Case Report

Bedside confirmation of a persistent left superior vena cava based on aberrantly positioned central venous catheter on chest radiograph

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This is a report of incidental diagnosis of a persistent left superior vena cava (PLSVC) based on an abnormal positioning of central venous catheter seen on chest radiograph and an abnormal pressure waveform. Non-invasive bedside tests included venography with simultaneous chest radiograph and a transthoracic echocardiography with an agitated saline microbubble contrast. These tests led to the diagnosis of PLSVC. Although PLSVC is the most common venous thoracic anomaly that produces a diagnostic dilemma, not many anaesthetists and intensivists are familiar with its appearance, diagnosis and implications. The clinical significance of PLSVC and diagnostic options are discussed.

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A 69-yr-old woman underwent an emergency laparotomy for intestinal perforation with faecal peritonitis. Intraoperatively the patient required i.v. infusion of inotropes. A left internal jugular venous catheter was inserted uneventfully after multiple unsuccessful attempts at cannulating the right internal jugular vein.

Postoperatively the patient required mechanical ventilation and i.v. infusion of inotropes and was transferred to the surgical intensive care unit. A chest radiograph taken after insertion of central venous catheter showed that the left internal jugular venous catheter followed a left para-mediastinal course instead of crossing the midline to the right to enter the superior vena cava; this would suggest an intra-arterial or extra-vascular placement (Fig. 1). The blood gas sample from the central venous catheter appeared venous and was distinctly different from the arterial sample, thus excluding an arterial placement. The transduced pressure wave showed an exaggerated central venous pressure waveform which could on postulation be attributable to the coronary sinus being so close to the right ventricle, or because of a hyperdynamic circulation (Fig. 2). This was not taken to be a diagnostic clue but rather an interesting observation as the transduced waveform would vary based on position of catheter tip.

Extravasation could not be excluded and there were two worrying issues. Firstly, when pressurized fluid was administered via the central venous catheter, the arterial pressure was noted to decrease dramatically but picked up when fluid was stopped or slowed down. In retrospect, this may have been because of a reduction in the coronary perfusion pressure as a result of an increase in coronary sinus pressure retarding coronary venous flow. Secondly, there was a decrease in postoperative haemoglobin from 11 to 7.9 g dl⁻¹ with worsening coagulopathy. There was concern whether this was a result of an initial vascular perforation and then re-entry into an anomalous or aberrant vessel thus causing oozing and bleeding at the perforation site, although worsening sepsis could be another likely reason. A repeat chest radiograph showed development of a new left pleural effusion raising the suspicion of infusate extravasation.

As we were unsure of the precise location of the central venous catheter, we inserted a new femoral venous catheter. We were wary of withdrawing the left internal jugular venous catheter as we had not excluded extravascular
placement and thus wanted to localize its position. A computed tomography (CT) of the thorax is the conventional method of investigation, but as our patient was unstable we opted for bedside tests which included a chest radiograph with simultaneous injection of contrast and a transthoracic echocardiography with an agitated saline microbubble test.

The chest radiograph with simultaneous injection of contrast is not routinely performed, and deserves elaboration.

Iodinated contrast 10 ml was gently injected into the left central venous catheter, with simultaneous exposure of a portable radiograph. This showed opacification of a tubular structure traversing a superolateral to inferomedial course. The position is characteristic for the course of the coronary sinus in the posterior atrioventricular groove (Fig. 3). There was no significant haemodynamic instability during or immediately after injection of contrast. Had the catheter tip not been in a vascular structure, contrast extravasation would have been obvious.

The transthoracic echocardiography showed a structurally normal heart with a dilated coronary sinus which was a clue to the existence of a persistent left superior vena cava (PLSVC). The tip of the central venous catheter was observed as a circular structure residing in the coronary sinus. Agitated saline infused via the central venous catheter showed initial opacification of the coronary sinus followed by the right atrium which helped in diagnosing that the PLSVC drained to the right atrium via the coronary sinus.

Discussion

In this case report, cannulation of the right internal jugular vein was attempted by a senior anaesthetist. On hindsight, ultrasound guidance for central venous catheter placement [as recommended in the National Institute for Clinical Excellence (NICE) report] would have helped in localizing the internal jugular vein for venepuncture or in this case would have alerted us to the absence of a patent vessel. This would have reduced the number of attempts and trauma to the patient. However, the course of the catheter once inserted in the vein cannot be seen subsequently.

Although PLSVC is a relatively rare vascular anomaly, it is the most common congenital anomaly of the thoracic venous circulation with a prevalence of 0.3–0.5% in healthy individuals and 1.3–4.5% in those with additional cardiac defects. The PLSVC may be present during early fetal life but usually becomes absorbed and attenuated during development. Persistence of the left anterior cardinal vein (which normally obliterates during embryogenesis) results in a PLSVC. Eighty-two percent of PLSVC co-exist with a right superior vena cava and so this anomaly is often missed as central line catheters are more commonly inserted on the right side. PLSVC itself does not cause any physiological derangements but may be associated with other congenital cardiac diseases (septal defects, Tetralogy of Fallot, situs inversus) that need to be excluded.

