**Persistent Left Superior Vena Cava – Accidental Finding**

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**Abstract.** Background/Aim: The presence of the superior left vena cava represents a rare anomaly of the thoracic venous system. Case Report: An asymptomatic case of this type of anomaly, discovered as an accident during investigations for a different pathology (superior left pulmonary lobe tumor), is presented. A 56-year-old, heavy smoker was admitted in our clinic with a tumoral mass in the left superior pulmonary lobe discovered during a routine chest x-ray. Physical and clinical examination was normal. However, transthoracic echography noted a coronary sinus enlargement, which led to the suspicion of a thoracic venous anomaly. Contrast chest computed tomography pointed out a venous anomaly at the level of the left hemithorax originating from the cervical region, crossing the aortic arch and draining in the coronary sinus. During the examination, contrast substance was not detected in the right superior vena cava, either early or late during the computed tomography. During surgery the presence of a persistent left superior vena cava was observed, coming from the cervical region, crossing lateral to the aortic arch and draining in the coronary sinus. Conclusion: The presence of an enlarged coronary sinus should warn the surgeon about the possibility of a thoracic venous anomaly. Identifying a persistent left superior vena cava is important due to its clinical implications, especially during certain procedures such as mounting central venous lines, cardiac cannulation or implantation of cardiac stimulators.

The persistence of the left superior vena cava (PLSVC) was noted even before 1787 (1), but the first case was described by Marshall in 1850 (2). Starting from 1975, a number of authors (3) have reported the presence of a persistent superior left vena cava in 2.1%-4.3% of patients which also had another congenital cardiac disease (2, 3). Even so, the persistence of the left superior vena cava is a less common vascular anomaly while still being the most frequent thoracic venous anomaly. It is found in 0.3-0.5% of cases, is usually asymptomatic and has no hemodynamic implications. In most cases, persistent left superior vena cava (80-90%) coexists with the right vena cava; however, in 0.09-0.13% of cases PLSVC exists in the absence of the right superior vena cava (4). In our case the venous anomaly was discovered accidentally, the patient presented no signs or symptoms, which could suggest this diagnosis.

In this presentation we will point out the clinical implications of this type of venous anomaly, as well as detailing the diagnostic approach and a perspective into the embryologic modifications which lead to this type of anomaly.

**Case Report**

A 56-year-old patient was addressed to the Department of Thoracic Surgery, “Marius Nasta” National Institute of Pneumopathology for persistent cough and minimal hemoptysis, weight loss (5 kg/30 days), loss of appetite and diffuse thoracic pain. Patient history revealed a surgically treated groin hernia as well as a previous history of heavy smoking.
smoking (-20 pack year). The blood tests revealed normal parameters except an elevated level of fibrinogen as well as an elevated erythrocyte sedimentation rate. The patient was submitted to an anterior chest x-ray which identified a slightly enlarged pulmonary hilum, a ¾ cm homogenous opacity with no clear margins situated in the subclavian area of the left upper lobe and a bilateral basal bronchiectasis; no other pathological finding was observed.

We went further with the investigation plan and we submitted the patient to bronchoscopy which revealed bilateral bronchiitis with no endobronchial proliferative elements; no new information towards the diagnosis was obtained. The thoracic and abdominal CT scan revealed the presence of a 3.5 cm/4.7 cm mass in the left superior upper lobe with poorly defined margins, in close contact and with possible infiltration of the parietal pleura. No hilar or mediastinal lymph nodes were observed; the abdominal computed tomography excluded the presence of any tumors at the level of the liver or adrenal glands. In the visceral compartment of the mediastinum a vascular structure was observed, with early filling of contrast which appeared to have a point of origin in the upper thoracic region, from the joining of the internal jugular vein with the left subclavian vein. It should be mentioned that the contrast media was injected through a peripheral vein on the left arm. This large venous structure had a caudal orientation, lateral to the left subclavian artery, going across the aortic arch and draining into the coronary sinus. Further computed tomography images revealed no contrast media in the venous brachiocephalic trunk in association with the presence of the superior vena cava (right), but also with no contrast media reaching it. Therefore, the computed tomography raised the suspicion of the persistence of the left superior vena cava as well as the absence of the venous brachiocephalic trunk (Figure 1).

The patient was further submitted to transthoracic echocardiography which showed an ejection fraction of 55%, preserved function of the left ventricle, normal pressures in the right atrium and a dilated coronary sinus which also pointed to a venous anomaly.

Surgery was performed under general anesthesia with selective intubation. Thoracic access was gained through the 5th left intercostal space by lateral thoracotomy. After opening the thoracic cavity, we observed the presence of the persistent left superior vena cava, entering the thorax through the apex, lateral to the left subclavian artery, descending towards the heart and crossing lateral of the aortic arch. At this level the PLSVC also drained another important venous structure – the left azygos – coming in from the posterior wall and entering the left vena cava near the aortic arch. It continued descending anteriorly to the left pulmonary artery and the left superior pulmonary vein, draining into the coronary sinus (Figure 2). Surgery consisted in left upper lobectomy with mediastinal lymphadenectomy. The histopathological examination confirmed the presence of a pulmonary adenocarcinoma with no mediastinal or hilar lymph node metastasis. The early postoperative evolution was uneventful, the patient being discharged in the seventh postoperative day.

