Brain abscess with persistent left superior vena cava

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ABSTRACT

إن بقاء الوريد الأجوف العلوي الأيسر (PLSVC) هو الشذوذ القلبي الولادي و الأكثر شيوعاً إذ يحدث في حوالي 0.4% من عامة الناس، ويشاهد كذلك بنسبة 1.3% – 4.5 في المرضى الذين يعانون من شذوذ قلبية إضافية. هناك تواجد مرافق في 82% من حالات بقاء الوريد الأجوف العلوي الأيسر (PLSVC) غير مترافق بأعراض، لكنه قد يسبب مصاعب مهمة خلال وضع القثاطر الوريدية المركزية من الجانب الأيسر. سنستعرض في هذه المقالة حالة مريض مصاب بخراج دماغي مع بقاء الوريد الأجوف العلوي الأيسر (PLSVC). تم تشخيص الحالة في قسم العناية المركزة (ICU) بمستشفى الملك فيصل –العاصمة المقدسة. ذكرت حالات قلبية قليلة في الأدب الطبي مشابهة للحالة المذكورة (PLSVC).

Persistent left superior vena cava (PLSVC) is the most common congenital cardiac anomaly occurring in around 0.4% of the general population, and 1.3-4.5% in those with additional cardiac defects. Eighty-two percent of PLSVC coexists with a right superior vena cava. Usually PLSVC is asymptomatic, but can cause difficulties during central venous cannulation from the left side. There are a few cases of brain abscess in the literature associated with PLSVC. Here, we describe a patient of brain abscess with PLSVC in the intensive care unit of King Faisal Hospital, Makkah, Kingdom of Saudi Arabia.

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Persistent left superior vena cava (PLSVC) is the most common congenital anomaly of the systemic veins of the thorax with a prevalence ranging from 0.3-2% in the general population,[1] and a higher prevalence of 4.4% among those with congenital heart disease.[2] The PLSVC drains into the coronary sinus and right atrium in around 80-90% of cases,[3] and these patients are usually asymptomatic. Rarely, in 10% of the patients,[4] the PLSVC will drain into the left atrium and is associated with an increased risk of cyanosis, heart failure, intracerebral abscess, and embolic cerebrovascular stroke. Here we report a case of brain abscess associated with PLSVC. Our objective is to make physicians aware of PLSVC, which is an anomaly to remember that can present sometimes with multiple brain abscesses. It is usually asymptomatic and associated with other anomalies. An abnormal course of central catheter from the left side should lead to suspicion.

Case Report. A 40-year-old male patient was admitted to our intensive care unit (ICU) with a history of fever and unconsciousness, with no history of trauma. His vital signs were as follows: temperature 39.5°C, pulse 100/min, blood pressure 90/55 mm Hg, and respiration 18/minute. Physical examination showed finger clubbing. There was no cyanosis. No lymph nodes were palpable. Cardiovascular examination was as follows: S1, S2 present, with sinus tachycardia, no gallop rhythm, and no murmur. Respiratory examination was as follows: bilateral equal air entry, and normal vesicular breath sounds with no added sounds. His abdomen was soft, with no palpable lymph nodes or organomegaly. The CNS examination showed hyporeflexia in all limbs, hypotonia, absent plantar reflex, pupils were bilaterally equal and reacting to light. An ECG showed only sinus tachycardia. Ophthalmic and funduscopic examinations were normal. There was no evidence of papilledema. An urgent brain CT scan was carried out which showed multiple brain abscesses. There was a right frontoparietal space-occupying lesion measuring 3x5x3.5 cm, which showed ring enhancement after contrast, suggestive of brain abscess, and another in the midline measuring 1.5x2 cm (**Figure 1**). Extensive perifocal edema with a midline shift was evident. There was effacement of ipsilateral ventricle and dilatation of contralateral ventricle (**Figure 2**). Laboratory investigations were as follows: white blood count -14,000/cmm, hemoglobin - 9.9 g/dL, platelets - 59,000/cmm, random blood sugar

