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RADIOLOGIC ANATOMY

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Anatomic variations of the hepatic arteries in 604 selective celiac and superior mesenteric angiographies

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Abstract In modern surgical and transplantation procedures the recognition of anatomic vascular abnormalities of the hepatic arteries is of greater importance than ever. The purpose of this study was to evaluate and classify these variations with respect to their impact on visceral surgery. A total of 604 selective celiac and superior mesenteric angiographies performed on patients with known or suspected liver cirrhosis or hepatic or pancreatic malignancies and on donors of partial liver grafts were analyzed retrospectively. The vascular anatomy of the liver was classified according to different established systems and with particular attention to rare variations. Hepatic arterial anatomy as considered normal in textbook descriptions was found in 79.1%, an aberrant or accessory left hepatic artery (LHA) arising from the left gastric artery in 3.0% and an aberrant or accessory right hepatic artery (RHA) branching off the superior mesenteric artery in 11.9% of the cases. In 1.4% of the cases there was a combination of anomalies of both the LHA and RHA. Variants of the celiac trunk, double hepatic arteries branching at the celiac trunk or hepatic arteries arising directly from the aorta, occurred in 4.1% of the cases. Further atypical branches of the LHA and RHA were found in 0.5% of the cases. Since the incidence and pattern of different types of hepatic arterial anatomy can require specialized preoperative diagnostic as well as intraoperative strategies, knowledge of these abnormalities and their frequency is of major importance for the surgeon as well as the radiologist.

Keywords Hepatic artery · Anatomy · Vascular anomalies · Angiography · Surgery

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Introduction

The vascular anatomy of the liver is variable. Modifications of the dominant scheme, in which the liver receives its total inflow from a common hepatic artery of the celiac trunk, occur in 12–49% of cases in general [7, 13, 22, 31, 33, 35]. Recognition and, if necessary, appropriate reconstruction of such abnormalities is of major importance in liver surgery and especially in liver transplantation, because the absence of an adequate hepatic arterial blood supply usually results in necrosis or graft loss due to ischemic biliary or parenchymal complications [4, 22, 36, 37]. Moreover, aberrant hepatic arteries can be of major surgical significance in operations of the upper intestinal tract, the gallbladder and pancreas [33, 40]. They can also become a technical problem for infusion therapy and transarterial chemoembolization of neoplasm in the liver [3, 5, 16].

The first description of aberrant hepatic arteries was published in 1756 by Haller [12]. However, later studies of the frequency of those variations required large series of anatomic autopsies, such as first performed in 1928 by Adachi [1]. In the radiologic literature, publications on selective angiographies dealing with accessory hepatic arteries date from 1958 [29]; extensive studies were performed by Lunderquist in 1967 [21]. Michels proposed an internationally recognized classification of these hepatic abnormalities in 1966 [24]. This classification was modified by Hiatt in 1994 [13]. Our study presents the findings of 604 selective angiographies and compares them with those obtained in other large series over the

past 35 years, paying particular attention to some rare variants not classified by Michels or Hiatt.

Materials and methods

Two radiologists retrospectively analyzed 502 consecutive angiographies that were performed at our institution between July 1997 and December 1999 on patients with biliary, toxic, infectious or metabolic cirrhosis and with known or suspected primary or secondary cancers of the liver or pancreas. Also included in our study were 102 angiographies performed on healthy donors of partial liver grafts prior to living donor transplantation in the years from 1992 to 1999.

The radiologic procedures performed included abdominal aortography (20 ml of 300 mg/ml iodine contrast medium administered at a rate of 15 ml/s), selective celiac (20 ml, 5 ml/s), hepatic (15 ml, 5 ml/s), splenic (25 ml, 5 ml/s) and superior mesenteric (25 ml, 6 ml/s) angiography in the sagittal projection. These were supplemented by oblique or lateral projections when necessary. Further hepatic or selective left gastric angiography was performed when the anatomic relationships remained uncertain. The percutaneous catheterization of the femoral artery was performed using the standard Seldinger technique. Digital subtraction angiograms were acquired on a Siemens Multistar TOP (Erlangen, Germany).

The anatomic findings were classified according to Michels, with the limitation that a description of the middle hepatic artery was omitted, since identification of this artery is often difficult in projection angiography. Arterial variants not illustrated by Michels were reviewed a second time.

In an alternative setting we also evaluated all hemodynamically relevant stenoses of the celiac trunk in all 604 cases.

