Clinical Case Report

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Ultrasound-guided catheterization of the left subclavian vein without recognition of persistent left superior vena cava

A case report

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Abstract

Rationale: A persistent left superior vena cava (PLSVC) is rare, but the most common thoracic venous anomaly. We report a case of PLSVC unrecognized during left subclavian vein catheterization using real-time ultrasound-guided supraclavicular approach.

Patient concerns: A 79-year-old man with history of hypertension presented with traumatic subdural hemorrhage, subarachnoid hemorrhage, and epidural hemorrhage. Before the operation, a central venous catheter (CVC) was placed into the left subclavian vein.

Diagnoses: A dilated coronary sinus on echocardiogram and subsequent agitated saline test confirmed the diagnosis of PLSVC.

Interventions: A CVC was placed into the left subclavian vein under real-time ultrasound guide, with supraclavicular approach. A postoperative chest X-ray revealed a left-sided paramediastinal course of the CVC; the CVC was removed under the impression of malposition.

Outcomes: The CVC functioned properly.

Lessons: Ultrasound is used for safe and correct placement of CVC; however, the presence of PLSVC could not be detected by ultrasonography in this case. When the chest radiograph shows the central venous catheter passing along the border of the left heart and a dilated coronary sinus detected on echocardiogram, we should have suspicion of a PLSVC.

Abbreviations: CVC = central venous catheter, PLSVC = persistent left superior vena cava.

Keywords: echocardiography, superior, ultrasonography, vena cava

1. Introduction

A persistent left superior vena cava (PLSVC) is rare, but the most common thoracic venous anomaly. It has an incidence of 0.3% to 0.5% in the general population. In the presence of other congenital heart diseases, the incidence of PLSVC increases to 3% to 10%.[1–3] In most cases, PLSVC blood return drains into the right atrium through the coronary sinus vein. Therefore, it itself causes no hemodynamic disturbance, is generally asymptomatic and usually detected incidentally during cardiovascular imaging, cardiac catheterization or heart surgery, performed for unrelated reasons.[4–6]

For a safe and effective procedure, ultrasound-guided approach was recommended during central venous catheterization. However, PLSVC, the anatomical variant and unusual catheter position, could not be detected by ultrasonography. We report a case of PLSVC unrecognized during left subclavian vein catheterization using real-time ultrasound-guided supraclavicular approach.

2. Case report

A 79-year-old man with history of hypertension presented with traumatic subdural hemorrhage, subarachnoid hemorrhage, and epidural hemorrhage. Immediately after brain computed tomography, he was moved to the operating room for an emergency decompressive craniectomy and hematoma evacuation. Upon arrival at the operating room his vital signs were as follows: blood pressure 124/77 mm Hg, heart rate 100 beats/min, oxygen saturation 95%, respiratory rate 20 rates/min, and body temperature 36.9°C. His trachea was intubated with 8.0 mm endotracheal tube and mechanical ventilation was started with the pressure control mode, a peak inspiratory pressure of 15 cm H_2O, no positive end expiratory pressure, a respiratory rate of 13 rates/min, tidal volume of 420 to 450 mL, and FIO_2 of 1.0. Before the operation, a central venous catheter (CVC) (Blue FlexTip

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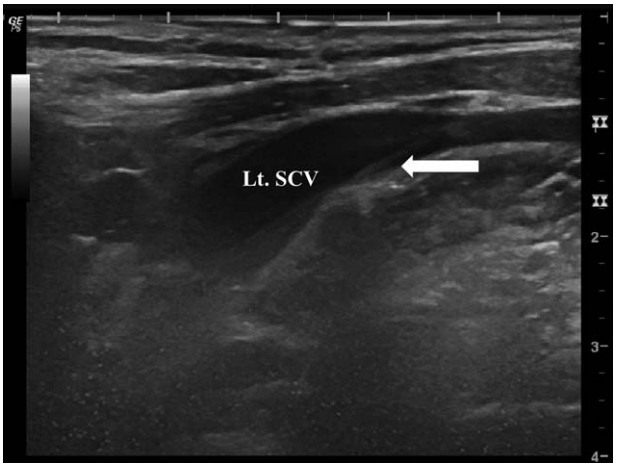

Figure 1. The ultrasonography of left subclavian vein. A needle (white arrow) inserted with supraclavicular approach. Lt. SVC = left subclavian vein.

