INTRODUCTION

Common bile duct (CBD) gangrene is an extremely rare cause of spontaneous biliary perforation leading to life-threatening biliary peritonitis. Only one such case has been reported, way back in the year 1951. Herein, we present an unusual case of massive CBD gangrene that also involved hepato-duodenal ligament. Moreover, we propose an algorithm for its preoperative diagnosis. To our knowledge, such a case is yet to be reported.

CASE REPORT

A 52-year-old male was admitted under the Department of Internal Medicine at our rural academic institute for evaluation of ascites and was referred to us for surgical consultation for his recent-onset painless jaundice. Chronic alcoholic since 15 years, he had noticed progressive abdominal distension and yellowness of his sclera over three days. He denied any recent abdominal pain, fever or trauma. With regular bowel habits, he passed yellow-brown stools and had no medical co-morbidities. On examination, he had normal vital parameters, was mildly icteric, and had no signs of chronic liver failure. His abdomen was uniformly distended, non-tender, with no organomegaly. Positive shifting dullness confirmed presence of free fluid in his abdomen. The hematological screen, except for raised serum bilirubin level, was uniformly normal.

On admission, his recent-onset painless jaundice was attributed to obstructive jaundice. However, with serial ultrasound imaging, the serum bilirubin content to that of the serum was as high as 10:1, suggesting CBD gangrene. His abdominal examination revealed a distended, non-tender abdomen. The hematological screen, except for raised serum bilirubin level, was uniformly normal.

The friable sub-serosal parenchymal layer of the CBD was exposed and the gangrenous and continuously leaking golden-yellow bile was sucked out, and the serum bilirubin level returned to the standard by day 3. He passed regular stools on 4th day and tolerated oral diet thereafter. As expected, his sub-hepatic drain yielded 200–300 ml typical golden-yellow bile everyday till day 11 (Figure 3).

Our case has several notable learning points: 1) massive CBD gangrene with its near-total disruption to cause choleperitonem, when most of the reported perforations are limited to <1 cm; 2) possible involvement of entire hepatoduodenal ligament in the gangrenous process which has not been reported so far; 3) clinical silence despite the severity for over three days; 4) with negligible amylase level. After adequate resuscitation he underwent emergency exploratory laparotomy.

At surgery, about 2.5 liters of non-turbid bile poured from the peritoneal cavity. The gallbladder, the entire bowel and the pancreas were all healthy, and the lesser sac was empty. Also, the whole hepato-duodenal ligament along with the CBD was gangrenous and continuously leaked golden-yellow bile (Figure 2). Here, any attempts to probe such a paper-thin CBD were restrained for avoiding further damage. The friable sub-hepatic tissues along with the impending ionotropic support discouraged even duodenal Kocher maneuver and signified “primum non nocere”.

Rigorous peritoneal lavage was followed by a drain each in the Morrison’s pouch and in the pelvis before abdominal closure.

With dramatic postoperative recovery, his jaundice subsided and the serum bilirubin level returned to the standard by day-3. He passed regular stools on 4th day and tolerated oral diet thereafter. As expected, his sub-hepatic drain yielded 200–300 ml typical golden-yellow bile everyday till day 11 (Figure 3).

A check abdominal contrast-enhanced computer tomogram (CECT) depicted a non-enhancing CBD having patchy mural loss without any calculi in its lumen or in the peritoneal cavity. Its axial blood vessels could not be delineated. The pancreas and its duct were normal, and both the drain-tubes were in situ. There were no residual fluid collections, and the oral contrast confirmed absence of duodenal perforation (Figure 4).

After optimizing his nutritional and performance status over three weeks, he underwent definitive surgery in the form of CBD excision followed by Roux-en-Y hepaticojejunostomy and had satisfactory recovery thereafter.

DISCUSSION

Our case has several notable learning points: 1) massive CBD gangrene with its near-total disruption to cause choleperitonem, when most of the reported perforations are limited to <1 cm; 2) possible involvement of entire hepatoduodenal ligament in the gangrenous process which has not been reported so far; 3) clinical silence despite the severity for over three days; 4)
policies; this could be of great concern at resource-limited settings. Postoperative external biliary fistula showing clear golden-yellow bile with negligible amylase further corroborated CBD pathology. And, its low-output nature suggested biliary-enteric continuity. Finally, postoperative CECT substantiated our diagnostic algorithm. However, as the exact cause of this vascular insult could not be identified, it was deemed idiopathic.

REFERENCES

1. Amberger M, Burton N, Tissera G, Baltazar G, Palmer S. Spontaneous common bile duct perforation—A rare clinical entity. Int J Surg Case Rep 2018;46:34-37.
2. Hamura R, Haruki K, Tsutsumi J, Takayama S, Shiba H, Yanaga K. Spontaneous biliary peritonitis with common bile duct stones: report of a case. Surg Case Rep 2016 Dec;2(1):103.
3. Hart DE. Spontaneous perforation of the common bile duct. Ann Surg 1951;133(2):280-282.
4. Paramhans D, Shukla S, Grover J. Spontaneous perforation of the common bile duct in an adult. Indian J Surg 2013 Jun;75(Suppl 1):376-8.
5. Subasinghe D, Udayakumara EA, Somathilaka U, Huruggamuwa M. Spontaneous Perforation of Common Bile Duct: A Rare Presentation of Gall Stones Disease. Case Rep Gastrointest Med 2016;2016:5321304.