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Left Side Gallbladder: Clinical and Anatomical Implication

Filippo Banchini and Patrizio Capelli

Abstract

Left side gallbladder is a rare anatomical anomaly reported in the literature. It is associated with various anatomical variations of the biliary way and intrahepatic portal supply. Most of the time, it is discovered as an incidental finding during intervention for cholecystectomy, exposing patients and surgeons to high risk of complication. To prevent this, we analyze the critical aspects that must be known to perform safe interventions either in the normal setting or in the emergency setting. Different theories are proposed to describe this anomaly, but a debate is still open. Reviewing the literature and analyzing the different processes of formation, we create a classification that can explain how this anomaly can occur, dividing into four variation types.

Keywords: left side, gallbladder, fusion of plans, liver resection, biliary, agenesis, abnormality, cholecystectomy, right umbilical vein, liver, hepatic

1. Introduction

The left side gallbladder (LSG) is a very rare alteration defined by the attachment of the gallbladder to the left lobe of the liver at the right side of the ligament tears.

Since Hochstetter's first description in 1886 [1], about 150 cases have been reported in the literature. The attempts to explain the cause of this anomaly have been different, but the numerous variations described do not allow a clear definition of its origin. Although this debate is still open, it is of considerable importance to know that LSG is frequently associated with alterations of both the portal branches and the intrahepatic biliary tree. The association of these anomalies, therefore, represents an important risk, especially if surgical treatment is necessary. There are two cases in which surgical treatment may be required: the first is gallbladder stones and in particular acute cholecystitis, and the second is the need for liver resection surgery. In the first case, the diagnosis of gallstones and cholecystitis is made only with ultrasound, but often this method does not describe the anomaly of the LSG. In fact, in most cases described, the diagnosis is made intraoperatively, making the surgical treatment problematic and risky due to the lack of correct anatomical knowledge. In the case of liver resection, the diagnosis is made before surgery, highlighting anatomical variations that require complex dissection strategies. Knowledge of the anomalies associated with a LSG can be of considerable help in preventing serious complications.

2. Estimation incidence and diagnosis

The real estimation of this anomaly is very difficult because most of the published articles are case reports and only a few of them have high numbers of cases. The literature analysis revealed 114 articles concerning LSG: 89 authors describe 1 case only, 11 present 2 cases, 3 present 3 cases, 3 present 4 cases, 3 describe 6 cases, 1 describes 7 cases, 2 describe 9 cases, 1 describes 10 cases, and 1 describes 26 cases, with a total of 211 cases (**Table 1**).

The incidence is variable and remains always below 0.3%: Idu et al. [109] describe 5 cases of LSG on 1764 cases of cholecystectomy (0.3%), Nagai et al. [90] 3 cases out of 1621 (0.2%), Sadhu et al. [43] 1 out of 1258 (0.08%), and Rozsos et al. [37] 1 out of 2536 (0.04%).

Naganuma et al. [87], in a series of 67.994 patients studied with ultrasound, found 18 cases of abnormal gallbladder position with an incidence of 0.026%. These included retrohepatic, supraphrenic, and floating gallbladder, and only nine cases of LSG, with an incidence of 0.013%. However, ultrasound is not the main exam to make the diagnosis. Pereira et al. [115] show that ultrasound has a positive predictive value of only 2.7% and that in 81.1% LSG is initially detected at surgery. Also, Lee et al. [36], in his series of 10 cases operated on for cholecystic stones or acute cholecystitis symptoms, describes the discovery: intraoperative in 8 cases and as an incidental finding on abdominal computed tomography (CT) in 2 cases.

The CT scan is the main examination capable of making a correct diagnosis. However, as reported in the review of Pereira et al. [115], CT has a positive predictive value of only 60%. This can be explained by the simultaneous proximity of the two hepatic lobes to the gallbladder, simulating the contact and not the adhesion to the left lobe of the liver. Considering the position of the gallbladder, if it is in the normal site, its right margin generally appears free (**Figure 1A**). If the gallbladder is positioned on the left, its left margin may appear free (**Figure 1B**) or included in the left lobe with a space between the two hepatic lobes (**Figure 1C**), making diagnosis easy. If the gallbladder appears between the right and left hepatic lobes, the diagnosis may remain unknown (**Figure 1D**) mimicking hypertrophy of the left hepatic lobe as also reported by Banchini et al. [20] and Iskandar et al. [6].

However, the CT remains the main examination as it can provide the anatomical portal and arterial variations that, as we will see, frequently occur in association with this anomaly.

3. Theories and embryology

The embryological theories of LSG development are numerous and complex, but we can distinguish two main ones. The first theory concerns an alteration that only concerns the development of the gallbladder and a second one that concerns the development of the central portion of the liver and consequently the malpositioning of the gallbladder.

In the theory of gallbladder development proposed by Gross [116], there are two ways in which gallbladder can develop:

- The first modality suggests a normal growth of the gallbladder migrating from the right side of the liver to the left side and attaching to the left lobe. In this case, the cystic duct originates from the right side of the bile duct and moves anteriorly and to the left of the liver pedicle.