The PLSVC can drain either into the right atria via the coronary sinus or into the left atria. Ninety-two percent of PLSVC drain into the right atrium via the left portion of the sinus venosum or coronary sinus and are usually asymptomatic and do not provoke clinical signs and symptoms. They are often considered an anomaly of the coronary sinus. Eight percent of PLSVC drain into the left atria and cause a right-to-left shunt and may have unexplained cyanosis and
clubbing. This hazardous variant is often associated with other cardiac anomalies and has the risk of systemic embolization of air or thrombus when cannulated.\(^2\)

Although a PLSVC draining to the coronary sinus and then to the right atria is of minimal clinical significance, it has important clinical implications during cardiac operations. It may be a problem to keep blood out of the field during cardiopulmonary bypass and thus the PLSVC may need to be ligated or separately cannulated to avoid its venous return causing distention of the right heart.\(^3\) Also it represents a contraindication to retrograde cardioplegia.\(^4\)

Other problems include arrhythmias, cardiac arrest and coronary sinus thrombosis. The coronary sinus thrombosis is attributed to catheter manipulation in the coronary sinus but this risk is substantially reduced by ensuring that the tip of the catheter is situated just cranial to the junction of the PLSVC and the coronary sinus. In doing so, the catheter can be left in situ and used for a few days ensuring the same precautions as for ordinary central venous catheters.\(^3,4\)

PLSVC can cause difficult left-sided central line insertion of pulmonary artery catheters or pacing wire attributable to orientation.\(^3\) Also, the abnormal catheter position on postinsertion chest radiograph may be mistaken for arterial or extravascular placement as in this report. Of note, if the catheter tip was situated in a mediastinal vein or in a vessel outside the central venous circulation the characteristic central venous pressure waveform would be absent.

If one suspects a PLSVC, clinical diagnosis based on physical examination and assessment is important. Clinical signs include jugular venous distention on the left, abnormal jugular venous waveform caused by direct transmission of left atrial contraction, atrial pressures and non-arterial blood gas analysis.\(^5\) Signs suggestive of PLSVC on chest radiograph (though inconsistent) include: widening of the aortic shadow, a paramedial bulge along the left heart border or a crescentic vascular shadow projecting from the left upper border of the aortic arch to the middle one-third of the clavicle.\(^2,3\)

Once arterial placement is excluded on the basis of the pressure waveform and blood gas analysis, diagnostic tests to determine exact localization of aberrant catheter placement may need to be done. The conventional assessment for exact catheter localization is the contrast enhanced computer tomogram. However, other easily performed bedside diagnostic tests include a chest radiograph performed with simultaneous injection of contrast (venography without fluoroscopy).\(^3,6,7\)

The chest radiograph during simultaneous injection of iodinated contrast has been performed in children\(^6\) and adults\(^37\) to detect PLSVC. It is uncommon, but can be replicated fairly easily. As described, the test is performed at the bedside, with the use of a conventional portable X-ray unit. The critical element in obtaining a diagnostic radiograph is the timing of film exposure to coincide with injection of radiopaque contrast. Once opacified, the course of the coronary sinus is characteristic. An added advantage of the venogram technique is that extravasation of contrast into the mediastinum or pleural space indicates catheter malposition. Radiation exposure is equal to that of a routine portable radiograph. To minimize radiation exposure, the operator performing the injection should wear a lead gown and stand as far away as practically possible, and also ensure that no part of the operator’s body is in the direct line of the radiographic beam. The injection in this case was performed under the supervision of a radiologist. The contrast radiograph (Fig. 3) showed opacification of the PLSVC and the coronary sinus, and also helped to exclude extravasation.

Another convenient and safe diagnostic option is the transthoracic echocardiography with an agitated saline microbubble contrast which has the advantage of being non-invasive, with less radiation and causing less haemodynamic instability in a sick patient compared with contrast imaging.\(^1,2\) As in this report, it was a fast, non-invasive and accurate method to diagnose the PLSVC. The unusually large coronary sinus on a 2-dimensional view is a clue to the presence of a PLSVC and it can be confirmed by a contrast technique (agitated saline microbubble) which demonstrates drainage of contrast into the coronary sinus.\(^1,4\) There are commercially prepared ultrasound-agitated saline contrast agents available. These commercial preparations ensure that the modified bubbles last long enough to be visualized during the echocardiography. However, in this case report we used sterile normal saline and agitated it in a 20 ml syringe just before injecting it into the catheter with the patient in a slight Trendelenburg position to reduce right-to-left shunt.

In conclusion, an aberrantly positioned central venous catheter on chest radiograph can be a diagnostic dilemma. It is important for anaesthetists and intensivists to be aware of, and to consider the possible existence of a PLSVC and understand its clinical implications. In order not to misdiagnose it as an arterial or extravascular placement, a blood sample should be obtained from the catheter to establish a venous placement followed by a confirmatory test. Non-invasive bedside tests such as venography with simultaneous chest radiography or transthoracic echocardiography using the agitated saline bubble test can help determine the precise location of an abnormal central venous catheter and can help diagnose a PLSVC. These tests are important diagnostic options when the conventional CT thorax is not an option.

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