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Case Report

A rare case of replaced right hepatic artery with direct aortic origin described angiographically during trans-arterial radioembolization☆,☆☆

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ABSTRACT

Normal hepatic arterial anatomy consists of the right hepatic artery and left hepatic artery branching from the common hepatic artery. Despite this being the most common configuration, many variations have been described. Here, we present a rare variant of hepatic arterial anatomy- a replaced right hepatic artery with direct aortic origin. Additionally, the patient was found to have a dorsal pancreatic artery originating from the replaced right hepatic artery This was angiographically identified during mapping for transarterial radioembolization for hepatocellular carcinoma. The unique anatomy in this case and the effect it had on transarterial radioembolization planning described herein demonstrates the necessity of understanding variant hepatic arterial anatomy in endovascular hepatic interventions.

Introduction

Hepatic arterial anatomy "normally" consists of the right hepatic artery (RHA) and left hepatic artery (LHA) arising from the common hepatic artery (CHA) arising from the celiac trunk [1]. RHA and LHA configurations can be replaced (having an origin separate from the CHA), accessory (existence of additional arterial supply), and aberrant (any deviation from "normal" including replaced and accessory) [2,3]. Michels in 1966 created a classification system for hepatic arterial variations that is still in use today [1]. He reported the "normal" configuration in 55% of cases while subsequent studies reported "normal" anatomy in 70%-80% of cases [1–5]. Since the introduction of Michels' classification and a later modification by Hiatt, rare variants that are not accounted for in these schemes have been de-

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scribed [1–3,6]. Understanding hepatic arterial anatomy and potential variations is crucial to the planning and performance of endovascular hepatic interventions. Herein, we report a rare variant of a replaced right hepatic artery (rRHA) with direct aortic origin, seen angiographically.

Case report

A 42-year-old male with cirrhosis complicated by hepatocellular carcinoma (HCC) who previously underwent Y90 transarterial radioembolization (TARE) and transarterial chemoembolization (TACE) of a segment 4 HCC presented for repeat TARE for a Liver Imaging Reporting & Data System- Treated (LR-TR) viable lesion in liver segments 4A and 4B seen on magnetic resonance imaging (MRI) (Fig. 1).

Angiography was performed for mapping with Tc-99m MAA prior to repeat planned Y90 TARE. A new arterial-arterial (A-A) shunt was noted between a segment 4B hepatic artery originating from the LHA to the segment 5 hepatic artery originating from the rRHA. This was thought to likely be a sequela of prior treatment. The A-A shunt was subsequently coil embolized with a single 2 × 20 mm detachable coil (EMBOLD, Boston Scientific, Marlborough, MA, USA) to prevent nontarget embolization. The gastroduodenal artery was also plug embolized with 2 detachable plugs (MVP, Medtronic, Dublin, Ireland). Given the repeat tumor recurrences this patient experienced, collateral supply outside the segment 4A and 4B hepatic artery branches was investigated. In doing so, the rRHA was found to directly originate from the aorta. The celiac trunk and superior mesenteric artery (SMA) also had separate origins. The right gastroepiploic artery and dorsal pancreatic artery (DPA) continuing into the transverse pancreatic artery (TPA) all originated from the rRHA. The patient was found to have a segment 4 hepatic artery originating distal to the gastroduodenal artery arising from the celiac trunk and also, a replaced left hep-

