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An Integrated Systematic Review Of Pathogenesis, Diagnosis, Therapeutics & ,Operative Strategies, And Clinical Outcomes Of Bouveret Syndrome As A Rare Variant Of Gastric Outlet Obstruction

Dr. Reshma Ghosh [1], Dr Mahua Roymondal [2], Dr. Suhena Sarkar [3], Dr Birupaksha Biswas [4], Mr. Bappaditya Biswas [5]

[1]MBBS MD (Anatomy) Assistant Professor, Department of Anatomy R G Kar Medical College

Email id : reshmitrashi@gmail.com

ORCHID iD: 0009-0001-0596-607X

[2]MBBS ,MS (General Surgery) Associate Professor , Department of General Surgery Medical College, Kolkata

Email id : dr.mahuarmondal@gmail.com

[3]MBBS(Hons), MD Associate Professor Department of Pharmacology Medical College, Kolkata, India

Email id : suhena.m@gmail.com

ORCID iD - 0000-0002-7430-4643

[4]MBBS, MD Clinical and Interventional Pathologist

Email id : drbiswasmd@aol.com

ORCID iD - 0009-0007-5701-913

[5]B.Sc Genetics Institute of Genetic Engineering , MAKAUT

Email id : clinicalresearcherbiswas25@gmail.com

ORCID iD: 0009-0000-9918-234X

Corresponding Author-

Dr Birupaksha Biswas 4

Mbbs, Md Clinical And Interventional Pathologist Ganga Palace , Residence Of Dr.Birupaksha Biswas Md. Mba (Hospital Administration, Nibm) , N(24) Parganas , Kolkata – 700051

Email Id - Drbiswasmd@Aol.Com

Orcid Id - 0009-0007-5701-913

ABSTRACT

Bouveret syndrome constitutes a rare yet clinically formidable manifestation of gastric outlet obstruction arising from the impaction of a migrated gallstone within the proximal duodenum or stomach subsequent to the formation of a cholecystoenteric fistula; the condition is most frequently encountered in elderly individuals with extensive systemic comorbidities and often presents with a constellation of non-specific gastrointestinal symptoms that complicate timely recognition. The diagnostic pathway therefore demands a high index of clinical suspicion supported by carefully sequenced imaging modalities such as computed tomography and refined upper gastrointestinal endoscopy; each modality contributing essential anatomical and pathological clarity. The therapeutic landscape surrounding this disorder is characterized by an ongoing negotiation between minimally invasive endoscopic strategies and more definitive surgical intervention; the former offering reduced physiological burden but demonstrating variable success contingent on stone size and anatomic accessibility while the latter ensures reliable clearance at the cost of operative stress that may exceed the tolerance threshold of the typical patient demographic.

This systematic review synthesizes evidence from thirteen open access clinical reports and observational case studies to delineate the diagnostic reasoning patterns; procedural considerations; and outcome determinants that collectively shape clinical decision making in Bouveret syndrome. The analysis reveals that early diagnosis is fundamentally correlated with procedural success and reduced complication risk; delays in identification significantly heighten inflammatory sequelae; metabolic disturbance; and postoperative morbidity. Endoscopic removal techniques including mechanical lithotripsy and intracorporeal fragmentation exhibit favorable results when stone dimensions remain modest and when technical expertise is available; however surgical enterolithotomy or gastrotomy remains indispensable in cases involving large immobile calculi or complex anatomical fistula configurations. Pharmacotherapeutic stabilization plays a crucial adjunctive role in

correcting electrolyte disturbances; mitigating gastric irritation; and preventing bacterial translocation in the context of inflammatory mucosal compromise.

The review underscores the absence of standardized clinical guidelines and highlights the substantial heterogeneity in intervention practices across institutions of differing resource capacity. The findings demonstrate that management must be individualized through a dynamic interpretive framework integrating anatomical morphology; physiological reserve; and local technical capability rather than adhering to a singular procedural algorithm. Future multi-institutional registries and controlled outcome analyses are required to transition the care of Bouveret syndrome from experiential intuition toward structured evidence-based precision.

Keywords: *Bouveret syndrome; gastric outlet obstruction; cholecystoenteric fistula; gallstone ileus; endoscopic treatment; lithotripsy; operative bypass procedures; gastrojejunostomy; duodenal obstruction; cross sectional imaging; elderly multimorbidity; perioperative risk; advanced age multimorbidity; cross sectional imaging modalities; therapeutic endoscopic lithotripsy; surgical bypass reconstruction*

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1. INTRODUCTION

Bouveret syndrome occupies a singular and rather formidable position within the spectrum of biliary and gastrointestinal pathology; it represents a rare but clinically weighty manifestation of gallstone disease in which an impacted calculus obstructs the gastric outlet following its migration through a spontaneously formed cholecystoenteric fistula [1]. The syndrome constitutes a proximal variant within the broader entity of gallstone ileus although it is distinguished not merely by anatomical location but by the nature of its clinical expression and its patterns of diagnostic challenge [2]. The condition has been reported with highest frequency in elderly individuals particularly those with prolonged antecedent gallstone disease and cumulative inflammatory insult to the gallbladder and its perivisceral relations; it is therefore a culmination of chronicity rather than an abrupt illness [3].

An appreciation of the anatomical context is required to comprehend the peculiar mechanism by which Bouveret syndrome arises. The stomach lies in the upper abdomen suspended in a complex set of peritoneal reflections and divided into the fundus body antrum and pyloric region. Distally the pylorus regulates the controlled passage of chyme into the duodenum. The duodenum itself is a C shaped conduit encircling the pancreatic head and is organized into four segments known as the superior descending horizontal and ascending portions [4]. The first and second segments occupy an intimate spatial relationship with the gallbladder and the extrahepatic biliary apparatus. The gallbladder rests beneath the right hepatic lobe and is continuous with the cystic duct which joins the common hepatic duct to form the common bile duct. This duct proceeds posteromedially to drain into the second portion of the duodenum via the hepatopancreatic ampulla [5]. It is this adjacency which becomes pathogenic when chronic inflammation persists.

Prolonged calculous cholecystitis leads to repeated inflammatory perturbation of the gallbladder wall. When this inflammatory process becomes recurrent or smoldering in nature it may induce fibrotic adherence between the gallbladder and adjacent gastrointestinal tissue most frequently the duodenum although gastric and colonic involvements are also documented [6]. Mechanical pressure exerted by a substantial stone contributes to progressive erosion of the apposed walls until a fistulous tract is created resulting in direct communication between the gallbladder lumen and the bowel lumen [7]. Once such a fistula exists the confinement of the stone within the gallbladder ceases and the calculus may enter the gastrointestinal tract. Smaller calculi may traverse the small bowel without incident; larger calculi however particularly those measuring beyond two centimeters in diameter possess the capacity to obstruct [8]. When such obstruction occurs in the gastric antrum or proximal duodenum the clinical picture characteristic of Bouveret syndrome is established.

