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A Rare Anatomic Variant of Double Replaced Hepatic Arteries: A Case Report and Brief Review of the Anomalous Hepatic Vasculature Literature

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Data Interpretation D
Manuscript Preparation E
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Patient: Male, 78-year-old
Final Diagnosis: Double replaced hepatic artery
Symptoms: None
Medication: —
Clinical Procedure: —
Specialty: Infectious Diseases • Transplantology

Objective: Congenital defects/diseases

Background: The liver is a frequent site of surgical resection for both benign and malignant lesions. Advanced knowledge of the hepatic arterial system and its variants is crucial to avoid incidental injuries during a resection procedure. Many variants have been previously described in the literature, yet extremely rare cases continue to be encountered in clinical practice. Documentation of these variants can thus allow for proper preoperative procedural planning when considering interventions involving the liver. Our aim is to present one such unique and extremely rare anomaly.

Case Report: During routine cadaveric dissection of a 78-year-old man who had died of acute myeloid leukemia, a rare anatomic variant of the hepatic vasculature was revealed: a replaced right hepatic artery (rRHA) coming directly from the celiac trunk, a middle hepatic artery (MHA) continuing from the common hepatic artery (CHA), and a replaced left hepatic artery (rLHA) branching from the left gastric artery (LGA). To the best of our knowledge, this anomaly has only been described once before in the literature.

Conclusions: We report a rare anatomical variant of the hepatic vasculature. The significance of this variant must be considered during preoperative planning and the intra-arterial infusion of targeted drugs. This case further emphasizes the importance of proper medical imaging and documentation to ensure the best course of treatment for each patient. Given that this variant has only so far been identified in 2 post-mortem subjects, further work should include attempts at characterizing its physiologic effects in a living patient.

Keywords: Hepatic Artery • Liver Neoplasms • Liver Transplantation

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Background

Knowledge of the hepatic hilar vasculature is critical to the practicing surgeon and interventional radiologist who frequently perform surgical resection and percutaneous interventional procedures for various pathologies within the liver and pancreaticobiliary systems. To provide anatomical context, the "standard" configuration for the hepatic arteries begins with the celiac trunk branching off the anterior aspect of the descending aorta at roughly the level of T12 into the splenic artery, left gastric artery, and the common hepatic artery. From there, the common hepatic artery gives off a branch as the gastroduodenal artery and another, smaller branch as the right gastric artery before continuing to become the proper hepatic artery. Lastly, the proper hepatic artery bifurcates into the right and left hepatic artery which supply their respective lobes of the liver, while the right hepatic artery supplies the gallbladder via the cystic artery [1]. Interestingly, this "conventional" hepatic vasculature is only seen in up to three-quarters of the public [2]. Therefore, there remains a sizable portion of the population with silent, yet clinically significant, anatomic variants. Identifying and discerning between these variations is fundamental in clinical scenarios, especially within the context of liver resections, transplantation, pancreaticobiliary surgery, and interventional radiologic procedures [3].

In contrast with the standard configuration of hepatic circulation, most known hepatic vasculature variations to date have been categorized under Michels' classification system, which was established in 1955 and later expanded in 1966 [4] (**Table 1**). Michels' initial taxonomy included categories for 5 different types of variants, but research has since then doubled that to at least 10 unique classifications [5]. Truly, the hepatic arterial system has a wide degree of variation, some with consequential clinical relevance. This report explores the clinical significance of a unique, atypical variation in hepatic vasculature that, to the authors' best knowledge, has only ever been identified once previously [6].

Case Report

The case presented here involves an incidental finding of hepatic circulation uncovered during routine cadaveric dissection of a 78-year-old man who had died of acute myeloid leukemia. Due to the nature of the cadaver donation, the patient's additional comorbidities, health history, surgical history, and family history were unknown. Furthermore, no medical imaging could be obtained post-mortem nor could a comprehensive chart review be performed to review past imaging that could highlight the relevant anatomy. However, given that the donor lived to the age of 78 and his liver was unremarkable on gross exam, it is not unreasonable to assume that this variant had little to no clinical impact on his health.

Table 1. The most common variants in hepatic arterial circulation as defined by Michels [4].

