Asian Journal of Cardiology Research

Volume 8, Issue 2, Page 1-6, 2023; Article no.AJCR.95598

A Case of Acute on Chronic Life Threatening SVC Stenosis Managed by Timely Intervention

Brajesh Kumar Kunwar [a++#*], Atul Ingle [b++†], Anshu Thakur [b‡], Nikhil Varge [b^], Subhashish Panda [b^] and Gautam Singh [b^]

[a] Department of Cardiology, Medicover Hospital Navi Mumbai, India.
[b] Fortis Heeranandani Hospital Vashi, Navi Mumbai, India.

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

Open Peer Review History:
This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: https://www.sdiarticle5.com/review-history/95598

Case Study

Received: 13/11/2022
Accepted: 19/01/2023
Published: 02/02/2023

ABSTRACT

Superior Vena Cava (SVC) obstruction is a very well recognized condition described in many diseases like tumour or malignancies of middle mediastinum, clots in SVC, aortic aneurysm, thyroid disorders, infections like fungal and tuberculosis of lungs and rarely constrictive pericarditis. Clinical presentation is usually chronic or sub-acute. Rarely, it can have acute presentation with symptoms of dyspnoea and stridor. Diagnosis is usually made in view of underlying disease and presence of puffiness of face and engorged neck veins. Timely intervention especially in acute presentation is often lifesaving. We present a case of acute on chronic SVC obstruction in a patient of chronic kidney disease (CKD) related to HD (Haemodialysis) catheter induced obstruction of SVC. We had a patient who presented with stridor and orthopnea in a SVC syndrome setting in an emergency.

[++] Head & Director;
[#] Senior Interventional Cardiologist;
[†] Senior Nephrologist and Transplant Physician;
[‡] Junior Consultant Cardiologist;
[^] Senior Physician;
**Corresponding author: Email: kunwar_brajesh@yahoo.com;*

Keywords: Superior vena cava obstruction; balloon dilation; stenting.

1. INTRODUCTION

Superior vena cava syndrome is a group of clinical signs and symptoms caused by a partial or total obstruction of blood flow through the SVC. According to the literature, the incidence of SVC syndrome ranges from 1 in 650 to 1 in 3100 people [1]. The SVC is a low-pressure venous channel with thin walls that can be damaged by a variety of pathogenic processes. These mechanisms are classified into three types: damaged vascular anatomy, impaired venous flow, and decreased vessel wall integrity. In patients with SVC syndrome, these processes frequently coexist. The most prevalent cause of SVC syndrome is extrinsic compression and blockage of the SVC by a mass in the mediastinum [2]. Occlusive venous thrombus development is now associated with an increasing proportion of SVC syndromes, limiting venous flow back to the heart. The increasing use of indwelling intravascular devices such as catheters and pacemakers, as well as implantable cardioverter-defibrillator (ICD) leads, has contributed significantly to this increase. The resulting venous wall inflammation, fibrosis, and eventual thrombus cause vascular stenosis. The most prevalent reason is thrombus development or cancer invasion of the artery wall [2]. The superior vena cava is created by the junction of the left and right innominate (brachiocephalic) veins and transports deoxygenated blood back to the heart from the head, neck, upper extremities, and torso. This syndrome is most usually encountered as a result of mediastinal malignancy, such as small cell bronchogenic carcinoma, non-lymphoma, Hodgkin's and metastatic malignancies. Other reasons, such as post-chemo-radiation and iatrogenic catheter caused, are also common. Venous congestion, combined with laryngeal oedema and cerebral congestion, might result in a serious clinical condition. Face or neck edoema, upper extremities swelling, dyspnoea, cough, and dilated chest vein collaterals are the most prevalent signs and symptoms. We had a patient who presented with stridor, orthopnea in a SVC syndrome setting in an emergency.

2. CASE PRESENTATION

A 73-year-old male presented with severe breathlessness and stridor. Patient was a known case of CKD on regular haemodialysis through right Internal jugular vein (IJV) Haemodialysis (HD) catheter. On examination patient was found to be in distress due to difficult breathing with stridor. Face, neck and chest wall was seen puffed up. After initial evaluation patient underwent emergency lifesaving balloon dilatation followed by stenting of SVC through left IJV. Post procedure patient improved immediately with complete resolution of symptoms with in 1 hour of procedure.