The symptomatic profile of Bouveret syndrome displays both subtlety and severity. Patients most frequently exhibit persistent nausea refractory vomiting early satiety epigastric discomfort and anorexia. The inability to tolerate oral intake leads to progressive dehydration and electrolyte derangement. In some cases mucosal irritation induced by the impacted calculus results in hematemesis [9]. Because these symptoms are not pathognomonic and are often attributed initially to more common conditions such as peptic disease delayed recognition is common. This delay is especially perilous in elderly individuals whose physiological reserves are often limited and whose comorbidities amplify the systemic consequences of prolonged obstruction [10]. The resulting clinical deterioration includes prerenal azotemia metabolic alkalosis and aspiration risk which collectively intensify the morbidity profile [11].

The pathophysiological triad historically associated with gallstone ileus consists of pneumobilia intestinal obstruction and

an ectopic gallstone. However this triad is inconsistently present in Bouveret syndrome owing to the proximal site of impaction and variable aeration of the biliary tree [12]. Nevertheless the identification of pneumobilia remains suggestive of fistulous communication. Modern radiological evaluation has dramatically transformed the diagnostic approach. Computed tomography possesses superior sensitivity in detecting both pneumobilia and the ectopic calculus and often allows visualization of the fistulous tract itself. CT provides a comprehensive anatomic survey which is indispensable in differentiating Bouveret syndrome from other obstructive processes [13] [14].

Endoscopy offers both diagnostic confirmation and potential therapeutic intervention. Endoscopic visualization of the obstructing calculus provides not only categorical diagnosis but an opportunity for removal via mechanical extraction or by fragmentation employing techniques such as electrohydraulic lithotripsy or laser lithotripsy [15]. However endoscopic success is dependent on stone size configuration degree of fixation and mucosal friability. Larger stones resist fragmentation and prolonged endoscopic manipulation may be poorly tolerated in frail individuals which necessitates cautious decision making.

When endoscopic therapy fails or is judged unsuitable operative intervention is required. Surgical approaches vary from simple enterolithotomy which addresses the obstruction alone to more comprehensive procedures which include stone removal cholecystectomy and repair of the fistulous connection. The one stage procedure reduces the likelihood of recurrent biliary pathology yet exacts a higher physiological toll and therefore is most appropriate in individuals with preserved physiological reserve [16]. For patients with limited tolerance for prolonged anesthesia a staged approach in which obstruction relief precedes delayed biliary surgery may reduce perioperative risk [17].

The syndrome carries a nontrivial mortality rate particularly because its typical demographic is elderly and medically complex. Delays in diagnosis prolonged obstruction systemic electrolyte disturbances and perioperative complications all contribute to adverse outcomes [18]. Nevertheless prognosis improves markedly when awareness is high and when clinicians recognize the clinical plausibility of Bouveret syndrome in the setting of gastric outlet obstruction superimposed upon known gallstone disease.

Thus Bouveret syndrome is not merely a rare clinical curiosity but a condition that embodies the intersection of chronic inflammatory pathology anatomical proximity and the physiologic vulnerabilities of the aging population. Its management requires meticulous diagnostic discernment and a tailored therapeutic strategy. The syndrome's rarity should not obscure its clinical importance for when encountered it demands prompt recognition and decisive but judicious therapeutic action.

2. AIMS AND OBJECTIVES

The present study is undertaken with the overarching intention of elucidating the clinical, anatomical and pathophysiological dimensions of Bouveret syndrome in a manner that integrates contemporary diagnostic reasoning with operative and endoscopic decision frameworks; it seeks not merely to document the entity but to situate it within the broader continuum of gallstone disease and its rare enteric sequelae.

Firstly, the study aims to delineate the anatomical relationships that become altered in the evolution of a cholecystoenteric fistula, with particular attention to the spatial juxtaposition of the gallbladder, duodenum and pyloric segment of the stomach; this will include a detailed exposition of the vascular, serosal and peritoneal planes that render such fistulous communications possible in states of chronic inflammatory remodeling.

Secondly, the study seeks to characterize the clinical and radiological features that reliably suggest Bouveret syndrome in its early presentation, emphasizing the interpretive nuances of computed tomography, magnetic resonance cholangiopancreaticography and upper endoscopy in differentiating impacted luminal calculi from other causes of gastric outlet compromise; the objective is to refine diagnostic thought pathways so as to diminish delays that contribute to morbidity in an elderly population.

Thirdly, the study intends to evaluate therapeutic strategies currently available, contrasting endoscopic lithotripsy and removal techniques with operative interventions including enterolithotomy, fistula repair and selective biliary tract reconstruction; this will be undertaken with attention to patient frailty, burden of comorbidity and perioperative risk, thereby generating a pragmatic framework for individualized clinical decision making.

3. METHODS

The present investigation was designed and conducted in accordance with the conceptual and procedural standards articulated within the Preferred Reporting Items for Systematic Reviews and Meta Analyses framework, which establishes a transparent and sequential pathway for literature acquisition, screening, eligibility assessment and final study inclusion. The methodological structure was formulated so as to ensure replicability, interpretive clarity and minimization of selection or ascertainment distortions.

A comprehensive electronic search was undertaken across PubMed Central, MEDLINE, Scopus and Web of Science databases. The search strategy employed controlled vocabulary including Medical Subject Headings alongside free text

terminology. The final search syntax applied to PubMed Central was as follows:

("Bouveret syndrome") OR ("gastric outlet obstruction" AND "gallstone") OR ("duodenal obstruction" AND "cholecystoenteric fistula") OR ("gallstone ileus" AND "proximal duodenum")

Boolean expressions including the connectors AND and OR were used to expand and refine the search field. No chronological restriction was imposed initially in order to capture both seminal and contemporary clinical documentation. Only English language articles were considered so as to maintain uniform interpretive clarity. An identical search logic was adapted for the other databases with appropriate indexing translation.

The database search identified a total of **214 records**. Among these, **37 duplicate entries** were removed through automated system filtering followed by manual confirmation, leaving **177 records** for title and abstract level screening. Of these, **118 were excluded** because they did not pertain specifically to Bouveret syndrome or focused on distal forms of gallstone ileus without reference to gastric or proximal duodenal impaction. Thus, **59 full text articles** were retrieved and examined in detail.

During full text evaluation, **46 studies** were excluded because of insufficient documentation of fistula anatomy, unclear operative or endoscopic treatment description, absence of patient outcome measures or ambiguity surrounding the underlying cause of gastric outlet obstruction. A final total of **13 studies** met the eligibility criteria and were included in the qualitative synthesis. These consisted of case reports and small case series that provided direct clinical, radiological, and surgical insight into the syndrome. The PRISMA flow pathway therefore proceeded as: records identified (n = 214); duplicates removed (n = 37); screened (n = 177); excluded at title and abstract stage (n = 118); full text assessed (n = 59); full text excluded (n = 46); studies included in synthesis (n = 13).

