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Rare anatomic variation of the right hepatic artery and accessory right hepatic artery supplying hepatocellular carcinoma

A case report and literature review

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Abstract

Introduction: Each hepatic artery is functionally essential for providing blood supply to the liver, and so are variant arteries. Variant arteries, including the accessory right hepatic artery (ARHA) and replaced right hepatic artery (RRHA) are commonly described in the literature. However, they usually occur independently. Here, we report an extremely rare case that involved both an ARHA and an RRHA arising from the gastroduodenal artery (GDA) and superior mesenteric artery (SMA), respectively. To date, this situation has never been reported in the literature.

They were preoperatively identified during magnetic resonance imaging (MRI) examination in a 69-year-old male patient with hepatocellular carcinoma. And they were further verified by following conventional angiography for transcatheter arterial chemoembolization (TACE) for the patient. In addition, the patient's tumor was primarily supplied by these 2 variant arteries. After the successful TACE procedure, the patient had a well postoperative recovery.

Conclusions: By analyzing this case and performing a systematic review of the literature, the important clinical implications of the ARHA and RRHA will be investigated and discussed. Main lessons learned from this case thorough understanding of the normal anatomy of the hepatic artery and its anatomic variation is crucial for surgeons and interventional radiologists; preoperative computed tomography, MRI, and intraoperative angiography play an important role in detecting the variant hepatic artery; identifying these anomalous hepatic arteries before operation can effectively avoid unintentional injury during surgery, such as massive hemorrhage or hepatic infarction.

Abbreviations: AFP = alpha-fetoprotein, ARHA = accessory right hepatic artery, CHA = common hepatic artery, CT = celiac trunk, GDA = gastroduodenal artery, HCC = hepatocellular carcinoma, IMA = inferior mesenteric artery, LGA = left gastric artery, LHA = left hepatic artery, MRI = magnetic resonance imaging, PHA = proper hepatic artery, RA = renal artery, RPA = right phrenic artery, RRHA = replaced right hepatic artery, SA = splenic artery, SMA = superior mesenteric artery, SPA = superior pancreaticoduodenal artery, TACE = transcatheter arterial chemoembolization.

Keywords: accessory right hepatic artery, anatomic variation, hepatocellular carcinoma, replaced right hepatic artery, transcatheter arterial chemoembolization

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1. Introduction

Liver resection and transplant is generally considered the criterion standard treatment for patients with hepatocellular carcinoma (HCC).[1] However, transcatheter arterial chemoembolization (TACE) is widely used in the treatment of patients with HCC where radical resection is not an option, and has also been considered a good alternative method to improve survival and help prevent postoperative recurrence of disease.[1] However, sometimes anatomic variations of the hepatic artery are encountered during liver resection, liver transplant, or TACE. Anatomic variation in the hepatic artery increases the risk of TACE for HCC. Knowledge and identification of the origin, course, and variations of the hepatic artery preoperatively are crucial for surgeons and interventional radiologists to perform hepatic surgery or TACE successfully and safely. Descriptions of anatomic variations in the hepatic arterial blood supply, especially rare variations, are very important, and many have been previously reported in the literature.

An accessory right hepatic artery (ARHA) and a replaced right hepatic artery (RRHA) are 2 of the most well-known anatomic variations of the hepatic artery. An (ARHA is defined as an additional artery supplying the right liver along with a normal right hepatic artery (RHA).[2] Various origins of the ARHA have been described, including the celiac trunk (CT),[3] splenic artery (SA),[4]

common hepatic artery (CHA),[5] proper hepatic artery (PHA), superior pancreaticoduodenal artery (SPA),[6] left hepatic artery (LHA),[7] left gastric artery (LGA), gastroduodenal artery (GDA), right phrenic artery (RPA), superior mesenteric artery (SMA),[8] renal artery (RA),[9] or directly from the abdominal aorta (AA).[10] The accepted "normal" RHA arises from the PHA, whereas RRHA arises from a different source.[11] The RRHA can arise from LGA, GDA, CHA,[9] SA,[12] RA, inferior mesenteric artery (IMA),[11] SMA,[13] CT,[14] right phrenic artery (RPA),[8] or directly from the AA.[15] However, these 2 variant arteries always occur independently in humans. Variant anatomy in patients involving both the ARHA and RRHA has never been reported in the literature.