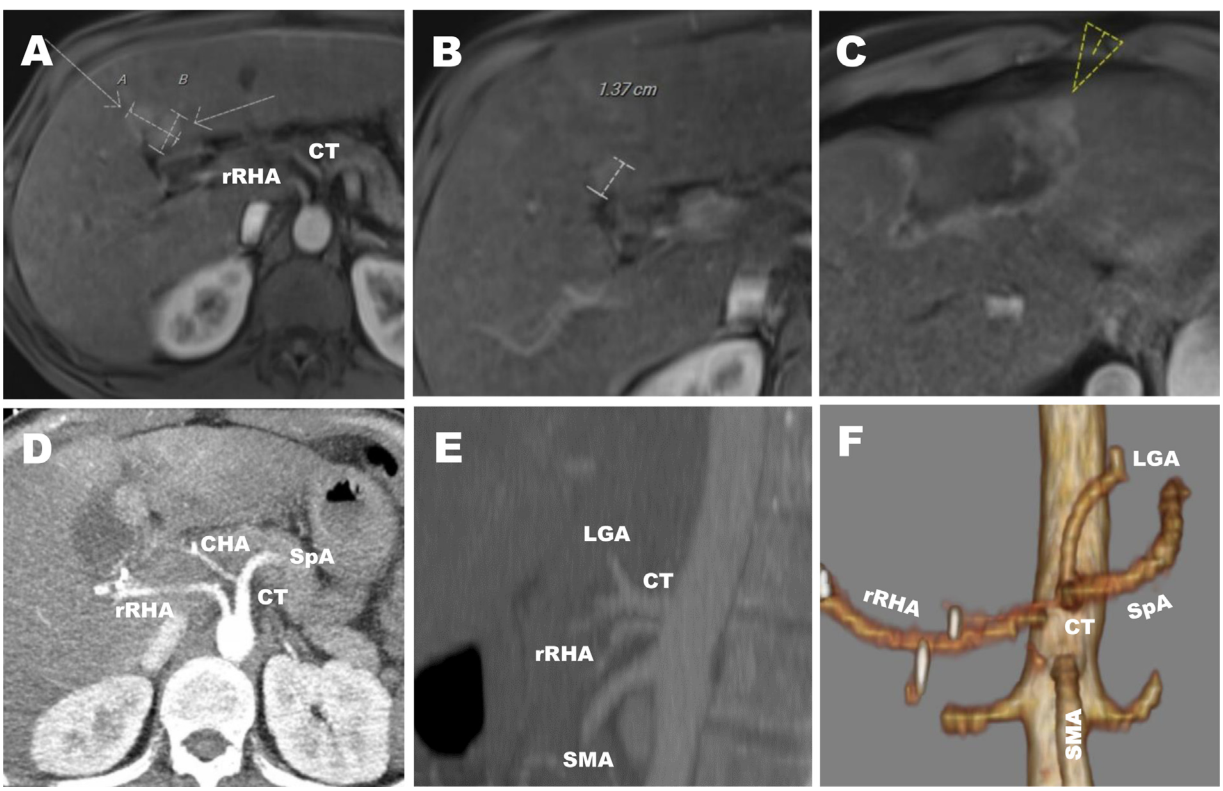

Fig. 1 – Cross-sectional imaging of the segment 4 hepatocellular carcinoma pre- and post-treatment and associated mesenteric vasculature. (A) Pretreatment T1 fat saturated arterial phase postcontrast abdominal MRI demonstrates the arterial phase hyperenhancement of the segment 4 hepatocellular carcinoma (HCC); CT, Celiac Trunk, rRHA, replaced right hepatic artery. (B) 5 month post trans-arterial chemoembolization T1 fat saturated arterial phase postcontrast abdominal MRI demonstrates nodular mass-like arterial phase hyperenhancement at the treatment site concerning for local recurrence. (C) 10 month post trans-arterial radioembolization T1 fat saturated arterial phase postcontrast abdominal MRI demonstrates nodular mass-like arterial phase hyperenhancement at the treatment site concerning for local recurrence. (D) Axial arterial phase postcontrast maximal intensity projection computed tomography images of the celiac region demonstrate the: celiac trunk's relationship to the rRHA with direct aortic origin. The SpA, splenic artery and CHA, common hepatic artery are also shown. (E) Sagittal arterial phase postcontrast maximal intensity projection computed tomography images demonstrating the CT, rRHA, LGA, left gastric artery, and SMA, superior mesenteric artery. (F) Computed tomography volume-rendered reconstruction of the aorta in the region of the CT, rRHA, and SMA. The SpA and LGA are also shown.