Results

There were no substantial differences in the rates of major variations between the patients with known or suspected abdominal disease and the donors of partial liver grafts. Normal anatomy, that is type I in the classifications of both Michels and Hiatt, was found in 478 cases (79.1%), while 126 cases (20.9%) showed anomalous arterial patterns (Fig. 1, Tables 1, 2). These anomalies consisted of a replaced left hepatic artery (LHA) arising from the left gastric artery (LGA) (Michels type II, Hiatt type II), a replaced right hepatic artery (RHA) arising from the superior mesenteric artery (SMA) (Michels type III, Hiatt type III), the combination of both these anomalies (Michels type IV, Hiatt type IV), and a dual arterial supply with an accessory left hepatic artery (Michels type V, Hiatt type II) or right hepatic artery (Michels type VI, Hiatt type III) that arose from the LGA or SMA in combination with the typical right or left hepatic artery originating from a common hepatic artery of the celiac axis.

Michels type VII, the combination of every other triple arterial supply, and Michels type VIII, the combination of every other triple arterial supply to the liver, were found in only one case, respectively. Michels type IX, the variant with the complete hepatic trunk replaced by the SMA, was found in 17 cases (2.8%). There was no case in which the hepatic trunk was found to be replaced by LGA, as described by Michels as type X.

As illustrated schematically in the lower part of Fig. 1, we found additional, previously unclassified variations with the hepatic arteries arising directly from the aorta and with replaced or accessory arteries arising separately from the celiac trunk or the gastroduodenal artery. The latter variation is also shown in the angiogram in Fig. 2.

The angiographies revealed 23 (3.8%) cases of relevant celiac trunk stenosis and retrograde filling of the celiac trunk from the SMA. More than half of these stenoses (13 of 23) were found in patients with pancreatic tumors or retroperitoneal lymphatic metastases.

Discussion

Michels' classic autopsy series of 200 dissections has served as a benchmark for many subsequent contributions in this area. However, our current angiographic series differs from his results in certain points. Table 1 gives a comparison with other angiographic studies [3, 5, 8, 24, 32, 35] based on Michels' classification. Like our study, most of these radiologic investigations show a higher rate of normal hepatic anatomy or reveal a large percentage of unclassified cases, as for example in the latest publication in 2000. These studies all share the finding of a relatively stable percentage of common hepatic arteries arising from the SMA (Michels type IX), which is in line with the extensive review by Lippert [20], although in other evaluations on cadaver anatomy this rate reaches 7% or 9% [21, 38].

Table 1 also demonstrates a general difference in the number of detected accessory hepatic arteries (Michels type V and type VI), with lower rates of these anatomic patterns in the recent angiographic series. In our experience, this difference is due to the small size of the accessory branches, resulting in lower detectability by angiography. The radiologic difficulty associated with the subdivision into accessory or replaced aberrant hepatic arteries may well result in underestimation of the accessory arteries in angiography in general.

Because of the methodologic difficulties in angiographic differentiation between accessory and replaced arteries, the classification introduced by Hiatt in 1994 collapses the distinction between these two types [13]. Hiatt distinguishes only six categories: the normal anatomy (Hiatt type I), the LHA arising from the LGA (Hiatt type II), the RHA arising from the SMA (Hiatt type III), every combination of a double-replaced pattern (Hiatt type IV), the common hepatic artery originating as a branch of the SMA (Hiatt type V), and the abnormality consisting of an isolated aortic origin of the common hepatic artery, which Hiatt introduced as type VI instead of including the rare variant with a common hepatic artery arising from the LGA.

Hiatt's modified and simplified classification has been applied in many subsequent series, as shown in Table 2, which presents three angiographic studies [13, 16, 27] and three studies on transplantation grafts [22, 34, 36].

