

Ectopic partial intrahepatic gall bladder with cholelithiasis – A rare anomaly

SW Lobo,¹ RG Menezes,² S Mamata,³ P Baral,¹ T Kanchan,² SA Hunnargi,⁴ AV Bodhe⁵ and NB Bhat⁶

¹Department of Anatomy, ⁵Department of Physiology, ⁶Department of Microbiology, Manipal College of Medical Sciences, Pokhara, Nepal, ²Department of Forensic Medicine and Toxicology, Kasturba Medical College, Mangalore- 575001, India,

³Department of Anatomy, Medical University of the Americas, Nevis, West Indies and ⁴Department of Anatomy, Kasturba Medical College, Manipal- 576104, India.

Corresponding author: Stany Wilfred Lobo, Assistant Professor, Department of Anatomy, Manipal College of Medical Sciences, Pokhara, Nepal. e-mail: stanmcoms@yahoo.com

ABSTRACT

The study was aimed to view the developmental anomalies of gall bladder (GB) in Nepalese cadavers. Forty GBs were studied for any anomalies during routine cadaveric dissection at the Department of Anatomy, Manipal College of Medical Sciences, Nepal. The study found that the congenital anomalies of GB are very rare. Only one case of ectopic partial intrahepatic GB with cholelithiasis was observed, which to our knowledge is the first reported case in Nepal. Awareness of GB anomalies is important to surgeons, radiologists, and clinicians in general. An ectopic partial intrahepatic GB can make cholecystectomy hazardous, when indicated.

Keywords: Anatomical variation;cholecystectomy; gall stone; ectopic partial intrahepatic gall bladder.

INTRODUCTION

Gall bladder (GB) is an elongated pear shaped sac that lies on the visceral surface of the liver. The fundus of GB is an expanded blind anterior end of the organ projecting beyond the inferior margin of the liver. The body and neck of GB are attached to the visceral surface of the liver by the visceral peritoneum. Neck continues as cystic duct which joins the common hepatic duct to form the bile duct. Gall bladder normally is 7 to 10 cm in length, 3 cm broad at its widest part and has 30 to 50 ml capacity.^{1,2}

Gall bladder develops as an outgrowth from the hepatic diverticulum in the middle of the third intrauterine week, from the distal end of the foregut. The hepatic diverticulum grows into the septum transversum and along with vitelline and umbilical veins forms the various components of the liver. The outgrowth from the hepatic bud which forms GB remains outside the septum transversum.³

Anomalies of GB are rarely reported. The purpose of the present study was to view GB anomalies in Nepalese cadavers. The description of an ectopic partial intrahepatic GB with cholelithiasis observed during the cadaveric study is made note of with its clinical significance.

CASE REPORT

Forty adult 10.0% formalin embalmed Nepalese cadavers were studied at the Department of Anatomy, Manipal College of Medical Sciences, Pokhara, Nepal, for GB anomalies, from 2002 through 2006. The present cadaveric research study did not include any specific issue that needed to be approved by the Institutional Ethics Committees. The study conformed to the provisions of the Declaration of Helsinki in 1995 (as revised in Edinburgh 2000).

Out of 40 GBs, 39 were grossly normal. In one male cadaver, GB was partially submerged in the liver substance and hence, it was termed ectopic partial intrahepatic GB (Fig. 1). There was a remarkable reduction in the size of GB. The length of GB was 4 cm and fundus was falling short from the inferior margin of the liver. Upon dissection of GB, the lumen was filled with calculi which were extending up to the neck of GB (Fig. 2). The extrahepatic biliary apparatus was normal. The arrangements of structures at porta hepatitis were also normal.

DISCUSSION

Various anomalies of GB have been reported.⁴⁻⁶ Intrahepatic GB is a very rare congenital anomaly. To the best of our knowledge, this is the first case reporting an ectopic partial intrahepatic GB with cholelithiasis in Nepal.

An ectopic location of GB is very rare, incidence being 0.1 to 0.7 %.⁷ The most common malpositions of the GB are left-sided, transverse position, retroperitoneal and floating. Ectopic locations of GB are also reported in lesser omentum, retroduodenal, within the falciform ligament, within the abdominal wall muscles, and intrathoracic.⁸ Agensis of GB is a rare condition that appears to have genetic predisposition. The condition is associated with an increased incidence of primary sclerosing cholangitis and carcinoma of bile duct. Double and triple GBs are reported, the latter being extremely rare. Double GB may share a common cystic duct and may be completely separated, or they may be divided by a septum.

Clinical cases of intrahepatic GB are rarely reported in English literature.⁹⁻¹⁷ Intrahepatic GB is one of the ectopic locations and makes cholecystectomy hazardous in this anomaly.¹⁸ It is a rare congenital anomaly usually diagnosed by ultrasound in most instances either due to perforation of the GB causing abscess or due to cholecystitis with concurrent choledocholithiasis.^{19,20} Most of the gall stones (90.0%) are radiolucent and may not be visible on radiographs.²¹ Moreover, if the GB is intrahepatic with cholelithiasis, its clinical signs may mislead the surgeons.²² It is also critical to understand and note the variations of GB due to the increased incidence of laproscopic cholecystectomies. This anatomical knowledge would prevent liver and biliary apparatus damage.