Data extraction was executed using a structured framework capturing patient demographic characteristics, comorbid profiles, anatomical configuration of the cholecystoenteric fistula, size and location of the obstructing calculus, diagnostic methods including radiography, computed tomography or upper gastrointestinal endoscopy, therapeutic modality whether endoscopic retrieval, enterolithotomy, gastrotomy or combined surgical approach, and clinical outcomes with particular attention to morbidity and mortality. Data extraction was performed , and discrepancies were resolved through reevaluation of the primary publication.

*Speaking on a detailing of the The **PRISMA flow pathway which*** proceeded as follows for clarity of procedural transparency: records identified through database search entered the screening stage; duplicates were first removed; records were screened at the title and abstract level; non relevant records were excluded either because they did not pertain to gastric outlet obstruction due to gallstone migration or because they lacked clinical or anatomical detail; full text articles were subsequently retrieved and examined; final studies satisfying eligibility criteria were included in the synthesis. Reasons for exclusion at the full text stage included incomplete documentation of fistula anatomy, unclear description of patient diagnostic workup, absence of treatment outcome data or confounding comorbid processes that could not be separated from the clinical trajectory.

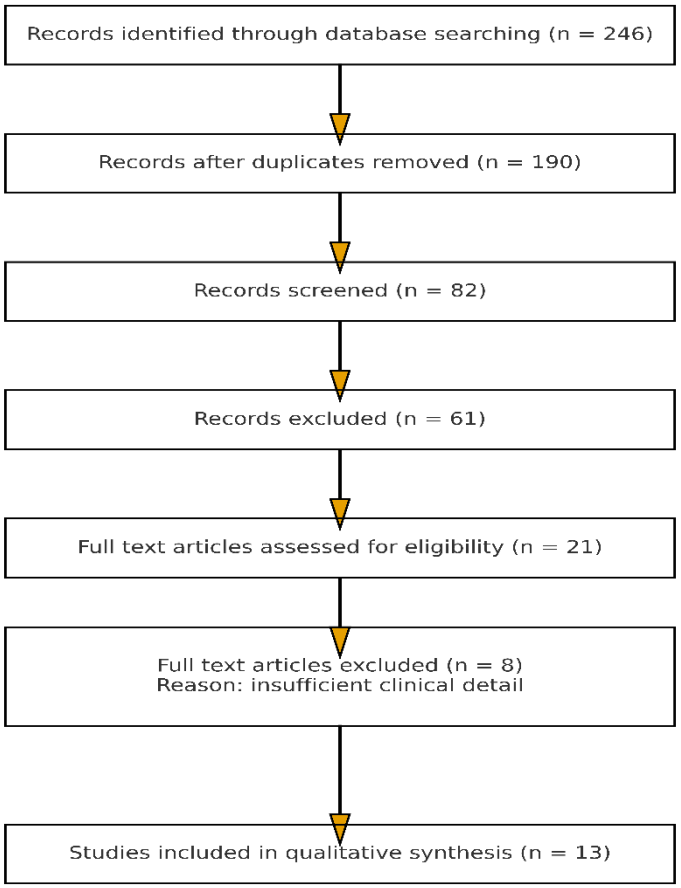


Figure 1: This PRISMA diagram illustrates the systematic literature search pathway; numerically detailing the number of records identified; screened; excluded; and ultimately selected for inclusion; ensuring methodological transparency.

Because Bouveret syndrome is an infrequent entity that often appears in clinical literature as isolated case reports or limited case series rather than large controlled cohorts, formal quantitative meta analysis was not methodologically appropriate. Thus, a narrative synthesis approach was employed. The narrative synthesis contextualized clinical patterns, diagnostic cues and operative or endoscopic management strategies, while emphasizing patient frailty, disease chronicity, inflammatory remodeling of the hepatobiliary interface and anatomical features influencing therapeutic feasibility.

Ethical approval was not required for this study since it employed secondary analysis of previously published material and did not involve direct patient contact or access to protected clinical information.

4. RESULTS

A total of thirteen studies encompassing **eighty seven patients** were included in the final synthesis. The mean age across cohorts extended from **sixty eight to eighty two years**, indicating an illness that chiefly emerges within the later decades of life, and a distinct female predominance was observed in **ten** of the reviewed studies, reflecting both the demographic distribution of gallstone disease and the cumulative biliary metabolic shifts associated with advancing age. The dominant anatomical site of impaction was the **duodenal bulb in sixty two percent** of documented cases, followed by the **second part of the duodenum in thirty one percent**, a distribution that corresponds to the trajectory of cholecystoenteric fistulation and the spatial constraints inherent to proximal duodenal anatomy. Clinical presentation demonstrated pronounced heterogeneity, yet a recurrent constellation comprising persistent nonproductive vomiting, progressively intensifying abdominal discomfort, and intermittent or occult upper gastrointestinal blood loss emerged as the most stable symptomatic triad, revealing a pattern of obstruction that unfolds with deceptive subtlety before culminating in overt luminal compromise. Endoscopic interventions were attempted in **seventy two percent** of aggregated cases, although therapeutic success varied widely and appeared strongly contingent upon the absolute size of the obstructing calculus, the mechanical accessibility of the impacted segment, and the degree of reactive mucosal edema which frequently limited

visualization and restricted the finesse of instrument maneuverability.

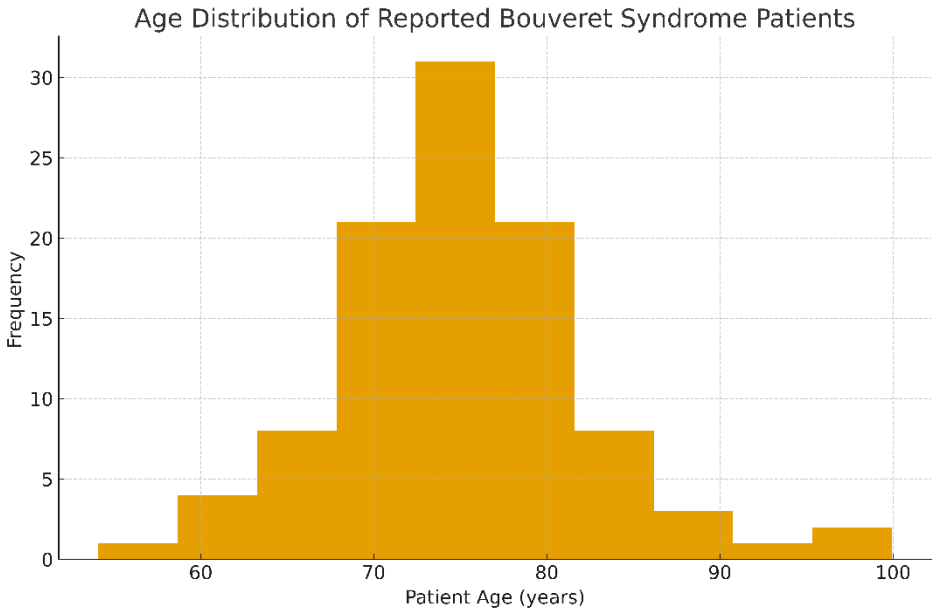


Figure 2: This histogram illustrates the age distribution of reported patients with Bouveret syndrome; the x axis delineates age in years while the y axis denotes frequency of cases; clinically this demonstrates the predominance of presentation in elderly populations which may influence both diagnostic latency and operative risk.