2.1 Diagnosis

Our patient's diagnosis of SVC syndrome was evident, based partly on a history of chronic kidney illness and a right IJV indwelling HD catheter. Face/neck swelling, distended neck veins, cough, dyspnea, orthopnea, upper extremity edoema, distended chest and neck veins, and conjunctival suffusion were all physical observations. In addition, the patient experienced stridor, hoarseness, dysphagia, pleural effusion, head plethora, headache, nausea, lightheadedness, syncope, change in vision, changed mental status, upper body edoema, cyanosis, papilledema, and was confused. The patient's vital signs were erratic, with a thready pulse and low volume. The blood pressure was low. The patient was orthopneic and could not lie down flat.

2.2 Treatment

CT angiogram prior to taking the procedure showed occluded right and left sided IJV, brachiocephalic vein and no flow across SVC. There was indwelling HD catheter on right IJV. Hence left IJV puncture was done under Doppler guidance. Peripheral angiogram showed occluded left IJV with no flow in brachiocephalic vein and SVC (Fig. 1). The GAIA 3 014 wire was used to cross the stenosis from left IJV to SVC. The whole path was dilated with NC trek 4x15mm coronary balloon at high pressure (Fig. 2). This was followed by dilatation with ULTRAVERSE 7x40mm peripheral balloon at 12 atmosphere (Fig. 3). Check angiogram showed fibrotic stricture at mid SVC with recoiling. Hence, SVC was stented using BARD ELUMINEXX 14X40mm self-expanding stent. Final shoot showed good flow from left IJV to SVC to right atria (RA) (Fig. 4). Right perm catheter (catheter used for dialysis) was removed and left IJV perm catheter (catheter used for dialysis) was inserted as patient had bilateral thrombosed lower limb veins and no fistula available for dialysis.

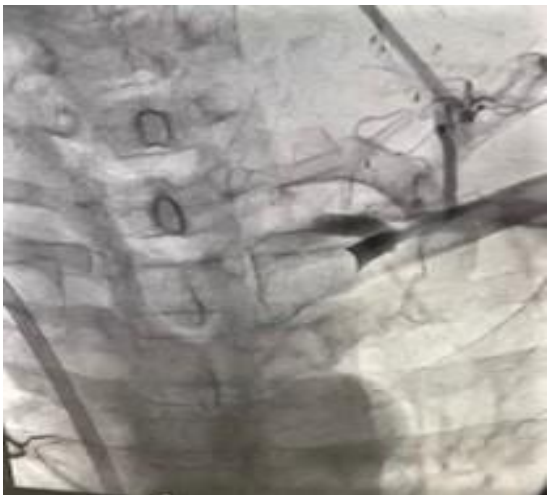

Fig. 1. Left Jugular Vein angiogram showing total occlusion of brachiocephalic & SVC

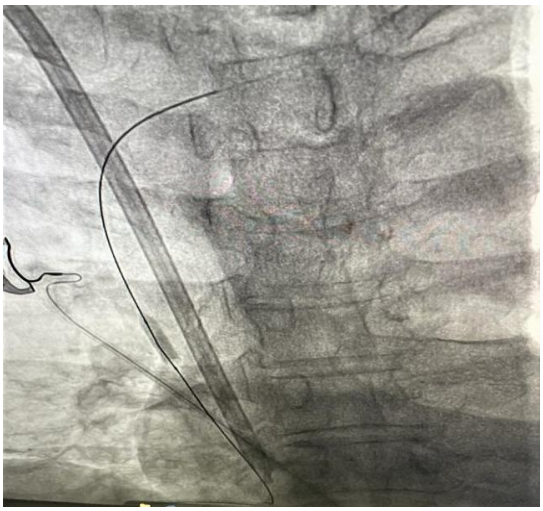

Fig. 2. Obstruction crossed with GAIA 3 wire and balloon dilatation with coronary balloon

