📕 Review Article 🐔

Superior Vena Cava Syndrome and Wallstent: A Systematic Review

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Purpose: To elucidate the indication, presentation, demographics, Stanford classification, technical efficacy, morbidity, mortality and long term patency of Wallstent for superior vena cava (SVC) syndrome.

Materials and Methods: A systematic review of literature in Pubmed and Embase, CINAHL and Cochrane Library in accordance to PRIMSA was conducted. Retrieval and extraction was performed by two independent reviewers with inter-rater reliability test. The hierarchy of the evidence was assessed through the National Institute for Health and Care Excellence Checklist. Data was subjected to pooled prevalence analysis, Cox regression, Kaplan–Meir survival and test of probability using log rank analytics. This review is registered with International prospective register of systematic review: CRD42021271009.

Results: A total of n=701 individuals with n=930 stents with median age of 60 (interquartile range (IQR): 26–89) years and male predominance 3.5:1 were identified in n=30 articles. The most common venographic classification was Stanford type II (n=344, 50%) and complete symptomatic resolution was achieved in 48 h. The 30-day morbidity was (n=62, 8%) and mortality was (n=21, 3%). Female gender was associated with higher 30-day morbidity (p<0.03). The cumulative median patency of Wallstent for non-malignant aetiology was [550 days (IQR: 14–1080)

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(C) BY-NC-SA ©2022 The Editorial Committee of Annals of Vascular Diseases. This article is distributed under the terms of the Creative Commons Attribution License, which permits use, distribution, and reproduction in any medium, provided the credit of the original work, a link to the license, and indication of any change are properly given, and the original work is not used for commercial purposes. Remixed or transformed contributions must be distributed under the same license as the original. vs. 120 days (IQR: 0–925)] for malignancy (p<0.03). **Conclusion:** The use of Wallstent for resolution of malignancy induced SVC syndrome as a first line therapy is feasible and associated with low mortality. Their use for non-malignant aetiology demands a more in depth review and advocates further investigation.

Keywords: superior vena cava (SVC), superior vena cava syndrome, malignancy, endovascular therapy (ET), Wallstent, systematic review

Introduction

Superior vena cava (SVC) syndrome refers to a groups of symptoms such as oedema (facial and arms), shortness of breath, conjunctival suffusion, coughing and stridor as a consequence of partial or complete SVC obstruction. In some series, sever neurological symptoms (stupor and coma) or airway compromise has also been reported.¹⁾ Their reported incidence ranges from 1 in 650–3100 cases and in USA alone 15,000 cases are reported annually. The first report of SVC syndrome dates back to 1757 when William Hunter described an extrinsic compression of the SVC by a large aneurysm secondary to syphills.²⁾ In order of prevalence, mediastinal malignancy (bronchogenic, lymphoma, metastatic) remain the most common aetiology (70%) followed by infectious and intragenic (Indwelling access and pacemakers) injuries.³⁾ The treatment aims at reduction of the venous pressure either by medical management or surgery (open or endovascular). Open repair using prosthetic (polytetrafluoroethylene [PTFE] or Dacron) or autogenous vein (spiral saphenous vein or femoral vein) is now reserved if endovascular approach fails to prevail as later is associated with lower morbidity and mortality.^{4,5)} Since early 1990s, Wallstent endo-prosthesis (self-expanding stent) has been routinely deployed for tackling the SVC syndrome amongst other stents such as Z stent. However, to date no systematic review has evaluated the independent outcome of Wallstents on their long-term technical efficacy, associated mortality and morbidity in the literature. In addition, there is no clear consensus or guidelines for their use that was originally designed for other purpose. We routinely use Wallstent in our unit and we could not suggest any long term outcomes to our patients due to lack of robust evidence. Therefore, the aim of this systematic review to is to establish the indication, classification, technical efficacy, morbidity, mortality and longevity of Wallstent for the treatment of SVC syndrome.