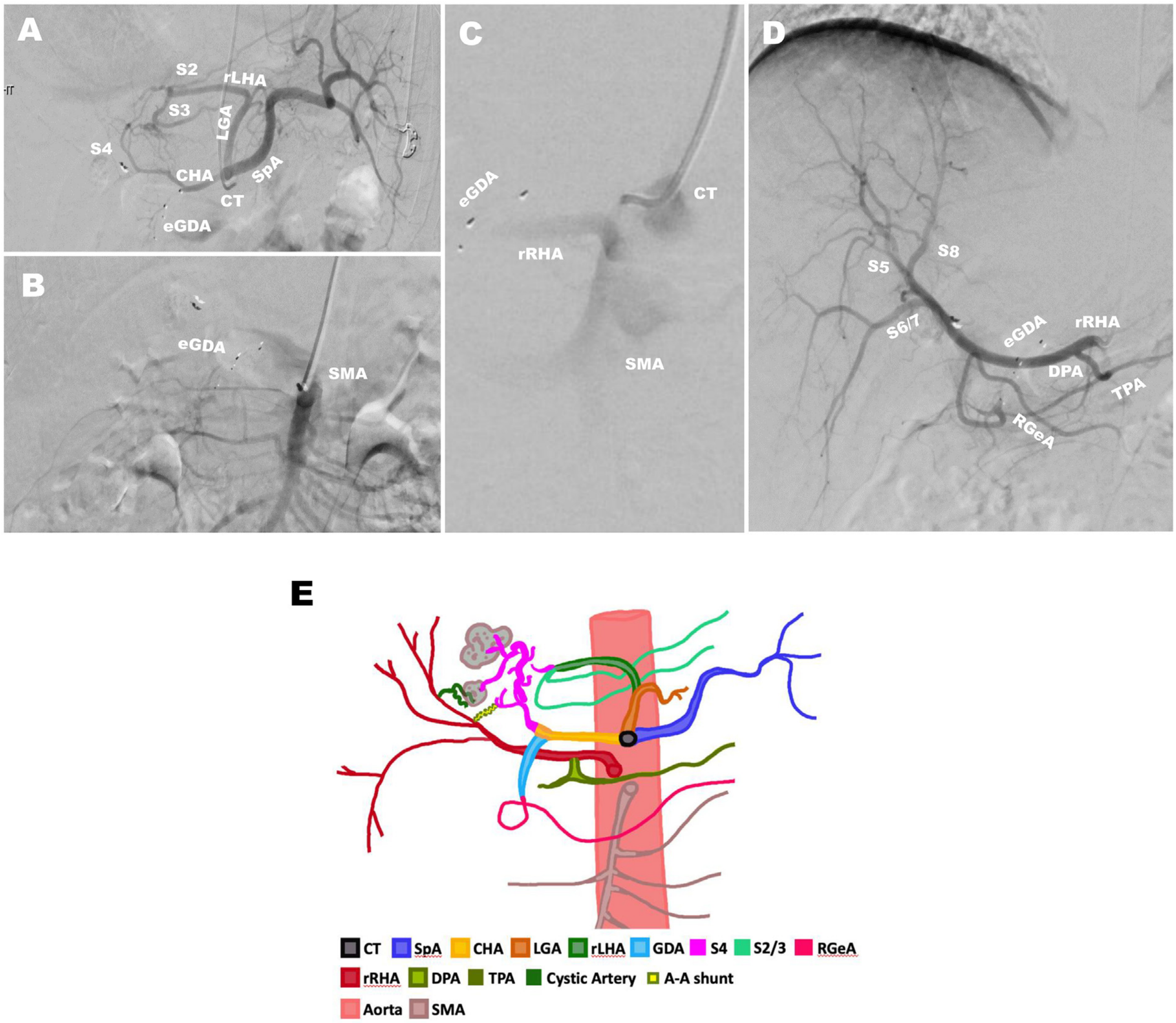

Fig. 2 – Angiographic images demonstrating replaced right hepatic artery with direct aortic origin anatomical relationship to the other mesenteric vessels. (A) Digital subtraction angiography (DSA) images with contrast injection of the Celiac Trunk and its branch vessels; CT, celiac trunk, SpA, splenic artery, CHA, common hepatic artery, LGA, left gastric artery, rLHA, replaced left hepatic artery, S4, Segment 4 hepatic artery, S2/3, Segment 2/3 hepatic arteries, eGDA, embolized GDA. During a prior procedure the GDA was embolized to prevent nontarget embolization. (B) DSA images with contrast injection of the superior mesenteric artery; SMA, superior mesenteric artery. The eGDA is again shown. (C) DSA images with contrast injection within the aorta depicting the separate origins of the rRHA, replaced right hepatic artery, SMA and CT. The eGDA is again shown. (D) DSA images with contrast injection within the rRHA. DPA, dorsal pancreatic artery, TPA, transverse pancreatic artery, RGeA, right gastroepiploic artery, S 6/7, Segment 6/7 hepatic artery, S5, segment 5 hepatic artery, S8, segment 8 hepatic artery. The eGDA is again shown. (E) Cartoon depicting anatomy seen on angiography and associated lesions.

atic artery (rLHA) arising from the left gastric artery (LGA) (Fig. 2).

The segment 4A hepatic artery which originated distal to GDA supplied the LR-TR viable lesion in segment 4A. It was determined that the segment 4B hepatic artery did not supply the LR-TR lesion. Upon injection of the rRHA, a prominent cystic artery was noted coursing to the region of the LR-TR viable lesion in segment 4B seen on prior MRI. Upon sub-selection and injection of a branch of the cystic artery with a microcatheter, a tumoral blush was noted corresponding to an area of LR-TR viable disease on MRI in segment 4B. The segment 4A component of the lesion was treated with Y90 microsphere injection from the segment 4A branch originating distal to GDA. Given that the segment 4B area of disease was supplied from vessels also supplying the gallbladder, percutaneous alcohol ablation was pursed for this lesion as opposed to TARE (Fig. 3).