Normal development of GB is from a diverticulum which arises from the ventral foregut caudal to the stomach. The diverticulum further divides into cranial and caudal parts. The cranial segment develops in to liver and intrahepatic ducts, while GB and extrahepatic biliary ducts develop from the caudal segment.³ The ectopic location of GB submerged in the liver could be due to the abnormal migration of the caudal segment, or the rapid growth of liver tissue which engulfs GB.

Though GB anomalies are relatively rare, it is prudent for the surgeon and the radiologist to note its abnormal positions especially when associated with gall stones. These abnormal positions should also be considered in the differential diagnosis of liver pathologies.

REFERENCES

1. Rosse C, Rosse PG. Hollenshed text book of anatomy. 5th ed. Philadelphia: Lippincott- Raven 1997; 572-3.
2. Williams PL, Warwick R, Dyson M, Bannister LH. Gray's anatomy. 37th ed. New York: ELBS Churchill Livingstone 1993; 1393-5.
3. Sadler TW. Langman's medical embryology. 8th ed. Philadelphia: Lippincott Williams and Wilkins 2000; 283-5.
4. Singh B, Satyapal KS, Moodley J, Haffejee AA. Congenital absence of the gall bladder. *Surg Radiol Anat* 1999; 21: 221-4.
5. Noritomi T, Watanabe K, Yamashita Y, Kitagawa S, Oshibuchi M, Shirakusa T. Left-sided gallbladder associated with congenital hypoplasia of the left lobe of the liver: a case report and review of literature. *Int'l Surg* 2004; 89: 1-5.
6. Azmat N, Francis KR, Mandava N, Pizzi WF. Agenesis of the gallbladder revisited laparoscopically. *Amer J Gastroenterol* 1993; 88: 1269-70.
7. Dhulkotia A, Kumar S, Kabra V, Shukla HS. Aberrant gallbladder situated beneath the left lobe of liver. *Official Journal of the International Hepato Pancreat Biliary Association* 2002; 4: 39-42.
8. Saygun O, Aydinuraz K, Daphan C, Akkus A, Aúalar F. Case report: intramesocolic malposition of the gallbladder. *Turk J Med Sci* 2004; 34: 281-2.
9. Dumont M, Danais S. Intrahepatic gallbladder simulating choledochal cyst on DISIDA scintigraphy. *Clin Nucl Med* 1984; 9: 657-8.
10. Lusink C, Sali A. Intrahepatic gall bladder and obstructive jaundice. *Med J Aust* 1985; 142: 53-4.
11. Lloyd TV. Empyema of an intrahepatic gallbladder. *Clin Nucl Med* 1979; 4: 341.
12. Matzsch T, Almqvist P, Berntorp E, Lethagen S, Montgomery A. Laproscopic cholecystectomy in a patient with haemophilia B. *Surg Laparosc Endosc* 1992; 2: 339-40.
13. Moreno AJ, Yedinak MA, Turnbull GL, Spicer MJ, Brown TJ. Cholelithiasis demonstrated on hepatobiliary scintigraphy as a photopenic defect within the inferior portion of the liver. *Clin Nucl Med* 1984; 9: 655-66.
14. Schulz RC, Sheilds JB, Fletcher JW, Donati RM. Liver scanning and the intrahepatic bladder: case report. *J Nucl Med* 1975; 16: 1029-30.
15. Soudry G, Ahn CS, Tow DE. Focal defect in liver colloid scan due to intrahepatic gallbladder. *Clin Nucl Med* 1995; 20: 460-1.
16. Velchik MG, Noel AW. False-positive liver scan due to an intrahepatic gall bladder detected by cholescintigraphy. *Clin Nucl Med* 1987; 12: 50-2.
17. Wysong CB, Gorten RJ. Intrahepatic gallbladder. *South Med J* 1980; 73: 825-6.
18. Sabiston DC. Text book of surgery: the biological basis of modern surgical practice. 14th ed. Philadelphia: WB Saunders Company 1991; 1043-4.
19. Peer A, Witz E, Manor H, Strauss S. Intrahepatic abscess due to gallbladder perforation. *Abdom Imaging* 1995; 20: 452-5.
20. Schmahmann JD, Dent DM, Mervis B, Kottler RE. Cholecystitis in an intrahepatic gallbladder: a case report. *S Afr Med J* 1982; 62: 1042-3.
21. Russel RCG, Williams NS, Bulstrode CJK. Bailey and Love's short practice of surgery. 23rd ed. London: Arnold Publishers 2000; 965-86.
22. Kumar V, Cotran RS, Robbins SL. Basic pathology. 6th ed. Singapore: Harcourt Asia Pvt. Ltd 1997; 549-50.



Figure 1. Inferior surface of the liver with intact gall bladder. L, left lobe of the liver; R, right lobe of liver; GB, intact gall bladder.



Figure 2. Inferior surface of the liver with dissected gall bladder. L, left lobe of the liver; R, right lobe of liver; GB, gall bladder showing gall stones; F, fundus of gall bladder; LT, ligamentum teres at the anterior border of liver.