.035” standard and hydrophilic guidewires with varying degrees of stiffness must be available**.**

3) 5- to 12-Fr vascular access sheaths in standard as well as longer lengths if needed.

4) Standard balloon dilatation catheters with diameters ranging from 6–20 mm.

5) High-pressure balloon catheters with diameters of 12–18 mm if needed.

6) A variety of large diameter self-expanding bare metal stents (12–24 mm), as well as covered stents, in case of venous rupture, must be available.

Appropriate access needles, guidewires and catheters or drains must also be available to perform emergency pericardiocentesis in case of pericardial tamponade due to rupture of the central veins.

Procedural features and technical variations

Prior imaging is critical in planning treatment for SVCO. The extent of underlying disease, length of venous obstruction, relationship with adjacent mediastinal structures, normal venous diameter, presence of thrombus and involvement of the brachiocephalic veins are important factors to assess on MDCT or MRI. Pre-procedural imaging can also be useful in determining the vascular access site.

SVC stenting is usually performed using local anaesthetic with conscious sedation if needed. Standard physiological monitoring (pulse, blood pressure, oxygen saturation and electrocardiogram) is carried out during the procedure. Vascular access to the superior vena

cava can be via the femoral, upper limb, internal jugular or subclavian veins. The subclavian route carries a slightly higher risk of pneumothorax and haemothorax [47], and is therefore not preferred as a primary access option by some operators. The route of access may be determined by factors such as relevant anatomy, operator and patient preference with many authors describing high success rates from a variety of approaches although the majority appear to be utilising femoral, internal jugular or upper limb veins with high success rates and minimal access complications [24, 25, 49, 57, 58].

A superior vena cavogram is carried out prior to stenting to confirm the extent of the disease, collateral formation, and coexisting thrombus. A bolus of heparin is regularly, but not universally, administered during the procedure. If extensive co-existing thrombosis is present in the SVC, local thrombolysis or mechanical thrombectomy may be considered [10].

Using pre-shaped catheters, a guidewire is placed across the obstruction under fluoroscopic guidance. If the lesion cannot be traversed from one approach (e.g. from femoral vein access), the opposite direction should be attempted (e.g. upper limb or internal jugular venous access). Dual access such as from femoral and internal jugular or upper limb veins with a snare to establish a ‘through-and-through’ or ‘body-floss’ wire is a technique that can be utilised to achieve increased wire stability in difficult cases although some operators do this routinely and have reported high technical success rates with no increase in access site complications [24].

Balloon pre-dilatation is required if the occlusive lesion precludes passage of the stent delivery system. There is no consensus on whether balloon pre-dilatation should be performed routinely or to what size of balloon to dilate up to. Some authors have suggested that routine serial slow pre-dilatation may help prevent SVC rupture although the evidence base for this claim is weak and this practice is not universal [24, 48, 49, 50, 51]. In cases of occlusion extending to the brachiocephalic or subclavian veins, some authors have advocated bilateral “kissing” or Y-shape stenting, however the majority of appear to agree that stent extension to one brachiocephalic vein is adequate for symptomatic relief and may be safer as well as being technically simpler [25, 51, 55, 56].

Documented venous rupture with catastrophic cardiovascular collapse, although rare, has been reported in the literature and the interventionalist should be prepared to insert a covered stent graft or perform pericardiocentesis if needed and there should be appropriate covered stent grafts available as well as arrangements for emergent cardio-thoracic surgical transfer if needed [18, 23, 24, 48].

Self-expanding bare metal stents are the most common type of stent usually deployed [10, 17, 25, 26, 51, 52]. Stents should be sized appropriate to the dimensions of the individual

patient with many operators over-sizing stents by up to 2mm reference vessel diameter, in a non-involved segment on MDCT or calibrated venography, to help reduce delayed stent migration [50, 51]. It is notable that, in the case series of Fagedet et al, patients treated with stents >16mm diameter had a significantly higher rate of caval rupture, pericardial tamponade or pulmonary oedema compared to patients with stents <16mm diameter [25]. However, there have also been recent series described with stent diameters up to 24mm and no significant increase in the rate of complications [51].

It has been suggested that optimum stent length should cover the lesion with approximately 10mm of free extension at the proximal and distal margins [18, 51]. However, authors reviewing cases of stent migration have advised that more of the stent should be positioned above the lesion than below, with approximately 60% of stent length above the lesion, extending to the brachiocephalic vein if needed, to reduce the risk of distal migration [50]. Of course, overlapping stents may be required to cover longer lesions.

There is no literature consensus on the role of post-stent dilatation although many operators do post-dilate with a balloon if there is a residual stenosis and this appears to be required in 70 – 78% of patients if no pre-dilatation has been routinely performed [50, 51, 52]. A completion venogram is usually performed to exclude venous rupture and confirm satisfactory position of the stent with free drainage and reduction of venous collaterals. Technical success in stent placement is usually indicated by complete coverage of the occlusive lesion with <30% residual stenosis [40, 51].

Medications and Peri-procedural care

Local anaesthetic and conscious sedation guidelines should be followed according to institutional practice. Facility for pericardiocentesis should be on hand in the case of SVC or central vein rupture with cardiac tamponade [23, 24]. Furthermore standard periprocedural physiological monitoring should be performed in all patients for at least 2 hours following the procedure. Patients should be advised to remain in bed for at least 2 hours after the procedure. Analgesia should be provided as needed on a symptomatic basis.