Materials and Methods

Search strategy

A systematic review of literature from the database inception to 1st of August 2021 in Pubmed, Embase, CINAHL and Cochrane Library in accordance to the Preferred Reporting Items for Systematic reviews and Meta-Analysis (PRIMSA) was conducted.⁶⁾ Medical Subject Headings (MeSH) terms or keywords included: "venae cavae" [MeSH Terms] OR vena cava [Text Word], Wallstent [MeSH Terms] OR Wallstent [text Word]. References of the retrieved articles were also manually evaluated for any additional literature not identified in the initial search. All abstracts were retrieved and reviewed by two separate investigators. Studies that appeared to fulfil the eligibility criteria but had an insufficient information in the abstracts were also retrieved and examined in full. The data extraction was also performed by two separate investigators and inter-rater reliability [Cohen Kappa Coefficient was (k)] was calculated. This systematic review was also registered with International Prospective Register of Systematic Review (PROSPERO) National Institute for Health Research, UK with registration (NIHR) number: CRD42021271009.

Selection criteria

All studies involving humans, pertained to the use of Wallstent for SVC syndrome for any given aetiology in English language were selected for inclusion. Published material that were experimental studies, narrative reviews and expert opinion were excluded.

Statistical analysis

To achieve an informed conclusion and evidence-based approach, the included articles were evaluated for their validity, bias, applicability and inference using critical appraisal tool provided by Oxford Critical Appraisal Skills programme (CASP). Due to a lack of consistency of data and its randomisation, a meta-analysis was not feasible. However, a pooled analysis was conducted by calculation of the median value along with their interguartile range (IQR). The data output was calculated and presented with percentile of each category. Sub-group analysis was performed using Cox regression to evaluate the impact of various attributes (age, gender, Stanford classification [Type I–IV]) over a median time on the endpoint of 30-day mortality and morbidity (binary) outcomes. In addition, a Kaplan-Meir survival analysis was used to see the difference between the survival (long-term patency) of Wallstent in malignant versus non-malignant aetiology with log rank test of probability (p-value).

Venographic classification

SVC syndrome is classified into four different subgroups according to Stanford. This venographic classification is primarily based on degree of obstruction, valve competency and collateral venous flow (intercostal, left accessory hemi-azygous, azygous, hemi-azygous, para-vertebral and internal mammary veins). Type I, is a partial stenosis up to 90% of the SVC with patent Azygous vein. Type II is near total occlusion (90%–100%) of SVC with flow from azygous vein to the right atrium. Type III is complete occlusion of SVC with reverse flow in azygous vein and finally, Type IV is complete obstruction of SVC with one or more than one collateral vein occlusion⁷ (**Table 1**).

Symptomatic classification

Symptomatology of SVC syndrome has been classified into four grades by Kishi et al. in 1993. According to this (a simplistic version) a grade I relates to any signs of venous congestion, grade II refers to nasal or fascial oedema,

 Table 1
 Venographic and symptom classification of SVC syndrome

Туре	Venographic features	
Stanford I	Partial stenosis up to 90% of the SVC with patent azygous vein	
Stanford II	Near total occlusion (90%–100%) of SVC with flow from azygous vein to the right atrium	
Stanford III	Complete occlusion of SVC with reverse flow in azygous vein	
Stanford IV	Complete obstruction of SVC with one or more than one collateral vein occlusion	
Grade	Kishi symptomatic classification	
Grade I	Any signs of venous congestion	
Grade II	Nasal or fascial oedema	
Grade III	Laryngeal oedema	
Grade IV	Central nerves related symptoms	

SVC: superior vena cava



Fig. 1 Preferred reporting items for systematic reviews and meta-analysis flow-chart.

grade III refers to laryngeal oedema and Grade IV is central nerves related symptoms. In this categorisation grade IV represents the most severe presentation that requires urgent assessment and management as delay in treatment could result in fatality⁸⁾ (Table 1).

