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ORIGINAL ARTICLE

Hepatic surface grooves in Trinidad and Tobago

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Abstract

Purpose Hepatic surface grooves (HSGs) are prominent depressions on the antero-superior surface of the liver. We sought to document the prevalence of HSGs in an Eastern Caribbean population.

Methods We observed all consecutive autopsies performed at a facility in Trinidad and Tobago and recorded the presence, number, location, width, length and depth of any HSG identified. Each liver was then sectioned to document intra-parenchymal abnormalities.

Results Sixty Autopsies were observed. There were HSGs in 9 (15%) cadavers (5 females and 4 males), at an average age of 66 years (range 48–83, Median 64, SD ± 10.4). The HSGs were located on the diaphragmatic surface of the right hemi-liver in 8 (89%) cadavers, left medial section in 4 (44%), left lateral section in 3 (33%) and coursing along Cantlie's plane in 3 (33%) cadavers. Eight (89%) cadavers with HSGs had other associated anomalies: accessory inferior grooves (5), parenchymal nutmeg changes (5), abnormal caudate morphology (4), hyperplastic left hemi-liver (3), lingular process (2), bi-lobar gallbladder (1) and/or abnormal ligamentous attachments (1).

Conclusions Approximately 15% of unselected Afro-Caribbean persons in this Eastern Caribbean population have HSGs. Every attempt should be made to identify HSGs on pre-operative imaging because they can alert the hepatobiliary surgeon to: (1) associated anatomic anomalies in 89% of cases, (2) associated hepatic congestion in 56% of persons, (3) increased risk of bleeding during liver resections and (4) increased technical complexity of liver resections. The association between HSGs, cardiovascular complications, hepatic congestion and nutmeg liver prompted us to propose a new aetiologic mechanism for HSG formation, involving localized hyperplasia at growth zones due to upregulation of beta-catenin levels.

Keywords Liver · Grooves · Variant · Anatomy · Surface · Surgery

Introduction

Hepatic surface grooves (HSGs) are prominent vertical depressions on the antero-superior surface of the liver [5]. Their presence is important to radiologists because they may lead to mis-interpretation of cross-sectional imaging studies [1]. They are also important to trauma surgeons as they may mimic liver lacerations, medical oncologists because they can be mistaken for liver metastases in cancer patients and also to hepatobiliary surgeons since they can be used as anatomic landmarks when planning liver resections [1, 5, 8, 14].

The global prevalence of HSGs ranges from 5% in Malaysia to 40% in Italy [8, 14]. One prior study reported a 12% prevalence in the Northern Caribbean [5], but there is no data on the prevalence of HSG in the Eastern Caribbean. This prospective study was designed to document the prevalence of HSGs in an Eastern Caribbean country. We also sought to correlate observational findings in cadavers with HSGs in an attempt to determine possible aetiology of these grooves. The clinical significance of HSGs is also discussed.

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Materials and methods

This study was performed in Trinidad and Tobago, a twin-island nation in the Eastern Caribbean with a population of 1,317,714 persons at the last population census [2]. The data was collected at the Port of Spain General Hospital—the main referral centre for pathology services across the nation [2]. Therefore, we expect the findings of this study to be representative of anatomy in the general population in the island nation of Trinidad and Tobago.

Ethical approval was secured from the local institutional review board and the study was carried out in keeping with the agreements reached in the Declaration of Helsinki, revised in October 2000 as drafted by the World Medical Association. We carried out a prospective study of all consecutive autopsies performed at this facility from September 1 to November 30, 2019. We observed all consecutive autopsies in unselected cadavers. We used the classic anatomic descriptions when evaluating the morphology of each liver [18]. In classic descriptions, 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 on the diaphragmatic surface of the liver in classic anatomic descriptions.