**Discussion**

Persistence of the left superior vena cava (PLSVC) can present in two forms depending on where the vessel drains into the heart: in up to 90% of cases it drains into the coronary sinus, while in up to 10% of cases it drains directly into the left atrium or one of the pulmonary veins (5, 6), in which case a right-to-left shunt may appear. When the venous drainage occurs directly into the coronary sinus, the PLSVC opens directly into the atrium, in close proximity to the insertion of the atrioventricular valves (7). In 82-90% of cases PLSVC appears as a duplicated superior vena cava (left and right). In this situation, the innominate vein connecting them is present in 30% of cases. In most typical cases, in the absence of other cardiovascular malformations, PLSVC does not associate with clinical implications or hemodynamic modifications (8). It has been reported (9) that this type of venous anomaly is more frequently associated with other cardiovascular anomalies such as: pulmonary veins anomalies (in 10% of cases), pulmonary atresia (in 6% of cases), tricuspid atresia (in 4-5% of cases), hypoplasia of the left ventricle (in 2.5% of cases) or Fallot tetralogy (in 2% of cases). Among these, arterio-venous fistulas have been described between the left circumflex coronary artery and the great cardiac vein which then drains into the persistent left superior vena cava (10). Others have reported the association of PLSVC with May-Thurner syndrome (consisting of compression of the right common iliac vein between the right common iliac artery and the lombar vertebrae) (11).

Isolated or doubled PLSVC (right superior vena cava) may be associated with cardiac defects, such as inter-atrial septal defect; in such cases a right-to-left shunt appears with hemodynamic implications (12). The association between PLSVC and aberrant venous drainages has also been reported, consisting of right subclavian vein draining into the left superior vena cava (13). PLSVC can also be associated with left subclavian vein stenosis and absence of the right superior vena cava (14). Moreover, cases in which PLSVC drains into the left superior pulmonary vein and the presence of usual signs of a right-to-left shunt have been also reported (15).

PLSVC has also been described in association with the absence of the hepatic part of the inferior vena cava (16) and also as part of the Raghib syndrome (PLSVC in association with coronary sinus atresia and atrial septal defect) (17). In
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Figure 1. Contrast CT view. SVC: Superior vena cava; PLSVC: persistent left superior vena cava; IVC: inferior vena cava; CS: coronary sinus.
other cases, different cardiac arrhythmias may appear along with PLSVC, such as: WPW syndrome or sick sinus syndrome and even sudden death (18). In rare cases, PLSVC is present in complete situs inversus or cardiac dextroposition in association with the absence of the right superior vena cava.

The normal development of the superior vena cava during fetal growth starts in the 4th week of pregnancy, when the anterior and posterior cardinal veins open into the venous sinus through the common cardinal veins. In the 7th week of pregnancy, communication between the anterior left and right cardinal veins leads to the anastomosis of the two segments. This anastomosis will then become the innominate venous trunk. The right superior vena cava forms from the right precardinal and the right common cardinal veins. On the left, the distal portion of the left precardinal vein, beyond the innominate vein anastomosis, atrophies and becomes the Marshalls’ ligament, while the cardinal common vein becomes Marshalls’ vein – collecting blood from the posterior part of the heart and draining it into the coronary sinus. The left innominate vein forms from the left precardinal vein (superior part) and provides the venous drainage towards the right superior vena cava (19). The left precardinal vein later becomes the internal left jugular and left subclavian veins.

PLSVC appears when there are anomalies in the development or anastomosis between the superior precardinal venous systems. Absence or underdevelopment of the innominate vein determines drainage persistence of the left precordial vein into the left common cardinal vein. The left internal jugular and the left subclavian veins drain venous blood through the PLSVC directly into Marshalls’ vein and from there into the coronary sinus, which opens into the right atrium.

Information regarding the embryologic development of PLSVC, either on its own or associated with other cardiac malformations is scarce. A study published in 1991 on 351 normally developed human embryos, followed according to the Carnegie development scale, compared the malformation aspects to 1208 human embryos with congenital malformations from the collection of embryological pathology of John Hopkins Hospital (20).

The Carnegie system of embryological development has 23 stages of evolution. According to this, the conclusion was that

Figure 2. Intraoperative view of PLSCV. PLSCV: Persistent left superior vena cava; LAV: left azygos vein.
in normally developed human embryos a symmetrical venous system appears in association with the cardiac tube during the Carnegie 9 stage. The relation between the left superior vena cava and the coronary sinus was established in the 12th stage. The functional left superior vena cava is present in the 20th stage, but its lumen is later obliterated by compression between the left atrium and pulmonary hilum. Of the 1208 embryos with congenital anomalies included in that study, 9% presented PLSVC. There were also frequent associations of PLSVC with atrio-ventricular canal defects, cor triatrium and mitral atresia. The late stage of embryologic development, when the left superior vena cava has its lumen obliterated, suggests its persistence is secondary to cardiac congestion, contributing to blood flow redistribution in a later stage (20).