Reference	Author	Number of cases	Reference	Author	Number of cases	Reference	Author	Number of cases
[2]	Abe	1	[3]	Ishii	1	[4]	Noritomi	1
[5]	Abongwa	1	[6]	Iskandar	1	[7]	Ogawa	1
[8]	Alharthi	1	[9]	Jona	1	[10]	Ogino	1
[11]	Almodhaiberi	1	[12]	Jung	1	[13]	Oshima	1
[14]	Aoki	1	[15]	Kanazumi	2	[16]	Ozeki	26
[17]	Asonuma	4	[18]	Kawai	1	[19]	Pradeep	4
[20]	Banchini	1	[21]	Kehr	1	[22]	Quah	1
[23]	Banzo	1	[24]	Kelly	1	[25]	Qureshi	1
[26]	Bender	1	[27]	Kim	4	[28]	Rafailidis	1
[29]	Bonomo	1	[30]	Kinoshita	2	[31]	Reddy	1
[32]	Chrungoo	2	[33]	Kubo	1	[34]	Rocca	1
[35]	Chuang	1	[36]	Lee	10	[37]	Rozsos	1
[38]	Chung	1	[39]	Leone	1	[40]	Saafan	1
[41]	Cirla	1	[42]	Lin	1	[43]	Sadhu	1
[44]	Colovic	2	[45]	Maetani	1	[46]	Sakihara	1
[47]	Dhulkotia	1	[48]	Makni	1	[49]	Schiffino	1
[50]	Donthi	1	[51]	Masood	1	[52]	Seiberman	1
[53]	Ergun	1	[54]	Matsumoto	1	[55]	Shen	1
[56]	Etter	1	[57]	Matsumura	1	[58]	Shimizu	1
[59]	Feldman	1	[60]	Matsuoka	1	[61]	Shirono	1
[62]	Fujita	1	[63]	Mazzamurro	1	[64]	Siebermair	1
[65]	Fujita	1	[66]	McGowan	1	[67]	Si-Youn	3

Reference	Author	Number of cases	Reference	Author	Number of cases	Reference	Author	Number of cases
[68]	Fukuda	1	[69]	Mendoza-Calderon	2	[70]	Strong	1
[71]	Funakoshi	1	[72]	Mizray	1	[73]	Surjan	1
[74]	Futamura	1	[75]	Mohammed	6	[76]	Szanto	1
[77]	Gondra	1	[78]	Moo-Young	1	[79]	Tachibana	2
[80]	Gui	1	[81]	Moravik	1	[82]	Takemura	1
[83]	Hasbahceci	1	[84]	Moriyama	2	[85]	Takiguchi	1
[86]	Herrington	1	[87]	Naganuma	9	[88]	Tomiyama	1
[89]	Hirohata	1	[90]	Nagay	3	[91]	Uesaka	2
[1]	Hochstetter	1	[92]	Nagendram	1	[93]	Velimezis	7
[94]	Hopper	1	[95]	Nakakubo	1	[96]	Watanabe	1
[97]	Hsu	9	[98]	Namikawa	1	[99]	Wong	1
[100]	Huang	6	[101]	Nastos	2	[102]	Wu	2
[103]	Hwang	3	[104]	Nemours	1	[105]	Yamazaki	1
[106]	Iaccarino	1	[107]	Newcombe	1	[108]	Yu	2
[109]	Idu	6	[110]	Nguyena	1	[111]	Zografos	1
[112]	Ikoma	1	[113]	Nishio	1	[114]	Zoulamoglou	1

Table 1.
Number of cases reported in the literature for each author.

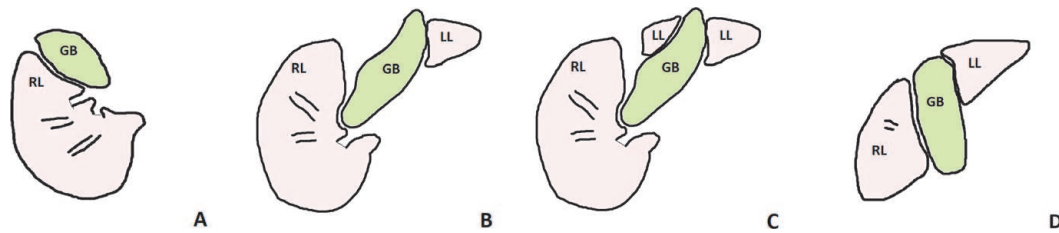


Figure 1.

CT scan visualization of the gallbladder. (A) Normal right side gallbladder attached to the right lobe; (B) left side gallbladder attached to left lobe with empty space between gallbladder fundus and right lobe; (C) left side gallbladder with fundus surrounded by left lobe and empty space between right lobe and left lobe; (D) left side gallbladder interposed between right lobe and left lobe, mimicking hypertrophy of right lobe. RL: right lobe; LL: left lobe; GB: gallbladder.

- The second modality hypothesizes the formation of the gallbladder directly on the left side of the bile duct with its positioning directly under the left hepatic lobe. In this case, the cystic duct originates in the left part of the bile duct.

The literature defines as a true LSG when it is located on the right side of the round ligament, and the cystic duct is inserted on the left side of the bile duct. The presence of true LSG, described in this way, represents a remarkably rare event, constituting 4.3% of the cases of left side gallbladder [18], and most of the cases described are therefore a different alteration.

The ways in which the cystic duct enters the biliary duct are numerous even in the absence of the left gallbladder, and as reported by Sarawagi et al. [117], the normal right conjunction is present in 51.1% of cases, while its medial (left) insertion can be presented in 16.1%. Pereira et al. [115], in his review, describe the insertion of the cystic duct on the right side 65.6%, on the left side of common hepatic duct 9.5%, left hepatic duct 9.5%, on the right hepatic duct 7.6%, and in a branch of the right hepatic duct 2.4%. Six patients had other minor biliary anomalies, and one had a duplicate common bile duct (CBD).