We observed all consecutive autopsies performed during the study period. A longitudinal midline incision was used to open the abdomen and the viscera were observed. Each cadaver with abnormal liver surface anatomy was selected for detailed analysis. Before the liver was explanted, we noted in situ relationships to nearby organs, orientation of the ribs and association with diaphragmatic slips. The liver was then explanted by incising the triangular and coronary ligaments, transecting the supra-hepatic inferior vena cava 2 cm above the superior liver border, transecting the hepatoduodenal ligament just above the pancreatic head and transecting the infra-hepatic inferior vena cava 2 cm below the lower border of the liver. The liver was then moved to the dissection bench where the unfixed liver was examined in detail.

At the dissection bench, two investigators independently recorded the presence, number, location, width, length and depth of any HSG identified. All measurements were taken with a standardized metal ruler and the average measurement was used at the final recorded dimension. Each liver was then sectioned to document the relationship of HSGs to intra-parenchymal structures.

Results

Autopsies were observed in 60 cadavers of Afro-Caribbean (49), Indo-Caribbean (8), Mixed (2) and Chinese (1) descent. There were 9 (15%) cadavers with HSGs, all in persons of Afro-Caribbean descent. These included 5 female and 4 male cadavers, with an average age of 66 years (range 48–83, Median 64, SD ± 10.4). None of the cadavers were known to have existing liver diseases. The documented causes of death included acute myocardial infarction (5), pulmonary embolism (2), strangulated internal hernia with bowel ischemia (1) and intra-abdominal sepsis (1).

The HSGs were located antero-superiorly on the convex (diaphragmatic) surface of the liver in all cases (Figs. 1, 2, 3). On in situ inspection of these livers, the diaphragm corresponding to the areas of the HSGs appeared normal. No muscular hypertrophy or fibrotic slips were noted in the diaphragm of any of these cadavers. One patient who had a prior cholecystectomy had post-surgical adhesions present between the liver and diaphragm at the operative site. The coronary and triangular ligaments were normal in all specimens. No anatomic abnormalities or supernumerary ribs were present in the thoracic wall.

There were 8 (89%) cadavers with HSGs in the right hemi-liver (Fig. 1), but there were wide variations in their number, length and depth as outlined in Table 1. The left medial section (segments 4a and 4b) was the second most common location (Fig. 2), with 4 (44%) cadavers having grooves in this section. Three (33%) cadavers had HSGs in the left lateral section (segments 2 and 3) of the liver (Fig. 2). Three (33%) cadavers had vertical grooves that roughly coursed along Cantlie's plane (Figs. 1, 2, 3).

Five (56%) cadavers had additional grooves on the visceral (inferior) liver surface, all at segments 5 and 6 (Figs. 4, 5). Interestingly, abnormal caudate morphology was present in 4 (80%) of the 5 cadavers with inferior HSGs. In these cases, there was an association with a caudate notch and well-developed caudate process (Fig. 4).

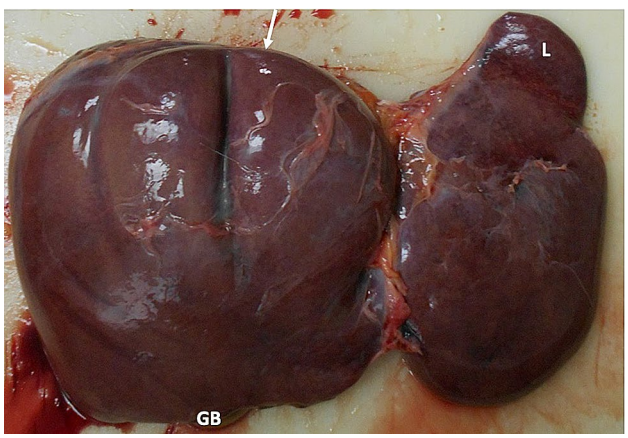

Fig. 1 The explanted liver of index case #2. A well-defined, deep groove can be seen coursing along Cantlie's plane, marked by the gallbladder (GB) inferiorly and the supra-hepatic cava superiorly (arrow). A second vertical groove is present in segment VIII. This specimen also demonstrates a lingular process of the left lobe (L)