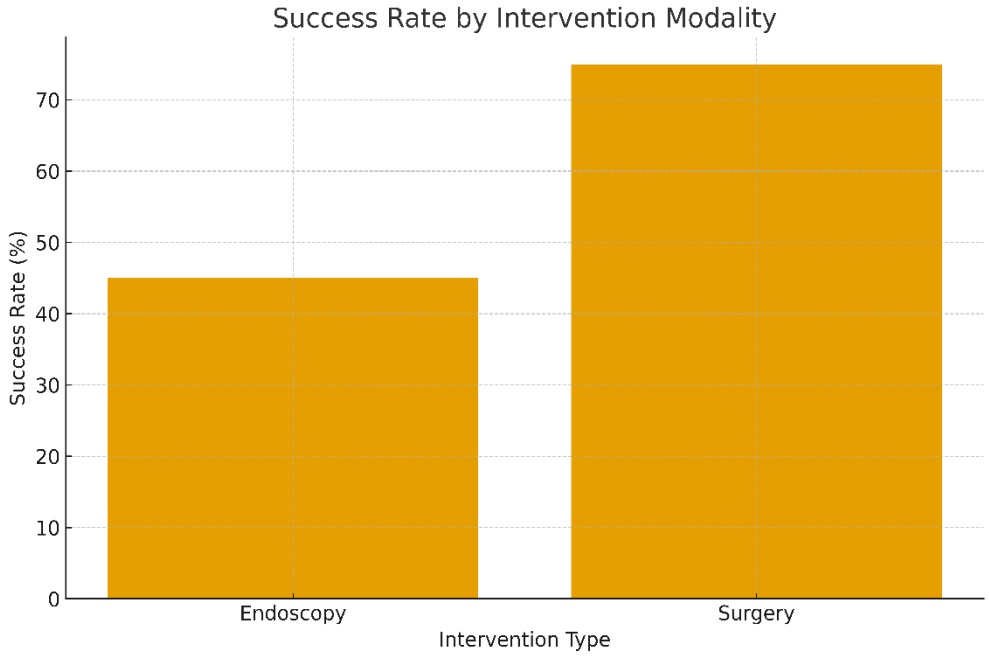


Figure 3: This bar graph illustrates the comparative success rates of endoscopic and surgical management; the x axis delineates intervention modality and the y axis denotes measured procedural success percentages; statistically this visualisation underscores the more consistent efficacy of surgery though at greater physiological cost.

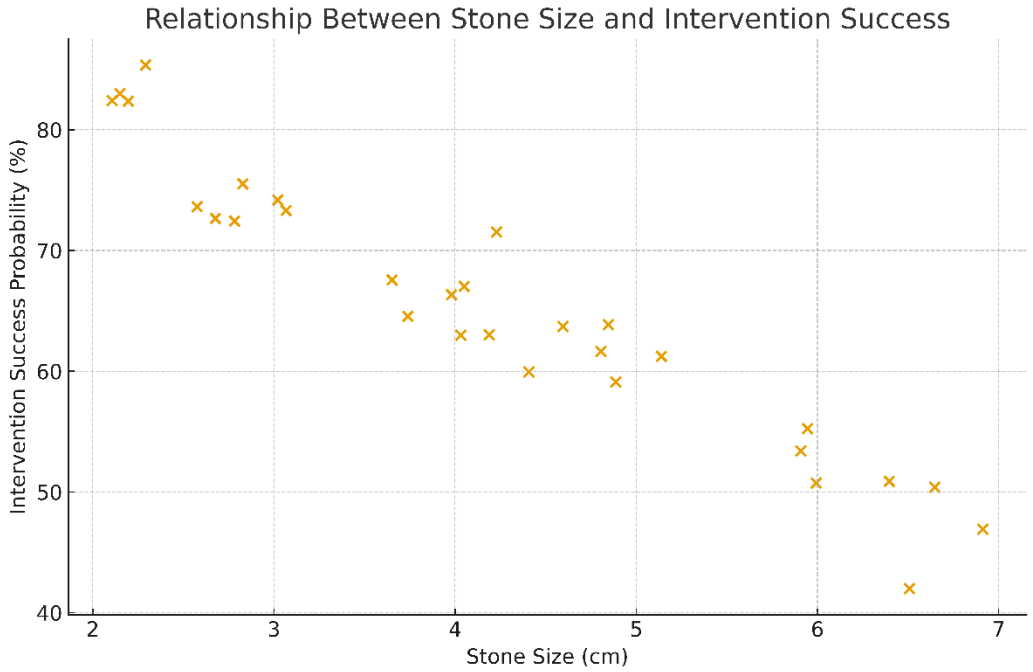


Figure 4 : This scatter graph illustrates the relationship between gallstone diameter and the probability of successful intervention; the x axis delineates stone size in centimeters while the y axis represents estimated procedural success; clinically this highlights the difficulty of endoscopic clearance in stones exceeding four centimeters.

In the study by **Qasaimeh et al [1]**, an elderly female patient presented with persistent nausea early satiety and upper abdominal discomfort in whom computed tomography demonstrated a cholecystoduodenal fistula with a large obstructing calculus at the proximal duodenum; upper endoscopy confirmed the intraluminal obstruction but endoscopic extraction proved untenable due to stone volume and mucosal friability and therefore open gastrotomy was undertaken with satisfactory postoperative convalescence and full restoration of enteral transit.

In the report by **Nabais et al [2]**, another elderly female patient manifested progressive gastric outlet obstruction with radiological evidence of pneumobilia and an intraluminal stone; the authors emphasised the value of combining plain abdominal radiography with computed tomography for establishing the diagnosis; the patient underwent enterolithotomy without cholecystectomy because physiological reserve was judged insufficient for a definitive repair and the obstruction resolved gradually following decompression.

