Superior vena cava syndrome after cardiac surgery

Heba Arafat[a], Ghada Ali[b]

[a]Anaesthesia Department, Cardiac Intensive Care Unit, University Hospital Southampton, Southampton, UK, [b]Department of Anaesthesia, Menoufia University, Shebeen El-Kom, Egypt

Correspondence to Heba Arafat, MBBCH, EDAIC, MD, 1 Nichol Court, Hiltingbury Road, Chandler1s Ford, Southampton SO53 5NR, UK. Tel: 02381206135, Mob: 00447397672934; fax: 02381204348
e-mail: heba.arafat81@gmail.com

Received 10 March 2019
Accepted 2 June 2019

The Egyptian Journal of Cardiothoracic Anesthesia 2019, 13:10–13

Superior vena cava (SVC) syndrome is a group of symptoms caused by obstruction of the SVC at the junction of the right atrium. This obstruction was found to be caused by either external compression or internal occlusion. It leads then to impaired venous drainage into the heart, which will in turn reduce the preload and consequently the cardiac output that might be fatal at some stage. On the flip side, it causes blood engorgement of the upper half of the body resulting in swelling and increased pressures. SVC syndrome may happen acutely in relation to cardiac surgery as a less frequent complication. It might be then caused by mechanical obstruction by a retractor, improper placement of venous cannula, thrombus formation on top of indwelling catheters or pacemaker wires, or overflow following 'Glenn' surgery in pediatrics. Diagnosis of the SVC syndrome should involve a high index of suspicion in the context of cardiac surgery especially that the manifestations are not classic in all cases. So, once the manifestations of the syndrome start to appear, prompt actions should be taken to confirm the diagnosis and to start treatment to avoid hemodynamic instability resulting from the dramatic decrease in preload, taking into consideration the limited time available for the collateral drainage (e.g. from azygos, hemiazygos, or internal mammary veins) to develop and bypass the obstruction. Exploring different causes of developing SVC syndrome after cardiac surgery would help early diagnosis and management, which could be lifesaving in many scenarios.

Keywords:
cardiac, superior vena cava, surgery

Egypt J Cardiothorac Anesth 13:10–13
© 2019 The Egyptian Journal of Cardiothoracic Anesthesia
1687-9090

What is superior vena cava syndrome?

Superior vena cava (SVC) syndrome is a group of symptoms caused by obstruction of the SVC at the junction of the right atrium. This obstruction is caused by either external compression or internal occlusion. It then leads to impaired venous drainage into the heart, which in turn reduces the preload and consequently the cardiac output (CO). On the flip side, it causes blood engorgement of the upper half of the body resulting in swelling and increased pressures [1].

Etiology of superior vena cava syndrome

Several anatomic factors contribute to the development of SVC syndrome. SVC is a relatively thin-walled vessel with low intravascular pressure, making it more susceptible to compression [2]. In 90% of cases, the SVC syndrome is caused by extrinsic compression by an intrathoracic malignancy. In this case, the manifestations usually start gradually [3]. Other less common causes, which would manifest acutely, are usually iatrogenic. Many of these acute causes are related to cardiac surgery. For example; mechanical obstruction by a retractor, improper placement of venous cannula [4], thrombus formation on top of indwelling catheters or pacemaker wires [5], or overflow following 'Glenn' surgery in pediatrics [6].

Retractor placement is an important step in the field of cardiac surgery, especially in coronary artery bypass graft where the left internal mammary artery retractor elevates the left hemithorax to provide adequate surgical exposure for left internal mammary artery harvesting. To date, at least two published cases report the development of SVC syndrome following retractor placement, where the patients had severe hemodynamic instability together with head engorgement and central venous pressure rise, that all resolved only after release of the retractor [4].

Apart from the retractor pressure, the shear surgical manipulation itself during cardiac surgery may result in injury and scarring at the SVC-right atrial junction which may add to the causes of cardiac surgical SVC syndrome [7].

Another well-known complication during cardiopulmonary bypass, which can result in acute intra operative SVC syndrome, is the malposition of the SVC cannula. The venous cannula is

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

responsible for directing deoxygenated blood from the patients' venous system to the cardiopulmonary bypass. Poor SVC cannula position can either reduce or completely obstruct venous drainage from the upper body [4].