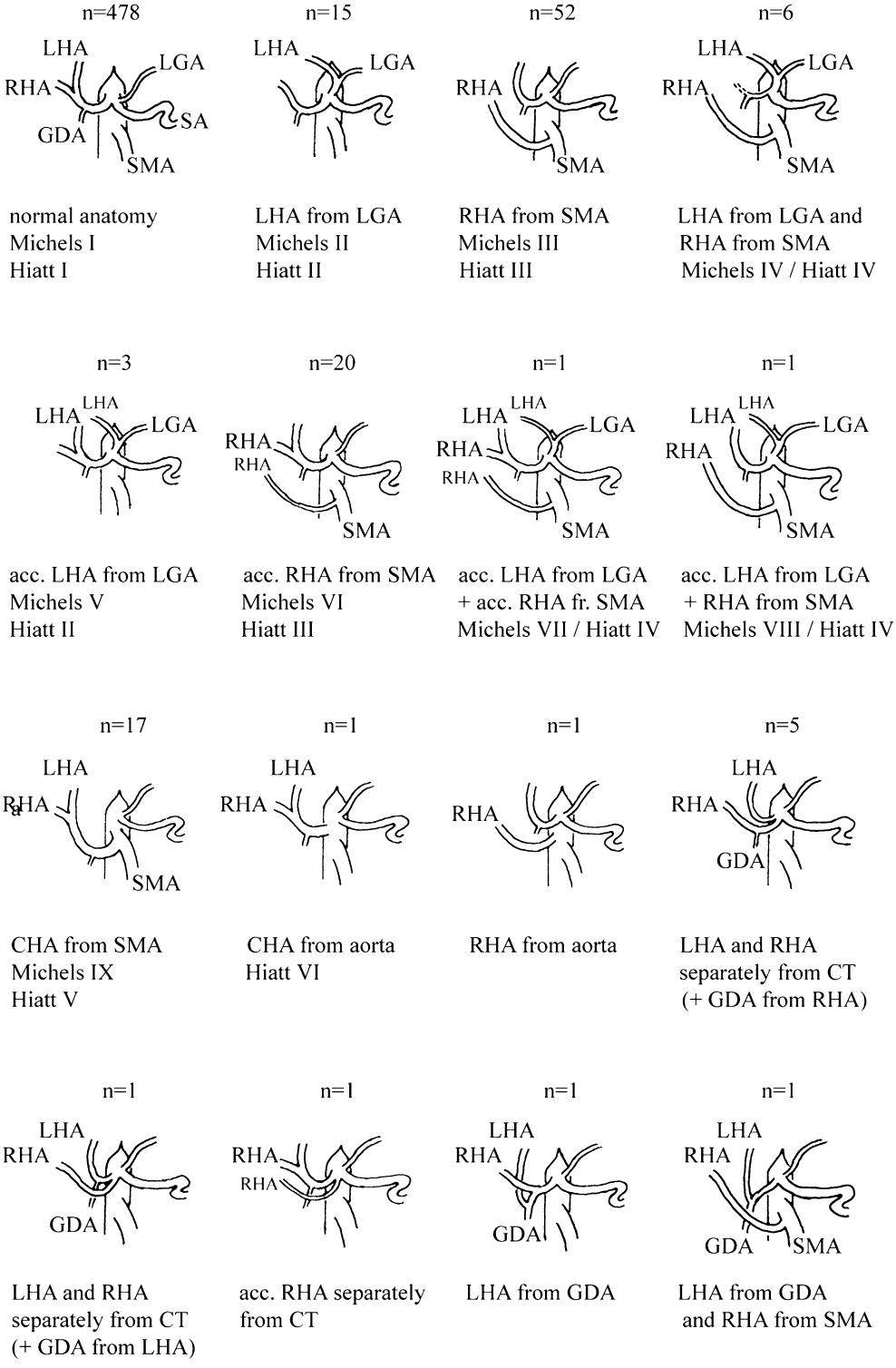

Fig. 1 Schematic anterior view of the upper abdominal aortic branches. Variants as found in our study ($n = 604$). *LHA*, left hepatic artery; *RHA*, right hepatic artery; *LGA*, left gastric artery; *SMA*, superior mesenteric artery; *GDA*, gastroduodenal artery; *SA*, splenic artery; *CT*, celiac trunk

By comparison, our current study shows a relatively low incidence of aberrant LHAs, which might be due to Hiatt type II patterns remaining unidentified among the cases of normal anatomy. Since there is a generally wide deviation in the rate of aberrant LHA from the LGA in radiologic publications, it is assumed that the reason lies in the more difficult angiographic identification of the LGA itself [28].

However, the incidences in our study of the rare anomalies not classified by either Michels or Hiatt do not differ from those reported in other publications. Cases of a separate origin of an aberrant hepatic artery

Table 1 Percentage of hepatic arterial variation according to Michels' classification

Type	Current series ($n=604$) Angiographies	Michels, 1966 ($n=200$) Autopsies	Suzuki, 1971 ($n=200$) Angiographies	Daly, 1984 ($n=200$) Angiographies	Rygaard, 1986 ($n=216$) Angiographies	Chen, 1998 ($n=381$) Angiographies	De Santis, 2000 ($n=150$) Angiographies
I	79.1%	55.0%	70.5%	76.0 %	75.5%	80.3%	52.0%
II	2.5%	10.0%	8.0%	4.0%	4.6%	7.8%	10.0%
III	8.6%	11.0%	3.5%	6.0%	13.4%	5.2%	15.5%
IV	1.0%	1.0%			0.9%	0.7%	0.6%
V	0.5%	8.0%	4.5%	3.5%	0	1.3%	0.6%
VI	3.3%	7.0%	4.0%	4.0%	0	1.5%	2.0%
VII	0.2%	1.0%			0.5%	0.5%	0.6%
VIII	0.2%	2.0%			0.5%	0	0
IX	2.8%	4.5%	3.0%	2.0%	1.4%	1.6%	4.0%
X	0	0.5%			0	0	0
Not classified	1.8%		6.5%	6.0%		1.1%	14.7%