ARROWg^{+}ard Blue, 14Ga, Arrow International Inc, PA) was placed into the left subclavian vein; the initial attempts on the right subclavian vein have been failed in the emergency department. The catheterization was performed under real-time ultrasound guide, with supraclavicular approach by an experienced attending anesthesiologist, uneventfully. The left subclavian vein and puncture needle inside the vein were well visualized with ultrasound (Fig. 1). We could aspirate blood easily through the catheter without pressure or pulsatile regurgitation. Central venous pressure was between 1 and 3 mm Hg with normal waveform and fluid infused freely through the catheter. The operation had finished within 3 hours. Total 8 units of packed RBC, colloid 1000 mL and crystalloid 4400 mL infused during the operation, and among them, packed RBC 2 unit, crystalloid 2700 mL, colloid 500 mL infused through the central venous catheter.

The patient was transferred to intensive care unit and maintained on controlled ventilation. The postoperative chest radiograph revealed the patch increased opacity in the right lower lung zone as preoperative examination and small amount of pleural effusion. Additionally, it showed central venous catheter passing straight along the border of the left heart and the tip in an unusual position (Fig. 2). Blood gas analysis (BGA) on the blood obtained from the catheter confirmed venous blood. The BGA was conducted in 37.0°C and revealed the following: pH 7.365, arterial CO_2 partial pressure ($PaCO_2$) 50.1 mm Hg, arterial O_2 partial pressure (PaO_2) 43.5 mm Hg, and base excess (BE) 2.6 mmol/L, and oxygen saturation 77.2%. However, we presumed the catheter malposition, so removed it and proceeded with femoral vein catheterization. The transthoracic echocardiogram, performed postoperatively by a cardiologist, revealed normal left ventricular systolic function, with no significant enlargement of the cardiac chambers. Additionally, the parasternal long axis view of transthoracic echocardiography showed a dilated coronary sinus (Fig. 3). There is no sign of elevated right atrial pressure or other reason for that. We considered the possibility of PLSVC, and to confirm the diagnosis, injected agitated saline contrast into the left antecubital vein. Following injection of contrast saline, echocardiography showed immediate opacification of the coronary sinus followed by subsequent opacification of the right atrium and the right ventricle (Fig. 4). Therefore, we could confirm the diagnosis of PLSVC without associated