In our case, we identified a rare variation of the ARHA and RRHA on magnetic resonance imaging (MRI) and conventional angiography during TACE for a patient with HCC. More importantly, these 2 variant arteries supplied the patient's tumor.

2. Case report

In August 2016, a 69-year-old man was diagnosed with multiple primary hepatic carcinoma lesions in the right hepatic lobe in our hospital. He had a medical history of hepatitis C virus for >10 years and had received antiviral treatment. His other viral hepatitis serology was negative. He underwent splenectomy in 1998 because of trauma. He also underwent radical gastrectomy for gastric cancer in 2005. He had no history of intravenous drug or alcohol abuse, smoking, or food or drug allergies.

The patient had no symptoms of discomfort, fever, abdominal pain or distention, or upper or lower gastrointestinal bleeding. On physical examination, his abdomen was soft and tender. His body mass index was approximately 18.5. There was no evidence of jaundice, caput medusae, or palmar erythema. Percussion of the hepatic region was positive for pain. His alpha-fetoprotein level was 133.8 ng/mL, and he had a moderate platelet count of 222×10^9 cells/L, red blood cell count of 3.31×10^{12} cells /L, and hemoglobin level of 92 g/L. Evaluation of his liver function revealed Child-Pugh class A liver disease.

MRI scan of the upper abdomen revealed multiple intrahepatic tumors, with the maximum tissue mass measuring 5.1×4.9 cm and the mean diameter of the other focal nodular lesions measuring approximately 2.3 cm. Radiography suggested primary hepatic carcinoma combined with multiple intrahepatic metastases (Fig. 1A and B). Contrast-enhanced MRI scan showed the mass and nodules within segments VI and VII of the right hepatic lobe, and demonstrated heterogeneous arterial enhancement with contrast, as well as washout on portal venous and delayed phases, compatible with a diagnosis of HCC. In addition, intrahepatic bile duct dilation and ascites were also present, and portal and splenic veins were dilated and tortuous. After carefully analyzing the MRI images, we discovered an RRHA arising from the SMA (Fig. 1C and D). However, the ARHA branching from the GDA was not visualized on the initial MRI images, but discovered later during angiography.

Owing to this patient's ascites, he was not a suitable candidate for partial hepatectomy. Therefore, the best alternative treatment plan in his case was TACE. After informed consent was obtained from the patient, TACE of the tumor was performed. The right groin was prepared aseptically and infiltrated with 2% lidocaine. Percutaneous access was obtained via the right common femoral artery using Seldinger catheterization. Conventional angiography was performed and multiple tumor blush was seen in the right hepatic lobe (Fig. 2B). Interestingly, during the procedure, we found that the tumor and surrounding nodular lesions were primarily supplied by both an ARHA arising from the GDA and an RRHA branching from the SMA (Fig. 2A and C). The CT divides into CHA, SA, and the LGA. The CHA divides into LHA and GDA (Fig. 2A). A schematic diagram is depicted in Figure 3.

When the selective catheter reached the CT, 0.25 mg of palonosetron, 10 mg of dexamethasone, 1.0 g of fluorouracil, and 200 mg of oxaliplatin were injected slowly. Superselective catheterization of the RRHA and ARHA was then performed, and the chemoembolization agent was prepared. This mixture consisted of 30 mg of doxorubicin and 15 mL of lipiodol emulsion and was successfully injected into the treated segments. This injection was followed by embolic microspheres. Finally, repeat arteriography was performed, and the imaging response of the vessels supplying the tumor disappeared because of iodized oil deposition, indicating successful chemoembolization (Fig. 2D). The catheters were removed without incident. A vascular closure device was successfully deployed, and hemostasis was achieved. The patient tolerated the procedure well without immediate complications and was discharged 5 days later.