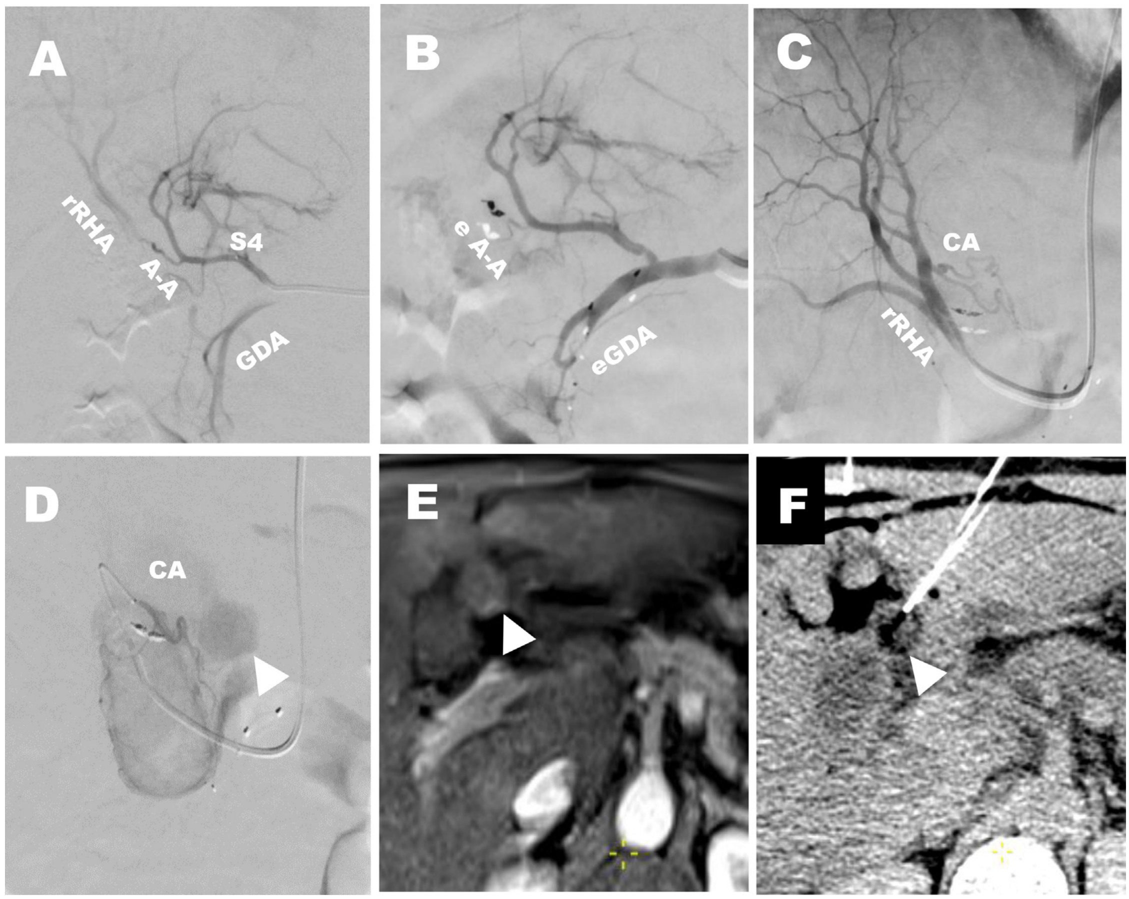

Fig. 3 – Replaced right hepatic artery (rRHA) supply via the cystic artery to a segment 4B mass adjacent to gallbladder fossa (A) Digital subtraction angiography (DSA) image with contrast injection of the CHA revealing an arterial-arterial shunt (A-A) between the S4 and the rRHA. GDA, gastroduodenal artery. (B) Postembolization of the A-A (e A-A) and the GDA (eGDA) with contrast injection from the CHA. (C) DSA image with contrast injection into the rRHA revealing a prominent cystic artery (CA) and possible mass like enhancement. (D) DSA image with selective contrast injection via a microcatheter through a branch of the CA revealing tumoral blush (arrowhead). (E) T1 fat saturated arterial phase postcontrast abdominal MRI revealing mass like enhancement (arrowhead) corresponding to that seen on DSA on image D. (F) Non contrast procedural CT images showing alcohol ablation of the mass (arrowhead).