Several authors have made multiple classifications of cases with this type of venous anomaly (PLSVC). An anatomical classification has been developed regarding the presence or absence of the azygos vein and the innominate anastomosis. Four types were described: the first type is represented by cases presenting an anastomosis between the left and right superior vena cava through the innominate venous trunk; the second type refers to cases presenting completely separated left and right superior vena cava (21); the third type refers to the absence or atrophy of the right superior vena cava, blood drainage being realized through the left superior vena cava, while the fourth case refers to separated left and right superior vena cava, each one presenting its own correspondent azygos vein (21).

Paraclinical examinations (thoracic contrast computed tomography, transthoracic echocardiography), as well as obvious intraoperative presentation, led us to consider our patient as being placed in the 4th category of the previously described classification. We confirmed the absence of the venous anastomosis represented by the innominate trunk due to the fact that the contrast medium injected into the left arm during the computed tomography did not reach the right superior vena cava, instead descending towards the heart through the persistent left vena cava. We also observed the presence of the right azygos vein (on the preoperative computed tomography) as well as of the left azygos vein (intraoperatively). However, transthoracic echocardiography did not identify increased atrial pressure, while computed tomography confirmed the venous drainage into the right atrium.

These data enabled us to conclude that there was no atrial right-to-left shunt, also confirmed by the patients’ clinical presentation with no hemodynamic anomalies.

Although rare, PLSVC may present severe associated malformations, requiring further cardiologic investigations (such as transthoracic contrast echocardiography, magnetic resonance imaging or cardiac computed tomography with contrast solution) (22). Transthoracic echocardiography with intravenous saline contrast solution can better indicate the blood flow through the coronary sinus via the left superior vena cava through the presence of microbulae (23). Some authors consider trans-esophageal echocardiography associated with contrast saline solution as being superior to standard transthoracic echocardiography (24). Recent studies have suggested that magnetic resonance imaging is the best means of investigation in the absence of a positive diagnosis, being significantly superior to both transthoracic and trans-esophageal echocardiography. Magnetic resonance imaging can offer a better view of a much wider field, while using gadolinium contrast makes it far superior to other investigations. Thoracic contrast computed tomography scan can also be useful; however, it should not be omitted that it is an invasive method due to radiation exposure, while iodine contrast solution has its’ own limitations.

PLSVC is a very important condition in cardiac surgery, being an absolute contraindication for retrograde cardioplegia (25). Furthermore, PLSVC in association with other cardiac malformations may increase postoperative mortality in children who undergo cardiac surgery, making its identification crucial in preventing intra and postoperative complications (26). Cannulation of PLSVC during surgery depends on several factors: the presence or absence of the innominate vein (between the two vena cava), the absence of the right superior vena cava, its caliber, blood flow through the left superior vena cava as well as the planned surgical procedure (27).

The presence of PLSVC may have important surgical implications, especially during cardiac transplantation, when cardiopulmonary bypass is required (28). Several authors consider that PLSVC may be associated with several cardiac arrhythmias, present in 36% of cases (29). Several mechanisms responsible for arrhythmia development in patients are represented by: possible histological modifications in the atrioventricular sinus, existence of multiple electric nodes between PLSVC, coronary sinus and right atrium (30), electric generating capacity of the PLSVC which may lead to tachyarrhythmia; interatrial conduction delay or atrial arrhythmia secondary to coronary sinus dilation (31).

The presence of PLSVC is important when the left subclavian vein is used for different catheterization procedures: Swan-Ganz catheterization, usage in renal dialysis (32), in oncological treatment (33) or in cardiac stimulator placement (34-36). In all of the abovementioned situations severe complications may occur: left subclavian vein thrombosis, cardiac arrhythmias, perforation of the coronary sinus, cardiac tamponade, cardiogenic shock or even death.

Conclusion

Transthoracic echocardiography is mandatory in all cases planned for thoracic or cardiac surgery, as well as in cases
proposed for different venous catheterizations. In the meantime, the presence of a dilated coronary sinus, observed during echocardiography must serve as a warning for the physician of a possible venous malformation. If PLSVC is suspected, the range of investigations must be broadened in order to confirm it along with any other anatomical malformations; by doing so we may avoid possible severe complications which may arise during the approach of the superior cavae system.

Conflicts of Interest

There are no conflicts of interest to declare regarding this study.

Authors’ Contributions

CS, CP, AM – performed surgical procedures; RP, IB – prepared the manuscript; IB, CD, NG – data analysis; NG – part of the surgical team; LI, NB – advised about the surgical oncology procedure, revised the final draft of the manuscript.

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