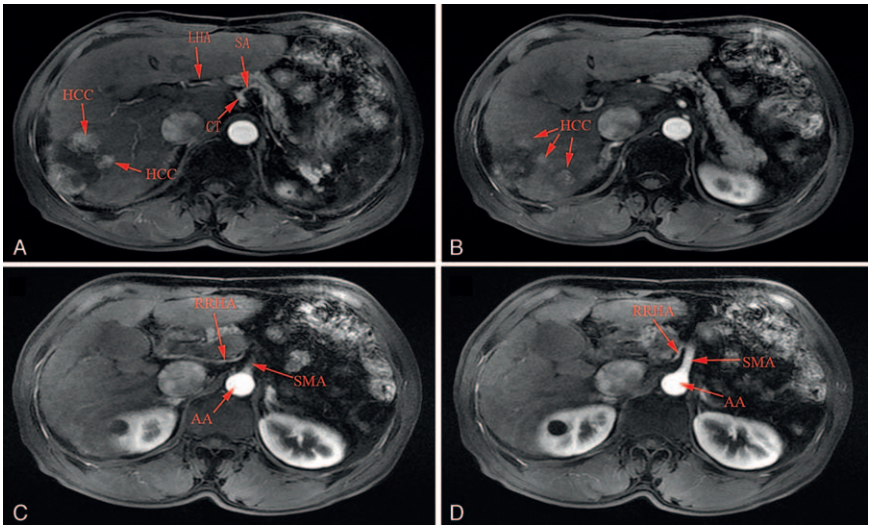

Figure 1. (A and B) Magnetic resonance imaging (MRI) scan showing multiple intrahepatic tumors (HCC) in the right hepatic lobe. (C and D) Contrast-enhanced MRI scan showing an RRHA arising from the SMA. AA = abdominal aorta, HCC = hepatocellular carcinoma, RRHA = replaced right hepatic artery, SMA = superior mesenteric artery.

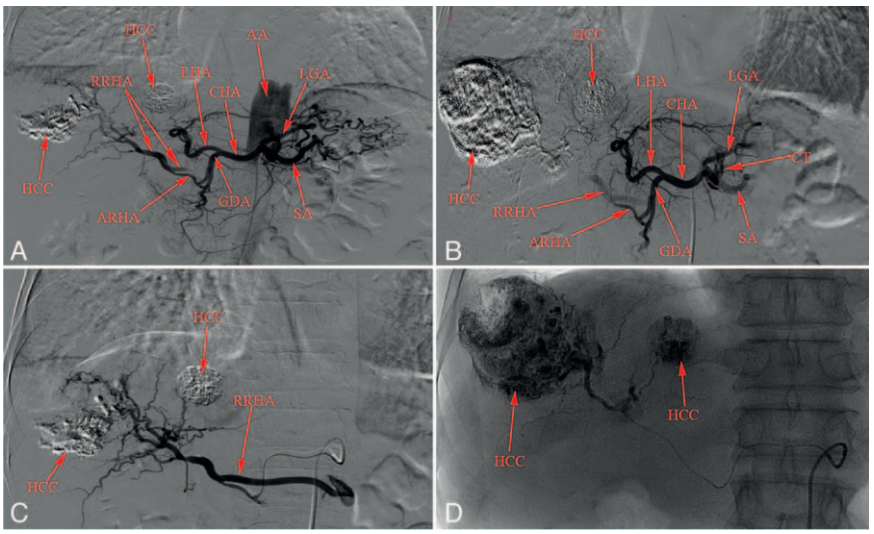

Figure 2. (A) Selective computed tomography angiogram showing the normal LGA, SA, CHA arising from the CT, and an ARHA arising from the GDA, as well as the RRHA. (B) An ARHA arising from the gastroduodenal artery and RRHA, as well as the tumor (HCC) blush. (C) Selective angiogram of the SMA showing an RRHA arising from the SMA. (D) HCC visualized as well-iodized oil deposition. AA = abdominal aorta, ARHA = accessory right hepatic artery, CHA = common hepatic artery, CT = celiac trunk, GDA = gastroduodenal artery, HCC = hepatocellular carcinoma, LGA = left gastric artery, LHA = left hepatic artery, PHA = proper hepatic artery, RHA = right hepatic artery, RRHA = replaced right hepatic artery, SA = splenic artery, SMA = superior mesenteric artery.

Three months following the TACE procedure, the patient remains feeling very well without significant clinical symptoms.

3. Discussion

Michels performed dissection of 200 cadavers and categorized the hepatic arterial supply into 10 basic types, which are considered the standard classifications.[16] His classification was further modified by Hiatt et al in 1994 who analyzed 1000 cases and classified them into 6 types.[17] Many subsequent studies have largely followed Michels' and Hiatt et al's classification schemes. We defined the standard anatomy in this study according to their description of the normal hepatic arterial anatomy. Thus, the CHA arises from the CT to form the GDA and PHA, which divide distally into the RHA and LHA, respectively.