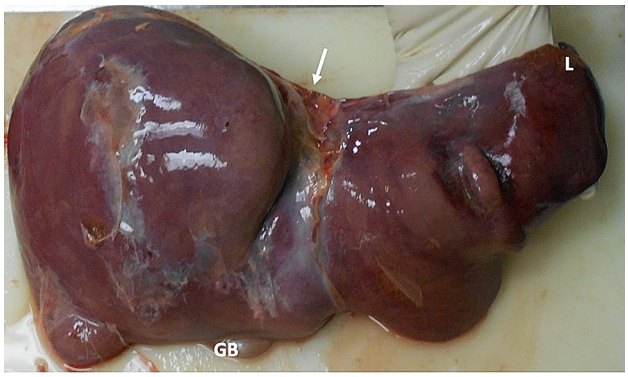

Fig. 2 The explanted liver of index case #1. Two grooves are seen on the diaphragmatic surface of segment 2/3. Another well-defined, deep groove can be seen coursing along Cantlie's plane, marked by the gallbladder (GB) inferiorly and the supra-hepatic cava superiorly (arrow). This specimen also demonstrates a lingular process of the left lobe (L)

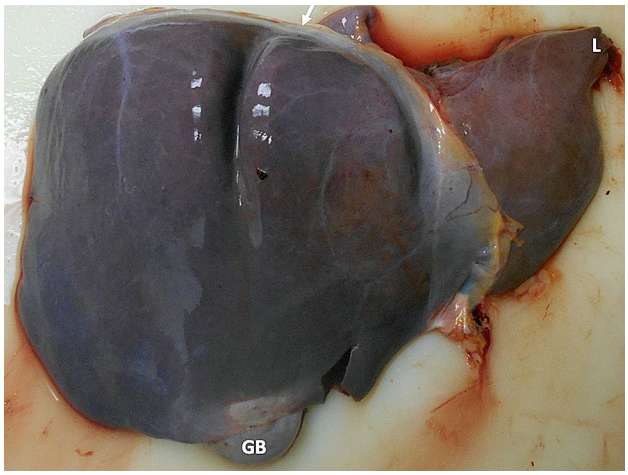

Fig. 3 The explanted liver of index case #9. A well-defined, deep groove can be seen coursing along Cantlie's plane, marked by the gallbladder (GB) inferiorly and the supra-hepatic cava superiorly (arrow). This specimen also demonstrates a lingular process of the left lobe (L)

Eight (89%) of the cadavers that bore HSGs often had other anomalies present: parenchymal nutmeg changes (5), abnormal caudate morphology (4), hyperplastic left hemiliver (3), lingular process (2), bi-lobar gallbladder (1) and/or abnormal ligamentous attachments (1).

One cadaver with multiple HSGs, also bore an associated bi-lobar gallbladder (Fig. 5) and an anomalous attachment of the falciform ligament to the caudal surface of the root of small bowel mesentery, creating an internal hernia (Fig. 6). This patient died from intra-abdominal sepsis after small bowel strangulated in the internal hernia (Fig. 1).

Discussion

In classic anatomic descriptions, there are no grooves on the diaphragmatic surface of the liver [18]. The classic anatomic pattern was present in 85% of unselected cadavers in this study. The prevalence of HSGs varies widely across the globe, ranging from 5% in Malaysia up to 40% in Italy [8, 14]. In our study, HSGs were present in 15% of unselected cadavers. This was reasonably consistent with the prevalence in international studies and similar to the 12% prevalence in Jamaica, as reported by Gardner et al. [5].

It is important for radiologists to be aware of the presence of HSGs in this modern era of readily available cross-sectional imaging. They should be careful not to misdiagnose these anomalies as liver lacerations in trauma patients or metastatic deposits in oncology patients [1, 5, 14]. There have also been reports of mis-interpretation of HSGs as Chilaiditi's syndrome on radiographs [4].