Definitions

Technical success within this review was defined as post endovascular venography evidence of patency with resolution of symptoms. Primary patency was defined as patency of the Wallstent endo-prosthesis requiring no further assistance for its luminal patency and symptomatic relief. Secondary patency was defined as any intervention (venoplasty, stent extension, thrombolysis, open surgery) to keep the original Wallstent prosthesis open following its primacy patency. Mortality was defined as death from the Wallstent endo-prosthesis placement within 30 days and complications as any event that arose from the procedure requiring further intervention.

Results

Total of n=99 articles were identified with no systematic or Cochrane review in the literature. All articles were found to be of case reports or cohort (grade and level of evidence: class III/ IIb, level C/D). The overall missing data was 2.5% (indication, gender, classification and follow up).⁹⁻¹¹) After application of the inclusion criteria, total of n = 30 articles was eligible. The PRISMA flow chart is highlighted in Fig. 1. Inter-rater reliability was 0.88 for study retrieval and 0.86 for data extraction.

A total of n = 701 individuals with n = 930 stents were identified of the n=31 articles. The aetiology for SVC syndrome was n = 643 (92%) for malignancy and rest for non-malignant condition (pacemaker, indwelling central lines, fibrosis) n = 36 (6%). There was a male predominance 3.5:1 [male n = 543 (78%) vs. female n = 152 (22%)]. The median age of the group was 60 years (IOR: 26-89 years). The most common venographic classification in the order of prevalence was Stanford type II (n =344, 50%), Stanford type III (n=219, 32%), Stanford I (n = 58, 9%) followed by Stanford IV (n = 57, 8%) which equates to life-threatening symptoms according to Kishi classification (grade III and IV). The most common stent diameter was 12mm (IQR: 10-16mm) and the median length of the lesion was 6 cm (IQR: 3-14 cm). The median length of follow up was 54 days (IQR: 1-1849 days) with mean of 331 days. The median time to complete

symptom resolution was 2 days (IQR: 0-5 days). There was an average of 1.5 stents per case in the entire series. The 30-day complication incidence was (n=62, 8%). This ranged from stent migration, malposition, failure to deploy, collapse (radial force) and immediate thrombosis. The 30-day mortality from the procedure was (n=21, 3%) from pericardial effusion, heart failure and rupture (**Table 2**).

Malignant versus non malignant

Overall mortality (n = 21, 3%) and complication was (n = 62, 8%) for both groups.

Amongst n = 36 treated non-malignant cases, mortality was 2.7% (n = 1/36) and complication incidence was 25% (n = 9/36). Amongst n = 643 treated malignant cases, mortality was 3% (n = 20/642) and complication incidence was 8.2% (n = 53/643).

The cumulative median patency of Wallstent for nonmalignant aetiology was 550 days (IQR: 14-1080 days)

 Table 2
 The overall information of all the cases that were subjected to Wallstent for SVC syndrome

Category	Outcome
Total cases	n=701
Total stents	n=930
Malignancy	n=642 (92%)
Benign	n=36 (5%)
Male	n=548 (78%)
Female	n=152 (22%)
Age (median)	60 years (IQR: 26–89)
Stanford Type I	n=58 (9%)
Stanford Type II	n=344 (50%)
Stanford Type III	n=218 (32%)
Stanford Type IV	n=57 (8%)
Median lesion length	6 cm
Stents per case (average)	1.5 stents
Common stent diameter	12mm (IQR: 10–16mm)
Time to resolution	2 days (IQR: 0–5 days)
Follow up (median)	54 days (IQR: 1–1849 days)
30-day mortality	n=21 (3%)
30-day complications	n=62 (8%)
Immediate thrombosis	n=22 (35%)
Premature thrombosis	n=20 (32%)
Stent malposition	n=10 (16%)
Failure to deploy	n=5 (8%)
Migration	n=5 (8%)
Malignant cases	
30-day complication	n=53/643 (8.2%)
30-day mortality	n=20/643 (3%)
Non-malignant cases	
30-day complication	n=9/36 (25%)
30-day mortality	n=1/36 (2.7%)

SVC: superior vena cava; IQR: interquartile range

versus malignant ones which was 120 days (IQR: 0-925 days).