Table 2 Percentage of hepatic arterial variations according to Hiatt's classification

Type	Current series ($n=604$) Angiographies	Hiatt, 1994 ($n=1000$) Angiographies	Niederhuber,1983 ($n=111$) Angiographies	Kemeny, 1986 ($n=100$) Angiographies	Todo, 1987 ($n=211$) Liver grafts	Mäkisalo, 1993 ($n=100$) Liver grafts	Soin, 1996 ($n=527$) Liver grafts
I	79.1%	75.7%	73%	59%	64.5%	76%	69.4%
II	3.0%	9.7%	10%	17%	12.8%	7%	14.2%
III	11.9%	10.6%	11%	18%	9.9%	7%	8.7%
IV	1.3%	2.3%	2%	2%	3.2%	3%	2.7%
V	2.8%	1.5%		3%	5.0%	3%	2.3%
VI	0.2%	0.2%		0%	0	0	0.2%
Not classified	1.7%		5%	1%	4.1%	4%	2.5%

arising directly from the aorta make up 1.0–1.7% of those reported in other angiographic studies [28, 31, 35]. In former anatomic studies this variation was mentioned in up to 7.5% [2, 6] of the cases examined, as a result of the more detailed visual image of the celiac trunk obtained in anatomic dissection compared with standard aortography displaying the celiac trunk in only one or a few projections.

The unusual pattern of aberrant or duplicated hepatic arteries originating separately in the celiac trunk is described by other authors as occurring in up to 2.0–5.4% of cases [1, 2, 6, 13, 20, 31, 38]. The reason for the complete lack of this variant in some studies may be that it is easily overlooked if the tip of the catheter is positioned too far in the celiac trunk in selective angiographies and the procedure lacks an adequate aortography [10, 39].

To our knowledge, the two anomalies with hepatic arteries arising from the GDA have not previously been reported. Only a few authors mention hepatic arteries branching off the GDA. Rong described one accessory RHA arising from the GDA in 120 angiograms [31], and Soin published the first case of an accessory LHA from the GDA with a typical common hepatic artery and an accessory RHA from the SMA [34]. A replaced LHA as a constituent of the distal GDA originating in the celiac trunk, in combination with the replaced RHA arising from the SMA, is shown in Fig. 2. This case provides an excellent example of the high surgical significance of this anomaly, since in gastric and especially pancreatic operations, when the GDA is usually ligated, infarction to the left liver lobe could be caused.

In determining the extent of possible ischemic complications related to the loss of hepatic arteries it is crucial to know whether an aberrant artery can be considered "accessory" or "replaced." Early studies [24] claimed that the intrahepatic arterial branches are essentially end-arteries with a lack of arterial collaterals when the extrahepatic anatomy is normal. But subsequent autopsies and embryologic studies have shown that even accessory arteries represent a specific territory of the liver [11, 25], which suggests that they should be considered as replacing arteries as well [4].

However, angiographic studies before and after therapeutic arterial ligation in primary liver tumors [17] and corrosion cast models have also demonstrated a limited collateral arterial supply by aberrant arteries at the hilus [30]. In addition, there are known anastomoses by capsular vessels and possible translobe collaterals. These collaterals are observed no later than 10 h after arterial ligation [23]. After left lateral hepatectomy in patients in whom liver segment IV was perfused via the resected LHA, atrophy developed in less than half the cases, which shows that there must be portal venous branches or collaterals that maintain perfusion [18].