The presence of HSGs is of particular interest to hepatobiliary surgeons because their presence was a marker of associated anatomic anomalies in 89% of our cases. Therefore, their presence can serve as a proxy for increased technical complexity during liver resections. Identifying HSGs on pre-operative imaging would allow hepatobiliary surgeons to adjust their assessment of operative risk and modify the informed consent procedure accordingly. They can also be used as landmarks to plan liver resections and/or transplantation [3].

In this study, the absence of diaphragmatic anomalies and the vertical alignment of the HSGs did not lend support to the "diaphragmatic slip" and "rib-compression" theories [5]. These theories essentially suggested that the HSGs were post-mortem findings that were due to the liver being compressed by fibrotic diaphragmatic slips and/or ribs [1, 6, 8, 12, 17, 21]. In addition, neither of these theories would explain the presence of associated inferior HSGs that were present in 56% of our cases.

Occasionally, HSGs may be encountered in patients with hepatic malignancies, especially those who have had prior chemotherapy. In these cases, it is postulated that they may be the result of tumour necrosis that leads to involution, desmoplasia and retraction of Glisson's capsule [16, 21, 22]. However, in our study there were no intra-hepatic tumours present on sectioning of the livers. Additionally, since the full ante-mortem medical history was available for each cadaver, we can confidently state that hepatotoxic drugs that may have led to hepatic desmoplasia were not aetiologic factors in these cases.

Sometimes, other pathologic processes may cause parenchymal injury, scarring and retraction of Glisson's capsule to produce a depression on the surface of the liver.

Table 1 Anatomic details of hepatic surface grooves

Case	Age	Sex	No. of sulci	Location (segment)	Length (cm)	Width (cm)	Depth (cm)	Special observations
1	61	F	4	II	4.5	1.3	1.6	Inferior HSG at sVI Caudate notch
				III	3.8	0.7	1.1	Caudate process
				IVa/b (C)	9.3	1.2	1.6	Hyperplastic left lobe lingular process left lobe
				VIII	3.5	0.5	0.6	
2	64	F	2	IVa	7.7	0.4	2.4	Hyperplastic left lobe lingular process left lobe
				VIII	5.6	0.3	0.3	Nutmeg changes
3	65	F	2	II	2.1	0.2	0.7	HSG inferiorly at sVI Caudate notch
				VII	6.7	0.4	0.9	Caudate process
								Nutmeg changes
4	61	M	2	IVb	1.3	0.3	1.5	HSG inferiorly at sVI Caudate notch
				V	7.3	0.7	1.8	Caudate process
5	78	M	3	IVa	3.8	0.5	0.5	HSG inferiorly at sVI Nutmeg changes
				VIII	5.5	0.3	0.2	
				VI	12.0	2.6	1.9	
6	83	M	3	IVb	3.5	3.3	1.8	Inferior HSGs at sV and VI
				V	7.0	0.3	1.1	Caudate notch
				VI	5.2	0.2	0.3	Caudate process Hypoplastic left lobe
								Bilobed gallbladder Anomalous attachment of falciform to omentum
7	48	F	1	VIa	1.3	0.6	1.0	None
8	72	F	2	II	7.2	1.3	2.5	Adhesions 2° to cholecystectomy
				III	3.9	0.6	0.6	Nutmeg changes
9	62	M	1	IVa	7.0	0.9	1.4	lingular process left lobe Nutmeg changes