Sub-group analysis

Data was further analysed for identification of attributes that might influence the endpoint of 30-day mortality and morbidity. The test of statistics on the endpoint of 30-day morbidity amongst all attributes was significant only on female gender (<0.03) (**Table 3**). This evaluation on the endpoint of mortality (30-day) demonstrated that no attribute is statistically significant. The survival analysis (Kaplan–Meier) demonstrated longer patency of the Wallstent in non-malignant cases in comparison to malignant ones (<0.03) (**Fig. 2**). The cumulative median patency of Wallstent for non-malignant aetiology was 550 days (IQR: 14–1080 days) versus malignant ones which was 120 days (IQR: 0–925 days).

Discussion

There is currently no consensus on the management of SVC syndrome to conform an evidence-based practice. The traditional modality of treatment in malignant cases, has been chemotherapy, radiotherapy, open surgery and bypass. Open approach using spiral vein graft, allografts or prosthetic graft have been promising, however their longevity due to further compression or low flow state remains poor.^{12,13)} In addition, most patients due to poor physiological reserve or function could not tolerate an open sternotomy and such could result in early mortality. SVC syndrome is also associated with multiple benign aetiologies as a consequences of intrinsic and extrinsic sequel. Despite their treatment, majority of benign cases still proceed to SVC syndrome and required resolution.^{1,8,14–20)}

In contemporary practice, endovascular therapy (ET) has gained significant attention as a first line option for the treatment of SVC syndrome as long as it does not preclude or impact the outcome of future open surgery.²¹⁾ This practice is not supported by robust evidence (systematic review or randomisation) and the use of Wallstent amongst other endo-prosthesis is of no exception.²²⁾ The outcome of this review demonstrates that wallstent within 48h as a first line option, could result in rapid and complete resolution of symptoms with relative low mortality (3%).²³⁻³¹⁾ This is an important outcome when majority of treated cases were of malignant nature (92%) categorised to Stanford type II (n = 344, 50%) and type III (n = 219, 32%) and secondly associated with life-threatening symptoms according to Kishi classification (grade III and IV).8,32-40) This review also showed that the type of venographic or symptomatic classification has no clinical implication on endpoint of mortality making it more desirable as a first choice of therapy.

Variables	Significance (p value)
Female gender	p=0.03
Male gender	p>0.5
Median age	p>0.5
Stanford I	p>0.5
Stanford II	p>0.5
Stanford III / Kishi III	p>0.5
Stanford IV/ Kishi IV	p>0.5

 Table 3
 Binary evaluation of attributes (variables) on the endpoint of 30-day mortality



Fig. 2 Cumulative patency of the Wallstent in non-malignant vs. malignant case was [550 days (IQR: 14–1080) vs. 120 days (IQR: 0–925)] Log Rank (<0.03). IQR: interguartile range

In this review, a total of sixty-two cases (8%) had complications within the 30-days of procedure with no reports of stent fracture. This ranged from immediate stenosis (n=22, 35%), premature thrombosis (n=20, 32%), stent malposition (n=10, 16%), failure to deploy (n=5, 16%)8%) and migration (n=5, 8%). Immediate thrombosis was overcome by successful percutaneous thrombolysis in all cases and stent re-stenosis with in-stent successful venoplasty. Migration retrieval was achieved in three cases (n=3), with one resulting in mortality and other with oversize stent placement. It is worth mentioning that Wallstent has weaker edges than its main body and its deployment within disease segment or under extrinsic compression makes it more susceptible to early collapse and premature thrombosis. In addition, this is a braided stent (matrix design) and its deployment lacks detailed accuracy making it technically challenging with short landing zones. Therefore, complications such as stent collapse, premature stenosis and migration are inevitable but should remain minimal specially in the female cohort where this is notable (Table 3). The reason behind this attribute is unclear and could be a type II error.