Nevertheless, ligation of minor accessory hepatic arteries can only be compensated to a limited degree. In liver surgery, the objective of keeping aberrant arteries

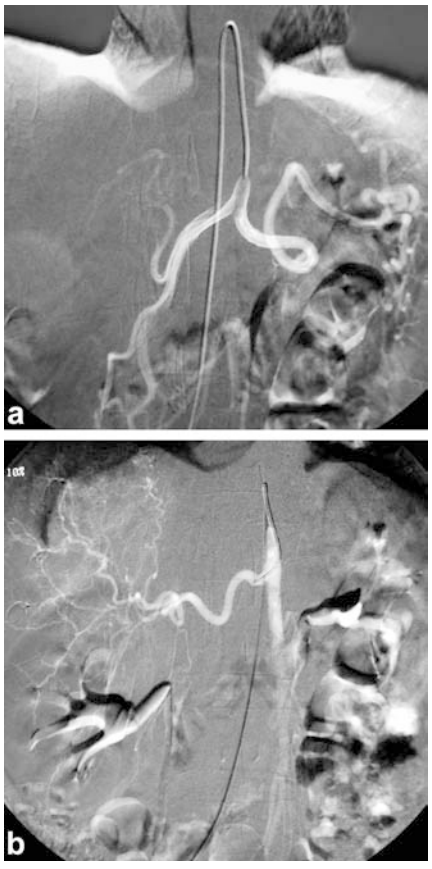

Fig. 2 Selective celiac (**a**) and mesenteric (**b**) angiograms of one patient. **a** Replaced left hepatic artery arising from a gastroduodenal artery that originates separately from the celiac trunk. **b** Replaced right hepatic artery from the superior mesenteric artery

or performing sufficient reconstruction is called into question by an increased risk of hepatic arterial thrombosis involved in the additional anastomoses of small vessels [37]. In living-related liver transplantation this problem has led to different attitudes in dealing with dual-artery supply to the left lobe. Some surgeons favor a reconstruction of all branches and have reported a reduction in the rate of hepatic artery thrombosis when using microvascular surgery [26]. Others reconstruct only the thickest branch if in the smaller branch pulsatile back-flow of blood is observed after revascularization of the main branch [14] or if there is back-flow inside the smaller branch while perfusing the graft with Ringer's solution during a back-table procedure [19]. In the comparison of their techniques with the reconstruction of all branches, these authors have reported no differences in the rates of hepatic artery thrombosis. Still other surgeons even choose a two-step strategy for an enlargement of the left arterial branch, ligating the smaller branch in a first surgical procedure 1 week before graft harvesting [9].

Anatomic variations consisting of replaced hepatic arteries with a larger diameter, however, can also be of advantage in liver surgery. For example, a replaced LHA may allow a rapid dissection of the porta hepatis or may, because of its length, serve as an ideal artery for anastomosis in left lobe liver transplantation.

In other surgical procedures the risks and consequences of aberrant hepatic arteries depend on the vessel's course. The LHA arising from the LGA typically runs to the right with the cranial part of the lesser omentum, entering the liver through the fissure of the venous ligament. Yet, often the abnormal artery takes a tortuous course, running along different paths relative to the esophagus, stomach or hilar structures [22, 34]. While passing the lesser omentum, it is endangered during gastrectomy and hernia repairs. Here, the recognition of the aberrant vessel is crucial to its protection, since even in gastrectomy because of gastric cancer it was shown that leaving the aberrant hepatic artery and the proximal LGA has the same oncologic effect as complete ligation of the LGA [40].

The aberrant RHA, which typically arises from the SMA, runs upward behind the pancreas and dorsal to the portal vein into the hepatic pedicle. The replaced vessel always branches off the cystic artery. Because the aberrant RHA often runs in a low and twisted manner close to the cystic duct and gallbladder, in laparoscopic cholecystectomy there is not only a risk of hepatic infarction due to accidental ligation or clipping, but also a risk of bleeding complications [33]. As the aberrant RHA from the SMA passes through the pancreatic head and as pancreatic resections often involve the peripancreatic vessels for oncologic reasons, injury to the hepatic blood supply is much more common in cases of arterial abnormalities. Consequently, in surgery of the pancreas recognition of hepatic arterial anatomy is important not only in resection but also in planning. As long as aberrant arteries are not involved in a tumor, preoperative angiography can lead to strategies to preserve or reconstruct the vessels [31].

Moreover, stenosis of the celiac trunk, which was found in our study as a peripheral aspect of arterial abnormality, becomes crucial in pancreatic surgery, because the retrograde collateral blood flow through pancreaticoduodenal arcades compensates celiac stenosis. The resection of these arcades, as required in Whipple's operation, could cause ischemia to all organs depending on the celiac axis. To avoid hepatic ischemia resulting from such celiac trunk stenoses in patients undergoing liver transplantation, transection of the median arcuate ligament or even an interposition of an iliac graft from the supraceliac aorta have been proposed [15].

Conclusion

Even if many of the described complications due to hepatic anatomic variations can be prevented or overcome by modern surgical techniques, knowledge of the range of arterial anomalies and their specific frequencies is of greater importance than ever for every visceral surgeon, as well as for the diagnostic and interventional radiologist.

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