

American Journal of Clinical Case Reports

Case Report

Massive Pleural Effusion with Bedside Echocardiographic Findings Suggestive of Pulmonary Thromboembolism after Left Internal Jugular Venous Catheterization: A Case Report

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Abstract

Bed-side echocardiography is critical for the diagnosis and management of undifferentiated shock. We report a case of massive hydrothorax after left internal jugular catheterization with suspected echocardiographic findings of massive pulmonary thromboembolism such as flattening of the intraventricular septum into the left ventricle and right ventricular dilation.

Keywords: Internal jugular catheterization; Pulmonary thromboembolism; Hydrothorax; Pleural effusion

Introduction

The rapid diagnosis and timely management of patients who are admitted to the Emergency Department (ED) with undifferentiated shock are critical in improving patient outcomes and preventing adverse events. Point-of-Care Echocardiography (POCUS) is volutionary tool to assess hemodynamic conditions and identify the cause of hypotension, thus improving diagnostic certainty and guiding targeted treatment plans [1]. Bed-side echocardiographic findings suggestive of acute Pulmonary Thromboembolism (PTE) in critically ill patients with shock include flattening or bowing of the intraventricular septum into the Left Ventricle (LV), right ventricular dilation (>1:1 Right Ventricle [RV]/LV ratio), right ventricular systolic dysfunction, and inferior vena cava (IVC)dilation without inspiratory collapse or evidence of free-floating thrombus in the right heart or pulmonary artery or deep vein thrombosis on compression ultrasound of the lower extremities [2]. Hydrothorax is a rare but well-known complication of central venous catheterization. The development of hydrothorax is attributed to the extra vasation of the intravenous infusion fluid through the eroded walls of the superior vena cava or in nominate vein by the Central Venous Catheter (CVC). In the present report, we described a case of massive hydrothorax after internal jugular vein catheterization, showing right ventricular dilatation and a D-shaped LV on bed-side echocardiography and chest Computed Tomography (CT).

Citation: Song S-J, Chai H-S, Kim Y-M, Kim S-C, Lee J-H, Park G-J, et al. Massive Pleural Effusion with Bedside Echocardiographic Findings Suggestive of Pulmonary Thromboembolism after Left Internal Jugular Venous Catheterization: A Case Report. Am J Clin Case Rep. 2021;2(3):1030.

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Publisher Name: Medtext Publications LLC Manuscript compiled: Apr 05th, 2021

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Case Presentation

A 72-year-old woman underwent manual reduction and fixation fora left elbow fracture under general anesthesia in a local clinic. The surgical procedure was uneventful and the patient was admitted to the ward for postoperative care. Her previous medical history included hypertension, diabetes mellitus, and asthma. On the second postoperative day in the local hospital, she collapsed suddenly with unstable vitals. Therefore, a 7 French double-lumen CVC was inserted via the left internal jugular vein; however, her vital signs remained unstable. At this time, troponin T and pro-B-type natriuretic peptide were elevated. Hence, she was transferred to our ED. On presentation to the ED, she appeared critically ill with generalized pallor, perioral cyanosis, a heart rate of 133 beats per minute (bpm), a blood pressure of 80/60 mmHg, and an oxygen saturation of 100% on 15 L O, via a non rebreather mask. Her examination was significant for grunting, with otherwise clear bilateral breath sounds, rapid but regular heart beats, weak femoral pulses, and symmetric, non-edematous lower extremities. Shortly after arrival, bedside echocardiography was performed, demonstrating a D-shaped LV and right ventricular enlargement (Figure 1). An arterial blood gas revealed hypoxia and combined metabolic and respiratory acidosis with pO, 41 mmHg, pCO₂ 55 mmHg, and pH 7.092. Oxygen saturation was70%. Hence, we performed chest CT angiography to differentiate possible PTE. Chest CT showed a collapsed left lung and greater accumulation of fluid in the left pleural space with left internal jugular venous catheter located in the left upper lung, without evidence of thrombus in the pulmonary artery (Figure 2). We also confirmed massive pleural effusion with bedside lung ultrasound and portable chest radiography, and we drained the pleural effusion by inserting a 12 French trocar (ultrasound-guided) along with removal of the internal jugular vein cannula. Up to 1200 cc of clear fluid was drained from the chest tube. Following this, the patient's condition improved with marked relief of respiratory distress and improvement in oxygenation.