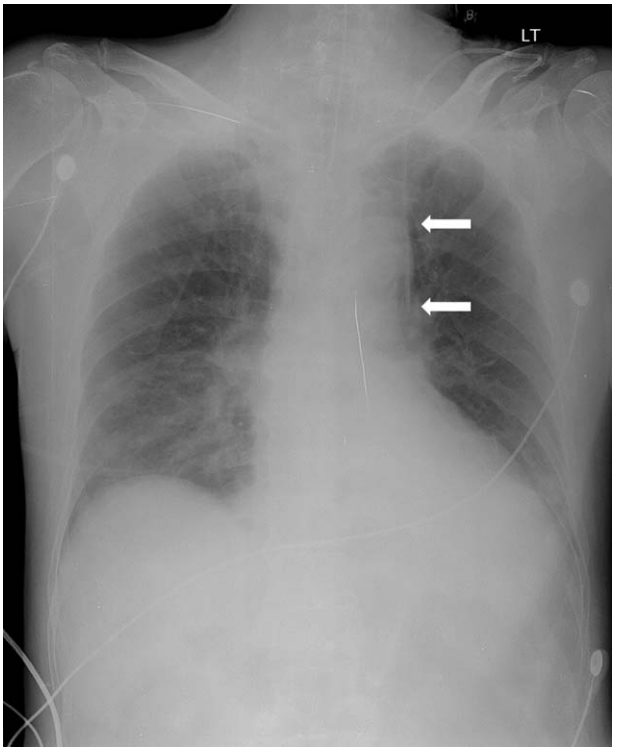

Figure 2. Postoperative chest radiograph revealed the central venous catheter (white arrows) passing along the border of the left heart and the tip in an unusual position.

cardiovascular anomaly. The patient expired at postoperative day 2 due to severe brain edema-induced cerebral infarction. The patient consent for a report could not be given.

3. Discussion

In this case, we used ultrasonography for left subclavian catheterization. The classical infraclavicular approach for subclavian catheterization causes a considerable number of malposition, most commonly misplacement in the ipsilateral

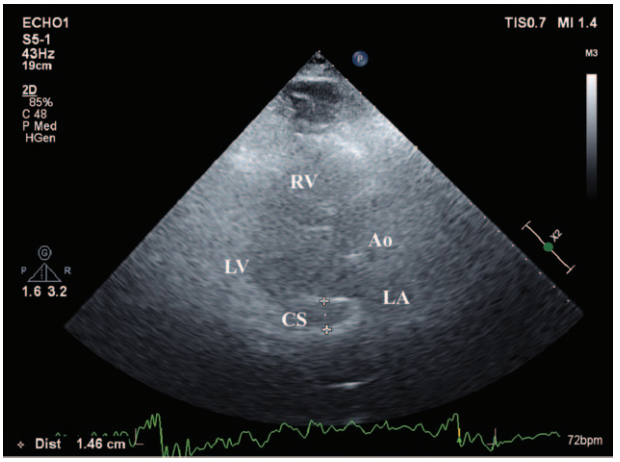

Figure 3. Parasternal long axis view of transthoracic echocardiography showed a dilated coronary sinus (1.46 cm). Ao = aorta, CS = coronary sinus, LA = left atrium, LV = left ventricle, RV = right ventricle.

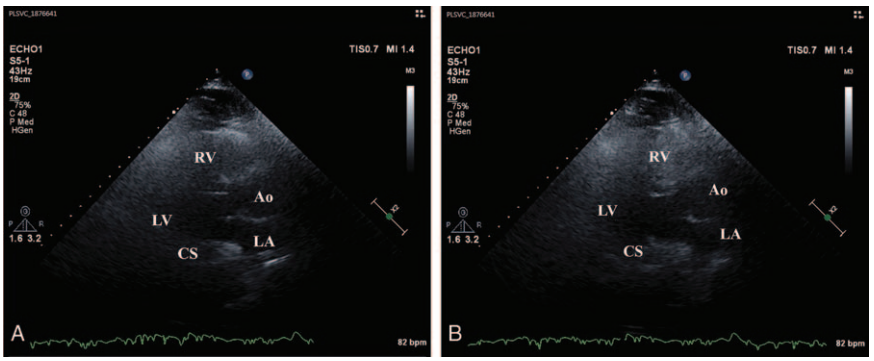

Figure 4. Following injection of contrast saline, echocardiography showed immediate opacification of the coronary sinus (A) followed by subsequent opacification of the right ventricle (B). Ao = aorta, CS = coronary sinus, LA = left atrium, LV = left ventricle, RV = right ventricle.

internal jugular vein.[7] Ultrasound-guided supraclavicular approach and confirmation may decrease the risk of misplacement. However, ultrasonography could not detect the PLSVC and could not prevent the abnormal placement of CVC.

