

Research article - Basic and applied anatomy

Hepatic surface grooves in an Afro-Caribbean population: a cadaver study

Michael T. Gardner¹, Shamir O. Cawich^{2*}, Ramanand Shetty¹, Neil W. Pearce³, Vijay Naraynsingh²

¹ Department of Clinical Surgical Sciences, University of the West Indies, St. Augustine Campus, St. Augustine, Trinidad & Tobago; ² Section of Anatomy, Basic Medical Sciences, University of the West Indies, Mona Campus, Kingston 7, Jamaica; ³ University Surgical Unit, Southampton General Hospital, Southampton, United Kingdom

Submitted January 20, 2015; accepted revised March 9, 2015

Abstract

Introduction: There have been no previous reports on hepatic surface grooves in an Afro-Caribbean population. This information is important to optimize radiology and hepatobiliary surgical services in the region.

Methods: Two investigators independently observed 69 cadaveric dissections performed over five years at the University of the West Indies. Variations in surface anatomy were described.

Results: In this Caribbean population the majority of patients had conventional hepatic surface anatomy (88%). However, we found a greater incidence of hepatic surface grooves (12%) than reported in international literature.

Conclusion: Abnormal surface anatomy is present in 12% persons in this population. Interventional radiologists and hepatobiliary surgeons practicing in the Caribbean must be cognizant of these differences in order to minimize morbidity during invasive procedures.

Key words

Liver, Variants, Grooves, Surface, Caribbean

Introduction

Reports of variations in ductal and vascular anatomy of the liver are commonplace, but few authors have reported the presence of hepatic surface grooves (HSG) in the medical literature (Auh et al., 1984; Lim et al., 1987; Macchi et al., 2003; Newell and Morgan-Jones, 2005; Othman et al., 2008). These are prominent vertical depressions that appear on the antero-superior surface of the liver. There is no consensus on nomenclature since they are known by several names, including accessory sulci (Othman et al., 2008), accessory fissures (Auh et al., 1984), hepatic furrows (Newell and Morgan-Jones, 2005), hepatic fissures (Lim et al., 1987) and diaphragmatic sulci (Macchi et al., 2003).

This study was carried out to describe the presence of HSG in an Afro-Caribbean population. This has not been previously reported and the information is important in light of the rapid progress in hepatobiliary services in the Caribbean over the past decade (Cawich et al., 2014).

* Corresponding author. E-mail: socawich@hotmail.com

Methods

The local institutional review board granted approval to carry out cadaver dissections as a part of anatomical teaching for post-graduate surgical residents at the University of the West Indies in Kingston, Jamaica. This institution is a major referral centre for northern Caribbean countries. It was previously established that these findings were representative of anatomy in the general population in the Anglophone Caribbean (Johnson et al., 2013).

Over five years, we observed the dissections of 69 consecutive cadavers. Upon opening the abdomen, the liver was inspected *in situ*. Cadavers were considered to have normal surface anatomy when the morphology was in keeping with classic descriptions (Sibulesky, 2013): the falciform ligament attaches to the anterior border of the liver and merges into the left and right superior coronary ligaments. There are no grooves present on the diaphragmatic surface of the liver. Cadavers with abnormal liver surface anatomy were identified and selected for detailed evaluation.

When abnormal HSG were observed, we documented the *in-situ* relationships to nearby organs, including ribs and diaphragmatic slips. The livers were then explanted by interrupting the triangular and coronary ligaments, transecting the supra-duodenal free edge of lesser omentum and transecting the inferior vena cava at least 2 cm above the insertion of hepatic veins and 2 cm below the lower border of the liver.

Each specimen was observed on the dissection bench. The number, location, depth and length of all abnormal HSG were recorded. All measurements were taken with electronic calipers (General Tools, MFG Co., New York, USA) and checked independently by two investigators. The average measurement was used as the final dimensions. The livers were then sectioned to document the relationship of HSGs to parenchymal structures.

Results

There were 69 cadavers dissected over the study period. Normal surface anatomy was present in 61 specimens and the remaining 8 (12%) had HSG present. The estimated mean age of cadavers with surface grooves was 68 years (range 55-85) and 7 (88%) were males.

The grooves were located antero-superiorly on the convex surface of the liver. All the specimens had HSG on the surface of segment IVa, five had HSG on the surface of segment VIII and only 1 specimen had an HSG at segment III. On *in-situ* inspection of these livers, the diaphragm corresponding to the areas of the HSGs appeared normal. No muscular hypertrophy, scarring or fibrotic slip were noted in the diaphragm of any of these cadavers. The coronary, triangular and falciform ligaments were normal in all specimens. No anatomic abnormalities or supernumerary ribs were present in the thoracic wall.