Another technical aspect which is operator dependent and subjected to open debate is, unilateral or bilateral (kissing) stent and so called "Y" stent placement. This modes operandi, is attributed to SVC diameter of more than 15 mm and concomitant bilateral brachiocephalic vein involvement.²⁵⁾ Amongst all retrieved articles, a complete comparative analytics was only available in one study that demonstrated lower complications in unilateral stent placement (p < 0.03) with better longevity.²⁵⁾ However, such practice continues to be case dependent and results are variable in practice.^{41–43)}

The main objective of Wallstent stent placement in malignant SVC syndrome, is longevity (patency) prior to patient secondment to death due to their malignancy. The median primary patency in malignant cohort was 120 days (IQR: 0-925) (4-months) (mean of 7.1 months) which is arguably an acceptable patency for palliative group of patients. In contrast, the primary patency in non-malignant group was clinically and statically longer [550 days (IQR: 14-1080) (18-months)]. This raises the clinical question as to whether open surgical repair instead of Wallstent in later cohort could possess a better longevity.44) This debate demands randomisation or comparative analysis which is not within the merit of this review however, it advocates further investigation.44) The details of secondary patency as a subsequence of stent thrombolysis and re-plasty was not detailed for an objective inference thus no conclusion could be drawn.

The role of anticoagulation or antiplatelet was not meticulously reported within the retrieved articles prior or following the Wallstent placement specifically in malignant cohort. There is currently no consensus on the dose or indication of the aforementioned therapies in malignant SVC syndrome.45,46) The lack of consensus is perhaps originated from the argument that angiogenesis within tumour could potentially result in procedural bleed and further re-intervention (thrombolysis or venplasty) could possibly be contraindicated.47) Finally, studies to date have failed to confer any benefit for the prophylactic or treatment dose of anticoagulation or antiplatelet in practice.48) In two studies, the use of antiplatelet did not demonstrate any benefit in terms of primary or secondary patency and finally in the study of Razton et al. the use of anticoagulation was associated with lower incidences of stent occlusion (hazard ratio 0.47, 95% confidence interval 0.2-1.13) with no statistical significance.^{18,19,48,49)}

Limitations

The standard of reporting within the retrieved articles lacked conformity. Amongst them, the details of secondary patency, symptom presentation, anticoagulation and antiplatelet were not available. In addition, the terminology of technical success and primary patency was commonly interchanged. In some series, it was not clear as the higher number of stents were due to technical failure, longer lesions or whether this was unilateral or bilateral stenting technique. Overall a meta-analysis would have been more optimal for the external validity, however, lack of comparative dataset created this limitation.

Conclusion

It appears that the use of Wallstent as a first line approach with median patency of 120 days, mortality of 3% and complications of 8% amongst other stents in the treatment of malignancy induced SVC syndrome might be justified. This might be an acceptable approach where an open intervention due to palliative nature of the malignancy is not feasible. Their use in benign cohort, demonstrates a longer patency (550 days) but higher associated complications (25%). Therefore, the question arises as to whether open procedure could be an alternative and a comparative analysis might be advocated (stented nonmalignant versus open surgery). The standard of reporting for endovascular therapy in SVC syndrome demands robust and universal definitions to achieve an objective clinical inference specially for benign cases.