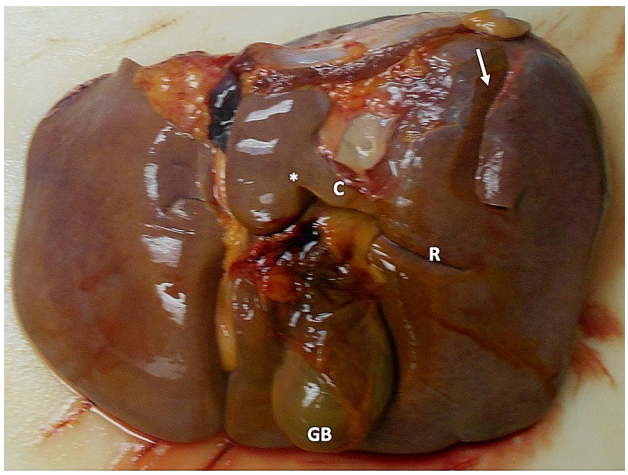

Fig. 4 The explanted liver of index case #9 viewed from the visceral surface. The arrow identifies an abnormal groove at segment VI, separate from the normal Rouviere's sulcus (R). Caudate lobe anomalies are also present, including a caudate notch (Asterix) and a well-developed caudate process (C). The gallbladder (GB) is labelled for orientation

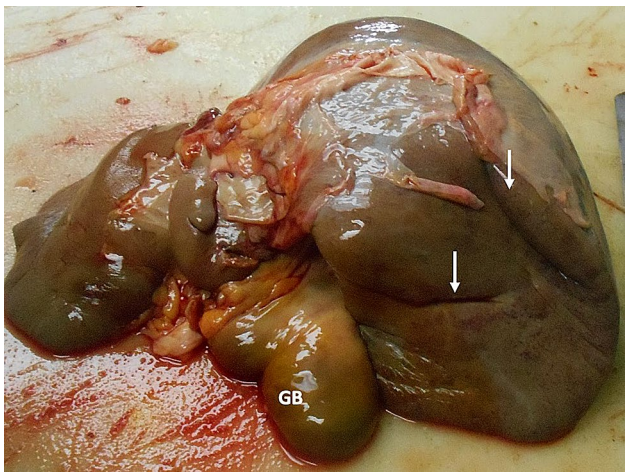

Fig. 5 Explanted liver of index case #6 demonstrating multiple grooves (marked by arrows) on the inferior (visceral) surface and a bi-lobed gallbladder (GB)

These include cirrhosis, hepatic abscesses and gallbladder empyema. Ohtomo et al. reported that these inflammatory processes may be present in up to 14% of patients with HSGs [13]. However, none of the specimens in our study had associated cirrhosis, abscesses, gallbladder empyema or any other infective of inflammatory diseases involving the liver. Therefore, these could not account for the presence of HSGs in our study population.

Interestingly, 56% of our specimens with HSGs had "nutmeg changes" in the liver on sectioning. Francis Kiernan

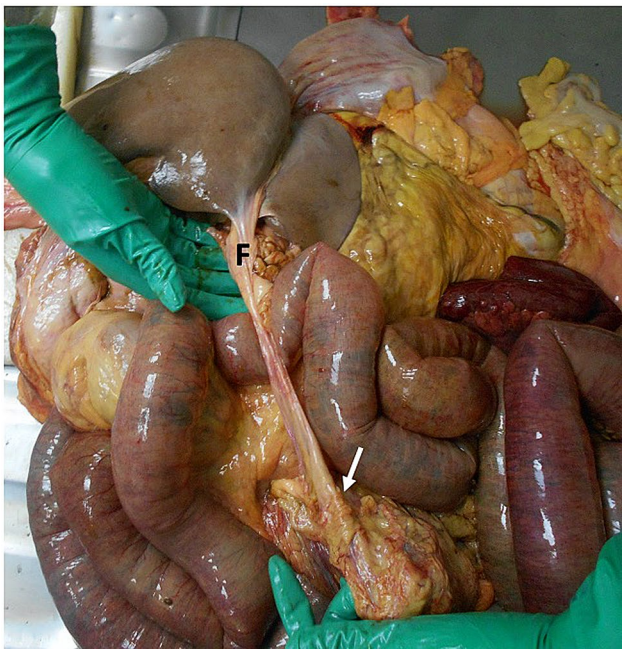

Fig. 6 Liver and gastro-intestinal system of index case #6. This photograph demonstrates an abnormal attachment of the falciform ligament (F) to the caudal surface of the root of small bowel mesentery (arrow), creating an internal hernia through which small bowel became strangulated and perforated