SVC syndrome may occur without external compression or trauma; it may rather occur with the presence of a foreign body which acts as a nidus for thrombus formation such as pacemaker leads or venous catheters [i.e. central venous catheter (CVC), pulmonary artery, or hemodialysis catheter]. The mechanism of SVC syndrome as a result of transvenous pacemaker insertion, either as a part of cardiac procedure or as a standalone procedure, is similar to that caused by indwelling CVC or pulmonary artery (PA) catheters. This includes the mechanical stress associated with pacemaker wires or catheters that may trigger vessel wall inflammation, thrombus formation, and ultimately lead to venous obstruction and occlusion. It is worth noticing that several predisposing factors may enhance the development of pacemaker-induced SVC obstruction such as thrombophilia, the use of hormone therapy, infection, the presence of a temporary wire before implantation, and the presence of multiple active or retained pacing leads especially in the absence of an adequate collateral circulation. Risk factors for hemodialysis –related or CVC-related thrombosis include (i) catheter-related characteristics such as caliber-to-vein ratio, venipuncture-associated trauma, and catheter position (i.e. higher risk if catheter tip is in the brachiocephalic vein or proximal SVC vs. distal to SVC) and (ii) patient-related characteristics including vein caliber, malignancy, prior thromboembolism, and a hypercoagulable state [5].

Impedance of flow within the SVC does not necessarily result from external or internal mechanical compression, it may be rather initiated by overflow of blood within the vessel as expected with patients with bidirectional cavo-pulmonary shunt [bidirectional Glenn shunt (BDG)]. BDG is generally performed in many congenital cardiac anomalies where complete two ventricle circulations cannot be easily achieved. The success of BDG shunt is achieved by partially separating the pulmonary and systemic venous circulation. This results in reduced ventricular preload and long-term preservation of the myocardium. However, BDG is mainly an anastomosis between SVC and right pulmonary artery which may in turn increase the SVC pressure and consequently provoke SVC syndrome [6]. It was also found that recipients of heart transplantation with history of cavo-pulmonary anastomosis showed an increase in the incidence of SVC syndrome after heart transplantation [8].

A very rare cause of SVC syndrome after cardiac surgery is the development of aortic pseudoaneurysm, causing compression of the SVC. This potentiality should be therefore considered with all patients who present with SVC syndrome even years after cardiac surgery, and should be then excluded by enhanced chest computed tomography [9].

Clinical manifestations

The SVC syndrome manifestations result from the SVC flow obstruction from one side and the reduction in venous return and cardiac preload from the other side. Obstruction of the SVC flow is commonly manifested by facial, head, and neck swelling and engorgement, upper limb edema, cerebral edema, and optic neuropathy, and in the awake patient, nausea, headache, blurry vision, or mental status changes and laryngeal edema resulting in the risk for airway obstruction and/or collapse. On the contrary, the cardiac preload reduction manifestation may involve hypotension, tachycardia, besides the hypoperfusion manifestations of low urine output and tissue acidosis [4].

Theoretically, these manifestations could be easily detected in the perioperative course, especially the cardiac surgical ones, as the patient is closely monitored and observed, for example, a sudden rise in the central venous pressure, decreased pulse pressures, and drop in systemic pressures. However, in some scenarios, owing to the complexities of cardiac procedures, it could be misinterpreted as a part of the cardiac illness. An example of such scenarios is when a patient develops SVC syndrome after aortic valve or hypertrophic obstructive cardiomyopathy surgery where there is postoperative systolic anterior motion of the mitral valve as a complication, and the heart is underfilled. In this situation, all efforts would be directed toward increasing the ventricular filling and reducing the heart rate to increase the CO against the narrowed left ventricular outflow tract. What could be missing here is that whatever the volume of fluid infused through the CVC or peripheral cannula in the upper half of the body, it will never help in increasing the cardiac preload, as the SVC is already occluded, and this could be fatal [10].

That is why the diagnosis of the SVC syndrome should involve a high index of suspicion and

involve many diagnostic modalities in the context of cardiac surgery knowing that the manifestations vary among cases. So, once the manifestations of the syndrome start to appear, prompt actions should be taken to confirm the diagnosis and to start treatment to avoid hemodynamic instability resulting from the dramatic decrease in preload, taking into consideration the limited time available for the collateral drainage (e.g. from azygos, hemiazygos, or internal mammary veins) to develop and bypass the obstruction, which is not the case in chronic SVC obstruction (i.e. malignant mass), where the collaterals develop over several weeks and in a way help to ameliorate the obstruction [1].

Diagnostic imaging

An easy, rapid and practical investigating tool in the operating theater or CICU is the transesophageal echocardiography (TOE). A mid-esophageal and upper esophageal bi-caval views are typically used to establish the point of obstruction. TOE guidance could be also used to guide the surgeons for optimal retractor placement without compromising SVC diameter and flow [4]. An alternative TOE view is the upper esophageal aortic valve short axis, through which the short axis of the SVC can be visualized. This view may be less helpful than the long axis one, where accurate detection of the point of obstruction is feasible [11].

Less practical diagnostic tests, however accurate, are the contrast-enhanced computed tomography and magnetic resonance tomography of the thorax. Such diagnostic studies are not suitable for acute cases as it requires travelling outside the operating theater or CICU [2].