Post procedural follow up care

The need for long-term anticoagulation after stent placement remains controversial, with no consensus among studies as to the type, duration or clinical efficacy of anticoagulation therapy [18, 25-27, 53, 54]. In a recent study of 172 patients treated for malignant SVCO,

long-term anticoagulant therapy, either with Aspirin, Heparin or Warfarin, did not appear to influence the risk of re-thrombosis and this was lower than the risk of bleeding [25, 53].

There is no literature consensus on the benefit of routine serial radiographic or CT surveillance of SVC stents although repeat imaging is indicated if symptoms recur. The utility of MDCT imaging in this context and also for imaging post-procedural complications is well established [28]. If symptoms of recurrent SVCO are manifest, repeat venography and intervention which may include thromboaspiration, thrombolysis and restenting are advocated.

Effectiveness (clinical and technical success)

Technical success rates for SVC stenting are high, ranging from 95-100% [17, 25, 29]. A systematic review of the literature published in 2009, showed that stents were 87-100% effective in relieving SVCO at initial presentation [30]. Reported re-obstruction rates following successful relief of obstructive symptoms range from 0-40%, however patency is restored in most patients with re-intervention. The only independent risk factor for endovascular therapeutic failure in a recent cohort analysis was thrombosis of the SVC [25]. Complication risk is statistically greater in stents >16mm, with other factors such as the use of bare metal stents, cases of occlusion, and initial associated thrombosis strongly associated with re-obstruction [25]. Recent evidence on the use of covered stents versus bare metal stents has suggested superior patency rates with covered stents after 12 months in malignant SVCO [31]. However, covered stents should be used with caution due to concerns of stent migration and covering important venous pathways or collaterals, particularly if placing a covered stent across the brachiocephalic confluence, although there is some evidence to suggest that custom-designed covered stents may not be prone to migration and coverage of a patent contralateral brachiocephalic vein is unlikely to be clinically evident [46]. At present, there is insufficient evidence to recommend a specific type of stent for SVC stenting.

The only comparative study evaluating open surgical repair versus endovascular stent placement for benign SVCO was performed in 2008 by Rizvi et al [40]. This was a retrospective study involving 70 consecutive patients. The results showed no early mortality in either group but peri-procedural morbidity was much higher (19%) in the open surgical repair group versus the endovascular group (4%). Additionally, the primary, assisted primary and secondary patency rates for the open surgical repair group were inferior to the endovascular group, being 45%, 68% and 75% versus 44%, 96% and 96%, respectively at 3 years follow-up [40]. 93% of patients from both groups had significant relief from symptoms.

The authors therefore concluded that SVC stenting is an appropriate first-line therapy for benign SVCO with surgery reserved for patients unsuitable for stenting or in whom stent insertion had failed [40].

Currently no randomised controlled study comparing SVC stenting with either radiotherapy or chemotherapy has been performed. There are 2 studies that have directly compared endovascular stenting with radiotherapy and/or chemotherapy [41, 42], with both suggesting SVC stenting offers more rapid and sustained relief of symptoms. Furthermore, a Cochrane database review showed that SVC stenting relieved SVCO in 95% of patients with bronchogenic carcinoma, compared to 60% (NSCLC) and 77% (SCLC) with chemotherapy and/or radiotherapy [36, 37]. However direct literature comparisons are challenging as a lack of consistency in defining underlying cause, reporting of clinical success, complication rates, repeat intervention rates, and follow up make definitive conclusions regarding efficacy difficult.

Complications and their management

Overall major complication rates in SVC stenting are approximately 4% [18, 30]. These include stent migration, bleeding, infection, thrombotic events, SVC rupture, pericardial tamponade, cardiac failure and arrhythmias [17, 24, 28, 30, 43, 44]. The minor complications rate was 3.2% and is attributable to puncture site haematoma, chest pain, epistaxis, infection and re-stenosis [17]. Overall, the complication rate for malignant SVC stenting compares favourably with chemotherapy and radiotherapy [36].

Recanalisation of the SVC may result in dramatic shifts in right heart filling pressures and venous return, resulting in an acute overload syndrome with pulmonary oedema. Treatment with diuresis, positive pressure ventilatory support and appropriate monitoring in the intensive care unit may rarely be required. Pre-procedural echocardiography may be of value in selected individuals with impaired cardiac reserve or known valvular disease.

Stent migration into the right atrium can be lead to cardiac arrhythmias causing significant morbidity, and occasionally mortality. Predisposing factors include poor patient selection, inadequate over sizing of the stents, inadequate positioning or deployment of the stents, cardiac motion and inadequate vessel measurement [50].

A recent review examining the safety of vascular endoprosthesis for malignant SVCO showed that in 32 studies, there was a 2% mortality rate. The commonest cause of death was severe haemorrhage (41%), followed by cardiac events (23%), respiratory failure (17%)

and pulmonary embolus (6%) [30]. Similar results have been reported in other cohort studies and case series [29].

It is a sobering reminder that despite effective endovascular therapy with high rates of technical and clinical success in alleviating symptoms of SVCO overall, median survival duration in patients with malignant SVCO is still only ~8-20 weeks [29, 30, 43, 45].

Conclusions

SVC stenting is the therapy of choice for rapid, safe and effective alleviation of significant symptoms due to SVCO. The success and complication rates compare favourably with traditional therapies such as targeted radiotherapy, chemotherapy and surgery.

Further research needs to focus on identifying the optimal stent design, procedural technique, role of anticoagulation, surveillance strategy and best medical therapies to achieve the best long-term results particularly as oncological advances and an increase in benign iatrogenic SVCO are likely to lead to longer life-expectancies in this cohort.

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