- 115 mg/dl, urea - 26 mg/dl, creatinine - 1.8 mg/dl, sodium - 160 mEq/L, potassium - 3.9 mEq/L, aspartate aminotransferase -159 IU/L, alanine aminotransferase - 63 IU/L, alkaline phosphatase - 84 IU/L, creatinine kinase - 709 units/L, calcium - 2.2 mg/dl, uric acid - 2.7 mg/dl, and erythrocyte sedimentation rate - 70 mm at the end of one hour. Low platelet count was due to sepsis. Coagulation profile was within normal range (prothrombin time - 10 sec, partial thromboplastin time - 25sec, INR - 1.2), which ruled out disseminated intravascular coagulation. C-reactive protein was positive (titre not available), due to infective process. Arterial blood gases showed a Ph of 7.305, oxygen pressure of 58.3 mm Hg, carbon dioxide concentration of 38.1 mm Hg, HCO_3 of 18.5 mmol/L, and O_2 saturation of 87.7%. Metabolic acidosis in arterial blood gases was due to lactic acidosis, because of persistent shock. Urine and sputum cultures were negative. Serology was found to be hepatitis B surface antigen positive, HIV negative, and negative titre for toxoplasmosis. Additional results showed that the ultrasound of the abdomen was normal. Lumbar puncture could not be carried out as he was critical and brain CT scan showed cerebral edema. He was scheduled for echocardiography, but this could not be carried out. He was hypotensive, and he required a central venous line and mechanical ventilation. A left subclavian central venous catheter was inserted. The post procedure chest x-ray showed an unusual route of the catheter to the left atrium along the left para mediastinum (**Figure 3**). Blood gas from that line proved that it was not

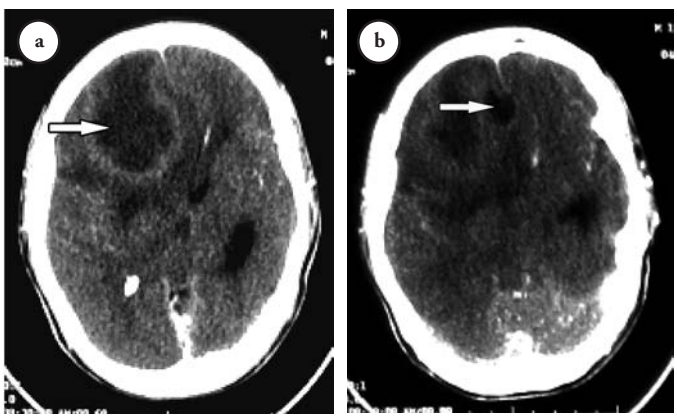

Figure 1 - Brain CT with contrast revealing **a**) right frontoparietal space occupying lesion measuring 3×5×3.5 cm, showing ring enhancement after the contrast, suggestive of brain abscess (plain arrow). **b**) Also showing another smaller lesion in the midline measuring 1.5×2 cm.

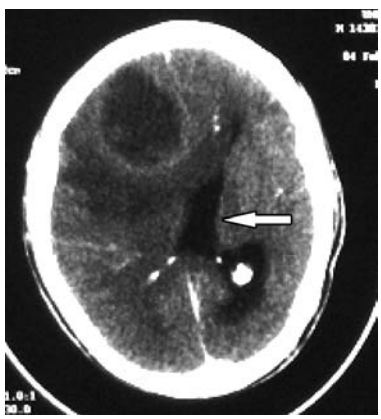

Figure 2 - Brain CT with contrast showing extensive perifocal edema with midline shift. There is effacement of ipsilateral ventricle and dilatation of contralateral ventricle (arrow).

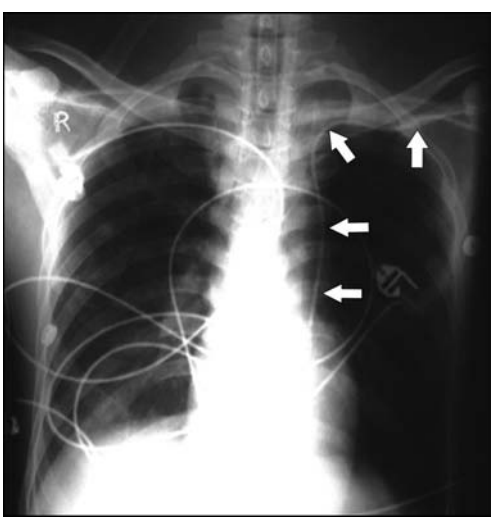

Figure 3 - A chest x-ray showing an unusual route of the left subclavian central catheter to left atrium along the left paramediastinum (arrows).