Discussion

In most patients, approximately 55%-80%, the RHA branches with the LHA from the CHA and as such is denoted “normal anatomy” [1–4]. The 2 most utilized classification schemes for hepatic artery variations are those of Michels and Hiatt (Table 1).

While these will encompass most cases, subsequent studies continue to report rare configurations that do not fit into these classification schemes. Noussios et al. reviewed 19,013 cases of reported hepatic arterial variations and found that 4.1% of cases did not fit into Michels’ classification. A retrospective study of 5625 patients found that 5.6% of cases did not fit into Hiatt’s classification [2]. While these rare variants and make up a small portion of reported anomalies, knowledge of their existence is important in possessing a comprehensive understanding of hepatic arterial supply that goes beyond these established classifications schemes.

This report presents a rRHA with direct aortic origin which is considerably rare in relation to other reported variants. The most common RHA variation is rRHA from the SMA with a reported prevalence of 3.7%-10.2% [2,3]. In comparison, the largest retrospective study of hepatic artery anomalies found rRHA with direct aortic origin in only 0.11% of cases while other studies reported a prevalence up to 1.7% [2,4]. Additionally, this case report presents this configuration angiographically which, to the best of our knowledge, is the first instance of this to be described in the literature. A case report by Tsoucalas et. al. described a rRHA with direct aortic origin on CT angiography. However, that patient lacked a CHA and their LHA originated directly from the celiac trunk while our patient had a rLHA arising from the LGA [7].

In addition to the rare nature of the RHA origin, this patient also possessed a DPA arising from the rRHA. A systematic review by Rousek et al. found that the DPA originated from aberrant hepatic arteries in 1.97% of cases. However, the authors did not elaborate on the nature of the aberrant arteries, so it

Table 1 – Michel and Hiatt classification of hepatic arterial variations.

Michel classification [1]	Hiatt [6]
I- normal anatomy	I- normal anatomy
II- rLHA from SMA	II- aberrant LHA
III- rRHA from SMA	III- aberrant RHA
IV- rLHA from LGA and rRHA from SMA	IV- aberrant LHA and RHA
V- aLHA from LGA	V- rCHA from SMA
VI- aRHA from SMA	VI- rCHA from aorta
VII- aLHA from LGA and aRHA from SMA	-
VIII- aLHA from LGA and rRHA from SMA	-
IX- rCHA from SMA	-
X- rCHA from LGA	-

aLHA, accessory left hepatic artery; aRHA, accessory right hepatic artery; LGA, left gastric artery; LHA, left hepatic artery; rCHA, replaced common hepatic artery; RHA, right hepatic artery; rLHA, replaced left hepatic artery; rRHA, replaced right hepatic artery; SMA, superior mesenteric artery.

remains uncertain if this configuration has been reported before [8]. The classic anatomy and variations of the TPA is less robustly described in the literature. One cross-sectional analysis of dissection specimens found that the TPA originated from the DPA in 73% of specimens [9]. A retrospective study of imaging from 226 patients similarly found the DPA to be the most common origin of the TPA at 56% [10].

Understanding hepatic arterial supply has obvious implications in planning and executing hepatobiliary procedures. Adequately establishing arterial blood supply during transplant and comprehensively ligating feeding vessels during a tumor or organ resection requires a thorough knowledge of a patient's arterial anatomy. As such, variations in hepatic arterial anatomy are associated with increased complications and worse prognosis in certain surgical circumstances [11]. During endovascular procedures such as TACE and TARE, access to target liver segments is dependent on the vessels that supply them. A particular consideration during endovascular procedures is the risk of nontarget embolization and or irradiation. Embolic agents may spread through nearby vasculature and cause ischemic damage to nearby structures. In this case, embolization was performed in the gastroduodenal and rRHA-segment 4 hepatic A-A shunt to decrease this risk. Selecting these targets was predicated on an understanding of the patient's particular anatomy and the risks associated with leaving these vessels patent. Additionally, accessory arteries may decrease the efficacy of embolization if additional vessels can collectively maintain blood flow to the target lesion.

Conclusion

This case report describes a rare variation of hepatic arterial anatomy, a rRHA with direct aortic origin. The patient also possessed a rLHA from the LGA and a DPA originating from the rRHA. To the author's knowledge, this case is the first to demonstrate a rRHA with direct aortic origin angiographically. Given that this variation does not fit into commonly utilized classification schemes, understanding this presentation is essential to the successful planning and execution of endovascular hepatic interventions.

Patient consent

This case report was deemed exempt from institutional review board review #24-0040 given that it does not constitute human studies research. All information in the report has been de-identified and anonymized. Written informed consent was obtained by the patient for use of case information in this publication.

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