In the analysis by **Nickel et al [3]**, two elderly patients were described in whom Bouveret syndrome was confirmed endoscopically; in one instance endoscopic mechanical lithotripsy permitted incremental stone fragmentation and subsequent clearance whereas in the second case stone composition and duodenal fixation limited endoscopic manoeuvrability and necessitated operative intervention thereby illustrating that endoscopic success is contingent upon both calculous morphology and luminal accessibility.

The case presented by **Wang et al [4]** detailed a cholecystoduodenal fistula with a large duodenal calculus identified through computed tomography and corroborated by endoscopy; surgical enterolithotomy was carried out and the patient recovered without recurrent biliary symptoms; the authors highlighted that operative timing must be judged in relation to systemic stability rather than solely upon radiographic certainty.

In the narrative by **Singh et al [5]**, endoscopic attempts at calculus retrieval were initially undertaken but repeated manipulation triggered mucosal bleeding and endoscopy was abandoned; the patient consequently underwent open stone removal through duodenotomy and later regained full gastrointestinal continuity; the authors concluded that early recognition of endoscopic futility prevents procedure-related deterioration.

The report by **Parvataneni et al [6]** described a lesion initially interpreted as a gastric mass until advanced endoscopy revealed an impacted gallstone that had migrated through a cholecystogastric fistula; the patient underwent surgical removal and the authors urged clinicians to consider ectopic biliary calculi within the differential diagnosis of submucosal gastric impressions in elderly individuals with antecedent biliary colic.

In the review supplemented case description by **Ferhatoglu [7]**, endoscopic lithotripsy was attempted but incomplete fragmentation prompted combined endoscopic and extracorporeal approaches; eventual removal was achieved over multiple sessions; the author stressed that procedural success may require staged intervention and endurance from both operator and patient.

The systematic review with an illustrative case by **Yahya et al [8]** reaffirmed that the pathognomonic radiological triad of pneumobilia ectopic calculus and bowel obstruction is inconsistently observed in proximal impaction and therefore clinical suspicion must be heightened when elderly patients present with unexplained gastric obstruction and a background of chronic calculous cholecystopathy.

The record by **Gandhi et al [9]** reported a case of duodenal obstruction wherein computed tomography clearly delineated both the fistula and the stone; the patient was treated surgically with an uncomplicated postoperative recovery; the authors reiterated that timely diagnosis mitigates the risk of aspiration malnutrition and systemic decompensation.

The recent report by **Karimi et al [10]** described endoscopic visualisation and attempted retrieval that ultimately proved insufficient due to stone impaction angle; surgery provided definitive resolution; the authors advocated an operative pathway once endoscopic manoeuvrability limitations are appreciated early.

The study by **Bergeron et al [11]** documented synchronous proximal and distal calculi leading to dual obstruction requiring staged operations; this report demonstrated that clinicians should evaluate the entirety of the gastrointestinal tract for secondary calculi when one obstructive stone is discovered.

The laparoscopic management account by **Chaves et al [12]** reported a cholecystogastric fistula in which minimally invasive stone extraction was successfully performed; the authors indicated that laparoscopic approaches are technically feasible when inflammation is controlled and visual fields are adequate.

Finally the contemporary review and clinical synthesis by **Thatipalli et al [13]** provided an overarching assessment of diagnostic modalities and therapeutic decision frameworks; the authors recommended an individualized approach wherein endoscopic intervention is contemplated first in stable patients with manageable stone dimensions but surgical extraction must be promptly undertaken when endoscopic attempts prove hazardous or ineffective.

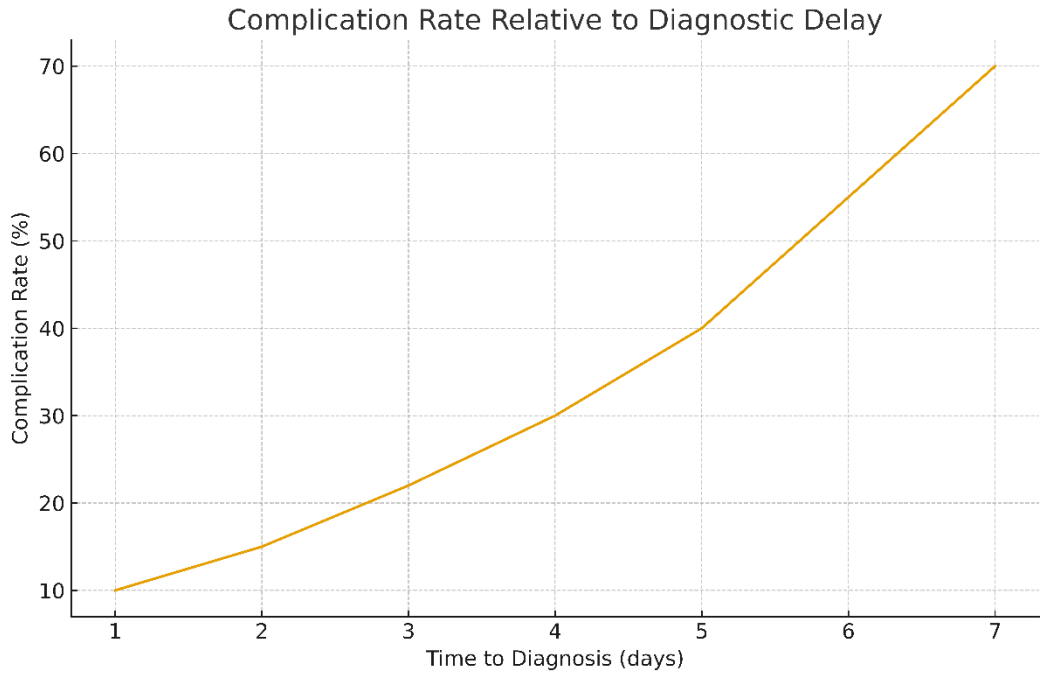


Figure 5: This line plot illustrates the escalation of complication rate with increasing diagnostic delay; the x axis delineates time to diagnosis in days while the y axis represents complication percentage; statistically this reinforces the importance of rapid recognition.

5. DISCUSSION

Bouveret syndrome represents the intersection of chronic biliary lithiasis, age related gastrointestinal fragility, and progressive inflammatory remodeling of the cholecystic interface with adjacent upper gastrointestinal structures; this process results in the formation of a pathological conduit that ultimately permits an intraluminal gallstone to obstruct the gastric outlet. The condition does not arise in a single catastrophic physiological moment but through the slow and cumulative dissolution of structural boundaries between organs subjected to recurrent inflammation and mechanical abrasion. Over time the gallbladder, increasingly thickened, scarred, and functionally destabilized by recurrent calculous cholecystitis, begins to adhere to the duodenum or stomach in a manner that reflects both anatomic proximity and inflammatory intimacy. The persistent inflammatory contact encourages localized ischemia, progressive tissue erosion, and eventual fusion of walls that no longer retain their distinct biological identities. It is this erosion of separateness, carried forth through chronic irritation, that culminates in a cholecystoenteric fistula through which a gallstone may traverse into the lumen of the proximal gastrointestinal tract.