The second theory about the development of the central portion of the liver was first described by Ozeki in 1987 [16] and later defined by Nagai in 1997 [90] as “right side round ligament.” This theory is associated with numerous alterations in intrahepatic anatomy and appears to account for over 95% of cases in which a left side gallbladder is present.

To understand this complex theory, it is necessary to investigate some moments of fetal evolution, in which the persistence of a right umbilical vein and/or hypertrophy of the left portion of the liver seem to be associated. According to the embryological studies of Arey [118] when the embryo measures 6 mm, there are at the same time two vitelline’s veins, one on the right side and one on the left side, everyone having a branch that enters the liver. The two veins inside the liver have branches that join them together. When the embryo reaches the size of 7 mm, the right vein goes into atrophy leaving the left side predominant. It is assumed that, if the right side vein does not atrophy, there is a persistence of this umbilical vein either extrahepatic or intrahepatic. In this case, the opposite process to the previous one occurs, with atrophy of the left side and hypertrophy of the right side (Matsumoto’s hypothesis) [119]. When this happens, we have the positioning of the gallbladder in the left portion of the liver.

Although this theory seems to be the most accreditable, there are cases in the literature of persistence of the right umbilical vein with the gallbladder positioned normally, showing how this theory does not always prove to be real [42].

Lucidarme et al. [120] describe this variation in portal anatomy as a defect in the evolution of the central portion of the liver, in which the right and left parts join

together in a variable way, renaming this as “Fusion of hepatic plans.” Considering these different descriptions, we can say that there is an anomaly in the mechanism of persistence or atrophy of the right umbilical vein and/or the liver surrounding it.

Based on these descriptions, we propose to combine the mechanism of atrophy of the central part of the liver with persistence of the right umbilical vein and the mechanism of fusion of the plans, trying to verify how these can give rise to different anatomical variations. In this way, we create a classification that can explain how this anomaly can occur.

As seen before, during fetal development, the right and left umbilical veins have a Y shape with one arm entering the liver and one passing laterally to it. At this stage, there is a set of vessels inside the liver that connect the two internal branches of the intrahepatic umbilical veins. This connection forms a venous conduit that we will call “intrahepatic right umbilical vein” (IRUV), which flows directly into the ligament of Arantius or enters the right umbilical vein at Rex’s recess. The extrahepatic portion of the right umbilical vein can enter the liver in a variable position between segment 5 and segment 4b. Imagine looking at the liver from its lower face, corresponding to segments 5 and 4b, we can divide this area into four parts as shown in **Figure 2**: the superficial part, corresponding to the acute margin of the liver, in the portion of segment 5 (**Figure 2**: yellow segment 5), and the portion of segment 4b (**Figure 2**: blue segment 4b) and a deep part toward the hilum of the liver (**Figure 2**: red segment 5; green segment 4b).

The involution of the right umbilical vein may affect the surrounding hepatic parenchyma differently, depending on its extension and site, determining unusual alterations.

If atrophy affects the superficial parts (**Figure 2**: yellow and blue), the result is a volumetric reduction in the corresponding liver segments. If the atrophy affects the deep part toward the hilum (**Figure 2**: red and green), we can have alterations that cause variations of the portal vein and the biliary tract. Complete or partial atrophy can also explain in various ways incomplete forms of gallbladder malposition such as the medioposition described by Hsu et al. [97].

The different possibilities with which the right umbilical vein can evolve are four, and we describe them as follows (**Figure 3**):

Type A: complete atrophy of the right umbilical vein and normal left umbilical vein.

Type B: atrophy of the right external umbilical vein and persistence of the IRUV.

Type C: persistence of the IRUV and extrahepatic umbilical vein and atrophy of the left umbilical vein.

Type D: persistence of the right extrahepatic umbilical vein and atrophy of the left umbilical vein.

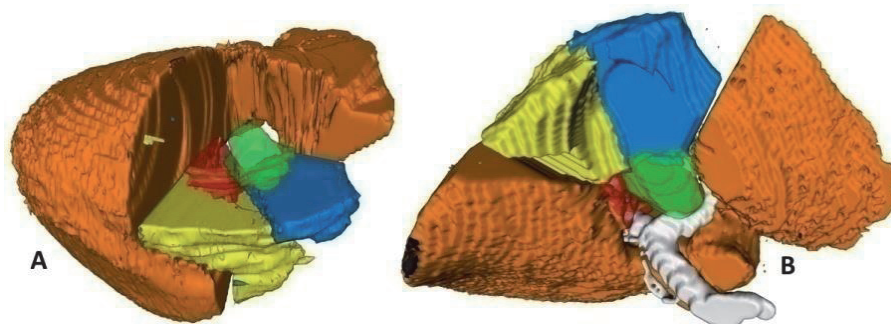


Figure 2.

Liver with the central portion divided into four parts: the superficial part of segment 5 in yellow; the lower part close to the hilum in red; the superficial part of segment 4 in blue; and the lower part of segment 4 close to the hilum in green. (A) Superior view and (B) inferior view.

