

Case Report

Bouveret Syndrome: A Rare Presentation- Managed With Combined Surgical And Endoscopic Approach.

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Abstract

Bouveret syndrome occurs when a gallstone migrates through a cholecystoduodenal (bilioenteric) fistula and obstructs the gastric outlet or proximal duodenum. We report a patient who presented with two months history of epigastric pain and recurrent vomiting for one month. Initial imaging (USG and CECT abdomen) showed cholelithiasis with a partially distended gallbladder, grossly distended stomach, and two calcific densities in the pyloric antrum suggestive of migrated stones. Four serial upper GI endoscopies were performed: the first revealed hard extrinsic compression at D1 with scope breakage; biopsies ruled out malignancy. Post-operative open subtotal cholecystectomy, duodenotomy, stone extraction, primary D1 repair, and feeding jejunostomy resolved the obstruction. Subsequent endoscopies documented progressive improvement—scope negotiation to D2 was possible by the fourth attempt, correlating with symptom resolution and tolerance of solid diet. This case highlights the value of multidisciplinary gastroenterology-surgical management in Bouveret syndrome, avoiding unnecessary re-laparoscopic intervention.

Keywords: Bouveret syndrome, Gastric outlet obstruction, Gallstone ileus, Endoscopy Cholecystoduodenal fistula, Surgery.

INTRODUCTION

Bouveret syndrome, a rare variant of gallstone related obstruction in the gastrointestinal tract, occurs when a gallstone migrates through a cholecystoduodenal (bilioenteric) fistula and obstructs the gastric outlet or proximal duodenum (1,2). Bouveret syndrome is an uncommon cause of gastric outlet obstruction (GOO), accounting for 2%-3% of all gallstone related obstruction in gastrointestinal tract, which by itself constitutes 1%-4% of all small bowel obstruction, thus rarity of this syndrome. It results from impaction of a gallstone at the pylorus or proximal duodenum following spontaneous cholecystoduodenal fistula formation. Diagnosis often requires multimodal imaging and endoscopy (3), while treatment typically involves surgical extraction—though endoscopic attempts are increasingly reported. We describe a challenging case managed with serial endoscopy and definitive surgery, emphasizing the role of repeated endoscopic monitoring in assessing resolution.

CASE REPORT

A 33-year-old male patient, presented with progressive epigastric pain for two months and vomiting for one month. He had no prior biliary symptoms or weight loss. On admission, abdominal ultrasound revealed a partially distended gallbladder with multiple calculi (6–8 mm) and normal common bile duct. Contrast-enhanced CT abdomen demonstrated mild hepatic steatosis, marked gastric distension (body and antrum), and two hyperdense calcific foci in the pyloric antrum, raising suspicion for migrated gallstones causing GOO. Upper GI endoscopy (first) showed severe extrinsic compression at the D1; the scope could not pass and fractured against some extrinsic hard obstruction, most probable stone. Multiple biopsies were taken—histology revealed only congestion, haemorrhage, and dense lymphocytic infiltration of lamina propria, excluding malignancy. Surgical exploration followed: open subtotal cholecystectomy, duodenotomy with extraction of gallstones,

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primary closure of the D1 defect, feeding jejunostomy, and abdominal drainage. There was gross edema at D1 and D2 level. Post-operative MRCP confirmed absent gallbladder and no residual biliary abnormality. Serial endoscopy documented recovery. The patient was put on proton pump inhibitors (PPI) with prokinetics. The second endoscopy done two weeks post-operatively, revealed passage of scope improved but still obstructed at D1 and at this point of time, patient had begun accepting oral liquids. The patient later on was able to take small amount of solid food, thus to visualize the obstruction part, third endoscopy was done after 6 weeks of surgery which showed residual food debris in stomach but scope negotiation became better and it reached D1-D2 junction but was still not able to reach D2. The patient was kept on PPI and prokinetics and was motivated to increase oral intake, in addition to support from feeding jejunostomy. Patient gradually improved and now was able to take normal diet orally, as he was taking before this illness episode. Thus, before removing feeding jejunostomy, fourth endoscopy was done at 10 weeks post-operatively during which endoscope was negotiated into D2 with minimal oedematous folds at D1. Thus, feeding jejunostomy was removed and patient remains well at follow-up with no recurrence of symptoms.