There was a wide variation in the number, length and depth of the HSG as outlined in table 1. Six (75%) specimens had 3 or more grooves on the diaphragmatic surface and most were well-defined deep grooves with a mean length of 4.6 cm and a mean depth of 1.8 cm (Figs. 1 and 2).



Figure 1 – Anterior view of an explanted cadaveric liver. Multiple vertical well-defined hepatic surface grooves are demonstrated.



Figure 2 – Superior view of the explanted cadaveric liver demonstrating the vertical well-defined hepatic surface grooves.

Table 1 – Accessory hepatic sulci in an Afro-Caribbean population

Index Case	Number of sulci	Location (segment)	Length (cm)	Depth (cm)	Special observations
1	2	IVa	4.0	0.5	None
		VIII	4.0	1.5	
2	4	IVa	5.0	2.0	Under-developed fissure for ligamentum teres
		VIII	6.0	1.9	
		VIII	8.0	2.5	
		VIII	3.0	0.8	
3	1	IVa	3.0	1.0	Macro-nodular cirrhosis
		IVa	5.0	2.0	
4	5	VIII	6.0	2.1	None
		VIII	8.0	2.9	
		VIII	3.0	0.5	
		VI	2.0	1.5	
		IVa	2.5	1.0	
5	3	VIII	3.0	2.0	None
		VIII	4.5	2.5	
		VI	2.0	1.9	
		IVa	3.0	0.9	
6	3	VIII	6.0	2.3	None
		VIII	4.0	2.0	
		VI	4.0	2.0	
		IVa	5.0	2.0	
7	3	VIII	6.0	2.9	None
		VIII	8.0	2.5	
		VI	1.5	1.0	
		III	2.0	0.5	
8	3	IVa	2.5	1.0	Left lateral section hypertrophied. No parenchymal lesions
		IVa	4.0	3.0	

Four cadaveric livers (50%) also had distinctive grooves on the visceral surface. Interestingly, the visceral grooves were all on the right, fewer in number and relatively consistent in their location. The distinctive grooves were located on the visceral surface of segment VI (Figure 3). When present, they were well developed with a mean depth of 1.5 cm and a mean length of 2.4 cm. On in-situ examination, there were no abnormalities of adjacent viscera in any of the specimens. The colic, renal and duodenal impressions appeared normal.

We had no information on the cause of death or about pre-morbid diseases for these cadavers. However, the cadaveric livers appeared grossly normal on sectioning in 6 cases. Two cadavers had notable clinico-pathologic abnormalities. One cadaver (Index case 3) had macro-nodular cirrhosis (Fig. 4). Another (Index case 8) had a markedly hypertrophic left lateral section but no other gross pathologic diseases (Fig. 5). None of the specimens had intra-parenchymal liver pathologies detected on sectioning.

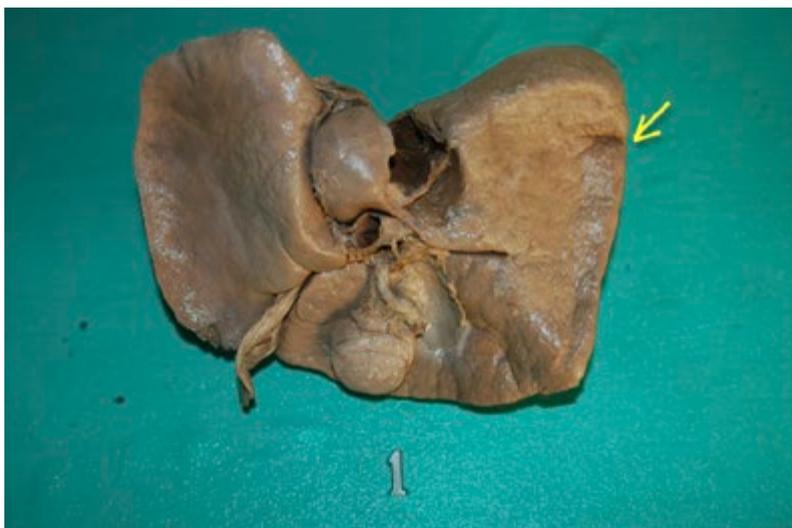


Figure 3 – Inferior view of the visceral surface of an explanted cadaveric liver. The yellow arrow demonstrates the consistent transverse groove that was present on the visceral surface of segment VI.