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Declaration of Conflict of Interests

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References

- 1) Rice TW, Rodriguez RM, Light RW. The superior vena cava syndrome: clinical characteristics and evolving etiology. Medicine (Baltimore) 2006; 85: 37-42. doi: 10.1097/01. md.0000198474.99876.f0.
- 2) Hunter W. The history of an aneurysm of the aorta with some remarks on aneurysms in general. Med Obs Soc Phys Lond 1757; 1: 323.
- De Potter B, Huyskens J, Hiddinga B, et al. Imaging of urgencies and emergencies in the lung cancer patient. Insights Imaging 2018; 9: 463-76. doi: 10.1007/s13244-018-0605-6.
- Kalra M, Sen I, Gloviczki P. Endovenous and operative treatment of superior vena cava syndrome. Surg Clin North Am 2018; 98: 321-35. doi: 10.1016/j.suc.2017.11.013.
- 5) Nossair F, Schoettler P, Starr J, et al. Pediatric superior vena cava syndrome: an evidence-based systematic review of the literature. Pediatr Blood Cancer 2018; 65: e27225. doi:

10.1002/pbc.27225.

- 6) Liberati A, Altman DG, Tetzlaff J, et al. The PRISMA statement for reporting systematic reviews and meta-analyses of studies that evaluate healthcare interventions: explanation and elaboration. BMJ 2009; **339** jul21 1: b2700. doi: 10.1136/bmj.b2700.
- Stanford W, Jolles H, Ell S, et al. Superior vena cava obstruction: a venographic classification. AJR Am J Roentgenol 1987; 148: 259-62. doi: 10.2214/ajr.148.2.259.
- Kishi K, Sonomura T, Mitsuzane K, et al. Self-expandable metallic stent therapy for superior vena cava syndrome: clinical observations. Radiology 1993; 189: 531-5. doi: 10.1148/radiology.189.2.8210386.
- Rusher AH, Tidwell K, Scroggin C, et al. Treatment of catheter-induced obstruction of the superior vena cava with Wallstent endoprosthesis. J Ark Med Soc 1997; 94: 299-300.
- 10) Irace L, Martinelli O, Gattuso R, et al. The role of selfexpanding vascular stent in superior vena cava syndrome for advanced tumours. Ann R Coll Surg Engl 2021; 103: 296-301. doi: 10.1308/rcsann.2020.7127.
- 11) Lau KY, Tan LT, Wong WW, et al. Brachiocephalic-superior vena cava metallic stenting in malignant superior vena cava obstruction. Ann Acad Med Singapore 2003; 32: 461-5.
- 12) Perez CA, Presant CA, Van Amburg AL 3rd. Management of superior vena cava syndrome. Semin Oncol 1978; 5: 123-34.
- Wilson LD, Detterbeck FC, Yahalom J. Clinical practice. Superior vena cava syndrome with malignant causes. N Engl J Med 2007; 356: 1862-9. doi: 10.1056/NEJMcp067190.
- 14) Barshes NR, Annambhotla S, El Sayed HF, et al. Percutaneous stenting of superior vena cava syndrome: treatment outcome in patients with benign and malignant etiology. Vascular 2007; 15: 314-21. doi: 10.2310/6670.2007.00067.
- 15) Hennequin LM, Fade O, Fays JG, et al. Superior vena cava stent placement: results with the Wallstent endoprosthesis. Radiology 1995; 196: 353-61. doi: 10.1148/radiology. 196.2.7617844.
- 16) Qanadli SD, El Hajjam M, Mignon F, et al. Subacute and chronic benign superior vena cava obstructions: endovascular treatment with self-expanding metallic stents. AJR Am J Roentgenol 1999; 173: 159-64. doi: 10.2214/ajr.173.1.10397119.
- 17) Alimi YS, Gloviczki P, Vrtiska TJ, et al. Reconstruction of the superior vena cava: benefits of postoperative surveillance and secondary endovascular interventions. J Vasc Surg 1998; 27: 287-301. doi: 10.1016/s0741-5214(98)70359-3.
- 18) de Gregorio Ariza MA, Gamboa P, Gimeno MJ, et al. Percutaneous treatment of superior vena cava syndrome using metallic stents. Eur Radiol 2003; 13: 853-62. doi: 10.1007/s00330-002-1489-9.
- 19) Lanciego C, Rodriguez M, Rodriguez A, et al. Permanent pacemaker-induced superior vena cava syndrome: successful treatment by endovascular stent. Cardiovasc Intervent Radiol 2003; 26: 576-9. doi: 10.1007/s00270-003-0070-5.
- 20) Bagul NB, Moth P, Menon NJ, et al. Migration of superior vena cava stent. J Cardiothorac Surg 2008; 3: 12. doi: 10.1186/1749-8090-3-12.
- 21) Rizvi AZ, Kalra M, Bjarnason H, et al. Benign superior vena cava syndrome: stenting is now the first line of treatment. J Vasc Surg 2008; 47: 372-80. doi: 10.1016/j.jvs.2007.09.071.
- 22) Lanciego C, Pangua C, Chacon JI, et al. Endovascular stent-

