

Hepatic arterial variations and liver-related diseases of 100 consecutive donors

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Abstract. We prospectively studied anatomical variations and diseases of the liver in 100 consecutive donor operations during a period of 1 year. The "normal" arterial anatomy with a single hepatic artery (HA) from the celiac trunk was seen in 76 % of all cases. Seven of twelve different major variations of the HA may be considered as "rare", one of which cannot be found in the earlier literature. During harvesting, 6 % of the livers were discarded, 3 % on the basis of infection and 1 % because of a polycystic disease. Two cases were rejected as the liver was found to be severely hypoperfused or hypoxic in an otherwise stable donor. Severe steatosis was macroscopically and histologically diagnosed in 3 % of the cases, and in three donors a benign tumour was found in the liver or in the gall bladder. Two primarily nonfunctioning livers in the present series of 94 recipient operations were retrieved from this group of severely steatotic livers. As the donor liver was totally "normal" in only 2 out of 3 of the cases, the present study underlines the importance of searching for extremely variable anomalies of the HA and for liver-related diseases during organ harvesting.

Key words: Hepatic artery – Liver transplantation – Liver donor, vascular anatomy

Introduction

Having established itself as a routine procedure, liver transplantation now involves two different operations performed by two different groups of surgeons. The one drawing increasing attention these days is that in which "local" teams retrieve livers for distant centres. At least as much attention needs to be drawn to the status, diseases and anatomy of the liver, especially as 10 % of all

grafts are still lost as a result of arterial complications [15]. With the increasing demand for liver transplantations, older and more unstable patients are now being accepted as donors, and the decision of whether or not to reject their organs is more often being made at the time of retrieval.

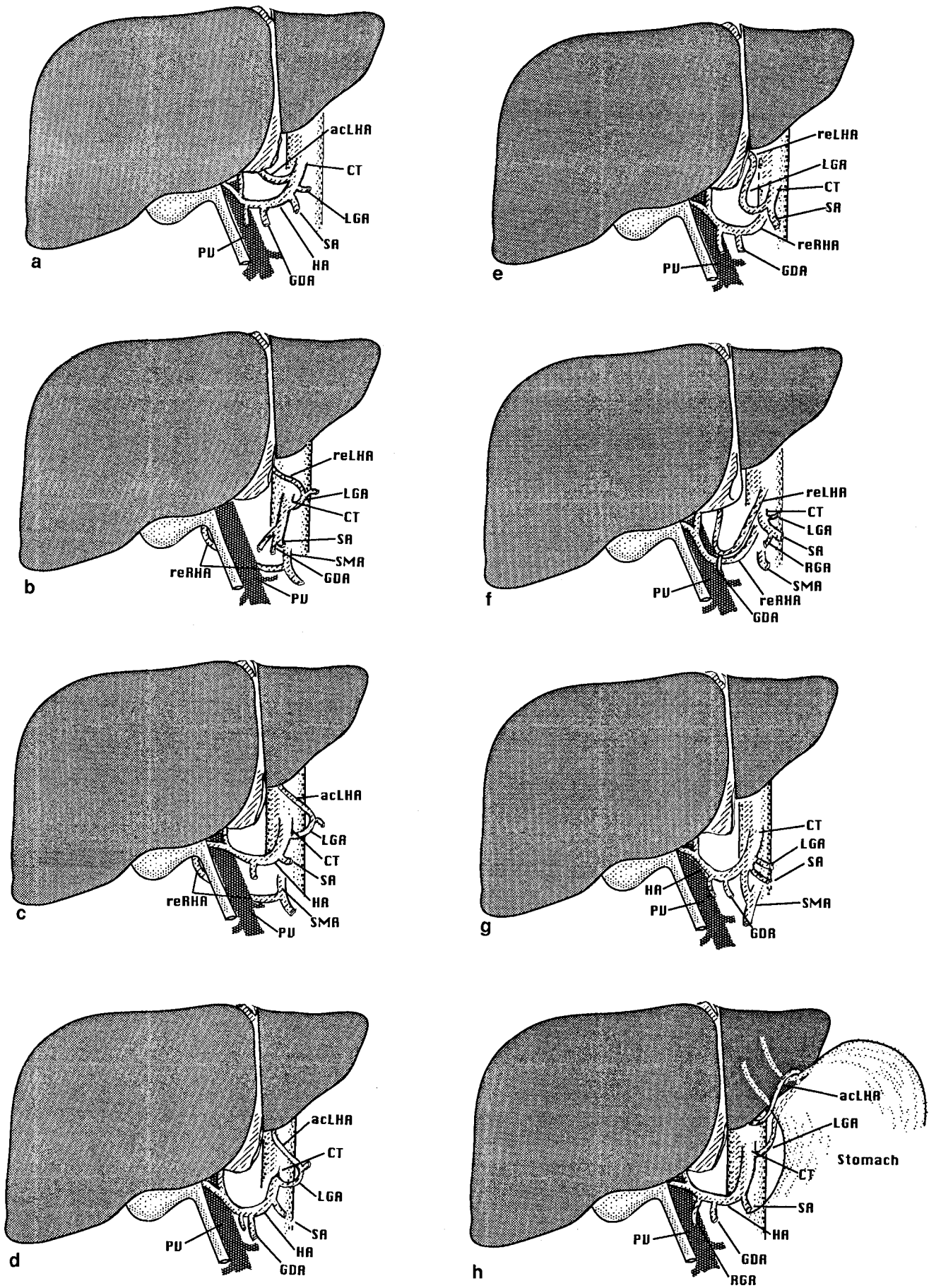
Because of the often detrimental consequences of a damaged hepatic artery (HA) during harvesting, the donor surgeon should be aware of all possible variations of the arterial blood supply to the liver. The major HAs are particularly prone to an anomalous course. In several analyses made on cadaveric dissections, arteriograms and liver donor operations [2, 3, 5–7, 9, 10, 12, 14], the arterial blood supply to the liver followed the "normal" textbook anatomy in 55 %–77 % of the cases. According to earlier studies, appropriately noted and reconstructed arterial variations in the HA of the donor liver did not affect graft survival [3–5, 14].