Figure 1. CT Scan abdomen showing Contracted and thickened gall bladder (green arrow) and dilated pyloric antrum (yellow arrow) and whole stomach with mild air in fundus.

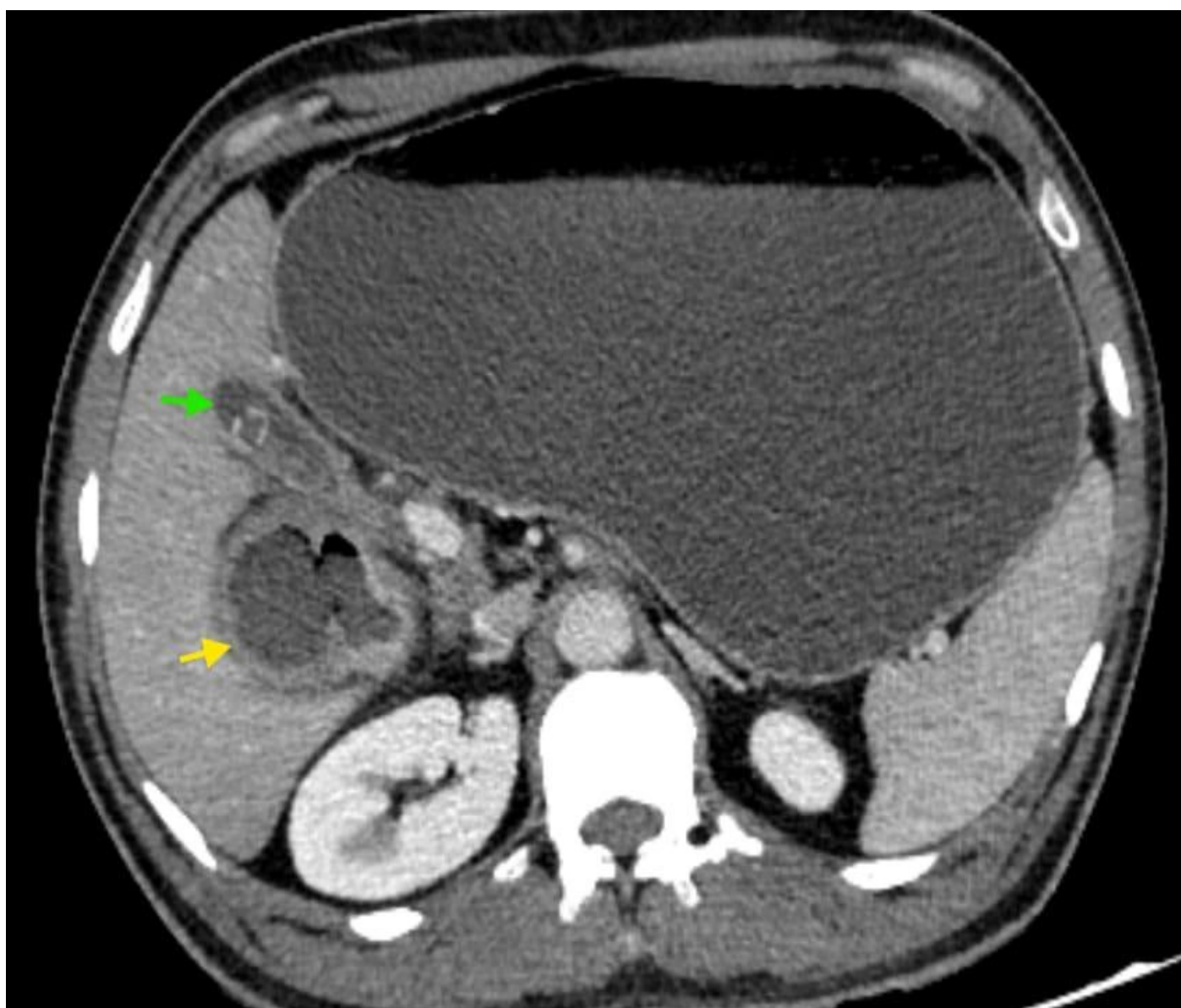


Figure 2. CT scan abdomen showing dilated duodenum and pyloric antrum (blue arrow) and impacted stone between duodenum and pyloric antrum (red arrow).

