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Case Report

Central Venous Pressure Catheter induced Superior Vena Cava Syndrome in a 2.7 kg Neonate after Patent Ductus Arteriosus (PDA) Ligation: Report of a Rare Case

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Abstract

Superior vena cava syndrome (SVCS) is an uncommon but severe complication following neonatal cardiac surgery. It is caused by severe obstruction or occlusion of the SVC. In neonates, SVCS is known to cause swellings of the face, head, neck, or upper chest; prominent vessels around the chest wall and neck; or even a significant airway obstruction and respiratory distress, cerebral edema, and significant morbidity and mortality. Recently, the growing use of SVC catheterization makes catheter-related thrombosis a potential cause of serious complications. Approximately 47 % of the SVCS occur in cardiac cases associated with congenital heart diseases. Following PDA ligation surgery, the SVCS can occur due to surgical trauma or direct injury to the SVC or surrounding veins during ligation under cardiopulmonary bypass (CPB) or CVP catheter or PICCs leading to the genesis of thrombus or stenosis. Prompt recognition and appropriate management are vital for improving outcomes in the affected neonates.

We report A 9-day-old, 2.7 kg neonate diagnosed with a PDA with left-to-right shunt and persistent respiratory distress, who underwent its emergency surgical ligation. Despite an initially unremarkable postoperative course, a progressive swelling of the neck, face (including the periorbital area and head), and upper chest, with engorged veins on the chest wall, was noticed. The baby slowly and progressively became lethargic and unresponsive to the stimulus and dusky in color and developed respiratory difficulty and arterial oxygen desaturation (87%), requiring high-flow nasal cannula (HFNC) oxygen therapy. Such a complication immediately following PDA ligation is uncommonly expected. The diagnosis of SVC syndrome in neonates can be challenging due to the subtle initial symptoms and the rarity of the condition. On the exclusion criteria, clinical picture and ultrasonography parameters, and the diagnosis of the SVC line (4.5Fg), induced SVCS was reached, and it was decided to remove it. There was spontaneous regression of the swelling and improvement in the mental status and respiratory efforts and the baby again became active within 2-3 hours of CVP catheter removal. The SPO2 was maintained at 97-99% with 2L oxygen flow through nasal cannula. The rest of the course was uneventful, and the baby was sent back to the referral hospital on the second postoperative day. We are of the opinion that cardiac-related SVCS has a faster onset and more symptoms, and so delay in diagnosis of acute complications would result in an increased rate of the primary outcome. To the best of our knowledge, SVC syndrome after PDA ligation caused by the obstruction due to a CVP line without thrombosis or injury has not been reported in the literature to date. Early diagnosis of SVC syndrome in neonates after cardiac surgery and prompt action is crucial in preventing the major complications and significant morbidity and mortality.

Keywords: CVP catheter; GA; left thoracotomy; PDA Ligation; SVC syndrome; swelling of chest; face; head; and neck

Introduction

Superior Vena Cava syndrome (SVCS) in neonates after cardiac surgery is a rare but potentially serious complication. It occurs due to obstruction of blood flow through the SVC, leading to swelling of the face, head, neck, and upper chest with venous engorgement particularly on the upper extremities and chest, or even significant morbidity and mortality due to increased cerebral venous pressure resulting in hydrocephalus or respiratory distress or low cardiac output. [1,2,3]

SVCS can be caused by thrombosis, external compression, or iatrogenic caused by improper placement of catheters or cannula during cardiac surgery or by direct injury to SVC. it can occur even after some heart procedure like Glenn or Senning cardiac repair.

Early diagnosis and treatment are crucial to prevent progression and potential morbidity and mortality. We describe a rare case of acute SVC syndrome in a 2.7 kg- female -neonate after successful PDA ligation, due to obstruction of the SVC by triple lumen CVP catheter inserted via right internal jugular vein (RIJV). Removal of the CVP catheter resulted in rapid recovery from the respiratory distress and lethargy within few hours.