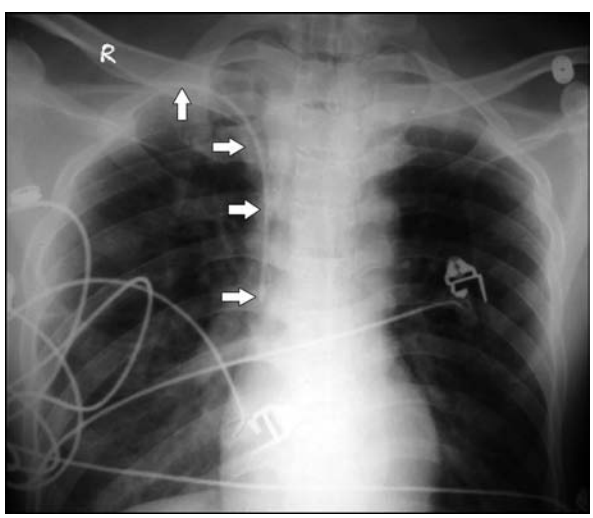

Figure 4 - A chest x-ray of the same patient showing right subclavian central line revealing the normal route going towards the right atrium through the right-sided superior vena cava (arrows).

an arterial insertion. A right subclavian central line was inserted, which showed a normal pathway going towards the right atrium through the right-sided superior vena cava (**Figure** 4). A diagnosis of PLSVC with a right superior vena cava was then made. He was persistently hypoxic and hypotensive, and required maximum doses of inotropic support and was given higher antibiotics. He was critically ill and was unfit to be moved to another hospital for the angiographic studies. Finally, he went into irreversible shock and died.

In the literature, only a few cases of brain abscess have been described associated with PLSVC.[5,6] Hence, we concluded that these multiple brain abscesses were associated with PLSVC possibly draining into the left atrium, causing shunting of blood between the right and left sides giving rise to intracerebral abscess. In the differential diagnosis, consideration should also be given to a coronary arteriovenous fistula, abnormal drainage of hepatic veins, and patent foramen ovale.

Discussion. A PLSVC is the most common congenital anomaly involving central venous return in the thorax. Anatomically it represents the counterpart of right SVC, formed by the union of the left internal jugular vein and left subclavian vein. Normally it disappears in the embryological stage.[7] Its incidence has been reported to be 0.3-0.5% in normal individuals, and around 3-10% in patients with other congenital heart anomalies. It remains asymptomatic in the majority of patients until they reach adulthood. It is most commonly detected coincidentally on a chest x-ray, following central venous catheter insertion through the left subclavian vein, where the catheter is seen to take an unusual route. The PLSVC usually drains the systemic venous blood from the left upper half of the body into the coronary sinus and right atrium.[7,8] However, in some it drains into the left atrium and is associated with increased risk of cyanosis, heart failure, intracerebral abscess due to shunting of blood between the right and left side giving rise to multiple brain abscesses. The anomaly develops in the eighth week of gestation as the main venous drainage system of the embryo's body develops. Paired anterior cardinal veins drain the cranial portions of the body, while the caudal portions of the body are drained by the paired posterior cardinal veins. The right anterior and posterior cardinal veins and the left anterior and posterior cardinal veins drain into the right and left common cardinal veins. At the eighth week of gestation, the innominate vein connects the left and right anterior cardinal veins. The internal jugular veins develop from the anterior cardinal veins cephalic to the innominate vein. Caudal to the innominate vein, the right anterior cardinal vein joins with a portion of the right common cardinal vein to form the normal right-sided superior vena cava. The left anterior cardinal vein, caudal to the innominate cardinal vein, normally regresses to form the ligament of Marshall. Failure of the left anterior cardinal vein to regress caudal to the innominate vein results in the development of a PLSVC. A normal right superior vena cava is present in 90% of cases where a PLSVC is present.

In conclusion, PLSVC is an anomaly to remember, which can present sometimes with multiple brain abscesses. It is usually asymptomatic and associated with other anomalies. An abnormal course of central catheter from the left side should raise suspicion.

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