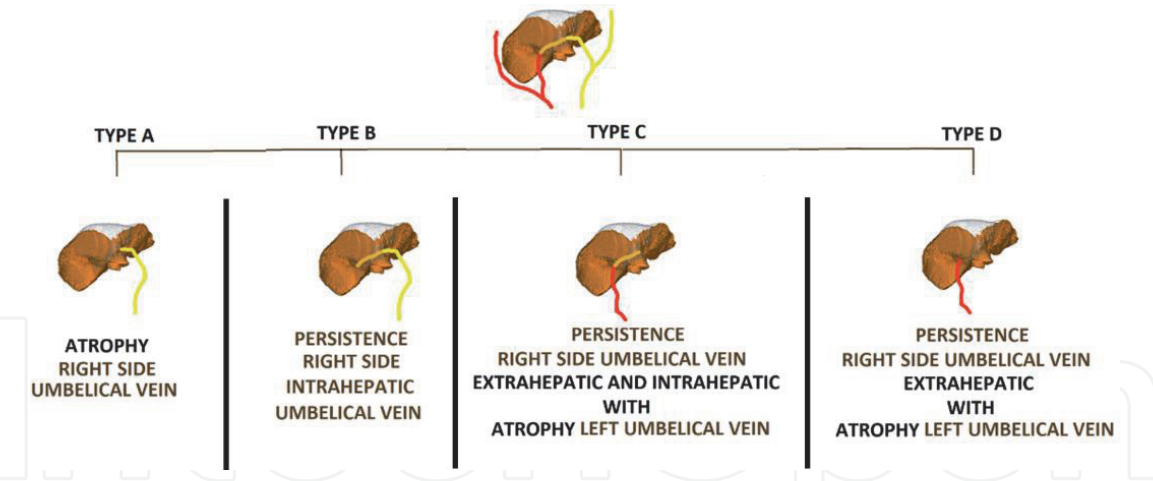


Figure 3. Schematic evolution of right and left umbilical veins with different evolution processes. (Type A) Normal evolution with the persistence of the left umbilical vein. (Type B) Persistence of the left umbilical vein and intrahepatic umbilical vein. (Type C) Persistence of the right umbilical vein and persistence of the intrahepatic umbilical vein with atrophy of left umbilical vein. (Type D) Persistence of the right umbilical vein only (in red: right umbilical vein; in orange: intrahepatic umbilical vein; in yellow: left umbilical vein).

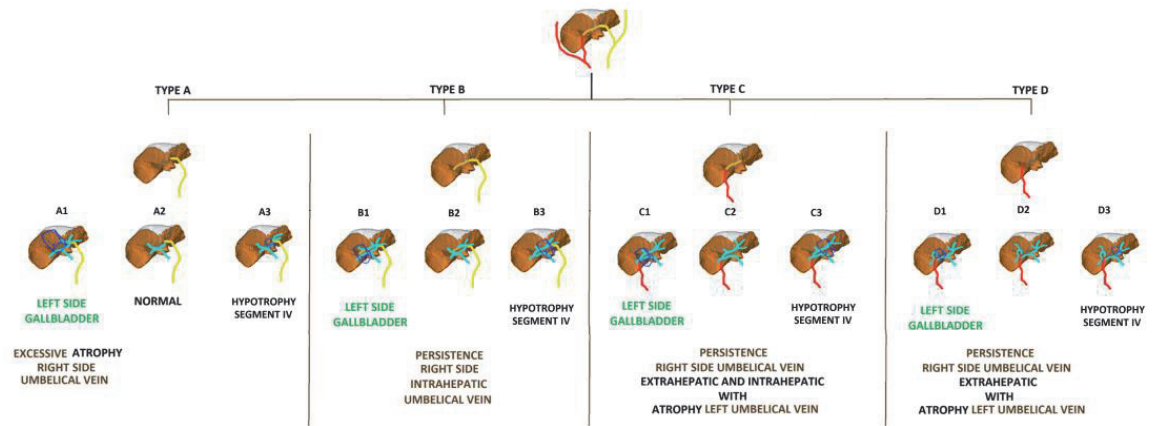


Figure 4. Combination of the evolution process of right and left umbilical veins with atrophy mechanism of segment 5 and 4 (in red: right umbilical vein; in orange: intrahepatic umbilical vein; in yellow: left umbilical vein; in light blue: portal vein and its branches; and in blue: area of hypotrophy). A1-B1-C1-D1 corresponding to atrophy of segment 5 area; A2-B2-C2-D2 absence of liver atrophy; A3-B3-C3-D3 corresponding to atrophy of segment 4 area.

Types C and D represent the cases in which a persistence of the right umbilical vein occurs instead of the normal left umbilical vein, classifying this as “true right side round ligament.”

Combining these variations with how lower liver atrophy can occur, the following classifications can be determined (**Figure 4**):

- Type A:
- A1: The right umbilical vein goes into complete atrophy and is accompanied by an excessive involution of segments 8-5-4b. In this case, the gallbladder will present to the left of the round ligament.
 - A2: The right umbilical vein goes into atrophy completely, and there is no parenchymal atrophy. This is the normal anatomy that is described.
 - A3: The right umbilical vein goes into atrophy completely and is accompanied by involution of segment 4b with hypotrophy of the latter.
- Type B:
- B1: The right external umbilical vein goes into atrophy, and the right intrahepatic umbilical vein persists. An involution in the segment 5 site is

associated. In this case, there is the presence of left side gallbladder with the persistence of the right portal branch only for segments 6 and 7 and hypertrophy of the portal branch of segment 4.

B2: The right external umbilical vein goes into atrophy, and the right intrahepatic umbilical vein persists. There is no involution of the hepatic parenchyma with normal portal trifurcation.

