

Inferior Vena Cava Agenesis in an Adult Organ Donor

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ABSTRACT

The inferior vena cava is the main organ of venous return from the lower extremities and abdominal organs to the right atrium. Congenital atresia of inferior vena cava is very rare. This anomaly can be surprising for transplant surgeons. The anomaly, if unknown, can cause procedural complications during interventional procedures or organ harvesting.

KEYWORDS: Caval agenesis; Organ donor; Anomalies

INTRODUCTION

Inferior vena cava (IVC) atresia is an extremely rare vascular anomaly. It is also called IVC agenesis or aplasia. Its etiology is controversial, but aberrant development during embryogenesis is the most commonly proposed mechanism. Patients with IVC anomalies usually develop compensatory circulation through the collateral veins with enlarged azygos veins. Herein, we report an unusual case of IVC atresia that discovered during organs procurement. To the best of our knowledge, IVC atresia has so far been reported in an adult case during organs harvest.

CASE PRESENTATION

Multiorgan donation was offered from a 20-year-old man due to brain death. The donor had been admitted three days previously with intracranial hemorrhage after motor vehicle accident. His past medical history and family history were insignificant. The test for human immunodeficiency virus and Epstein-

Barr virus were negative. He had no history of intravenous drug abuse. On physical examination, he had a blood pressure of 100/60 mm Hg, pulse rate of 80/min, and oral temperature of 36.8 °C. The serum electrolytes and liver function tests were within the normal range. Electrocardiogram showed no significant changes. Cardiac assessment for heart donation was made by transthoracic echocardiography (TTE). Given the young age, coronary angiography was not performed. TTE displayed normal findings with no inotropic support. During the organ procurement, we noted that a suprarenal IVC was atretic (Fig1). After surgical evaluation, the grafts were retained suitable for liver, kidneys, and heart transplantation. The donor liver was prepared and the suprahepatic vena cava was over sewn. Then, a latero-lateral anastomosis to the recipient IVC was made. Heart transplantation was done without any problem. Ultrasonography was unable to identify IVC atresia.

DISCUSSION

Congenital anomalies of the IVC are seen in 0.5–3% of the general population [1]. However, congenital absence of IVC is exceedingly rare with a prevalence of 0.0005–0.01 [2]. These anomalies are usually detected incidentally during surgery, radiologic procedure,

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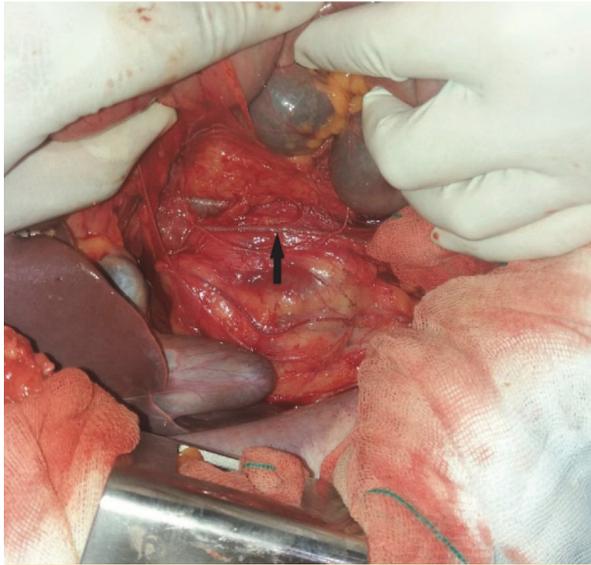


Figure 1: Intra-operative imaging of absent of IVC (black arrow)

and autopsy [3], or after an episode of deep vein thrombosis (DVT). The IVC has four segments: hepatic, suprarenal, renal, and infrarenal [4]. The IVC develops between the 6th and 8th week of embryonic life as a composite structure formed from three paired embryonic veins: the posterior cardinal, the subcardinal, and supracardinal veins [4]. The infrahepatic absence or interruption of IVC is caused by the absence of fusion of the right subcardinal vein to the liver and it is characterized by the presence of IVC from the common iliac vein up to the renal veins, from which point it continues posteriorly to the diaphragmatic crura as dilated azygos vein [5]. This congenital anomaly is often a part of a more complex syndrome such as cardiac malformation, asplenia, polysplenia, or situs inversus. In our case, no additional congenital anomalies were seen. IVC

atresia is commonly asymptomatic but may present with symptoms of the lower extremity DVT, especially in young males. There are only two reports on IVC agenesis in an adult and a pediatric organs donation [6, 7]. Presence of this anatomical anomaly in the donor is not a contraindication for liver transplantation [6]. Prior knowledge of this anomaly is vital, especially if these individuals are undergoing cardiac procedures via femoral vein access.

CONFLICTS OF INTEREST: None declared.

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