Discussion

The unique feature of the present case is that the patient developed massive pleural effusion after left internal jugular catheterization and showed classical findings mimicking massive PTE on bed-side

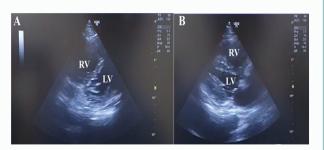


Figure 1: Point-of-care echocardiogram in a case of suspected pulmonary thromboembolism demonstrates the flattening of the interventricular septum (D-shaped left ventricle) and right ventricle enlargement on a parasternal short axis view (A) and parasternal long axis view (B). LV: Left Ventricle; RV: Right Ventricle

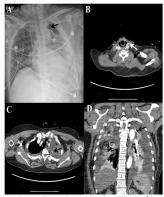


Figure 2: Internal jugular vein catheter malposition. Chest X-ray (A) shows the tip of the line at the left lung (arrow) with a large amount of left pleural effusion. Contrast chest computed tomography scan demonstrated the tip of the left internal jugular catheter placed in the left pleural cavity (B and C, arrows) with bilateral pleural effusion (B–D).

echocardiography. The diagnosis and initial management of patients with hypotension in the ED must be prompt and accurate in order to improve patient outcomes. POCUS is typically devoted to answering very specific clinical questions with respect to a particular differential diagnosis, with a rapid bedside structural assessment of the heart, while using the most efficient and minimal echocardiographic views and techniques in critically ill patients [3]. In particular, it has been widely used in difficult cases to assess hemodynamic instability and exclude its possible causes in patients with undifferentiated shock in ED [1,4]. There are multiple bed-side echocardiographic findings that support the diagnosis of acute PTE, which can be classified into direct and indirect signs. Direct signs included visualization of a free-floating thrombus in the right heart or pulmonary artery [2,5]. Indirect signs include right ventricular dilation (RV/LV ratio >0.6-1:1), flattening or bowing of the intraventricular septum into the LV, right ventricular systolic dysfunction, McConnell's sign, and IVC dilation without inspiratory collapse [5,6]. Our patient had all these direct signs present on echocardiography, including flattening/ bowing of the intraventricular septum into the LV, right ventricular dilation (>1:1 RV/LV ratio), right ventricular systolic dysfunction, and IVC dilation without inspiratory collapse, as well as other unstable signs such as chest pain, dyspnea, and hypotension. Therefore, we considered massive PTE as the first differential diagnosis and performed an urgent chest CT. On chest CT, we identified malposition of the internal jugular vein catheter and a massive pleural effusion. The CT scan revealed an incorrectly placed CVC outside the jugular vein. The tip of the catheter had followed an abnormal path, passing beside the esophagus and trachea and ultimately entering the left pleural cavity—with direct consequences such as pneumothorax and hydrothorax. Central venous lines represent an imperative tool in the ED for unstable patients to maintain fluids, nutrients, and drugs and to monitor the volume status. However, complications of central venous catheterization are well-known; the immediate complications are pneumothorax, hemothorax, or arterial puncture. Hydrothorax caused by malposition of CVC is rare but could possibly be very serious. Catheterization via the internal jugular vein may result in fewer cases of malposition than catheterization via the subclavian vein [7]. In general, catheterizations via the left internal jugular vein result in more malpositions and vascular perforations than catheterizations via the right internal jugular vein, as the right internal jugular vein runs into the right brachiocephalic vein with a fairly straight course, whereas the left internal jugular vein forms a greater bend when it becomes the left brachiocephalic vein [7]. Reports of ipsilateral pleural effusion following misplaced CVSs are not unusual. However, a small amount of right hydrothorax was also identified along with massive left hydrothorax on chest CT in our case. The most likely explanation of this bilateral effusion is the passage of normal saline from the displaced catheter into the mediastinum and then into both the pleural cavities via anatomical communications. However, only a few similar cases of bilateral pleural effusion following central venous catheterization could be found in the literature. This is a rare complication that is yet to be satisfactorily explained. In conclusion, we report the first case of acute pleural effusion after left internal jugular vein catheterization mimicking PTE on echocardiography. Our findings suggest that in cases of suspected PTE on echocardiography, acute pleural effusion should be included in the differential diagnosis and bed-side pulmonary ultrasound should be performed.

Acknowledgements

This research was supported by the Basic Science Research Program through the National Research Foundation of Korea funded by the Ministry of Education, Science and Technology (2015R1A2A2A01002303& NRF-2016R1D1A1B04930247).

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