B3: The right external umbilical vein goes into atrophy, and the right intrahepatic umbilical vein persists. It is associated with segment 4b hypotrophy. This determines the absence of the left portal branch and vascularization of segments 2 and 3 from the right portal branch through the persistent right umbilical vein portion.

Type C:

C1: The right extrahepatic and intrahepatic umbilical veins persist with atrophy of the left umbilical vein. An involution in the segment 5 site is associated. In this case, there is the presence of left side gallbladder with the persistence of the right portal branch only for segments 6 and 7 and the absence of segment 5 or 5 and 8. The vascularization of segments 2 and 3 and 4 occurs through the persistent right intrahepatic umbilical vein portion.

C2: The right extrahepatic and intrahepatic umbilical veins persist with atrophy of the left umbilical vein. No involution of the hepatic parenchyma occurs. The vascularization of the liver is arched from right to left giving, in sequence, the branch for segments 6 and 7, that of segments 5 and 8 and ending with the vascularization of segments 2 and 3 and 4 through the persistent intrahepatic right umbilical vein.

C3: The right extrahepatic and intrahepatic umbilical veins persist with atrophy of the left umbilical vein. An involution in the segment 4b site is associated. The vascularization of the liver is arched from right to left giving the branch for segments 6 and 7, the branch for segments 5 and 8 and ending with the vascularization of segments 2 and 3 through the persistent intrahepatic right umbilical vein.

Type D:

D1: The right extrahepatic umbilical vein persists with atrophy of the left umbilical vein. An involution in the segment 5 site is associated. In this case, there is the presence of left side gallbladder with the persistence of the right portal branch that vascularizes segments 6 and 7 and the absence of segment 5 or 5 and 8, and the left portal branch that vascularizes segments 2 and 3 and 4.

D2: The right extrahepatic umbilical vein persists with atrophy of the left umbilical vein. There is no involution of the hepatic parenchyma. In this case, the right portal branch that vascularizes segments 6–7 and 5–8 and the left portal branch that vascularizes segments 2 and 3 and 4 persist.

D3: The right extrahepatic umbilical vein persists with atrophy of the left umbilical vein. An involution is associated with segment 4b. In this case, the right portal branch that vascularizes segments 6–7 and 5–8 and the left portal branch that vascularizes segments 2 and 3 persist.

The principle of the fusion of the planes is necessary to be added at the classification performed by rotating or increasing the volume of one of the two hepatic lobes.

In this way, we can reposition the ligament tears in the position in which it is located once the complete fetal development has taken place. To clarify, we have modified the left liver portion by rotating it counterclockwise as shown in **Figure 5**.

We can see how this classification allows us to catalog the cases of literature described or represented with images, the portal modifications found: Kawai et al. [18] describe the absence of the left portal vein and a branch for the left liver originating from the right portal branch that we identify as an atrophy of left

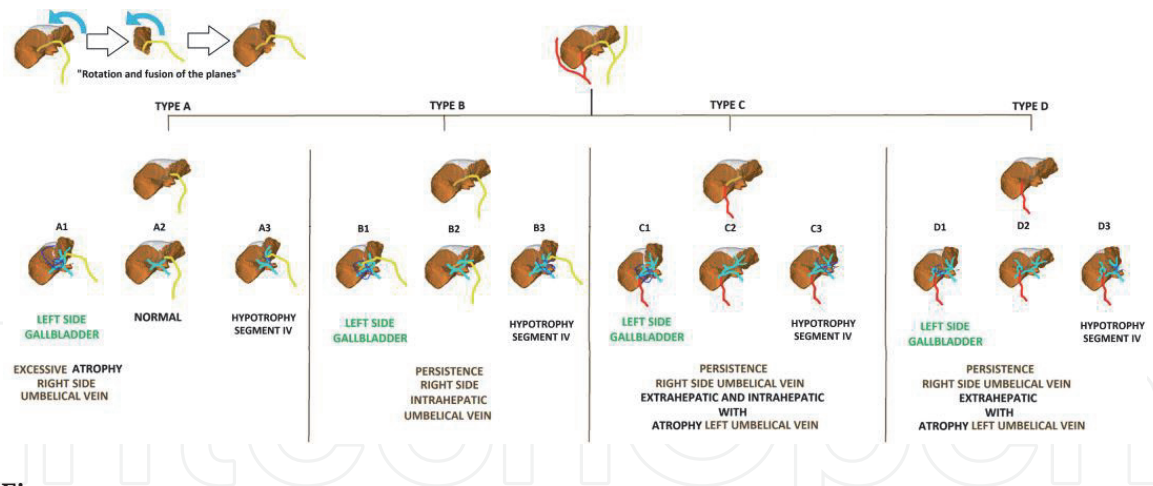


Figure 5.
Combination of the evolution process of right and left umbilical veins with atrophy mechanism of segments 5 and 4, associated rotation and fusion of the right and left liver.