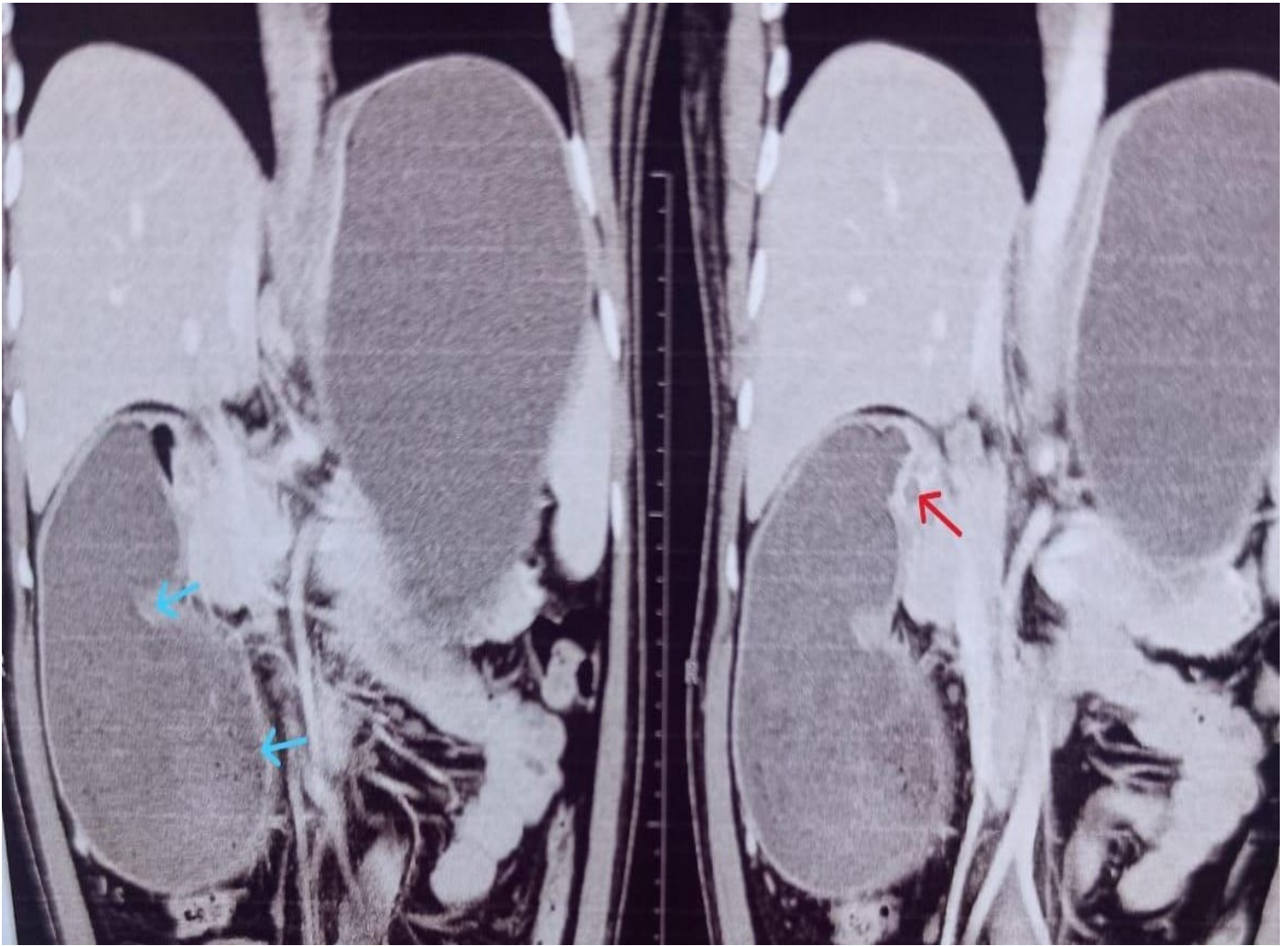


Figure 3. Barium meal and follow through showing dilated stomach and narrowing at duodenal level.