Figure 4 – Explanted cadaveric liver from index case 3. Macro-nodular cirrhosis is present and a well-defined hepatic surface groove is noted.



Figure 5 – Explanted cadaveric liver of index case 8. A markedly hypertrophic left lateral section can be seen. No pathologic lesions were noted on sectioning.

Discussion

In classic descriptions (Sibulesky, 2013), the falciform ligament attaches to the anterior border of the liver and merges into the left and right superior coronary ligaments. There are no sulci present on the diaphragmatic surface of the liver (Fig. 1). This pattern was seen in 88% cadavers.

There were variations in surface anatomy in 12% unselected cadaveric livers. The awareness of HSGs has become increasingly relevant since cross-sectional imaging of the abdomen is now routine in modern medicine, and especially in light of the rapid progress in hepatobiliary services in the Caribbean in the past decade (Cawich et al., 2014). Their presence may lead to misinterpretation of imaging because HSGs can mimic pathologic liver lesions (Auh et al., 1984), liver lacerations in trauma patients (Othman et al., 2008), normal sulci when planning liver resections (Johnson et al., 2013), and increase the complexity of liver transplantation (Alonso-Torres et al., 2005). Therefore, it is relevant to radiologists for image interpretation, surgeons for planning resections and oncologists during post-operative surveillance.

The prevalence of HSG in this Afro-Caribbean population is comparable with that in published series, ranging from 5% in Malaysia (Othman et al., 2008), 6% in India (Joshi et al., 2009), 25% in New York, USA (Auh et al., 1984), to 40% in Italy (Macchi et al., 2003).

The majority of HSGs occurred at the supero-anterior surface near the midline, most commonly involving segments IVa and VIII. Similar patterns have been reported in medical literature where the HSG are commonest on either side of Cantlie's line (Auh et al., 1984; Lee et al., 2012). Some have postulated that this may be due to pressure on the hepatic surface from pulmonary emphysema (Schumaker, 1997) or diaphragmatic slips. Macchi et al (2003) described the presence of "weak zones"

on the superficial aspect of hepatic parenchyma that offer low resistance to external pressure. The prevailing theory is that projections from the diaphragm, known as diaphragmatic slips, exert pressure on this surface of the liver, creating HSGs (Yang et al., 2002; Macchi et al., 2003; Macchi et al., 2005; Auh et al., 2008; Joshi et al., 2009). Newell et al (2005) noted that diaphragmatic slips were usually located between the right and the anterior surface, with a course and concavity towards the falciform ligament, corresponding to 'radial' grooves from the diaphragm. However, we could not identify any diaphragmatic abnormalities during our in situ examinations on cadavers with HSGs. If they were related to diaphragmatic slips, we would have expected to see focal or radial thickening of the diaphragmatic muscle in relation to the grooves. These were not present in any of our specimens. No fibrotic bands, scarring or hypertrophic bands were noted to account for these sulci. The ligament attachments were also normal in all cadavers, ruling out the possibility of indentation by ligaments.

Others have suggested that these are post-mortem changes due to external compression from the ribs, explaining the increased frequency in cadaveric studies (Auh et al., 1984; Newell and Morgan-Jones, 1993) and the increased prevalence on the diaphragmatic compared to the visceral surface of the liver (Ono et al., 2000; Othman et al., 2008). This explanation did not seem plausible in our series as the sulci were not related to the oblique path of the overlying ribs. The sulci on the diaphragmatic surface were vertically oriented in all cadavers.

Furthermore, even if the "diaphragmatic slip" and "rib-compression" theories could explain the HSGs on the diaphragmatic surface, neither can explain visceral grooves. The fact that 50% of our cadavers had co-existing visceral grooves suggests that another etiology is responsible.

Compared to the diaphragmatic HSGs, those on the visceral surface were remarkably consistent. Specifically, there was a constant transverse groove at segment VI. Othman et al (2008) described a transverse fissure posteriorly in segment VI "near the colic impression". Their paper contains photographs of the groove that bear a remarkable similarity to the consistent segment VI HSG that was present in 50% of our cadavers with surface grooves. They suggested that "pressure exerted by the colon" was responsible for this groove. However, we observed that the colon was relatively mobile and contacted a large surface area of the liver. A single consistent fissure as a result of colonic pressure does not seem feasible either. Furthermore, there was no corresponding HSG at the renal, duodenal or gastric impressions. We could not find any obvious relations to this fissure on in-situ examination. Joshi et al (2009) reported a similar groove at segment VI being present in 30% of cadavers with vertical surface grooves. The incidence in our cadavers was higher.