An ARHA is defined as an additional artery supplying the right liver along with the RHA.[2] The normal RHA arises from the PHA, whereas an RRHA arises from other sources.[11] ARHA and RRHA are well described in the literature by Michels,[16] Suzuki et al,[18] Mäkisalo et al,[19] Hiatt et al,[17] Gruttadauria et al,[9] Covey et al,[8] Varotti et al,[20] Kishi et al,[21] Koops et al,[22] Ahn et al,[6] Abdullah et al,[23] López-Andújar et al,[24] Winston et al,[25] De Cecco et al,[26] Egorov et al,[27] Saba and Mallarini,[28] Gümüs et al,[29] Loschner et al,[10] Dandekar et al,[15] Mugunthan et al,[11] Thangarajah and Parthasarathy,[30] as well as some case reports (Tables 1 and 2).[3–39] By systematic review of these articles and case reports, we identified the various origins of ARHA and RRHA, as well as their incidence. We drew the conclusion that an ARHA can arise from the SMA, CT, SA, LGA, GDA, LHA, CHA, RPA, RA, PHA, SPA, or directly from the AA. Meanwhile, the origins of an RRHA can be the SMA, CT, SA, LGA, GDA, CHA, RPA, RA, IMA, or directly from the AA.

In total, we reviewed 21 articles and 16 case reports. The presence or absence of a middle hepatic artery (MHA) was regarded as normal. It was also considered normal anatomy regardless of where the MHA originated from (either the LHA or RHA). Regardless of whether the ARHA and RRHA occurred alone or accompanied by other arterial variations, all were taken into account. In addition, in Winston et al's article,[25] 3 cases of ARHA were documented without a description of their origins,

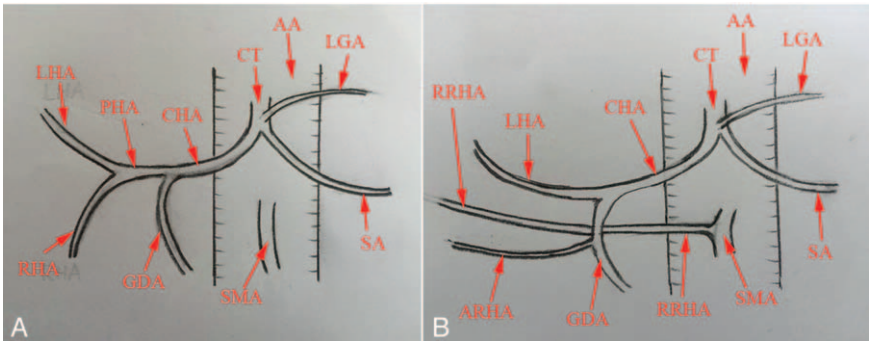

Figure 3. (A) Schematic diagram showing the standard anatomical pattern of the hepatic arteries. (B) Schematic diagram showing an ARHA arising from the GDA and an RRHA arising from the SMA. AA = abdominal aorta, ARHA = accessory right hepatic artery, CHA = common hepatic artery, CT = celiac trunk, GDA = gastroduodenal artery, LGA = left gastric artery, LHA = left hepatic artery, PHA = proper hepatic artery, RHA = right hepatic artery, RRHA = replaced right hepatic artery, SA = splenic artery, SMA = superior mesenteric artery.

Table 1

The origins and incidence of ARHA and RRHA.