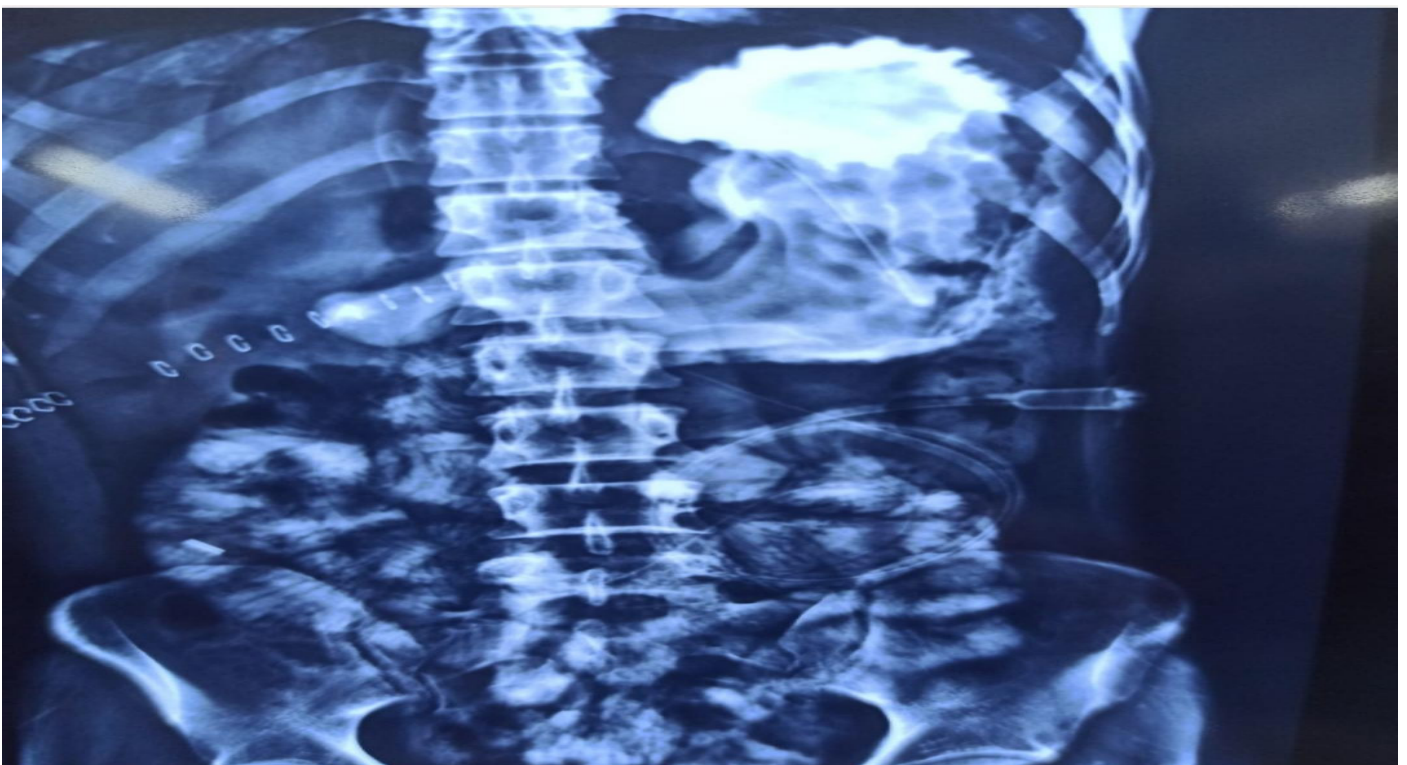


Figure 4. Second Endoscopy done post-operatively showing gross edema at D-D2 Junction which was non-negotiable.



Figure 5. Fourth Endoscopy done post-operatively showing mild edema and puckering at D1-D2 Junction.