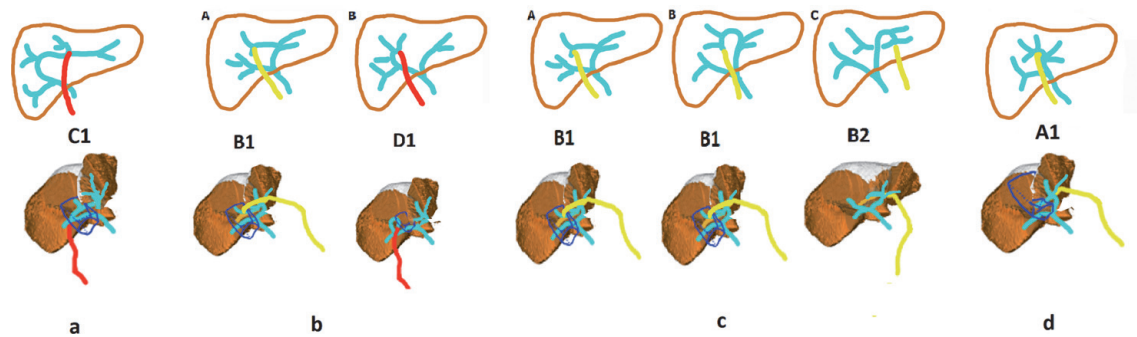


Figure 6.
Schematic representation published in the literature with comparison and reclassification with our nomenclature. (a) Kawai description (12) corresponding to Type C; (b) Nagai description (3) corresponding to Types B1 and D1; (c) Maetani description (20) corresponding to Type B1-B1-B2; and (d) Banchini description (9) corresponding to Type A1.

umbilical vein Type C1 (**Figure 6a**); Nagai et al. [90] describe a case with the absence of the right anterior portal branch, corresponding to Type B1, and the other absence of the left portal branch that we call Type D1 (**Figure 6b**); Lin [42] describes tree cases of the absence of the right anterior portal branch, corresponding to Type B; Maetani et al. [45] also describe two cases that we classify as Type B1 and one case with the absence of the right anterior portal branch and a vascularization of segments 5 and 8 from the left portal one that we consider Type B2 (**Figure 6c**); Banchini et al. [20] describe the agenesis of segments 5 and 8 with the absence of the right anterior portal branch, Type A1 (**Figure 6d**). On the contrary, the classification we propose differs from Shindon’s classification [121] in defining the true right side ligamentum teres. Comparing the three types of portal bifurcation listed by Shindon, we consider the “independent right lateral” one as Type A1, the “bifurcation” one as Type D, and “trifurcation” one as an intermediate of Types A1–A3 (**Figure 7**), concluding that only the “bifurcation type” could be considered as “true right side ligamentum teres.” It is evident that these three variables of fusion, atrophy, and umbilical vein evolution can be combined in a considerably higher number of ways and may result in intermediate presentations. Likewise, using this principle, we can also hypothesize how the biliary tract variations occur. Even if we will not deal in this chapter, we underline that, as evidenced by Nishitai et al. [122], the Biliary tree could differ a lot from the corresponding portal branches, making this a further challenge.

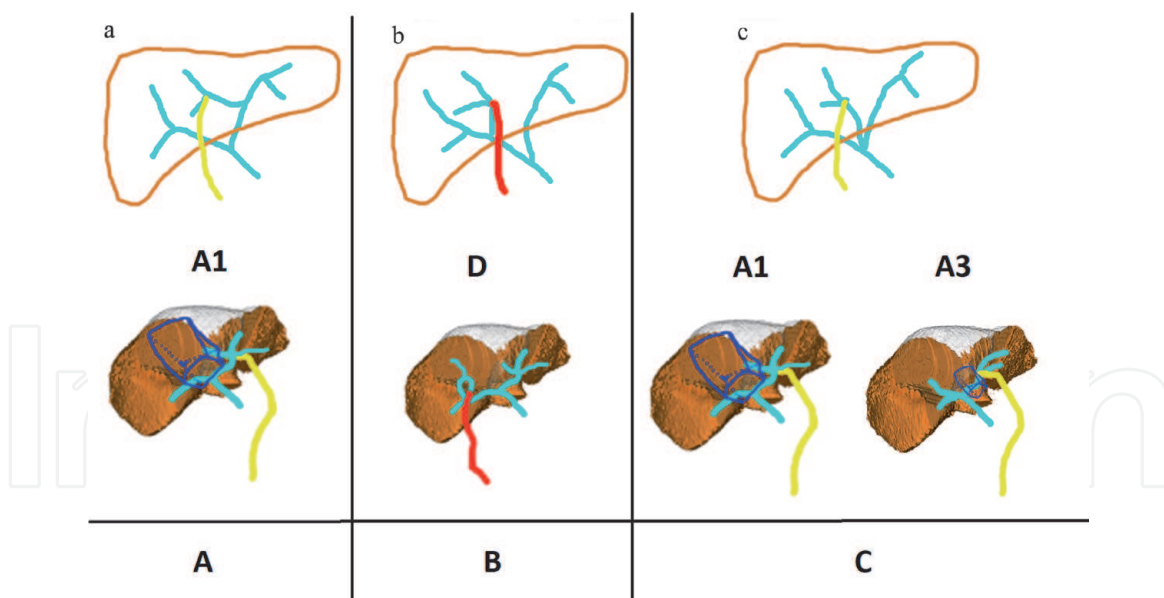


Figure 7.

Schematic representation of Shindon's classification (21) with comparison and reclassification with our nomenclature. (A) "Type A Shindon" corresponding to Type A1; (B) "Type B Shindon" corresponding to Type D; (C) "Type C Shindon" corresponding to intermediate Types A1–A3.

Considering the numerous possibilities with which a left gallbladder can present, we recommend an accurate study of the entire hepatic anatomy, with the need to recognize the portal, arterial, and biliary changes, in all patients presenting this diagnosis in the preoperative setting.

4. Clinical implication

As pointed out in the previous paragraph, the presence of LSG is associated with a high number of variations both internal and external to the liver, regardless of the type of classification we want to use.