Although earlier studies on the hepatic arterial anatomy were based on large samples, the data were often collected over a considerable period of time or the donor operations were performed by several surgeons. We decided to estimate the quality of our present donor livers by evaluating 100 consecutive donors operated on by the same team of surgeons during a period of 1 year. Special attention was paid to the anatomical variations and diseases of the liver.

Materials and methods

One hundred consecutive liver donor patients in the Cambridge liver transplantation series from April 1991 to March 1992 were prospectively analysed with regard to anatomical variations and liver-related diseases. To avoid any confusion about the origin of the anomalous arteries, the HA was dissected free in situ up to the celiac trunk and to the aorta as variations were noted. The anatomy was regarded as "normal" when a single proper HA entered the liver through the celiac trunk. Variations of arterial branches distally from the gastroduodenal artery (GDA) were not considered in the present study. The size of an anomalous artery determined whether it was regarded as "replaced" (re-) or as an "accessory" (ac-).

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Although all of the macroscopically abnormal findings of the donor livers were biopsied during harvesting, the decision to discard the organ during the procedure was always based on inspection of the organ.

Results

The "normal" arterial anatomy, with a single HA from the celiac trunk, was seen in 76 % of the cases. The most commonly seen arterial variations – an accessory or replaced right hepatic artery (RHA) from the superior mesenteric artery (SMA) or a left hepatic artery (LHA) from the left gastric artery (LGA), as isolated variations – were seen in only 7 % and 4 % of all cases, respectively (Table 1). If combined arterial variations of the HA are also included in the figures, an artery to the liver originated in 13 % of the cases from the SMA and in 11 % of the cases from the LGA (Tables 1, 2). The whole arterial blood supply to the liver originated from the SMA in 3 % of all cases (Table 1). According to earlier literature, nearly half of the arterial abnormalities seen in the study may be considered as "rare" (Table 1) and they are illustrated in Fig. 1.

During the harvesting, the liver was discarded in 6 % of all cases (Table 4). In 3 % the reason was infection and in one case a polycystic disease. In two preoperatively stable

donors, the liver was gravely dark coloured, a sign of severe hypoxia, from the beginning of the harvesting (Table 3). In three cases the liver seemed to be macroscopically severely steatotic. These livers weighed over 2 kg and histologically more than 50 % of the hepatocytes were filled with large, fat droplets. Two non-functioning livers noted among the 94 recipients in the present series belonged to this group of severely steatotic donor livers. None of the three liver-related benign tumours diagnosed led to rejection of the organ.