first coined this term in 1833 to describe a change in the parenchyma that becomes similar in appearance to a transected nutmeg seed [7]. There is a variegated, red-tan, mottled appearance of the parenchyma that is due to hemorrhagic, engorged sinusoids contrasting with the yellow colour of unaffected liver [7, 20]. The nutmeg appearance results from hepatic congestion due to any cause, such as right heart failure or veno-occlusive liver disease. Since the full ante-mortem history was available for each cadaver in our study, we noted that all cadavers with "nutmeg liver" had a long-standing history of hypertension with cardiovascular complications. Incidentally, 3 of these cadavers had HSGs coursing roughly along Cantlie's plane.

These findings raise the possibility of a new aetiologic mechanism for HSG formation. Consider the fact that animal studies demonstrate that beta-catenin expression stimulates hepatocyte proliferation, and regulation of beta-catenin produces changes in liver size, shape and accessory lobe formation [11, 19]. On this background, Suksaweang et al. theorized that specific areas in the liver, known as growth zones, are susceptible to changes in beta-catenin and Wnt activity [19]. To date, the prevailing aetiologic theory is that reduction in beta-catenin expression at the growth zones causes hepatocyte involution and capsular retraction, producing HSGs [8].

It has also been shown that beta-catenin is up-regulated in humans with right heart failure [9, 15]. These are the patients who would have nutmeg changes in the parenchyma. Therefore, we purport a new mechanism to consider is that hepatic congestion leads to up-regulation of beta-catenin levels, leading to hyperplasia at the growth zones. When hyperplasia occurs at the localized growth zones, they produce protuberant areas relative to the less-well vascularized watershed areas between hepatic sections. This localized hyperplasia in isolated grown zones then produces HSGs. This could explain why 60% of cadavers with nutmeg changes had HSGs coursing along Cantlie's plane, known to be a poorly vascularized watershed area separating left and right hemi-livers. We acknowledge, however, that more research is necessary before this can be proven as a cause of HSGs.

The numbers in this study are too small to ascribe statistical significance to the association between hypertension, cardiac pathology and HSGs. However, we believe that this is a clinically important association. As a marker of hepatic congestion, HSG may herald of increased bleeding during liver resection. There is robust data demonstrating that excessive bleeding is an independent predictor of morbidity and mortality after major liver resections [10].

Since both HSG and nutmeg changes can be identified on cross-sectional imaging, they can be used as a proxy for increased operative complexity, allowing hepatobiliary surgeons to modify the informed consent process accordingly. On cross sectional imaging, the nutmeg liver changes can be appreciated as in-homogenous parenchymal enhancement during the portal venous phase with return to normal in the delayed phase [20]. The areas of lower attenuation correspond to areas with decreased portal blood flow due to hepatic congestion [20].

Conclusion

Approximately 15% of unselected persons in this population have hepatic surface grooves present. Every attempt should be made to identify hepatic surface grooves on pre-operative imaging because they can alert the hepatobiliary surgeon to: (1) associated anatomic anomalies in 89% of cases, (2) associated hepatic congestion in 56% of persons, (3) increased risk of bleeding during liver resections and (4) increased technical complexity of liver resections.

The association between HSGs, cardiovascular complications, hepatic congestion and nutmeg liver prompted us to propose a new aetiologic mechanism for HSG formation, involving localized hyperplasia at growth zones due to upregulation of beta-catenin levels. More research is needed to further investigate this aetiologic relationship.

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Author contributions SOC: protocol development, data management, data analysis, manuscript writing.RAA, JC, SS: data collection, data analysis, manuscript editing. MTG, NWP, VN: data analysis, manuscript editing, protocol development.

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Availability of data and material All data will be made available to the editorial board upon request.

Compliance with ethical standards

Conflict of interest The authors have no competing interests to declare that may serve as conflicts of interest.

Ethics approval This research was approved by the local institutional board.

Consent to participate The research does not include living patients and therefore consent to participate is not necessary.

Consent for publication The authors all give consent for publication of this work.

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