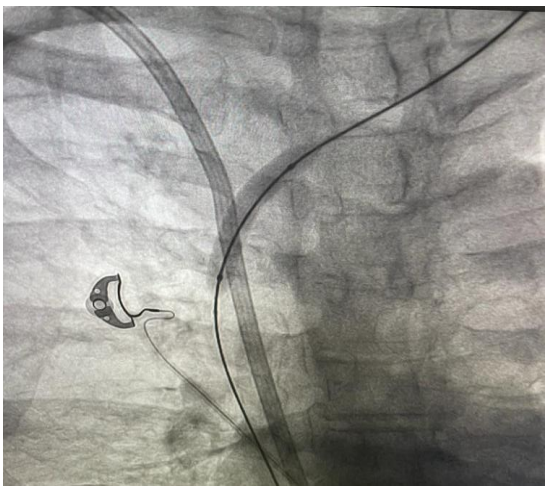

Fig. 3. Obstruction dilated with peripheral balloon

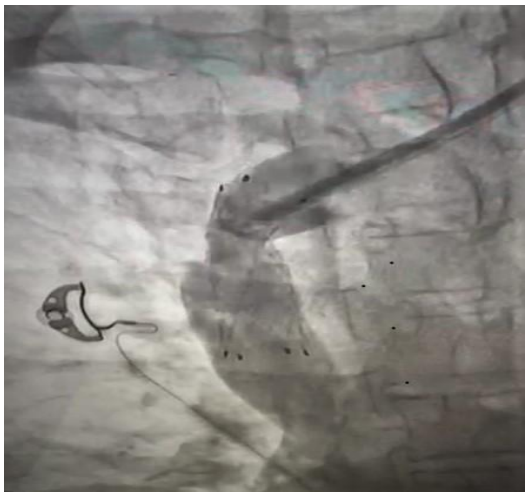

Fig. 4. Post Stenting SVC shows good flow

2.3 Follow up

Post procedure patient started improving immediately on cath lab table. He was shifted to ICU and his puffiness of face was reduced to minimal in 1 hour time. Vitals were stabilized and was able to lie flat with total disappearance of respiratory distress and stridor. Next day patient underwent routine HD throght the left IJV HD catheter and was discharged following that. Timely skilled intervention saved a life with quick reduction in morbidity.

3. DISCUSSION

William Hunter reported SVC syndrome in a patient with a massive syphilitic aortic aneurysm compressing the SVC in 1757.(3) About 15,000 Americans experience it every year in the United States.(1) SVC syndrome, which results from external compression, thrombosis, or vein invasion, is the clinical manifestation of SVC obstruction. Today, malignancy is nearly always (more than 90%) the secondary cause of SVC syndrome.(2) Non-small cell lung cancer, which affects 50% of patients, is the most frequent malignant cause. Catheter-related thrombosis could result with HD catheters put into the IJV. A higher risk of thrombosis-related problems is also thought to be present with HD catheters in comparison to conventional central venous catheters (CVCs) [4]. It is unclear exactly what causes HD catheter-associated thrombosis, however factors such recurrent vascular access, platelet dysfunction, endothelial factors, inflammation, and aberrant clotting have all been proposed.(5) Other characteristics of catheters (such as catheter caliber-to-vein), venipuncture-related trauma (now reduced with ultrasound assistance), and catheter position are risk factors for HD thrombosis (i.e., higher risk if catheter tip is in the brachiocephalic vein or proximal SVC versus distal to SVC). Practice guidelines said that the catheter to vessel ratio (CVR), which can range from 33% to 45% of the vessel's diameter, can help doctors choose the most suitable-sized for device vessel. This is supported by the fact that using larger diameter catheters can increase the risk of thrombosis [6,7]. Catheter placement can damage the endothelium wall, the catheter can be positioned inappropriately, or the catheter can obstruct blood flow within a vein, resulting in blood stasis, which are all risk factors for catheter-related thrombi. These are parts of the Virchow triad and are thought to be contributing elements to thrombosis in iatrogenic SVCS [8]. In our case, the patient had an HD catheter in the right IJV for the previous two months.

Supportive therapy and medical management are frequently undertaken following a clinical diagnosis. This entails elevating the patient's head as a simple procedure to reduce venous pressure. The patient's underlying SVC syndrome aetiology guides future therapy. In patients with thrombus caused by an indwelling intravascular device, removal, coupled with anticoagulant medication and catheter-directed thrombolysis, should be considered. Multidisciplinary treatment planning is essential for people with obstruction due to cancer, as tumour kind and staging can assist guide suitable chemotherapy or radiation therapy. To overcome SVC obstruction, open surgical repair with bypass grafting using spiral saphenous vein, femoral vein, polytetrafluoroethylene (PTFE)