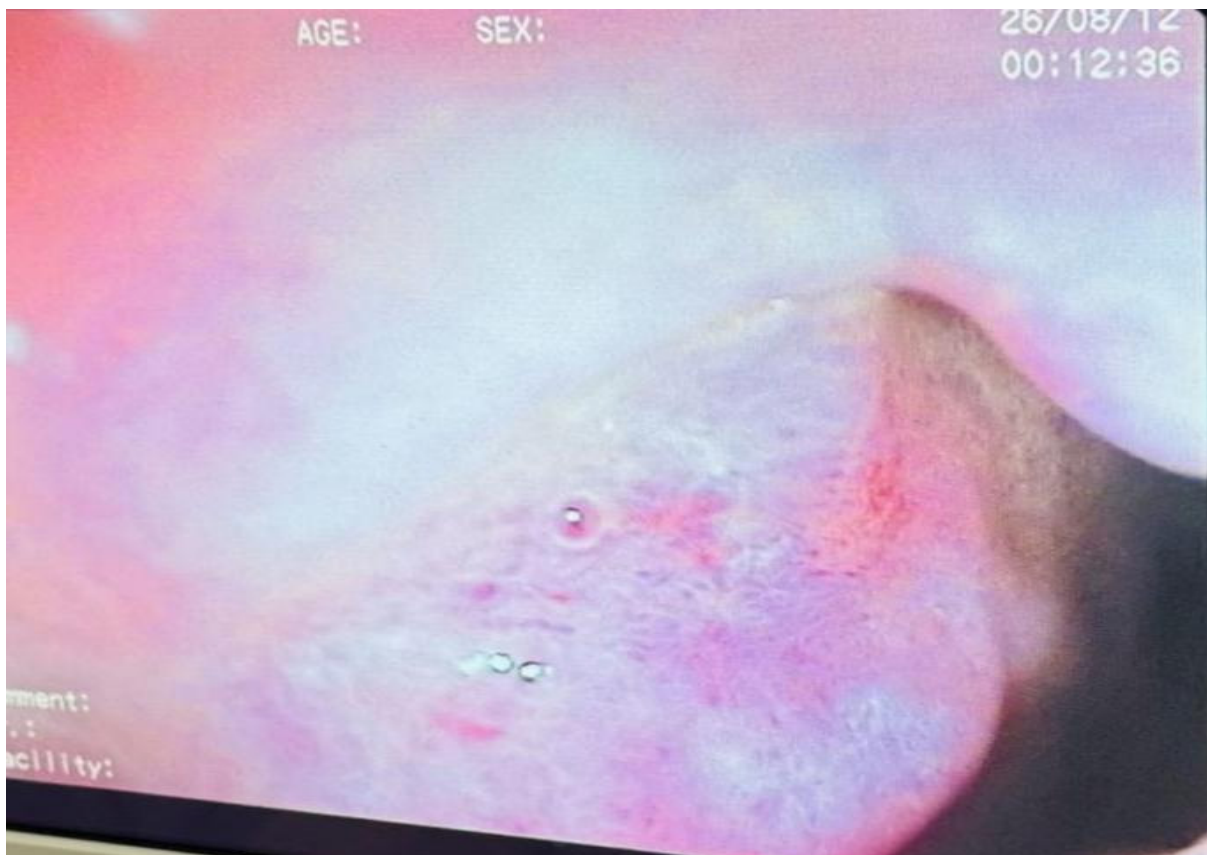


Figure 6. Fourth Endoscopy done post-operatively showing endoscope reaching Duodenum second part (D2).



Figure 7. Normal Barium Meal and Follow through after complete recovery of patient.



DISCUSSION

The pathophysiology of Bouveret syndrome involves chronic inflammation within the gallbladder, usually in the setting of longstanding cholelithiasis, resulting in an erosion into adjacent gastrointestinal structures, most commonly the duodenum (4). This erosion creates a fistulous tract that permits large gallstones to pass into the bowel, where they may become lodged in the duodenum or pylorus, causing an obstruction (5). The size of the gallstone, typically over 2-3 cm, plays a crucial role in determining the likelihood of obstruction, as stones of this size rarely pass spontaneously through the bowel (5). This obstruction presents unique challenges in diagnosis and management due to its rare occurrence and overlap in symptoms with other causes of gastric outlet obstruction (6). Bouveret syndrome classically presents with GOO symptoms and is diagnosed by imaging showing Rigler's triad (pneumobilia, ectopic gallstone, bowel obstruction)—though pneumobilia was absent here, likely due to early fistula closure. Repeated endoscopy proved crucial: it ruled out malignancy, monitored post-operative edema resolution, and avoided re-operation. Surgical stone removal remains gold standard, but our case supports adjunctive endoscopic follow-up as a safe, low-cost tool for confirming patency (7-9).

CONCLUSION

This report underscores the diagnostic and therapeutic synergy between gastroenterology and surgery in Bouveret syndrome. Serial endoscopy can guide management, prevent unnecessary re-exploration, and document complete resolution—offering a practical template for similar rare cases

Conflict of interest

The authors declare that there is no conflict of interest and no financial support was taken for the same.

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