Author	Year	Total cases (no.)	Standard anatomy (no./%)	ARHA (no./%)	Origins (no./%)												RRHA (no./%)	Origins (no./%)										ARHA + RRHA (no./%)
					SMA	CT	SA	LGA	GDA	LHA	CHA	AA	RPA	RA	PHA	SPA		SMA	CT	SA	LGA	GDA	CHA	AA	RPA	RA	IMA	
Michels [16]	1966	200	110/55.0	34/17.0	34/17.0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	24/12.0	24/12.0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Suzuki et al[18]	1971	200	107/53.5	8/4.0	8/4.0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	28/14.0	19/9.5	2/1.0	(–)	(–)	1/0.5	5/2.5	1/0.5	(–)	(–)	(–)	0
Mäkisalo et al[19]	1993	100	76/76.0	7/7.0	7/7.0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	6/6.0	3/3.0	1/1.0	(–)	(–)	(–)	(–)	2/2.0	(–)	(–)	(–)	0
Hiatt et al[17]	1994	1000	757/75.7	106/10.6	106/10.6	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	23/2.3	23/2.3	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Gruttadauria et al[9]	2001	701	405/57.8	170/24.3	160/22.8	3/0.4	(–)	(–)	(–)	(–)	(–)	6/0.8	(–)	1/0.1	(–)	(–)	11/1.6	2/0.3	2/0.3	(–)	1/0.1	2/0.3	2/0.3	2/0.3	(–)	(–)	(–)	0
Covey et al[8]	2002	600	368/61.3	15/2.5	11/1.8	1/0.16	(–)	1/0.16	1/0.16	(–)	(–)	(–)	1/0.16	(–)	(–)	(–)	74/12.3	73/12.2	(–)	(–)	(–)	(–)	(–)	(–)	1/0.1	(–)	(–)	0
Varotti et al[20]	2004	96	68/70.8	3/3.1	3/3.1	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	12/12.5	12/12.5	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Kishi et al[21]	2004	223	181/81.2	4/1.9	(–)	1/0.5	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	2/0.9	1/0.5	15/6.7	15/6.7	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Koops et al[22]	2004	604	478/79.1	22/3.6	21/3.4	1/0.2	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	67/11.1	60/9.9	6/1.0	(–)	(–)	(–)	(–)	1/0.2	(–)	(–)	(–)	0
Ahn et al[6]	2005	192	173/90.1	7/3.6	3/1.6	(–)	(–)	(–)	(–)	2/1.0	(–)	(–)	(–)	(–)	2/1.0	(–)	12/6.20	12/6.2	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Abdullah et al[23]	2006	932	635/68.1	95/10.2	95/10.2	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	67/7.2	61/6.6	4/0.4	(–)	(–)	(–)	1/0.1	(–)	(–)	(–)	1/0.1	0
López-Andújar et al[24]	2007	1081	761/70.3	17/1.6	17/1.6	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	118/10.9	118/10.9	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Winston et al[25]	2007	371	188/50.7	3/0.8	NG	NG	NG	NG	NG	NG	NG	NG	NG	NG	NG	NG	73/19.7	54/14.6	13/3.5	(–)	(–)	5/1.3	(–)	1/0.3	(–)	(–)	(–)	0
De Cecco et al[26]	2009	250	165/66.0	15/6.0	15/6.0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	30/12.0	30/12.0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Egorov et al[27]	2010	350	197/56.3	19/5.4	18/5.1	(–)	(–)	(–)	(–)	(–)	(–)	1/0.3	(–)	(–)	(–)	(–)	54/15.4	54/15.4	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Saba and Mallarini[28]	2011	1629	998/61.4	146/9.0	146/9.0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	205/12.5	205/12.5	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Gümüs et al[29]	2013	820	548/66.8	38/4.6	38/4.6	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	108/13.2	108/13.2	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Loschner et al[10]	2015	1297	937/72.2	27/2.1	26/2.0	(–)	(–)	(–)	(–)	(–)	(–)	1/0.1	(–)	(–)	(–)	(–)	108/8.3	105/8.1	3/0.2	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Dandekar et al[15]	2015	60	47/78.3	2/3.4	(–)	(–)	(–)	(–)	1/1.7	(–)	1/1.7	(–)	(–)	(–)	(–)	(–)	11/18.3	8/13.3	2/3.3	(–)	(–)	(–)	1/1.7	(–)	(–)	(–)	(–)	0
Mugunthan et al[11]	2016	60	52/86.6	3/5.0	3/5.0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	5/8.3	5/8.3	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Thangarajah et al[30]	2016	200	114/57.0	7/3.5	7/3.5	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	18/9.0	18/9.0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Total	—	10966	7365/67.2	748/6.8	718/6.5	6/0.05	0/0	1/0.01	2/0.02	2/0.02	1/0.01	8/0.07	1/0.01	1/0.01	4/0.04	1/0.01	1069/9.7	1009/9.2	33/0.3	0/0	1/0.01	8/0.07	8/0.07	8/0.07	1/0.01	0/0	1/0.01	0

(–) = negative, AA = abdominal aorta, ARHA+RRHA = variation involving both ARHA and RRHA, ARHA = accessory right hepatic artery, CHA = common hepatic artery, CT = celiac trunk, GDA = gastroduodenal artery, IMA = inferior mesenteric artery, LGA = left gastric artery, LHA = left hepatic artery, NG = the concrete data isn't given in article, No. = the number of each item, PHA = proper hepatic artery, RA = renal artery, RPA = right phrenic artery, RRHA = replaced right hepatic artery, SA = splenic artery, SMA = superior mesenteric artery, SPA = superior pancreaticoduodenal artery.