Fig. 5. Pre & Post Procedure Picture (Note: Marked reduction of Face & Neck swelling and reduced distress)

graft, or Dacron graft has historically been explored. Endovascular therapy is now commonly considered the first-line treatment for SVC syndrome, with expanded treatment options for both benign and malignant causes. Endovascular therapy that is less intrusive might provide patients with rapid relief from symptoms. In some circumstances, SVCS can be fatal and necessitates immediate treatment. If there is laryngeal edoema producing laryngeal constriction or cerebral edoema, these medical problems must be managed immediately, and therapy of the underlying cause of SVCS may be necessary [9-11]. If left untreated, these clinical SVCS consequences may result in long-term morbidity or mortality, and empiric treatment with radiation, stenting, and/or chemotherapy may be necessary even before biopsy results are available. To help practitioners classify the urgency of SVCS situations, multiple rating systems have been devised. Grade 4 SVCS, according to Yu et al., indicates life-threatening disease caused by one or more of the following: "significant cerebral edoema (confusion, obtundation) or significant laryngeal edoema (stridor) or significant hemodynamic compromise (syncope without precipitating factors, hypotension, and renal insufficiency)." Despite the fact that only 5% of SVCS patients have grade 4 illness, any of the aforementioned problems would warrant an urgent venogram, stent installation, and thrombolytic medication if required [12]. In our case report, the patient likewise had grade 4 SVCS and was taken for emergency catheter intervention, which resulted in a good rewarding immediate result (Fig 5).

4. CONCLUSIONS AND RECOMMENDATIONS

Acute SVCS produces severe patient distress, usually signals the presence of a serious underlying disease, and demands prompt treatment. Percutaneous stenting can relieve symptoms quickly. Stenting has the added benefit of providing strong radial strength to combat the fibrosis that causes recoiling of the post-balloon dilatation segment of conduit. Emergent indications for immediate therapy include laryngeal and cerebral edoema, as well as associated symptoms, for which endovascular stenting should be performed as soon as possible.

Most patients with SVCS, particularly grade 4, should be offered stent insertion as soon as possible. When the fundamental reason is identified, as in our instance, the procedure becomes straightforward. In competent hands, the procedure is simple and usually risk-free. Therapeutic anticoagulants can help patients who have recurrent catheter failure or thrombosis.

CONSENT

As per international standard or university standard, patient(s) written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Higdon ML, Higdon JA. Treatment of oncologic emergencies. American family physician. 2006;74(11):1873-80.
2. Seligson MT, Surowiec SM. Superior vena cava syndrome. 2017.
3. Azizi AH, Shafi I, Shah N, Rosenfield K, Schainfeld R, Sista A, et al. Superior vena cava syndrome. Cardiovascular Interventions. 2020;13(24):2896-910.
4. Cohen R, Mena D, Carbajal-Mendoza R, Matos N, Karki N. Superior vena cava syndrome: A medical emergency? International Journal of Angiology. 2008; 17(01):43-6.
5. Baldari D, Chiu S, Salciccioli L. Aortic pseudoaneurysm as a rare cause of superior vena cava syndrome: A case report. Angiology. 2006;57(3):363-6.
6. Szokol JW, Alspach D, Mehta MK, Parilla BV, Liptay MJ. Intermittent airway obstruction and superior vena cava syndrome in a patient with an undiagnosed mediastinal mass after cesarean delivery. Anesthesia & Analgesia. 2003;97(3):883-4.
7. Gaslin M, Seymour P, Willcox TO. Superior vena cava syndrome presenting as serous otitis media. Otolaryngology-Head and Neck Surgery. 2005;2(133):P243.
8. Guijarro Escribano J, Antón R, Colmenarejo Rubio A, Sáenz Cascos L, Sainz González F, Alguacil Rodríguez R. Superior vena cava syndrome with central venous catheter for chemotherapy treated successfully with fibrinolysis. Clinical and Translational Oncology. 2007;9(3):198-200.
9. Baker G, Barnes H. Superior vena cava syndrome: etiology, diagnosis, and treatment. American Journal of Critical Care. 1992;1(1):54-64.
10. Sofue K, Takeuchi Y, Arai Y, Sugimura K. Life-threatening cerebral edema caused by acute occlusion of a superior vena cava stent. Cardiovascular and interventional radiology. 2013;36(1):272-5.
11. Taguchi J, Kinoshita I, Akita H. Superior vena cava syndrome. Gan to Kagaku ryoho Cancer & Chemotherapy. 2011;38(4):518-23.
12. James BY, Wilson LD, Detterbeck FC. Superior vena cava syndrome—a proposed classification system and algorithm for management. Journal of Thoracic Oncology. 2008;3(8):811-4.

Peer-review history:
The peer review history for this paper can be accessed here:
https://www.sdiarticle5.com/review-history/95598