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Case Report

Pericardial tamponade: Rare complication of subclavian vein cannulation

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Introduction

Most commonly reported complications during central venous catheter (CVC) insertion are cardiac arrhythmias and conduction blocks.^{1–3} Others include looping, knotting, fragmentation, embolization and rarely, intravascular entrapment of the wire.^{2–4}

Pericardial tamponade (PT) following internal jugular vein (IJV) or subclavian vein (SCV) cannulation have been rarely reported.^{4–7} In one case, the SCV was cannulated on the 10th day following peritonitis-induced septic shock with resultant needle-stick induced ascending aorta wall discontinuity.⁴ Eleven cases of PT have been reported following forceful threading of larger diameter (11–12/14–15 F diameter)

haemodialysis catheters over the guide wire.⁵ None mention difficulty with the passage of guide-wire.^{1,3–5,7}

We report a patient with rapid haemodynamic deterioration due to the guide-wire of a 7F CVC catheter in an otherwise healthy individual. The patient gave a written consent for publication without disclosing identity.

Case report

A 28-year-old, 60 kg, male patient, ASA 1 patient was scheduled for craniotomy and excision of right infra-tentorial meningioma in semi-lateral Janetta's position. Patient was anaesthetized, intubated and mechanically ventilated uneventfully, as per institutional protocol. Post-intubation, blood pressure (BP), heart rate (HR) and oxygen saturation (SpO₂) were normal at 110/70 mmHg, 82/min and 99%, respectively.

The right SCV was successfully punctured through the infra-clavicular approach, 1 cm inferior to the junctions of the middle and medial third of the clavicle following USS localization using the short axis view by a senior resident. However, the guide-wire of a 7Fr, Seldinger-type 15 cm, double-lumen CVC did not advance beyond 4 cm in spite of mild pressure and rotation of the needle. The needle was withdrawn and repositioned with the same skin puncture. Venous blood was freely aspirated but again slight resistance to guidewire insertion was met at 10 cm which was overcome with gentle pressure. Subsequent advancement was smooth. Dysrhythmias were not observed. The CVC was threaded

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uneventfully and after confirming non-pulsatile blood return in both lumens, it was sutured at 14 cm from the insertion site.

While positioning for surgery, the HR suddenly dropped to 50 beats/min with a BP of 60/40 mmHg, unresponsive to head repositioning. Inj Atropine 0.6 mg and Inj Mephentermine 12 mg IV in 3 mg aliquots was given. Ringer Lactate 1000 ml was rushed in. BP increased to 90/60 mmHg. Supraventricular tachycardia (HR 160–170/min) was seen on the monitor. Presuming the catheter tip was irritating the sino-atrial node (SA Node), the catheter was pulled out by 1 cm and resutured after confirming its intravascular position. After 10 min, the HR settled to 110–115/min but BP dropped again to 70/40 mmHg. Dopamine infusion was started at 5 mcg/kg/min. The HR increased to 130–135/min but BP did not rise more than 85/45 mmHg. Simultaneously, pneumothorax was ruled out clinically. A presumptive clinical diagnosis of PT was made in view of the transient response of hypotension to fluids, although the heart sounds were not muffled and there was no rise in Central Venous Pressure (CVP).

Urgent 2-D transthoracic echocardiography revealed a large pericardial effusion, measuring 20 mm all around in diastole with diastolic collapse of right ventricle (RV). Increased respiratory blood flow velocity variation across the tricuspid valve was demonstrated (Figs. 1 and 2). Percutaneous pericardiocentesis was performed under ultrasonographic guidance (USmart 3200T, Teratech Corporation, Burlington) through the left sternocostal margin. Approximately 80 mL of haemorrhagic fluid was aspirated, following which BP improved to 110/60–120/70 mmHg. The catheter was removed after observation for any re-collection of blood for 30 min. Surgery was abandoned and patient observed in the ICU for another 24 h for any re-accumulation of PT. Post-extubation chest roentgenogram was normal and a CT Chest did not reveal any breach in the walls of the great vessels. Vitals and serial 2D echocardiography remained normal. The patient underwent an uneventful craniotomy a week later.

Fig. 1 – Echocardiography of the patient, apical four chamber (A4C) view.

Fig. 2 – Echocardiography of the patient, subcostal view.

Discussion

Guide wire-related PT is usually due to perforation of the great vessels lying within the pericardial sac (ascending aorta, brachiocephalic and subclavian veins), the right atrium (RA), or, rarely, the right ventricle. The shorter dilator does not reach the vessels within the sac and cannot cause PT in adults.^{1,2} Time from CVC insertion to onset of typical clinical features varies from minutes to days^{1,3,6} with PT that results from ascending aorta perforation presenting acute and dramatic. Low pressure venous system perforations have an insidious onset, although few suggest that administering pressurized fluid infusion for the hypotension aggravates the problem.^{2,3,6,8} The classical Beck's triad (elevated CVP, hypotension, pulsus paradoxus) may not always be apparent. Awake patients may complain of chest pain (pericardium stretch through phrenic nerve), nausea (vagus nerve), and abdominal pain (splanchnic nerves).^{1,3} Diagnosis may be difficult under anaesthesia and a strong clinical suspicion is warranted. An emergent echocardiogram followed by pericardiocentesis can be life-saving in such situations. Other diagnoses with similar presentations such as tension pneumothorax and air embolism should be ruled out.^{1,6}

In this case, since the PT was moderate but rapid in onset, without recurrence or any evidence of vascular rupture, it is theorized that the guidewire caused a RA perforation. This may occur if the J-tip gets lodged longitudinally in the deep sub-eustachian sinus lying antero-inferior to the inferior vena caval orifice in the RA. In comparison to the coarse trabeculae present in the walls of the RA, the medial part of the right atrial appendage and the sinus are smooth-walled, non-uniform, with a minimal median thickness of 2 mm only (range 1–4 mm). Excessive pressure, especially if the wire is less malleable, may cause perforation. The fibrous Chiari network, existing across the cavity of the RA may direct the J-tip into the sinus.^{8,9}

To avoid puncture of the great veins, it is generally recommended that J-tip wires should be used rather than straight- or angle-tip wires. Also, force should never be applied

during insertion. Any resistance felt during guidewire insertion should prompt wire withdrawal by several millimetres and/or needle reinsertion.^{1,2,4–6} In the current case, mild pressure on the guidewire overcame the resistance. The clinician should be aware of this rare but potentially fatal complication of PT. To prevent this, it is recommended that the J-tip guidewire should not be inserted beyond the upper fold of the pericardial sac which lie just below the level of the carina.^{6,7} The sternal angle (also known as the angle of Louis or manubriosternal junction) should be used as guide for the maximum depth of insertion of the wire.¹⁰ This requires operator awareness as the guidewires are usually not calibrated or graded. It is also recommended that all CVC cannulations should be done under real time ultrasonographic guidance, if available. A timely diagnosis requires a high index of suspicion in the setting of unexplained hypotension in a recently placed or attempted CVC catheterization.

Conflicts of interest

The authors have none to declare.

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