ing as the first step in the overall management of malignant superior vena cava syndrome. AJR Am J Roentgenol 2009; **193**: 549-58. doi: 10.2214/AJR.08.1904.

- 23) Sasano S, Onuki T, Mae M, et al. Wallstent endovascular prosthesis for the treatment of superior vena cava syndrome. Jpn J Thorac Cardiovasc Surg 2001; 49: 165-70. doi: 10.1007/BF02913595.
- 24) Leung ST, Sung TH, Wan AY, et al. Endovascular stenting in the management of malignant superior vena cava obstruction: comparing safety, effectiveness, and outcomes between primary stenting and salvage stenting. Hong Kong Med J 2015; 21: 426-34. doi: 10.12809/hkmj144363.
- 25) Dinkel HP, Mettke B, Schmid F, et al. Endovascular treatment of malignant superior vena cava syndrome: is bilateral Wallstent placement superior to unilateral placement? J Endovasc Ther 2003; 10: 788-97. doi: 10.1177/ 152660280301000416.
- 26) Stock KW, Jacob AL, Proske M, et al. Treatment of malignant obstruction of the superior vena cava with the self-expanding Wallstent. Thorax 1995; 50: 1151-6. doi: 10.1136/thx.50.11.1151.
- 27) Gross CM, Kramer J, Waigand J, et al. Stent implantation in patients with superior vena cava syndrome. AJR Am J Roentgenol 1997; 169: 429-32. doi: 10.2214/ajr.169.2. 9242747.
- 28) Martin M, Baumgartner I, Kolb M, et al. Fatal pericardial tamponade after Wallstent implantation for malignant superior vena cava syndrome. J Endovasc Ther 2002; 9: 680-4. doi: 10.1177/152660280200900520.
- 29) Smith SL, Manhire AR, Clark DM. Delayed spontaneous superior vena cava perforation associated with a SVC Wallstent. Cardiovasc Intervent Radiol 2001; 24: 286-7. doi: 10.1007/s00270-001-0022-x.
- 30) Oudkerk M, Kuijpers TJ, Schmitz PI, et al. Self-expanding metal stents for palliative treatment of superior vena caval syndrome. Cardiovasc Intervent Radiol 1996; 19: 146-51. doi: 10.1007/BF02577610.
- 31) Warren MJ, Sen S, Marcus N. Management of migration of a SVC Wallstent into the right atrium. Cardiovasc Intervent Radiol 2008; 31: 1262-4. doi: 10.1007/s00270-008-9389-2.
- 32) Kuo TT, Chen PL, Shih CC, et al. Endovascular stenting for end-stage lung cancer patients with superior vena cava syndrome post first-line treatments—a single-center experience and literature review. J Chin Med Assoc 2017; 80: 482-6. doi: 10.1016/j.jcma.2017.04.005.
- 33) Lanciego C, Chacon JL, Julian A, et al. Stenting as first option for endovascular treatment of malignant superior vena cava syndrome. AJR Am J Roentgenol 2001; 177: 585-93. doi: 10.2214/ajr.177.3.1770585.
- 34) Watkinson AF, Hansell DM. Expandable Wallstent for the treatment of obstruction of the superior vena cava. Thorax 1993; 48: 915-20. doi: 10.1136/thx.48.9.915.
- 35) Baltayiannis N, Magoulas D, Anagnostopoulos D, et al. Percutaneous stent placement in malignant cases of superior vena cava syndrome. J BUON 2005; 10: 377-80.