Table 2

The origins and incidence of ARHA and RRHA in these case reports.

Author	Year	Total cases (no.)	Standard anatomy (no.)	ARHA (no.)	Origins (no.)												RRHA (no.)	Origins (no.)										ARHA + RRHA (no.)
					SMA	CT	SA	LGA	GDA	LHA	CHA	AA	RPA	RA	PHA	SPA		SMA	CT	SA	LGA	GDA	CHA	AA	RPA	RA	IMA	
Madhu and Harish[32]	2013	1	0	1	1	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Bastos-Neves et al[3]	2016	1	0	1	(–)	1	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Al Zahrani et al[4]	2017	1	0	1	(–)	(–)	1	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Panagouli and Venieratos[33]	2011	1	0	1	(–)	(–)	(–)	1	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Yamashita et al[34]	2015	1	0	1	(–)	(–)	(–)	(–)	1	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Polguj et al[35]	2014	1	0	1	(–)	(–)	(–)	(–)	1	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
de Albuquerque Martins[7]	2010	1	0	1	(–)	(–)	(–)	(–)	(–)	1	(–)	(–)	(–)	(–)	(–)	(–)	0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Polguj et al[5]	2010	1	0	1	(–)	(–)	(–)	(–)	(–)	(–)	1	(–)	(–)	(–)	(–)	(–)	0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Troupis et al[36]	2008	1	0	0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	1	1	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Rebibo et al[37]	2014	1	0	0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	1	1	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Felli et al[13]	2016	1	0	0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	1	1	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Wang et al[38]	2016	1	0	0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	1	1	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Sayyed et al[39]	2016	1	0	0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	1	1	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Katagiri et al[14]	2016	1	0	0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	1	(–)	1	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Caruso et al[12]	2016	1	0	0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	1	(–)	(–)	1	(–)	(–)	(–)	(–)	(–)	(–)	(–)	0
Braun et al[31]	1991	1	0	0	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	1	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	1	(–)	0
This report	2017	1	0	1	(–)	(–)	(–)	(–)	1	(–)	(–)	(–)	(–)	(–)	(–)	(–)	1	(–)	1	(–)	(–)	(–)	(–)	(–)	(–)	(–)	(–)	1
Total	—	17	0	9	1	1	1	1	3	1	1	0	0	0	0	0	9	5	2	1	0	0	0	0	0	1	0	1

(–) = negative, AA = abdominal aorta, ARHA + RRHA = variation involving both ARHA and RRHA, ARHA = accessory right hepatic artery, CHA = common hepatic artery, CT = celiac trunk, GDA = gastroduodenal artery, IMA = inferior mesenteric artery, LGA = left gastric artery, LHA = left hepatic artery, NG = the concrete data isn't given in article, No. = the number of each item, PHA = proper hepatic artery, RA = renal artery, RPA = right phrenic artery, RRHA = replaced right hepatic artery, SA = splenic artery, SMA = superior mesenteric artery, SPA = superior pancreaticoduodenal artery.

so it was marked "NG" in Table 1. Some publications have noted additional, previously unclassified variations of hepatic arterial supply that are not included in Michels' classification. However, no published detailed description of these exits, so they were not taken into account. Three points require further illustration. First, in Table 1, an ARHA arising from the LHA or PHA was defined as a kind of anatomic variation based first upon the existence of a normal RHA. When a normal RHA exists, then another artery supplying the right hepatic lobe is called an ARHA, and can be considered a variant artery. Without the presence of an RHA, the artery supplying the right hepatic lobe originating from the LHA or PHA cannot be classified as an ARHA, rather, it is an RHA. Second, in Kishi et al's[21] article, an accessory hepatic artery branch supplying segment VI was termed as "A6," but is actually an ARHA, which can arise from the CT, PHA, or SPA. Third, additionally, Ahn et al[6] has mentioned that a right posterior hepatic artery arising from the LHA or SMA is also an ARHA owing to the fact that it supplies the right liver but arises from arteries other than the RHA.

