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CORRESPONDENCE

Cannulation of a persistent left superior vena cava or a pericardiophrenic vein?

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To the Editor,

We read with interest the recently published case report by Schreiber et al.,[1] and in our view, the diagnosis of a persistent left superior vena cava (PLSVC) is unclear.

The diagnosis appears to have been based on the features seen on the anterior chest *x-ray* and the findings at mini-sternotomy. The differential diagnosis of a cannula observed to be passing straight down from the left internal jugular vein to the mediastinum on an anterior chest *x-ray* would include both extravascular and intravascular placement with intravascular siting, including intra-arterial (aortic) and intravenous (left internal thoracic, pericardiophrenic, PLSVC, and left superior intercostal veins) siting. Features that may help differentiate between the various veins include the lateral arching of the superior intercostal vein near the aortic arch in the upper mediastinum before proceeding caudally, the lateral turning of the pericardiophrenic vein lower in the mediastinum along the left heart border, and the medial turning of the PLSVC near the left atrium. A simple way to demonstrate this is to repeat the anterior chest *x-ray* following the injection of intravenous contrast via the cannula to outline the vessel. Schummer recommends a lateral chest *x-ray* with injected contrast to help determine the position of the cannula where the lateral thoracic vein lies anteriorly passing to the chest wall, the pericardiophrenic vein and PLSVC lie centrally, while the superior intercostal vein lies more posterior.[2,3]

The mini-sternotomy did not define the lower limit of the cannulated vessel, but it did confirm its origin at the caudal junction of the left internal jugular vein and the left subclavian vein. However, all four of the abovementioned veins could arise from this site, with the pericardiophrenic vein and the PLSVC occupying similar positions in the upper mediastinum. Although the pericardiophrenic vein is usually a small vein in patients with portal hypertension (as is the case with liver transplant patients), it may be involved with gastroesophageal varices and portosystemic shunting causing enlargement.[4]

The authors reasoned that the hemodynamic instability with the institution of veno-venous bypass (VVB) and its reversibility on clamping the VVB circuit was due to blood loss into the left pleural cavity. However, blood loss sufficient to cause such a sudden change would presumably need volume resuscitation to restore hemodynamic stability. Furthermore, no overt damage to the cannulated vessel was observed, blood could be withdrawn easily from the cannula, and the patient was stable up to the institution of VVB. At the time, transesophageal echocardiography detected a large left pleural collection; however, the chest tube was not inserted until the completion of liver transplantation and only 1 L of dark blood was drained via the chest tube. Although it is true that a dilated coronary sinus may cause left ventricular inflow tract obstruction due to its proximity to the mitral valve, the coronary sinus was not dilated on the preoperative transthoracic echocardiography.

An alternative explanation could be that the cannulated vessel was a dilated pericardiophrenic vein involved in portosystemic shunting, and during VVB, blood would flow down this vessel in a retrograde manner and would not be returned to the heart. This would have caused an immediate drop in venous return, which would have been immediately reversible on clamping the VVB circuit. Blood in the left pleural cavity may have originated from several sources, including the contralateral diaphragm during surgery.

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Reply,

We thank Drs. Verniquet and Kakel for their thoughtful analysis of our case.[1] Their argument for other possible vessels that may have been cannulated in the left vascular tree only reinforces the assertion that one should avoid the left-sided insertion of a veno-venous bypass (VVB) cannula because of the potential sources of catheter malposition in this area.[2-4] The following two issues are raised; 1) whether the vein in this case was indeed the persistent left superior vena cava (PLSVC), and 2) whether the hemodynamic instability was indeed caused by exsanguination due to malposition of the VVB return cannula. They suggest an alternative explanation, i.e., the VVB cannula may have been inserted in the dilated left pericardiophrenic vein, and the hemodynamic instability occurred due to a simple recirculation of the VVB blood flow into the venous system, not due to bleeding into the left thoracic cavity.

First, in our view, the hemodynamic instability was due to exsanguination of the blood into the left thorax at the attempted VVB. One of the authors (B.C.B.) had examined the bilateral thoracic cavity with transesophageal echocardiography (TEE) prior to the VVB (it is a routine examination for VVB in our institution) and confirmed there had not been a fluid collection immediately preceding initiation of bypass. Upon attempting to initiate VVB, when the patient became hypotensive, a repeated TEE examination revealed the new fluid collection in the left chest cavity. Therefore, the two incidences were temporally associated. Of note, it is extremely unlikely that the surgical blood in the right upper abdomen where hepatectomy was performed would migrate into the left thoracic cavity. Second, it is unlikely that the VVB return cannula could be inserted in either the left internal thoracic vein (which runs anteriorly on the anterior mediastinum and along the retrosternum)[3] or the left superior intercostal vein (which runs sharply posteriorly at the level of the aortic arch to join the left hemiazygos vein)[4] since, during the surgical exploration, the VVB cannula was found in the vein which ran straight on the lateral surface of the mediastinum and toward the posterior aspect of the pericardium. We acknowledge that the definitive confirmation of PLSVC could have been possible with venography. This could have ruled out the possibility of placement in the pericardiophrenic vein, which might be abnormally dilated in portosystemic shunting and large enough to accommodate the 18 Fr. VVB return cannula as Drs. Verniquet and Kakel indicate. However, our findings (i.e., the development of the left-sided hemothorax upon initiation of VVB; subsequent relative hemodynamic stability upon termination of the VVB with fluid resuscitation; and a negative finding of "back-bleeding from the distal end" of the vein after ligation of the proximal part of the vessel during the surgical exploration) strongly suggest that the vein in question was the PLSVC with possible obstruction/severe stenosis at the junction of the left oblique cardiac vein.

Competing interests None declared.

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