- 36) Dyet JF, Nicholson AA, Cook AM. The use of the Wallstent endovascular prosthesis in the treatment of malignant obstruction of the superior vena cava. Clin Radiol 1993; 48: 381-5. doi: 10.1016/s0009-9260(05)81105-5.
- 37) Shah R, Sabanathan S, Lowe RA, et al. Stenting in malignant obstruction of superior vena cava. J Thorac Cardiovasc Surg 1996; 112: 335-40. doi: 10.1016/S0022-5223(96)70259-3.
- 38) Miller JH, McBride K, Little F, et al. Malignant superior vena cava obstruction: stent placement via the subclavian route. Cardiovasc Intervent Radiol 2000; 23: 155-8. doi: 10.1007/ s002709910033.
- 39) Srinathan S, McCafferty I, Wilson I. Radiological management of superior vena caval stent migration and infection. Cardiovasc Intervent Radiol 2005; 28: 127-30. doi: 10.1007/s00270-003-0183-x.
- 40) Ghanem A, Tiemann K, Nickenig G. Gone with the flow: percutanous retrieval of a migrated wallstent trapped in the right ventricle. Eur Heart J 2009; 30: 717. doi: 10.1093/ eurheartj/ehn547.
- 41) Bardet J, Fabre D, Brenot P, et al. Kissing stents for superior vena cava syndrome due to mediastinal fibrosis. Open J Cardiovasc Surg 2018; 10: 1179065218771900. doi: 10.1177/1179065218771900.
- 42) Cordial R, Moussavian MR, Corvalan J, et al. Percutaneous endovascular Y-stenting of a malignant superior vena cava and innominate vein obstruction. Vasc Endovascular Surg 2014; 48: 77-9. doi: 10.1177/1538574413507982.
- 43) Amin P, Sharafuddin MJ, Laurich C, et al. Anatomic bifurcated reconstruction of chronic bilateral innominate-superior vena cava occlusion using the Y-stenting technique. Ann Vasc Surg 2012; 26: 276.e5-9. doi: 10.1016/j.avsg.2011.10.005.
- 44) Kalra M, Gloviczki P, Andrews JC, et al. Open surgical and endovascular treatment of superior vena cava syndrome caused by nonmalignant disease. J Vasc Surg 2003; 38: 215-23. doi: 10.1016/s0741-5214(03)00331-8.
- Andersen PE, Duvnjak S. Palliative treatment of superior vena cava syndrome with nitinol stents. Int J Angiol 2014; 23: 255-62. doi: 10.1055/s-0034-1383432.
- 46) Marcy PY, Magne N, Bentolila F, et al. Superior vena cava obstruction: is stenting necessary? Support Care Cancer 2001; 9: 103-7. doi: 10.1007/s005200000173.
- 47) Pabba K, Rojas-Hernandez CM. Concurrent presentation of a hemorrhagic pericardial effusion and venous thromboembolism in malignancy: a systematic review of case studies. J Thromb Thrombolysis 2019; 48: 454-8. doi: 10.1007/ s11239-019-01884-z.
- 48) Ratzon R, Tamir S, Friehmann T, et al. Thrombosis, anticoagulation and outcomes in malignant superior vena cava syndrome. J Thromb Thrombolysis 2019; 47: 121-8. doi: 10.1007/s11239-018-1747-6.
- 49) Nagata T, Makutani S, Uchida H, et al. Follow-up results of 71 patients undergoing metallic stent placement for the treatment of a malignant obstruction of the superior vena cava. Cardiovasc Intervent Radiol 2007; 30: 959-67. doi: 10.1007/s00270-007-9088-4.