However, the presence of this anomaly does not seem to be associated with either a particular clinical manifestation or cancer, thus representing a simple anatomical variation presents in the population. The absence of symptoms makes the diagnosis of LSG an occasional event, consequent to its finding during investigations performed for other factors. Likewise, as in the normal population, gallstones follow the physiological mechanism of formation. However, gallbladder malpositioning does not seem to change the afferent pain pathways, and, as reviewed by Iskandar et al. [6] on 32 articles, the related symptoms are those of biliary colic or classic acute cholecystitis, with pain in the right upper quadrant or epigastric pain.

The diagnosis of gallbladder gallstones is mostly performed with ultrasound, but it has a very low diagnostic capacity in case of unknown LSG. Therefore, patients presenting with symptoms of biliary colic or acute gallbladder cholecystitis have a high probability to find this anomaly only during surgery. This condition exposes the patient and the surgeon to considerable risk. In the literature, cases of biliary lesions during cholecystectomy in LSG range from 4.4 [115] to 7.3% [5].

Laparoscopic cholecystectomy does not seem to be contraindicated, but some precautions are necessary to avoid risks of complications. Many authors advocate different techniques in trocar placement or in the patient's position, but this strategy could be applicable only in the case of preoperative diagnosis.

Anyway, the main risk factor seems to be the passage of the gallbladder anterior to the liver pedicle. This rotation moves Calot's triangle from horizontal and lateral to a vertical and anterior position, bringing the gallbladder closer to the biliary tract (Figure 8).

In normal cholecystectomy, the opening of the Calot allows moving the gallbladder and cystic duct away from the biliary tract. This isolation is performed with a dissection directed from the superficial to the deep plane and is done by pulling the gallbladder laterally. The dissection finishes posterolaterally by finding an area free of tissue, corresponding to the posterior side on Calot's triangle. In the case of LSG, if we perform the dissection of the Calot using this method, we risk finding a posterior plane occupied by the biliary tract and liver peduncle instead of a free one (Figure 9).

Taking into account this condition, it is necessary to look for a dissection modality that allows maintaining the distance from the biliary tract and the hepatic peduncle. We advocate performing laparoscopic cholecystectomy with fundus first technique [24] to achieve a proper distance from the hepatic pedicle. Once the body of the gallbladder is detached from the liver surface and Calot's triangle is joined, we recommend to follow the dissection close to the gallbladder border. The border dissection allows minimizing the removal of peripedicle fat tissue, avoiding unintended biliary duct discovery. In order to augment the distance from the pedicle and the biliary way, once the peritoneum of the Calot has been opened, it is advisable to pull the gallbladder on the lateral side to horizontalize the triangle itself. This traction can move the gallbladder on the right side of the hepatic pedicle, repurposing the normal anatomy. After this mobilization, it could be useful to apply the Strasberg criteria for the visualization of all structures [123], so that the cystic duct and cystic artery can be dissected and clipped distant from the biliary tract, after their recognition. In case of doubt, it is useful to perform an intraoperative cholangiography or visualize the biliary tract with the indocyanine green or finally proceed with the conversion to have a direct view.

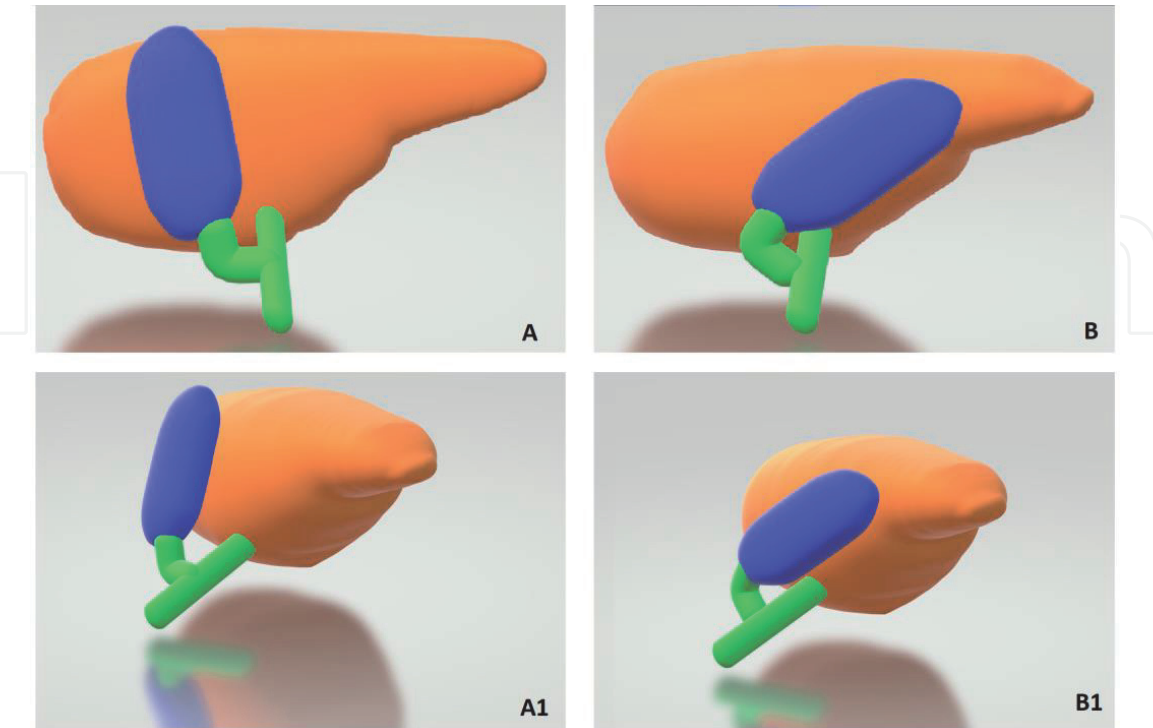


Figure 8.
Blue gallbladder; green cystic duct and biliary way; brown liver. (A) Normal gallbladder frontal view; (A1) normal gallbladder left lateral view; (B) left side gallbladder frontal view; and (B1) left side gallbladder left lateral view.