PLSVC is a congenital venous anomaly. Normally, at the eighth week of gestation, the proximal part of the left cardinal vein regresses with development of the left innominate vein, and the right anterior and common cardinal veins become the right superior vena cava. The incomplete regression of the left cardinal vein results in the formation of a PLSVC.[8] In most cases, the PLSVC runs to the right atrium via an enlarged coronary sinus. Therefore, a PLSVC does not cause hemodynamic disturbance, and does not require any specific treatment. However, the presence of a PLSVC has several clinical implications. It can create difficulty during transvenous placement of pacemaker or implantable cardioverter-defibrillator leads, because the left subclavian approach is a common site of access. It may also complicate central venous and pulmonary artery catheterization via the left subclavian vein. More importantly, catheterization of the coronary sinus through a PLSVC has been associated with chest pain, collapse, and electrocardiographic changes consistent with myocardial ischemia.[9] In 1 series [10], the incidence of supraventricular tachycardia during catheterization was high in patients with left superior vena cava (38% vs 7.9% in patients with only the right superior vena cava). Other significant clinical consequences associated with PLSVC catheterization include venous stenosis, coronary sinus thrombosis, vascular erosion, cardiac tamponade, and cardiac arrest from coronary sinus irritation.[11–13] Additionally, the size of the 2 vena cava is complementary (the larger the left superior vena cava, the smaller the right), sometimes there is atresia of the right superior vena cava, and the congenital atrioventricular conduction defects and arrhythmias may occur in this setting.[3,4] These are the reasons why we should recognize this anomaly, even though it is generally considered that overall cardiac hemodynamics is not significantly affected. Also, there is a case report about the paradoxical hypotension during fluid resuscitation in a patient with PLSVC.[14] In that case, fluids were infused through CVC residing PLSVC. Rapid infusion of fluids under pressure induced the paradoxical hypotension and it resolved when fluids were infused slowly without pressure. The exact mechanism remains unknown, but they hypothesized that the high pressure infusion of fluids into the PLSVC caused rapid dilatation of the coronary sinus which encroached on the left atrium with resultant reduction in left ventricular preload. This reduction in preload together with elevated coronary venous pressure that arises from rapid and high pressured infusion may account for transient hypoperfusion and ischemia at the microvascular level. Although short-term or intermediate-term catheterization of the PLSVC variant with right atrial drainage is safe and effective, this could be another clinical consideration.

We suspected the presence of PLSVC according to the large coronary sinus without any sign of elevated right atrial pressure. PLSVC is the most frequent cause of enlargement of the coronary sinus.[2] Echocardiography with agitated saline (or other ultrasound contrast) can confirm the diagnosis of PLSVC. In this case, the diagnosis of PLSVC was also made by echocardiography with agitated saline. In normal condition, the diameter of coronary sinus is about 1 cm and injected contrast into left antecubital vein appeared at right atrium and right ventricle first. In PLSVC, the left side venous flow drain to coronary sinus, so echocardiography showed immediate opacification of the coronary sinus followed by subsequent opacification of the right atrium and right ventricle. Diagnosis of PLSVC by echocardiography has 100% specificity and 96% sensitivity.[2,5]

Clinicians should possess a thorough knowledge of not only normal venous anatomy, but also of relevant anatomic variation for safe practice. Although PLSVC is uncommon, we should not miss this anomaly. Misinterpretation of imaging in patients with PLSVC catheterization may lead to the incorrect diagnosis of catheter malposition, which may result in unnecessary subsequent intervention, as well as delaying initiation of necessary therapy, such as hemodialysis or intravenous administration of medication. In this case, we also misinterpreted malposition of CVC, even we confirmed vessel puncture with ultrasound image and excluded arterial catheterization through blood gas analysis.

When the chest radiograph shows the functioning central venous catheter passing along the border of the left heart and a dilated coronary sinus detected on echocardiogram, we should have suspicion of a PLSVC. It may be difficult to detect the PLSVC even during ultrasound-guided catheterization. And it could be confirmed by a simple diagnostic method using the echocardiography with agitated saline.

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