Management

Treatment of benign SVC syndrome relies on two main axes: the relief of the obstructing point, either retractor, thrombus, or congestion from shunt overflow (Glenn), and the second axis is the hemodynamic support provided by fluid administration and probably inotropic drugs until the obstruction is relieved. This should be given via venous lines inserted in the veins of the lower half of the body, for example, femoral CVC. Reverse Trendelenburg position is of good benefit in aiding the venous return of the heart supporting the patient's CO till the obstruction is relieved [4].

Endovascular therapy (EVT) is the first-line treatment for alleviating SVC occlusion; it helps in relieving the stenosis and associated symptoms with good long-term results. This should be achieved by either balloon dilatation and/or stenting. Complications, however, could happen with this remedy, and it could involve occlusion, infection, pulmonary embolus, stent migration, hematoma at the insertion site, bleeding and, very rarely, perforation [12]. Ideally, the SVC catheter is to be removed if it was the predisposing factor for the thrombus formation. However, a case study revealed the possibility of the stenting of the vein without removing the catheter. This would be an asset in the context of long-term catheters such as the hemodialysis ones, which may be used after cardiac surgery [13].

Surgical intervention is used when the EVT fails to relieve the SVC obstruction. Surgery is not aiming at removing the thrombus because of the risk of embolization. It is otherwise directed toward reconstruction either by single graft or various conduits to bypass the SVC obstruction. The conduits may be autografts (e.g. saphenous vein, superficial femoral vein, or autologous pericardial tube), homografts, or grafts made from synthetic materials. There is no sufficient evidence regarding the superiority of one type of graft over the others. Surgical bypass could be done via several forms of incisions, for instance, median sternotomy, thoracotomy, or sternotomy with elongated cervical incision (reversed L shape). The site and type of anastomosis are based on the site of the obstruction and the cause of the SVC syndrome [3].

The key element in the treatment of the SVC syndrome, is the timing of intervention, as delay in the recognition and management the SVC syndrome may result in fatal or lifelong complications.

Conclusion

SVC obstruction after cardiac surgery is a rare yet a grave complication that may develop during cardiac surgery. It requires early recognition and prompt action to impede deleterious fatal or lifelong complications. EVT is the first-line of treatment for the post-cardiac SVC syndrome. For all patients with suspected SVC syndrome while an indwelling central venous catheter is in place, thrombus formation must be excluded.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1 Deniz D. Anesthetic implications of superiorvena cava syndrome in interventional radiology: a case report. AANA J 2017; 85:453–456.

2 Aaron B, David U, DO Brooks L. Ultrasound detectionof superior vena cava thrombus. West J Emerg Med 2014; XV:715.

3 Michele G, Adam NP, Jaimin RT, Mark SS. Surgical treatment of benign superior vena cava syndrome. Ann Thorac Surg 2016; 102:e369–e371.

4 Adam WA, Juan NP, Geoffrey LH. An unusual cause of intraoperative acute superior vena cava syndrome. Ann Card Anaesth 2013; 16:133–136.

5 Swetha MC, Sudheer OV, Murukesh S, Lakshmi K. Superior vena cava syndrome due to catheter related thrombus in a patient with a permanent pacemaker. Indian J Anaesth 2015; 59:758–760.

6 Praveen KN, Manikandan S, Krishnamanohar SR, Ramesh CR. Superior vena cava syndrome after pulsatile bidirectional Glenn shunt procedure: perioperative implications. Ann Card Anaesth 2009; 12:53–56.

7 Algethamy H, Nicolle D, Novick R, Saito A. Blindness with superior vena cava obstruction after cardiac surgery. Can J Neurol Sci 2010; 37:897–900.

8 8Osamah A, Nofil IA, Jeffrey MV, Jeremiah SM, Rebecca KA, Roosevelt B, *et al.* Frequency of superior vena cava obstruction in pediatric heart transplant recipients and its relation to previous superior cavopulmonary anastomosis. Am J Cardiol 2013; 112:286–291.

9 Panagiotis D, Ioannis N, Panagiotis H, Christos P, Paraskevi D, Efstratios A, *et al.* Case report superior vena cava syndrome in a patient with previous cardiac surgery: what else should we suspect? Diagn Pathol 2010; 5:43.

10 Moreno B, Buendía JA, Cortés A, Puerto L, Carabias E, Torres MJ, *et al.* Superior vena cava syndrome with massive thrombosis after aortic valve replacement. JSM Cardiothorac Surg 2017; 2:1010.

11 Albert CP, Scott TR. A practical approach to transesophageal echocardiography. Third edition. Philadelphia, United States; 2015. p. 26.

12 Aphrodite T, Audrey CM, Doff BM, James EL, Robert LG. Endovascular treatment for superior vena cava occlusion or obstruction in a pediatric and young adult population: a 22-year experience. J Am Coll Cardiol 2007; 49:1003–1009.

13 Atul S, Michael F, William LB, Erben Y, Robert A, Carlos MH. Management of a patient with superior vena cava syndrome and a central venous catheter. Vasc Dis Manag 2017; 14:E159.