Aim

To report on a rare case of acute superior vena cava syndrome (SVCS) developed in cardiac ICU immediately after the cardiac surgery in neonates. The nearly complete blood flow obstruction due to mismatch of the size of SVC cannula and the IJV or SVC should be suspected the cause of SVCS after ductus ligation, and its removal causes a swift recovery from the signs and symptoms of SVCS not only the edema but respiratory distress and consciousness level.

Case presentation

A 9-days- 2.7 kg Female, born at 37 weeks as normal delivery in a general hospital, presented with shortness of breath since 3rd day of life. The baby required oxygen therapy and diuretics round the clock. On examination, she was conscious, respiratory rate was 45 cycles /min, HR 144bpm. Chest auscultation revealed generalized bilateral basal crepitations. CVS examination revealed normal S1 and S2 sounds and a machinery murmur on left parasternal and sub clavicular region suggestive of a PDA. The attempt of ductus pharmacological closure with intravenous indomethacin (0.1-0.2 mg/kg) slowly over a minimum of 30 minutes and even 3 doses were unsuccessful. Therefore, she was referred to CTVS department of the super speciality tertiary care centre for the surgical PDA ligation. Echocardiography revealed situs solitus, levocardia, a PFO of 3mm with L-R shunt, PDA of 6mm with L-R shunt and PG of 38 mmHg, no PAH, good biventricular functions and no other congenital anomalies. Chest X-Ray (AP) view showed cardiomegaly with lung plethora suggestive of increased lung blood flow. Her HB was 16.8 gm%, TLC- 8800/cmm, platelets -1.64 lacs/cmm, total bilirubin of 14.9mg/dl with direct 2.58mg/dl and indirect of 12.32 mg/dl, total bilirubin decreased to 10.2 on the day of surgery. CRP value was in decreasing order from 15mg/l to 7.02mg/l suggestive of decreasing the body infection, PT of 13.7sec, INR of 1.06 and PTT of 32 sec. A detailed informed consent was obtained from the parents, and the baby was posted for emergency surgical PDA ligation.

In OR, a standard ASA monitoring including ECG, SPO2, ETCO2, were attached, The SPO2 revealed a baseline arterial oxygen saturation of 93%. General anaesthesia was induced with sevoflurane 8% in oxygen in air with a FIO2 of 60% until venous access with 24 G cannula was achieved and then its concentration was reduced to 1% and anaesthesia was further supplemented with fentanyl (5 mcg), midazolam (0.25 mg) and atracurium bromide 2mg was used as a muscle relaxant to facilitate the endotracheal intubation with 3mm cuffed tube, fixed at 9cm. A 22G leader Cath was inserted in the left femoral artery for continuous BP monitoring and serial arterial blood gas analysis. 4.5 Fg triple lumen catheter was inserted via the right internal jugular vein (RIGV) for CVP monitoring and administration of anaesthetic drugs, fluids, and inotropes as well as vasodilators.

The patient positioned for left lateral thoracotomy and the ductus was approached through the left 4th intercostal space. The aortic arch vessels, descending aorta, pulmonary artery and PDA were dissected. The PDA was confirmed by temporary clamping resulting in increase in diastolic pressure from 29 mm hg to 43 mmHg without any drop in the arterial saturation. Following appropriate hemodynamic assessment, the BP was reduced to MAP of 40 mmHg with administration of NTG (1mcg/kg/min) and sevoflurane 5-8% to facilitate the smooth ligation of the ductus and to avoid its rupture, a catastrophic complication. After ligation, Milrinone (0.3mcg/kg/min) was started to counter the systolic dysfunction following ductus ligation. The haemostasis was secured, and chest incision was closed in layers as usual fashion, and intercostal chest tube was also inserted. Incision and chest tube sites were infiltrated with bupivacaine (0.125%, 3ml) for post operative pain relief. Patient was extubated successfully in the OR and showed full recovery with good cry and saturation of 99-100% with oxygen support at 2L/min by nasal prongs. However, the slow and progressive swelling on the face around eyes, upper extremity, neck and upper chest and engorged veins on the chest and respiratory distress with decrease in arterial oxygen saturation to 87% was noticed.