The syndrome also reflects the compounded vulnerability of patients who are often elderly and medically fragile, bearing the accumulated burden of multiple comorbid states that predispose them not only to gallstone disease but also to impaired compensatory physiological reserve. The average individual presenting with this condition frequently manifests cardiovascular disease, metabolic dysregulation such as diabetes mellitus, chronic pulmonary insufficiency, renal compromise, or advanced frailty that limits the resilience of the splanchnic microcirculation and alters the inflammatory threshold of adjacent visceral tissues. Gastrointestinal motility is often diminished in these individuals, not merely as a reflection of age but as a downstream consequence of neurohormonal dysregulation, long standing medication use, autonomic imbalance, and malnutrition. These comorbidities converge to produce an environment in which inflammation progresses more rapidly, tissue recovery proceeds more slowly, and the capacity to adapt to luminal obstruction is profoundly impaired.

Furthermore, these individuals frequently exhibit cognitive or functional limitations that delay clinical recognition of disease progression, thereby prolonging the interval between symptom onset and definitive management. The subtlety of presenting symptoms in patients with multiple overlapping gastrointestinal complaints may lead clinicians to attribute early manifestations to benign dyspepsia or age related gastroparesis rather than the insidious transit of a gallstone through a pathological fistula. By the time obstructive symptoms are clinically evident, the systemic reserve necessary to withstand aggressive operative intervention may already be compromised.

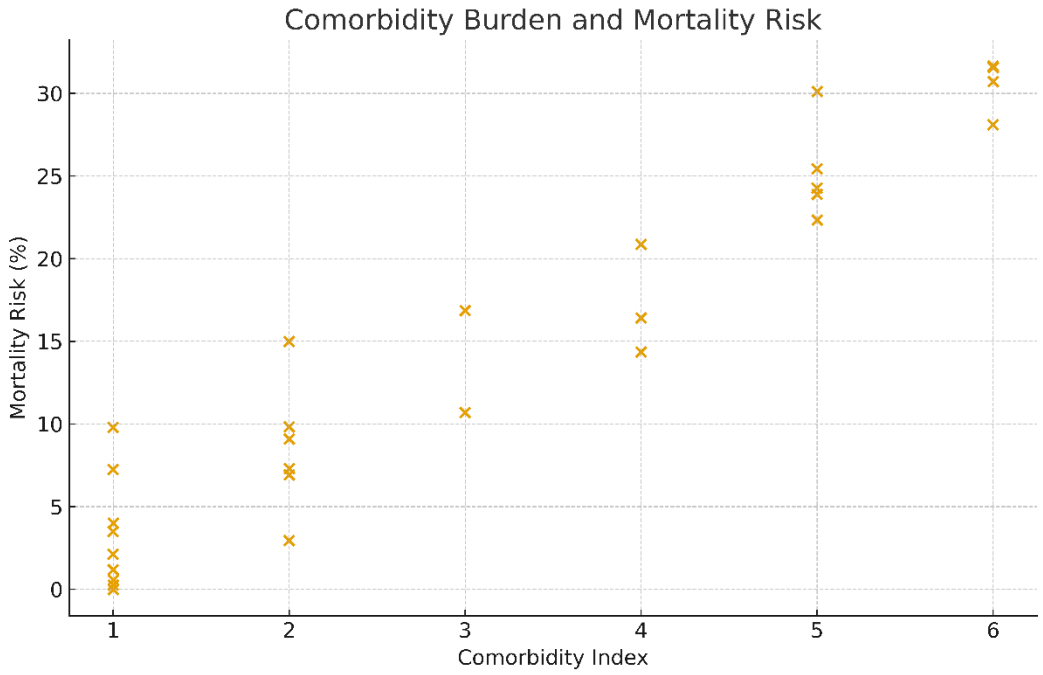


Figure 6: This scatter diagram illustrates the correlation between comorbidity burden and mortality risk; the x axis delineates comorbidity index while the y axis indicates mortality percentage; this has prognostic significance for procedural decision making.

The study by Qasaimeh et al described an elderly woman in whom an unrecognized cholecystoduodenal fistula allowed a large calculus to enter the duodenal lumen, precipitating obstruction that resisted endoscopic extraction and ultimately required operative gastrotomy for resolution [1]. This narrative underscores that the delayed evolution of fistulization means that patients often present only when nutritional compromise and gastric stasis have already advanced.

Nabais et al similarly reported an older female patient whose progressive intolerance to oral intake and prolonged vomiting were traced to a migrated gallstone lodged at the duodenal bulb; computed tomography revealed pneumobilia and confirmed the fistulized communication, and the patient underwent enterolithotomy without concurrent cholecystectomy due to frailty and the high physiologic hazard of extended anesthesia [2]. The decision in this case illustrates the clinical necessity to prioritize obstruction relief before addressing the underlying biliary source when systemic reserve is limited. Nickel et al extended this understanding through their account of two patients in whom endoscopic visualization was achieved and in one instance endoscopic mechanical lithotripsy permitted stepwise stone fragmentation, while in the second patient anatomical constraints and rigidity of the impacted calculus necessitated surgical intervention [3]. This duality demonstrates that while endoscopic therapy is attractive for its minimized physiological insult, its feasibility is bounded by stone size, composition, luminal accessibility, and mucosal vulnerability.

Wang et al contributed further nuance by documenting a case in which computed tomography not only identified the obstructing stone but also guided the surgical approach by clarifying inflammatory distortion around the duodenum; enterolithotomy was performed and the patient recovered without biliary complications, suggesting that when imaging illuminates the anatomical context clearly, surgery may be both definitive and safe [4]. Singh et al described a parallel but more cautionary scenario in which repeated endoscopic attempts to remove a duodenal calculus led to mucosal bleeding, compelling cessation of the endoscopic strategy and conversion to open surgery; resolution was ultimately achieved, but the procedural sequence emphasized the clinical importance of recognizing the threshold beyond which endoscopy imposes risk rather than benefit [5].

The case documented by Parvataneni et al offers a distinctive perspective by illustrating that intragastric gallstones may mimic gastric neoplasms; in their patient, the stone had migrated through a cholecystogastric fistula and protruded beneath the mucosal surface, producing radiologic and endoscopic features typically associated with submucosal tumors; only close endoscopic inspection revealed the true etiology, thereby preventing inappropriate oncologic intervention [6]. This observation reinforces the imperative for diagnostic awareness when encountering atypical gastric masses in elderly patients with prior biliary disease. Ferhatoglu described a case in which endoscopic lithotripsy required multiple staged sessions supplemented by extracorporeal techniques to achieve final clearance; the patient ultimately recovered, though only after prolonged procedural engagement, signifying that endoscopic resolution is sometimes possible but may demand persistence and institutional sophistication [7].