Genetic factors have also been suggested to be responsible (Suksaweang et al., 2004; Macchi et al., 2005; Othman et al., 2008). Suksaweang et al (2004) theorized that the span of activity and shift of growth zones in the liver are dependent on beta-catenin/Wnt activity. They theorized that a failure of genetic guidance at this stage may be responsible for development of HSGs. Unfortunately the observational nature of this study did not allow us to determine whether genetic or developmental factors played a part in the occurrence of HSGs in the Caribbean population. Johnson et al (2013) were the first to propose that ethnicity could play a role in morphologic variations of the liver since the Caribbean population is predominantly black while pub-

lished reports from North American and European centers have predominantly Caucasian populations. However, these theories of a genetic predisposition to anatomic variance in Afro-Caribbean populations would require further research.

An alternate mechanism for HSGs may be a disorder at the embryonic stage during development of the hepatic diverticulum in the fourth week of fetal life. During this period, the hepatic bud grows into the septum transversum and divides dichotomously forming the hepatic lobes (Hassan et al., 2013). This relies on a process known as dynamic signaling (Wandzioch and Zaret, 2009) where hepatocyte proliferation is dependent on the presence of several growth factors including bone morphogenic proteins, transforming growth factor- β , fibroblast growth factors and VEGF-Flk-1 (Wandzioch and Zaret, 2009; Sugiyama et al., 2010). Additionally, through Notch signaling (Hassan et al., 2013) cells adjacent to the developing hepatocytes are stimulated to differentiate into duct cells. Notch signals are required for bile duct morphogenesis, and activation of Notch signaling in the hepatic lobule promotes ectopic biliary differentiation and tubule formation in a dose-dependent manner (Zong et al., 2009). In the absence of these mediators, there would be discoordinated Notch / dynamic signaling leading to disordered growth patterns.

Pathologic grooves may also occur in the liver associated with a variety of malignancies when there is tumor necrosis that leads to involution, desmoplasia and retraction of the hepatic capsule (Yang et al., 2002; Sans et al., 2009). A similar process can occur in patients who have had systemic chemotherapy (Young et al., 1994) and/or transarterial chemical embolization (TACE; Matsui et al., 1993). However, this was not thought to be likely in our setting as there were no parenchymal lesions present on sectioning in any of our cases.

There are instances where a similar process of capsular retraction can occur without tumor. Patients with cirrhosis may develop areas of focal confluent fibrosis in 14% cases (Ohtomo et al., 1993). The resultant fibrosis with parenchymal atrophy may cause capsular retraction leading to wedge shaped areas of retraction, especially in the medial segment of the left lobe or anterior segment of the right lobe (Ohtomo et al., 1993). There was one cadaver in our series with a cirrhotic liver. While it could be postulated that this may have been the cause, the HSG did not have the characteristic wedge-shaped, irregular, desmoplastic appearance that is typical of capsular retraction in cirrhosis. It, in fact, appeared similar to the other HSG that we encountered except that it was on the background of a cirrhotic liver.

Conclusion

Hepatic surface grooves are present in 12% of an unselected Afro-Caribbean population. In this population, there was a consistent groove at the inferior (visceral) surface of the liver at segment VI. This information is important to surgeons, radiologists and oncologists who encounter liver diseases.

Conflict of interest statement

The authors confirm that there are no conflicts of interest to disclose.

Authors' contributions

MG, SOC, RS, NP and VN conceptualized the study. MG and RS carried out observations and collected data; MG, SOC, RS, NP and VN checked the manuscript and endorsed the academic content.