Hepatic arterial anatomy	Michels' Class
Normal anatomy	Type I
LHA branching from LGA	Type II
RHA branching from SMA	Type III
LHA branching from LGA & RHA branching from SMA	Type IV
aLHA branching from LGA	Type V
aRHA branching from SMA	Type VI
aLHA branching from LGA & aRHA branching from SMA	Type VII
aLHA branching from LGA & RHA branching from SMA	Type VIII
CHA branching from SMA	Type IX
CHA branching from LGA	Type X

Unlike typical hepatic hilar vasculature, our donor had 3 hepatic arteries inserting into the liver from different origins: a replaced right hepatic artery (rRHA), a middle hepatic artery (MHA), and a replaced left hepatic artery (rLHA) (**Figure 1**). Other local structures, including the portal vein and biliary tree, appeared unremarkable. Akin to typical anatomy, our donor's common hepatic artery diverged into the gastroduodenal artery and a proper hepatic artery (PHA), but the PHA provided no additional branches before it inserted as the MHA in the fissure between the right and left lobes of the liver. The rLHA was smaller in diameter than the MHA and inserted into the left lobe of the liver medial to the MHA. The rLHA originated from the left gastric artery, about 2 cm from its origin at the celiac trunk (**Figure 2**). The rLHA was otherwise unremarkable.

The origin of the rRHA was directly from the celiac trunk, effectively forming a quadfurcation between itself, the left gastric artery, the splenic artery, and common hepatic artery. Due to its unique origin, the rRHA traversed deep to the portal vein and common bile duct before inserting into the right lobe of the liver. Despite this unique path, the rRHA gave off a branch for the cystic artery before inserting into the liver in the right lobe, consistent with typical hepatic vasculature (**Figure 3**).

Discussion

The exact mechanism leading to the variations in hepatic arterial circulation is not well understood but is hypothesized to stem from incomplete development of the celiac axis during embryonic development. In utero, the 10th-13th vitelline

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Figure 1. The three anomalous hepatic arteries can be seen branching directly off the the celiac trunk and inserting into the liver in this photograph. In this view, the portal vein and biliary tree are present to highlight the anomalous vasculature relative to local anatomic structures.

arteries form connections between the dorsal (descending) aorta and the ventral longitudinal anastomosis. Typically, the ventral anastomosis will obliterate along with the 11th and 12th vitelline arteries. Thus, the 10th vitelline artery remains and becomes the celiac trunk supplying the foregut and the 13th vitelline artery remains to become the SMA supplying the midgut [7]. Failure of these obliterations and separations can potentially be the causative source for various arterial anomalies, thereby forcing the necessary circulation to arise from unconventional origins [8,9].

Since Michels' original classification scheme has been published, multiple new variants have been discovered that do not quite fit within his original criteria. For example, Michels' classification system only accounts for the RHA arising from its usual origin and the SMA. A replaced RHA arising from the SMA is by far the most common origin for a rRHA, being present in an estimated 3.7-9.2% of the population [10-13]. Since then, there have been documented cases demonstrating that a RHA can also arise from the inferior mesenteric artery [10], splenic artery [14,15], celiac trunk [16], right renal artery [17], right gastric [18], left gastric artery [19], gastroduodenal artery

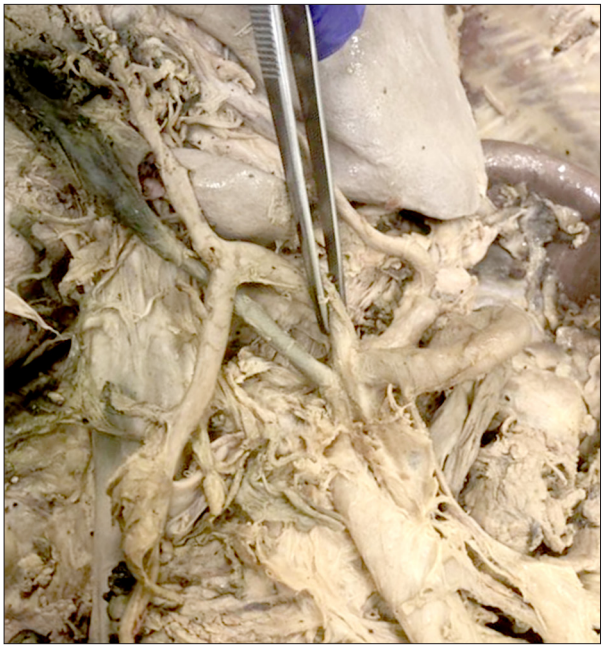

Figure 2. This is another view of the anomalous vasculature but with more local structures dissected out to better highlight the exact course traversed by each artery.