Discussion

As in earlier studies on liver donors [3–5, 14], we found anomalous main HAs in 24 % of the cases, which is clearly a lower figure than that seen in series based on autopsy material or arteriograms [2, 6, 7, 9, 10]. This fact is somewhat difficult to explain, although the origin of a single HA may be misinterpreted by the donor liver surgeon, especially as the rapid technique for liver harvesting is used and the anatomy is clarified on the back table [11]. However, the most common variations in autopsy and angiographic studies [7, 9, 10] are the LHA originating from the LGA, in up to 25 % of all cases [9, 10] and the RHA off the SMA, in up to 20 % of all cases [7] and these anomalies are very unlikely to be overlooked during the donor operation. In addition, it is suggested that spontaneous cerebral haemorrhage, which is clearly over-represented in the donor material (40 % in the present material), is associated with arterial variations of intra-abdominal parenchymal organs. An arterial variation was noted in 29 % of this group, compared to 20 % among brain-dead patients from other causes.

In spite of the relatively small number of cases included in the present study, attention should be paid to the comparatively high number of "rare" variations of the HAs. Seven of 12 different extrahepatic arterial variations and one gall bladder abnormality in the present material may be considered rare according to descriptions in the earlier literature. One of the hepatic arterial branches or the common HA has been found to originate directly from the aorta, like the RHA in one of our cases (Fig. 1 e), in 0.5 %–2 % of all cases [3, 5, 12, 14]. Yet, both the RHA and LHA coming directly from the aorta (Fig. 1 f) is something that seems to be found far less frequently.

Earlier studies [10] have clearly shown a lack of arterial collaterals inside the liver when the extrahepatic arterial anatomy is "normal". Reduction of our liver, shown in Fig. 1 i, revealed a strong intrahepatic connection between abnormally located right and left HAs [9].

The celiac trunk and the SMA may have a common origin from the aorta (incidence approximately 1 % [9, 14]), or the trunk may have become a fibrotic string, as in our case (Fig. 1 d). The arc of Bühler, i.e. a link vessel between the HA and the SMA, is not so unusual [1, 9]; a seemingly more extraordinary situation is the case in Fig. 1 g, where a strong communicating artery between the celiac trunk and the SMA was found.

A review of the literature would make it seem as if ours is the first report of the case described in Fig. 1 h.

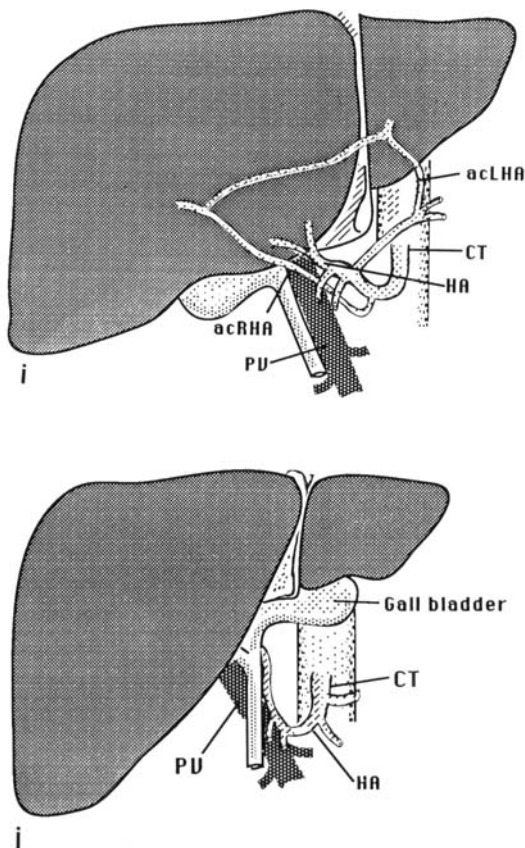


Fig. 1a–j. "Rare" variations of extrahepatic arterial tree. *re*, Replaced; *ac*, accessory; *HA*, hepatic artery; *RHA*, right hepatic artery; *LHA*, left hepatic artery; *RGA*, right gastric artery; *LGA*, left gastric artery; *SMA*, superior mesenteric artery; *CT*, celiac trunk; *SA*, splenic artery; *GDA*, gastroduodenal artery; *PV*, portal vein

Table 1. Arterial and gall bladder variations of 100 liver donor patients. HA, Hepatic artery; RHA, right hepatic artery; LHA, left hepatic artery; LGA, left gastric artery; SMA, superior mesenteric artery; GDA, gastroduodenal artery

| "Common" variations | No./% |
|--|-------|
| re/acRHA off SMA | 7 |
| re/acLHA off LGA | 4 |
| HA off SMA | 3 |
| "Rare" variations | |
| RHA off SMA, LHA off LGA | 2 |
| RHA off SMA, LHA off celiac trunk, acLHA off LGA | 1 |
| RHA off aorta, LHA off aorta or LGA | 2 |
| RHA off celiac trunk, LHA off LGA, intrahepatic communication of RHA and LHA | 1 |
| acLHA off celiac trunk | 1 |
| acLHA off LGA around the oesophagus | 1 |
| HA off anastomosis connecting celiac trunk to SMA | 1 |
| Celiac trunk occluded, blood supply via GDA (SMA), acLHA off LGA | 1 |
| Total | 24 |