References

- Alonso-Torres A., Fernandez-Cuadrado J., Pinilla I., Parron M., de Vicente E., Lopez-Santamaria M. (2005) Multidetector CT in the evaluation of potential living donors for liver transplantation. *Radiographics* 25: 1017-1030.
- Auh Y.H., Rubenstein W.A., Zirinsky K., Kneeland J.B., Pardes J.C., Engel I.A., Whalen J.P., Kazam E. (1984) Accessory fissures of the liver: CT and sonographic appearance. *Am. J. Roentgenol.* 3: 565-572.
- Cawich S.O., Johnson P.B., Shah S., Roberts P., Arthurs M., Murphy T., Bonadie K.O., Crandon I.W., Harding H.E., Abu-Hilal M., Pearce N.W. (2014) The multidisciplinary team approach to hepatobiliary diseases: a working model in the Caribbean setting. *J. Multidisciplinary Healthcare* 7: 1-4.
- Hassan G.M.A., Sliem H.A., Ellethy A.T., Salama M.E. (2013) Hepatic embryonic development and anomalies of the liver. *J. Gastroenterol. Hepatol. Res.* 2: 489-493.
- Johnson P.B., Cawich S.O., Roberts P., Shah S., Gardner M.T., Gordon-Strachan G., Pearce N.W. (2013) Variants of hepatic arterial supply in a Caribbean population: a computed tomography based study. *Clin. Radiol.* 68: 823-827.
- Joshi S.D., Joshi S.S., Athavale S.A. (2009) Some interesting observations on the surface features of the liver and their clinical implications. *Singapore Med. J.* 50: 715-719.
- Lee J.H., Lee S.Y., Hwang D.W., Park K.M., Lee Y.J. (2012) Practical usefulness of minor grooves of the hepatic surface for liver resection. *Hepatogastroenterol.* 59: 458-460.
- Lim J.H., Tae Ko Y., Han M.C., Kim C.W., Choi B.I., Im J.G. (1987) The inferior accessory hepatic fissure: sonographic appearance. *Am J Roentgenol.* 149: 495-497.
- Macchi V., Feltrin G., Parenti A., De Caro R. (2003) Diaphragmatic sulci and portal fissures. *J. Anat.* 202: 303-308.
- Macchi V., Porzionato A., Parenti A., Macchi C., Newel R., DeCaro R. (2005) Main accessory sulcus of the liver. *Clin. Anat.* 18: 39-45.
- Matsui O., Kadoya M., Yoshikawa J., Gabata T., Arai K., Demachi H., Miyayam S., Takashima T, Unoura M., Kogayahi K. (1993) Small hepatocellular carcinoma: treatment with subsegmental trans- catheter arterial embolization. *Radiology* 188: 79-83.
- Newell R.L.M., Morgan-Jones R. (1993) Grooves in the superior surface of the liver. *Clin. Anat.* 6: 333-336.
- Ohtomo K., Baron R.L., Dodd III G.D., Federle M.P., Ohtomo Y., Confer S.R. (1993) Confluent hepatic fibrosis in advanced cirrhosis: evaluation with MR imaging. *Radiology* 189: 871-874.
- Ono M.L., Murakami G., Sato T.J., Sawada K. (2000) Hepatic grooves and portal segmentation. *Kaibogaku Zasshi* 75: 517-523.

- Othman F.B., Latiff A.A., Suhaimi F.H., Das S. (2008) Accessory sulci of the liver: An anatomical study with clinical implications. *Saudi Med. J.* 29: 1247-1249.
- Sans N., Fajadet P., Galy-Fourcade D., Trocart J., Jarlaud T., Chiavassa H., Giron J., Railhac J.J. (1999) Is capsular retraction a specific CT sign of malignant liver tumor? *Eur. Radiol.* 9: 1543-1545.
- Schumaker U. (1997) Groovy livers. *Clin. Anat.* 10: 144-145.
- Sibulesky L. (2013) Normal liver anatomy. *Clin. Liver Dis.* 2: 1-3.
- Sugiyama Y., Takabe Y., Nakakura T., Tanaka S., Koike T., Shiojiri N. (2010) Sinusoid development and morphogenesis may be stimulated by VEGF-Flk-1 signaling during fetal mouse liver development. *Dev. Dyn.* 239: 386-397.
- Suksaweang S., Lin C.M., Jiang T.X., Hughes M.W., Widelitz R.B., Chuong C.M. (2004) Morphogenesis of chicken liver: identification of localized growth zones and the role of beta-catenin/Wnt in size regulation. *Dev. Biol.* 266: 109-122.
- Wandzioch E., Zaret K.S. (2009) Dynamic signaling network for the specification of embryonic pancreas and liver progenitors. *Science* 324: 1707-1710.
- Yang D.M., Kim H.S., Cho S.W., Kim H.S. (2002) Various causes of hepatic capsular retraction: CT and MR findings: Pictorial review. *Br. J. Radiol.* 75: 994-1002.
- Young S.T., Paulson E.K., Washington K., Gullive D.J., Vredenburgh J.J., Baker M.E. (1994) CT of the liver in patients with metastatic breast carcinoma treated by chemotherapy: findings simulating cirrhosis. *Am. J. Roentgenol.* 163: 1385-1388.
- Zong Y., Panikkar A., Xu J., Antoniou A., Raynaud P., Lemaigre F., Stanger B.Z. (2009) Notch signaling controls liver development by regulating biliary differentiation. *Development* 136: 1727-1739.