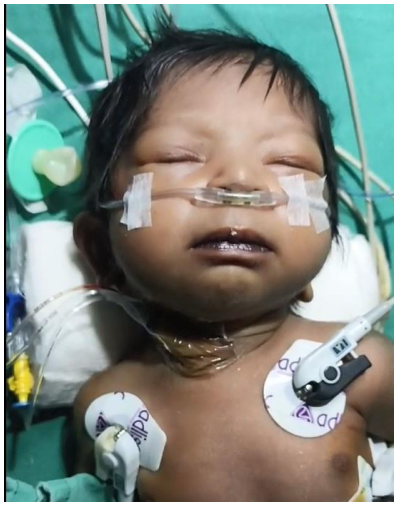

Figure 1: the postoperative photograph of the baby showing the swelling of head, neck, chest along with bluish discoloration of the face and chest, the features suggestive of the SVCS. In addition, a right sided triple lumen catheter and the HFNC oxygen therapy for respiratory distress and arterial O_2 desaturation can also be noticed.

The baby, became lethargic and developed and bluish discoloration of the lips and tongue and therefore, she was put on HFNC with a 4L oxygen flow and fluid was restricted and furosemide (1mg) was given, however, there was no relief occurred and swelling kept on increasing over the next 12 hrs., but hemodynamic were still maintained, BP systolic/ diastolic70-80/45-55 mmHg, CVP of 2mmHg, HR 150bpm. Postoperative, Chest X-ray (AP) view revealed only cardiomegaly with normal lung fields, without any gross widening of the mediastinum or any mass looking(thrombus) opacity on the right side of the chest, a right sided CVP line was confirmed, reaching almost to the RV so withdrawn 2-3 cm. (Figure-2)

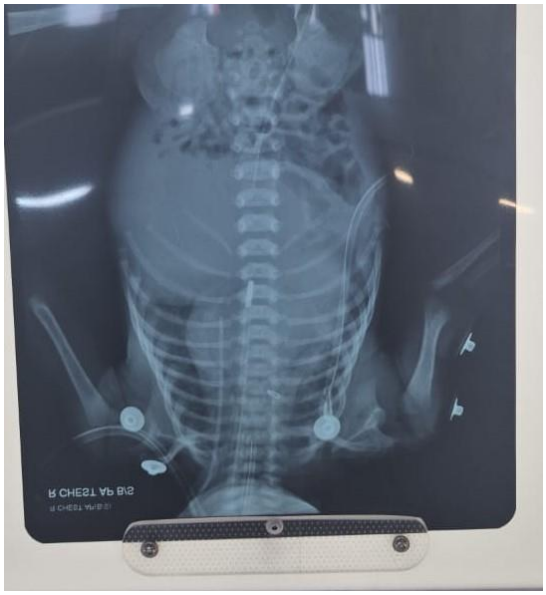

Figure-2. Chest X-ray (AP) view show - cardiomegaly with normal lung fields, without any gross widening of the mediastinum or any mass looking(thrombus) opacity on the right side of the chest, a right sided CVP line is seen reaching almost to the RV. So primarily excludes SVC thrombus with dilatation or compression by any mass

The ultrasound revealed noncompressible and nondistended IJV and SVC, with catheter in situ, there was a scanty blood flow, and the S, D, AR and VR waves could not be appreciated suggestive of severe SVCS. Finally, on the bases of clinical picture and USG parameters, the diagnosis of SVC syndrome was established and CVP catheter was the culprit for it, and so, it was decided to remove it. Following that the swelling started regressing and a gross improvement was observed over the next 2hr, and a complete recovery with recovery of alertness, normal colour of the face and saturation of 97% even without any oxygen support (Figure 3, &4).