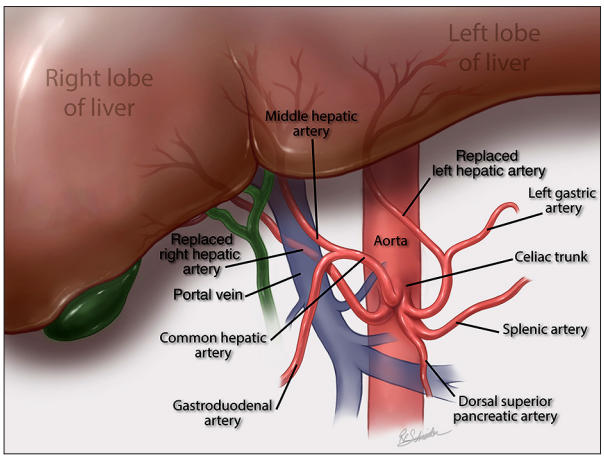

Figure 3. Illustration depicting the vascular anomalies.

[20], common hepatic artery [21], right phrenic artery [22], or the abdominal aorta [18]. These cases tend to be exceedingly rare, occurring in only a fraction of a percent of cases documented in the literature [13]. Similarly, the left hepatic artery is aberrant in an estimated 13.5-20% of patients, with a replaced LHA having an estimated prevalence of 3.8-10% of [6,12,23]. While originating from the LGA is the most common variant of the LHA [24,25], there have also been other isolated incidents where the LHA has been shown to originate from the gastroduodenal artery [26], the splenic artery, and the aorta [18].

The clinical significance of properly identifying the hepatic circulation is especially important to transplant specialists. One

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study found that patients with variant hepatic arteries experienced post-liver transplantation complications at a rate of 20.8% vs 3.3% for patients with classic hepatic arterial anatomy. Complications included stenosis, thrombosis, liver abscesses, and biliary strictures; with some cases being so severe as to require re-transplantation. The largest contributing risk factor for complications was the significantly smaller diameter for the arteries supplying the liver in those who experienced complications vs those who did not: 5.2±0.8 mm vs 6.2±0.9 mm, respectively (P<0.01) [28]. Hepatic variants generally increase the number of arteries supplying the liver or supply them from different origins. These variants tend to stem from smaller trunks, which lead to smaller caliber vessels supplying the liver. In the context of transplant surgeries, this makes for a more difficult anastomosis and increases the resistance of blood flow to the liver, potentially leading to increased complications.

Interventional radiologists also have a vested interest in the variants of the hepatic hilar vasculature. One of the mainstay treatments for non-resectable hepatic metastases of colorectal cancers is intra-arterial hepatic chemotherapy, also known as hepatic artery infusion. The advantage of this approach is its ability to provide a concentrated dose of a chemotherapy agent to a local area that cannot otherwise be obtained via systemic injection. Arterial variants pose a challenge to this technique since anomalous vascularization can result in incomplete or incorrect perfusion of the target lesions [29]. Therefore, it is essential to properly assess each patient's vascular architecture and create a unique preoperative plan that accounts for any potential anomalies while still optimizing drug delivery.

Conclusions

We report a rare anomaly of the hepatic vasculature with clinical significance for transplant surgeons, interventionalists, and other specialist who work closely with the liver. While this variation, and many others, may be exceedingly rare, it still bears clinical significance and is therefore worth noting in the literature. This is only the second ever documented case of this variant, with both cases being incidentally discovered in post-mortem cadaveric dissections. Further studies should be done if this variant is ever discovered in a living patient so that the full physiologic impact can be assessed further, characterized, and documented.

Acknowledgements

Illustration by Roy E Schneider.

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