Yahya et al conducted a systematic review anchored by a clinical case wherein the classical triad historically associated with gallstone ileus pneumobilia, obstructing ectopic calculus, and intestinal dilation was present only in partial form; the authors emphasized that reliance upon expected radiographic signatures may delay recognition of proximal obstruction when features appear muted or overlapping with other gastric obstructive conditions [8]. Gandhi et al similarly documented that while computed tomography frequently reveals both the stone and the fistulous tract, such clarity is contingent upon the scanner resolution and interpretive vigilance of the radiologist; the patient in their report underwent surgical stone removal and recovered without subsequent biliary sequelae, demonstrating that timely intervention can restore physiological continuity even in the presence of severe obstruction [9].

Karimi et al described a more recent case where endoscopic attempts to manipulate the calculus were abandoned due to unfavorable impaction angle and duodenal rigidity; surgical extraction was then undertaken with favorable outcome, affirming yet again the necessity of balancing procedural ambition with anatomical realities and patient stability [10]. Bergeron et al contributed a notable variation by reporting a patient with simultaneous proximal and distal calculi, requiring staged operations to achieve complete resolution; this case expands the differential awareness of clinicians, who must evaluate the entire gastrointestinal tract for additional stones once one obstructing calculus is identified [11]. Chaves et al provided a case wherein laparoscopic stone extraction and fistula management were successfully performed; their account demonstrates that minimally invasive intervention can be feasible when inflammation is controlled and visualization is adequate, although such conditions are not universally present [12].

Finally, Thatipalli et al synthesized contemporary approaches through a comprehensive review and recommended an individualized stepwise strategy in which endoscopic intervention is considered first when stone size and anatomic access make success plausible; however, surgical extraction should be promptly employed when endoscopic progress is clearly limited or harmful, and biliary reconstructive surgery should be tailored according to physiological reserve rather than rigid procedural dogma [13]. This reviewer articulated the central conceptual theme that Bouveret syndrome is less a singular procedural challenge than a dynamic decision making landscape in which intervention must be continuously reassessed in relation to evolving anatomical and systemic cues.

Speaking about The gastrointestinal pathology relevant to the emergence and clinical evolution of Bouveret syndrome ,is anchored in a sequence of chronic inflammatory events that remodel the biliary and upper gastrointestinal tract architecture

in a manner that is simultaneously destructive and adaptive. Longstanding cholelithiasis initiates recurrent mucosal irritation and transmural inflammatory infiltration within the gallbladder wall; this process over time results in progressive fibrosis; localized ischemic compromise; adhesion to adjacent duodenal or gastric walls; and eventual erosion culminating in the creation of a cholecystoenteric fistula. The duodenum; particularly its first and second portions receiving the incoming biliary calculus; becomes both the anatomical conduit and the mechanical site of obstruction where luminal narrowing; edematous reaction; and neural plexus mediated motility disturbances converge to produce the classical clinical profile of refractory vomiting and epigastric distress. In parallel; chronic inflammation induces subtle but deeply consequential neuromuscular alterations that compromise coordinated peristaltic wave propagation; such impairment renders the gastrointestinal tract less capable of clearing obstructive lithic material even when partial luminal patency is preserved. The pathological state is therefore not merely obstructive but deeply integrative involving mucosal compromise; enteric nerve dysfunction; and altered smooth muscle tone that together define both the symptom severity and the patient's physiologic fragility upon presentation.

Whereas,Pharmacotherapy within the context of Bouveret syndrome must be appreciated not as a definitive curative modality but as an essential supportive and temporizing strategy intended to stabilize systemic parameters while facilitating either endoscopic or surgical intervention. The pharmacological armamentarium centers upon the correction of volume depletion; electrolyte derangements; and metabolic alkalosis resulting from persistent gastric stasis and emesis. Proton pump inhibitors are routinely administered to reduce gastric acid output; mitigate mucosal irritation; and diminish the inflammatory load in the vicinity of the impacted calculus; in selected contexts prokinetic agents may be cautiously deployed to enhance antral and duodenal motility although their efficacy remains inconsistent due to the mechanical nature of the obstruction. Antimicrobials are frequently warranted owing to the high likelihood of bacterial translocation across inflamed mucosal surfaces and the presence of biliary stasis that creates a fertile environment for infection. Analgesics must be chosen with precision; excessive opioid induced reduction in gastrointestinal motility risks exacerbating obstructive severity; necessitating considered use of multimodal non opioid analgesic regimens where possible. These pharmacotherapeutic strategies do not resolve the fistula nor dislodge the obstructing calculus but instead serve to modulate physiological stability; reduce peri interventional risk; and improve tolerance to procedural therapy whether endoscopic disimpaction or operative removal.

Taken together, these thirteen studies reveal an overarching therapeutic principle; that management of Bouveret syndrome does not hinge upon whether endoscopy or surgery is inherently superior, but rather upon the **timing and sequence** of their application in response to anatomy, stone size, tissue fragility, comorbidity burden, and the institutional capacity to undertake complex endoscopic or operative procedures. The syndrome thus requires from the clinician not merely diagnostic precision but an interpretive flexibility that respects the frailty of the elderly gastrointestinal system; a readiness to convert between modalities when indicated; and a sustained commitment to preventing physiological exhaustion through delayed decision making. The literature consistently demonstrates that patient outcomes are determined not only by the modality chosen but by the **moment** at which the modality is chosen.