Figure 3: The postoperative photograph of the neonate shows regression of the swelling of the body. Normal colour of the skin and completely weaned off the oxygen after removal of the CVP catheter. Now there is no CVP line on the right side of the neck as it was noticed in the figure 1a.

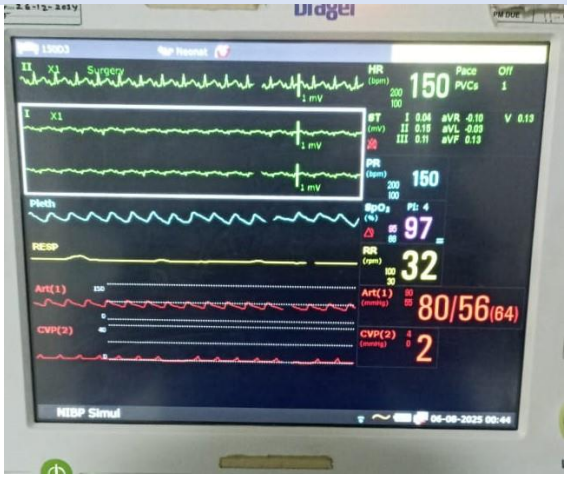

Figure 4: The monitor screen shows BP of 80/56mmHg, CVP of 2, SPO2 of 97% and HR of 150bpm without oxygen and milrinone therapy. At this point, the arterial line and PICC lines were also removed.

The femoral arterial cannula and chest tube were also removed before the neonate was transferred back to the referral hospital on the 2nd postoperative day and the rest of the course was uneventful as informed by the treating physicians.

Discussion

Superior Vena Cava syndrome (SVCS) after cardiac surgery in neonates is a rare but serious complication. SVCS results from partial or complete obstruction of blood flow through the SVC. This can result in the rapid development of symptoms of blood flow obstruction, respiratory compromise, and neurologic manifestations. SVCS in cardiac surgical patients can occur due to several causes like mechanical obstruction, thrombosis and iatrogenic factors related to the cardiac surgery or leads of pacemaker or defibrillators devices. Mechanical obstruction from mediastinal masses, improper positioning of the retractors, or purse-string suture above the SVC cannula during management of CPB, even SVC obstruction due to CPB cannula before and after the CPB, even long use of CVP catheter can cause inflammation and thrombosis genesis. [4,5,6]

Several cardiac surgical procedures can directly precipitate the SVCS like Glenn procedure (superior vena cava to right pulmonary artery anastomosis), Fontan surgery (SVC and IVC directly route to the PA), SVC repair in partial anomalous pulmonary venous connection (PAPVC), senning or mustard due to baffle obstruction or even congenital heart diseases post-surgical repair. [7,8]

The internal jugular veins (IJV) are the primary venous outflow channels of the head and neck. The union of internal jugular vein and subclavian vein forms the brachiocephalic veins. The IJV joins with the subclavian vein posterior to the sternal end of the clavicle and forms the brachiocephalic vein and drain the upper extremities and head and neck and lymph from the thoracic duct. These veins drain the blood from the head neck and upper limb. The SVC is formed by the union of the right and left brachiocephalic veins, and even azygos vein draining blood from posterior thoracic and abdominal walls, as well as the mediastinal viscera also joins the SVC. [9,10] So, on the basis of the anatomy the SVC syndrome will affect the venous drainage of these areas and present with clinical sign and symptoms of swelling of the head and neck and upper extremities, chest and thorax along with engorged chest wall vessels. In addition, in neonates, SVC is known to cause significant airway obstruction and respiratory distress, cerebral edema with loss of consciousness, chylothorax.[1]. On the contrary, the cardiac preload reduction manifestation may involve hypotension, tachycardia, along with the hypoperfusion manifestations of low urine output and tissue acidosis.[11]