Table 2. Origin of the anomalous hepatic artery in 100 liver donors. Abbreviations as in Table 1

| | No./% |
|------------------------------|-------|
| SMA (RHA/HA) | 13 |
| LGA (LHA) | 11 |
| Aorta | 3 |
| SMA + celiac trunk (1 trunk) | 1 |

Table 3. Diseases discovered during the operation of 100 liver donor patients

| | No./% |
|---------------------------------------|-------|
| Severe steatosis | 3 |
| Gangrenotic cholecystitis (no stones) | 2 |
| Cholecystolithiasis | 1 |
| Polycystic disease | 1 |
| Haemangioma | 1 |
| Hamartoma in gall bladder | 1 |
| Adenomyoma in gall bladder | 1 |
| Total | 10 |

Table 4. Donor livers rejected after inspection ($n = 100$)

| | No./% |
|------------------------------|-------|
| "Hypoperfused/hypoxic" liver | 2 |
| Gangrenotic cholecystitis | 2 |
| Polycystic disease | 1 |
| Purulent pleuritis | 1 |
| Total | 6 |

The situation would be extremely dangerous for a gastric surgeon to face. An acLHA, typically situated in the hepatogastric ligament, was surprisingly found to come behind and around the oesophagus, originating from the LGA. Both of the last two arterial variations seem to have an obvious embryological explanation. According to the theory of Tandler [13], the celiac trunk and the

SMA develop as cephalic roots of vitelline or omphalomesenteric arteries, which are joined with a ventral para-aortic anastomosis between the 4th and 7th gestational weeks. In contrast to normal development, in the case illustrated in Fig. 1g, this anastomosis seems to have persisted all the way into adulthood. The embryological development of the liver starts in the 3rd gestational week as a hepatic diverticulum of the foregut [8]. The dorsal arteries join to form the aorta, from which paired branches provide a supply of blood to both the foregut and the liver bud. Since the primitive vitelline arteries form the celiac trunk and the SMA, one of these paired branches to the left of the foregut seems to have persisted in our case (Fig. 1h), creating a branch surrounding the gut. Following the development of the stomach during the 5th gestational week, the artery may have dislodged to the oral direction, thus surrounding the oesophagus. In our case, the obvious solution for retrieving the liver was transection of the oesophagus to preserve a single common artery to the liver.

Although one does not expressly look for variations of the biliary tree during the donor operation, gall bladder abnormalities are usually noticed. The variation in our material – the gall bladder attached to the left liver lobe (Fig. 1j) – is not exceptional [9] but may cause trouble if it is connected to a segmental abnormality and needs to be cut down, as was the case in our series.

Because of the infrequency of silent liver-related diseases in the normal population, usually only liver function tests of donors are determined preoperatively. Thus, liver tumours or other abnormalities may be found only during the operation. Even infections in brain stem-dead patients may be very insidious and difficult to diagnose with routine blood tests. In 10% of our cases, a liver-related disease was noted during the harvesting procedure. The discovery of two benign gall bladder tumours and one haemangioma did not change plans for transplantation but advanced polycystic disease did lead to our discarding both the kidneys and the liver of one patient. Two other donors, one of whom was 3.5 years old, had gangrenous cholecystitis after spending 5 days in the intensive care unit. These livers were also discarded.

Three out of 100 donor livers looked macroscopically gravely steatotic, and histologically over 50% of the hepatocytes in these livers were filled with fat droplets. These three livers were used because of an urgent need and two of them were primarily non-functioning, something which was not observed among the other 92 recipients. Although the biopsies had established the grave hepatic steatosis in these cases, the diagnosis and the decision not to use these livers could have been done macroscopically as well.

The present study underlines the importance of searching for extremely variable anomalies of the HA during the process of organ harvesting. In spite of a relatively high number of liver-related diseases in the present material, excluding a fatty liver they caused only little difficulty in deciding whether or not to use the organ. Thus, special preoperative examinations of the donor liver using methods such as ultrasound or CT may not be thought appropriate.

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