Congenital absence of gall bladder is a rare anomaly of the biliary system and is important in clinical practice as it may cause some clinical, surgical and diagnostic problems. Approximately 400 cases have so far been reported in the literature with a reported incidence ranging between 0.013-0.075%.

Subjects with gall bladder agenesis are classified into 3 categories, symptomatic (55.5%); asymptomatic (31.6%) and multiple fetal anomalies (12.9%). The CAGB may cause feeling of stuffed and stuck after eating, irritating gas symptoms, dyspepsia, pain in epigastrium and upper right quadrant, diarrhea, biliary colic and jaundice. The present communication describes the first case of CAGB to be reported in the Kingdom of Saudi Arabia (KSA) with special emphasis on clinical, hematological findings and body composition.

In this study, the subject was a 20-year-old Saudi student at the College of Pharmacy, King Saud University, Riyadh, KSA. One day after a discussion on the functions and applied physiology of gall bladder in the lecture hall, the student came forward and asked some questions regarding the functions of liver and gall bladder then gave a history of pain in right hypochondrium. Physical examination showed a pulse rate of 80 beats/min; respiratory rate 18 times/min; blood pressure 120/70 mm Hg and temperature was 98.4°F. In addition, subject was also examined for anemia, cyanosis, jaundice, edema, clubbing, koilonychia and lymph node enlargement but no abnormal findings were observed. Ultrasonography of the abdomen with special emphasis to biliary system showed the absence of gall bladder. The ultrasonography of the abdomen was repeated in a different radiology division for second opinion and results were confirmed. Computerized tomography scan of the biliary system was also examined for the gall bladder. Laboratory investigations revealed complete blood count (CBC) and showed no abnormal findings; RBCs 5.5x10^6/µL, hemoglobin 16.0 g/dl, HCT 47.3%, MCH 28.7 pg, MCHC 33.7 g/dl, WBCs 5.8x10^3/µL; neutrophils 64%, eosinophils 2%, basophils 0.3%, lymphocyte 27.8%, monocytes 5.9%, platelets 211x10^9/µL, bleeding time 9 minutes and 45 seconds; clotting time 6 minutes and 15 seconds.

Similarly, serum biochemistry was normal; Na+ 140mmol/L, K+ 4.8mmol/L, Cl− 100mmol/L, albumin 46g/L, triglycerides 1.08mmol/L, cholesterol 4.3mmol/L, total protein 77g/L, BUN 4.4mol/L, creatinine 79umol/L, glucose 3.6mmol/L, total bilirubin 15umol/L and uric acid 224.3umol/L. Serum enzymes: Aspartate aminotransferase (AST) 15U/L, lactate dehydrogenase (LDH) 184U/L, creatine kinase (CK) 137U/L, alanine aminotransferase (ALT) 32U/L, alkaline phosphatase (ALP) 104U/L, amylase 72U/L and glutamyl transpeptidase (GT) 26U/L.

In the present case study an additional step was taken and body composition was analyzed, using body composition analyzer (Biospace, Korea). The results showed body mass index (BMI) 23.5kg/m², basal metabolic rate (BMR) 1822.7kcal, total body fluid 40.2L, extra cellular fluid 12.7L, intra cellular fluid 27.5L, protein mass 11.0kg, soft lean mass 51.5kg, fat mass 14.6kg, lean body mass 54.8kg; Percent body fat 21.1%; Mineral mass 3.51 and obesity degree was 108%. However, the overall body fitness was 75%. In addition to these investigations, clinical examination of all body systems including, central nervous system, cardiovascular, respiratory and gastrointestinal systems showed no abnormalities except that during deep abdominal palpation, the subject complained of pain in right hypochondrium. The causes of the embryonic injury, which could result in CAGB and the malformations associated with it, are unknown. However, the reasons for symptoms of CAGB are still obscure. If there is associated biliary pathology
such as common duct or intrahepatic duct stone, the resulting symptoms are readily justified. But in the present case, the subject present a pain in right hypochondrium.

Meshkinpour et al\(^5\) demonstrated that a subject with normal pancreatic biliary tree had a significantly higher sphincter of oddi resting pressure, as well as increased retrograde propagation of phasic musculature contraction may cause pain. Sing et al\(^2\) classified the subjects with gall bladder agenesis into 3 categories; 1. Multiple fetal anomaly (12.9%), 2. Asymptomatic (31.6%) and 3. Symptomatic (55.5%). The functions of gall bladder is storage and concentration of the bile though its impaired functions can cause a feeling of stuffed and stuck after eating, irritating gas symptoms, dyspepsia, diarrhea. In subjects with CAGB, Richards et al\(^4\) found that, the dyspepsia was the predominant symptom in 15 of 44 patients (34%), 24 of 44 (54%) had symptoms suggestive of biliary colic, 12 of 44 (27%) had jaundice. Chang et al\(^3\) reported that all the 9 cases with congenital absence of gall bladder complained of one or more biliary symptoms such as jaundice, epigastric pain, and pain in upper right abdominal quadrant. In addition, it has been also suggested that the awareness of the possibility of agenesis of gall bladder may allow the surgeon to attempt confirmation of diagnosis by non-operative methods and avoid surgical exploration in specific instances.

In conclusion, CAGB is a rarely encountered condition, however, extensive diagnostic workup including abdominal ultrasonography and computerized tomography should be performed in situations when CAGB is suspected and unnecessary laparotomy should be avoided. In addition, it should be keep in mind that CAGB does not cause serious problems related to the general health of the subject in early decades of life. However, follow-up examination should be conducted to find out the delayed effects of CAGB.

Acknowledgment. We are highly thankful to Prof. AM Abdel Gader, Department of Physiology and Dr. Sohail Sabir, Department of Radiology, College of Medicine, King Khalid University Hospital, Riyadh, Kingdom of Saudi Arabia, for their critical review of the manuscript. We also extend our thanks to our student (Subject) for his patience and cooperation.

Received 5th April 2004. Accepted for publication in final form 21st June 2004.

From the Department of Physiology, College of Medicine, King Saud University, Riyadh, Kingdom of Saudi Arabia. Address correspondence and reprint requests to Dr. Sultan A. Meo, Assistant Professor, Department of Physiology (29), College of Medicine, King Khalid University Hospital, PO Box 2925, Riyadh 11461, Kingdom of Saudi Arabia. Tel. +966 (1) 4671604. Fax. +966 (1) 4671046. E-mail: sultanmeo@hotmail.com

References