PDA ligation is unlikely involved directly in the development of acute SVCS, however, the CVP line used for the administration of various anaesthetic drugs, inotrpes or vasodilators to achieve the desired hemodynamic (hypotension or hypertension) to facilitate the ductus ligation and to counter the systolic dysfunctions can obstruct the SVC flow due to disproportionate large size as compare to the internal jugular or SVC size. The diameter of the IJV is 3.1 mm(median), so it may be completely blocked by the pediatric triple lumen catheter size of 4.5 FG.[12] The cardiac related SVCS presents as more acute, symptomatic and with increased morbidity and mortality.[2,3] Our previous publication on aortic flow obstruction and SVC and IVC obstruction by the CPB cannulas and clinical manifestation draw our attention on the SVCS by the CVP catheter itself and ultrasonography helped in reaching to the diagnosis of the syndrome.[6]

Diagnosing SVCS in the Cardiac ICU should involve a combination of clinical presentation, imaging, and potentially invasive procedures that are crucial to confirm the obstruction and determine its cause. X-ray chest will show the widening of the mediastinum with mass looking opacity on right side of mediastinum, and enlarged SVC. But chest X-Ray may appear normal with SVC thrombus. Ultrasound can be used to diagnose the thrombus or stenosis or obstruction with colour flow acceleration and loss of appreciation of S,D, AR and VR waves in severe SVCS.[13] MRI or contrast CT scanning may provide detailed anatomical information about SVC and surrounding structures and be useful in assessing the extent and nature of the obstruction.[13,14] Transthoracic or Trans esophageal echcardiography has a vital role in diagnosing the SVCS after congenital cardiac anomaly repair such as Glenn reversed flow, baffle obstruction in atrial switch operations etc. In this patient, the high index of suspicion due to mismatch of the SVC diameter and CVP line led to the conclusion that CVP line is the culprit for SVC syndrome and its removal resulted in tremendous improvement in the signs and symptoms of SVCS. [15]

Treatment modalities for SVCS involves anticoagulation in proven thrombosis, removal of the tumour in tumor compressed SVCS. Cardiac onset of SVCS may require anticoagulants or thrombectomy or surgical or interventional therapy. But acute bleeding in cardiac surgical infants can occur secondary to the anticoagulation therapy.[2] In neonates CVP line should be observed and removal may be implemented as a conservative management. In the presented patient removal of CVP line led to rapid resolution of sign and symptoms of SVCS. In post- cardiac surgery SVC stenosis; catheterization, stenting, balloon dilatation, and even surgical intervention with open heart surgery for thrombectomy, endovascular surgical repair may be used to treat the SVCS. [16,17] SVCS secondary to cardiac surgical procedures like Glenn procedure, Fontan repair or post PAPVC or even due to Senning procedure with

baffle obstruction induced SVCS require urgent revision of the cardiac procedure. The hemodynamic management should also be the priority as syndrome decreases the preload and so cardiac output. Thampi et al have reported an acute onset SVCS in a 40 days infant after previously bilateral IJV cannulation placement, presented with rapid severe face and neck swelling and cyanosis requiring emergent intubation to maintain the airway.[3] Following proper care and treatment of the SVCS, the symptoms resolve with in few days, but complete resolution may take even I month (median 2 days).[2]

Conclusion

SVC syndrome affects a small proportion of neonates, but its effects can be devastating. It has been reported after congenital cardiac surgical procedures, but not because of direct impact of PDA ligation. This single case report suggests that acute SVCS can develop in cardiac ICU immediately after the cardiac surgery in neonates. The mismatch of the size of SVC cannula and the IJV or SVC should be suspected the cause of SVCS after ductus ligation, and its removal causes a swift recovery from the signs and symptoms of SVCS not only the edema but respiratory distress and consciousness level also. We are of the opinion that cardiac related SVCS, have faster onset, more symptoms, and so delay in diagnosis of acute complications would result in an increased rate of the primary outcome. Use of anticoagulation therapy is not the first choice in such cases due to risk of fresh bleeding.

Declarations

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Conflict of interest: declare no conflict of interest

Ethical approval: Not required

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