For the 21 articles, according to our statistical results, the total number of cases included was 10966, of which 7365 (67.2%) had standard anatomy. An ARHA was identified in 748 (6.8%) cases. The most common origin of an ARHA was the SMA (718 cases, 6.5%), followed by the AA (8 cases, 0.07%), CT (6 cases, 0.05%), PHA (4 cases, 0.04%), GDA (2 cases, 0.02%), LHA (2 cases, 0.02%), LGA (1 case, 0.01%), CHA (1 case, 0.01%), RPA (1 case, 0.01%), RA (1 case, 0.01%), SPA (1 case, 0.01%), and SA (0 cases, 0.00%) (Table 1). An ARHA arising from the SA was not been reported in these articles, but was reported separately by de Albuquerque Martins[7] in 2010 and Al Zahrani et al[4] in 2017 (Table 2).

An RRHA was identified in 1069 (9.7%) cases. As with an ARHA, an RRHA most commonly originated from the SMA (1009 cases, 9.2%), and followed by the CT (33 cases, 0.3%), GDA (8 cases, 0.07%), CHA (8 cases, 0.07%), AA (8 cases, 0.07%), LGA (1 case, 0.01%), RPA (1 case, 0.01%), IMA (1 case, 0.01%), SA (0 cases, 0.00%), and RA (0 cases, 0.00%) (Table 1). An RRHA originating from the SA or RA was not been reported in these 21 articles, but Braun et al[31] reported a rare anatomic variation of an RRHA arising from the RA in 1991, and Caruso et al[12] reported a similar case in 2016 (Table 2). Among this research, the incidence of ARHA and RRHA is very different. For ARHA, the incidence ranges from 24.3% to 0.8%. For RRHA, the incidence ranges from 19.7% to 1.6% (Table 1). This heterogeneity may be related to a difference in the number or characteristics (races and ethnicities, region, or genetic traits) of the patients included in the studies. A literature search did not reveal any relevant studies regarding a correlation between hepatic artery variation and any such characteristics.

According to our statistics, an RRHA has 2.9% higher incidence than an ARHA, corresponding to 1069 cases (9.7%) and 748 cases (6.8%), respectively. For both an ARHA and an RRHA, they most usually originate from the SMA. Interestingly, an RRHA originating from the CT is significantly more common than an ARHA originating from the same artery, corresponding to 33 cases (0.3%) and 6 cases (0.05%), respectively. The other origins are relatively rare. More detailed data are shown in Table 1.

The origins of the ARHA have been well-described in previous articles except for the SA and LHA. Similarly, except for an RRHA originating from the SA and RA, the other origins of RRHA have also been previously described. More detailed data are shown in Table 2.

In reviewing the literature, we found that ARHA and RRHA typically exist independently. Anatomic variation involving both an ARHA and an RRHA has not been reported. Furthermore, Caruso et al[12] conducted an extensive literature review in 2010 of 27 articles with detailed descriptions of anatomic variation in hepatic arteries. In their article, the variation involving both an ARHA and an RRHA was still not reported. According to the above publications, the most common variation reported is the presence of an RRHA without an ARHA, or the presence of an ARHA with a normal RHA. To the best of our knowledge, there have been no published reports to date describing an ARHA branching from the GDA with an RRHA arising from the SMA in the same patient. This is the first report about this type of variation in hepatic arterial anatomy.

The origins and course of the hepatic artery, especially anatomic variations, have important clinical significance for gastric resection, liver resection, liver transplantation, and TACE. Each hepatic artery is functionally essential for providing blood supply to the liver, and so are variant arteries. Inadvertent ligation or embolization of an accessory or replaced hepatic artery during surgery could cause ischemic necrosis of the liver with fatal results. Early in 1989, Brown et al[40] reported a case of a patient who underwent LGA embolization and suffered complications of hepatic ischaemic necrosis owing to a replaced left hepatic artery arising from it. Miura et al[41] also documented 11 patients in 2010 who had postoperative hepatic infarction after undergoing pancreato-biliary surgery and analyzed the underlying causes. In 2 of the 11 patients, the cause of hepatic infarction was inadvertent ligation of the RHA during surgery, and one patient 1 a variant RRHA originating from the SMA. Such serious complications may have been avoided if the variant arterial supply was identified before surgery.

Therefore, a thorough understanding of the normal anatomy of the hepatic artery and its anatomic variation is crucial for surgeons and interventional radiologists to avoid inadvertent injury while performing hepatic surgery and arteriography. These anomalous hepatic arteries should be identified preoperatively and carefully avoided during surgery to prevent unintentional injury, massive hemorrhage, or hepatic infarction. Before liver transplantation, the extrahepatic arterial anatomy must also be precisely understood to assure safety and prevent recipient complications.

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