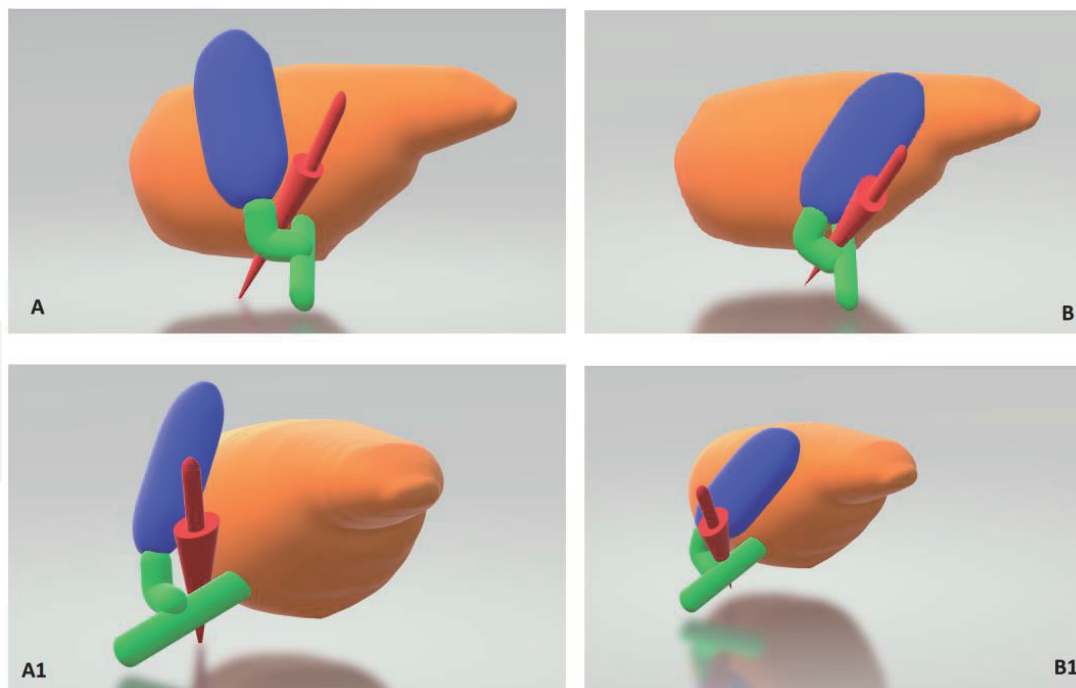


Figure 9. (A) Normal gallbladder frontal visual; (A1) normal gallbladder left lateral visual; (B) left side gallbladder frontal vision; (B1) left side gallbladder left lateral. Visual red arrow: direction of dissection in Calot's triangle.

While accurate dissection and recognition of structures in cholecystectomy can prevent iatrogenic lesions, it is different in the case of liver resections. The anatomical variations that can occur are so high that a detailed study of portal, arterial, and biliary structures is mandatory. This necessity stems from the fact that the portal variations may not correspond to arterial or biliary anomalies, and therefore, all three of these structures must be considered separately. CT scan could be sufficient for portal and arterial study, and in particular, 3D CT could be particularly effective. On the opposite, the CT scan is not sufficient to demonstrate biliary variations, and for this reason, a magnetic resonance cholangiopancreatography is mandatory before elective major hepatectomy to ensure patient's safety [122].

Defining the types of resection is too complex, and therefore, each patient will require an on-demand treatment depending on the anomalies found. The main issue to keep in mind is that, in the case of LSG, liver supply can be sustained by only a single portal branch, as pointed out by Hsu et al. [97]. This eventuality is characterized by an arch shape of the portal vascularization visualized on CT scan, exposing the risk of extending resection to most of the liver itself.

The approach to LSG seems to have a significant clinical implication, augmenting the risk of complications in both liver resection and cholecystectomy. On one side, even if the probability to perform liver resection in LSG is very low, the risk is related to the major intrahepatic modification, on the other, considering cholecystectomy one of the most frequent interventions in surgery, the risk is related to the high probability to treat LSG as symptomatic gallbladder discovered intraoperatively.

5. Conclusion

Left side gallbladder is a rare and little known anomaly that is diagnosed, in most cases, during cholecystectomy for biliary colic or cholecystitis symptoms. The disposition of the gallbladder over the liver pedicle and the simultaneous presence of

variations in the liver vascularization result in an increased risk during surgery. To prevent complications, we recommend performing a cholecystectomy with safety criteria, starting from the fundus and isolating Calot's triangle along the edge of the gallbladder. In the case of hepatic resection, an accurate study of the portal, arterial, and biliary branches should be done before surgery. This makes possible to plan an intervention tailored to the patient's anatomical condition.

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Conflict of interest

The author declares no conflict of interest.

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