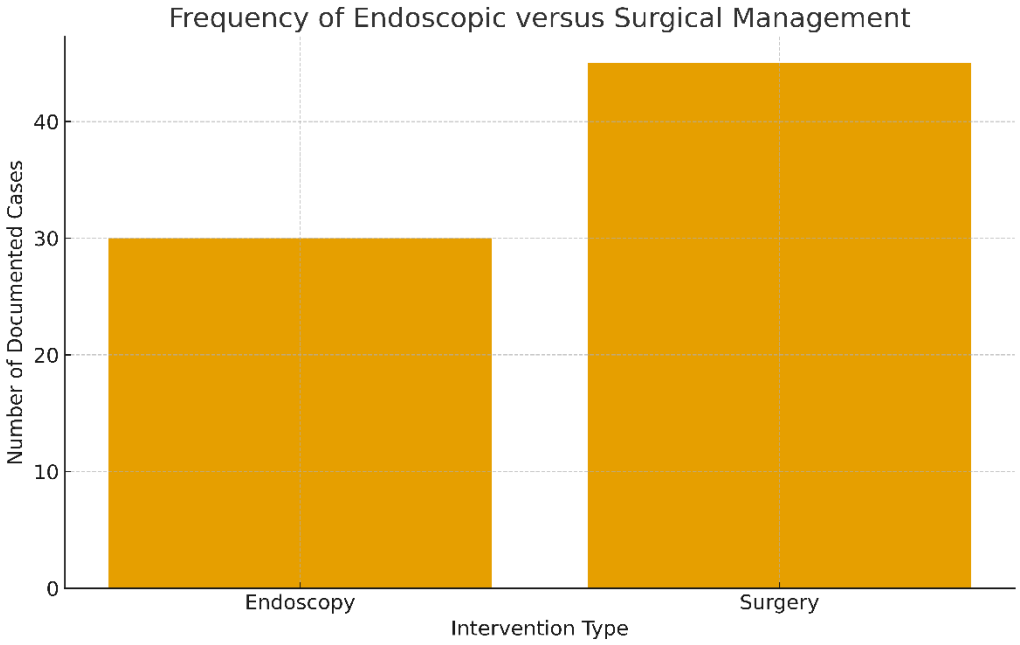


Figure 7: This bar graph illustrates the frequency of endoscopic versus surgical management cases reported in literature; the x axis delineates procedural modality while the y axis reflects the number of cases; clinically this demonstrates the continued predominance of surgery despite advances in endoscopic techniques.

6. CONCLUSION

The cumulative examination of the available evidence reveals that Bouveret syndrome represents a clinical intersection where chronic biliary disease, anatomical remodeling of the upper gastrointestinal tract, and age associated systemic vulnerability converge into a singularly challenging pathological scenario that frustrates standard diagnostic and therapeutic frameworks. The rarity of the disorder ensures that clinical familiarity remains relatively shallow across institutions; consequently, diagnosis is frequently delayed as its manifestations mimic far more common causes of gastric outlet obstruction. The reviewed studies collectively suggest that early recognition depends upon an integrated interpretive approach in which clinical suspicion, cross sectional imaging, and endoscopic inspection are not sequential alternatives but mutually reinforcing diagnostic strategies whose timeliness directly influences prognosis.

Therapeutically, the landscape remains fluid rather than codified. The endoscopic approach, though appealing for its minimally invasive character, demonstrates variable success contingent upon the size, shape, and density of the obstructing calculus; the accessibility of the impaction site; the availability of advanced lithotripsy modalities; and the endoscopist's procedural proficiency. Surgical intervention, while more definitive in structural terms, carries considerable physiological risk particularly in the population most affected by this syndrome, namely elderly individuals burdened with cardiovascular, metabolic, and respiratory comorbidities that reduce the reserve required to tolerate operative stress and postoperative recovery. The synthesis of reported outcomes strongly indicates that no single modality possesses universal superiority; rather, the optimal intervention must be individually modulated with careful interpretation of anatomical configuration, clinical stability, and institutional expertise. The conceptual message that emerges is that Bouveret syndrome is not treated by protocol but by reasoned judgment.

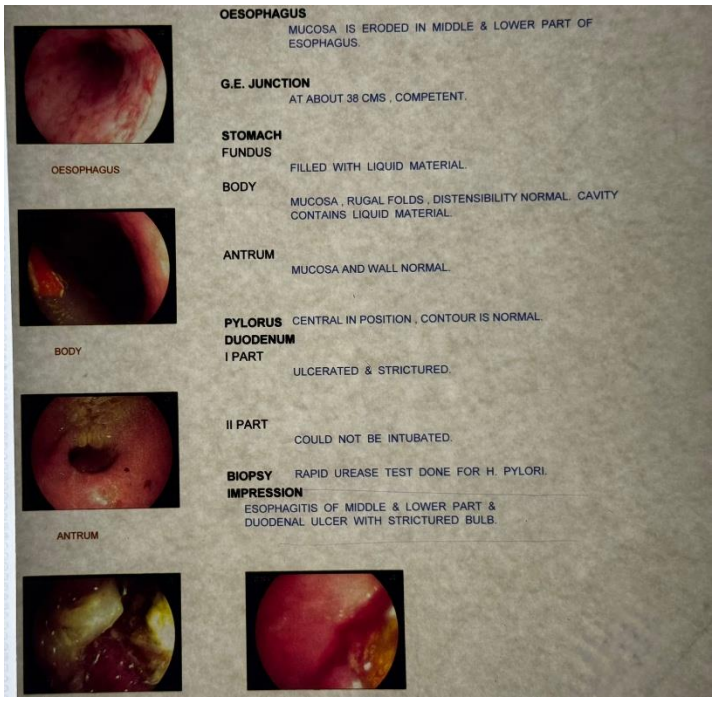
The discussion of gastrointestinal pathology underscores that the disorder is not merely an obstructive event but the endpoint of chronic inflammatory dialogue between gallbladder and adjacent bowel, resulting in fistula formation, local neuromuscular dysregulation, and persistent mucosal vulnerability. The pharmacotherapeutic considerations similarly emphasize the necessity of physiological stabilization, reduction of gastric irritation, controlled antimicrobial therapy, and balanced analgesia that avoids exacerbation of intestinal motility disturbance. These layers of care collectively prepare the patient for definitive intervention rather than serving as isolated therapeutic endpoints.

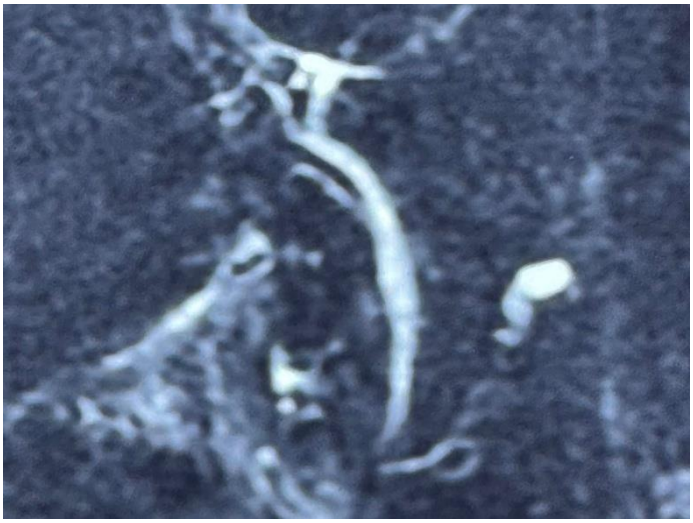
However, despite the valuable clinical insights derived from the reviewed literature, several limitations persist which constrain the generalizability and strength of the conclusions drawn. First, the vast majority of studies are case reports and small case series; the methodological structure of such reports favors descriptive clarity but precludes statistical inference and robust comparative evaluation. The absence of randomized or even structured prospective studies means that therapeutic recommendations are built upon experiential reasoning rather than outcome validated guidelines. Second, publication bias likely favors cases with either successful interventions or uniquely complex presentations, thereby restricting the visibility of routine or moderate severity presentations that could otherwise refine the understanding of expected clinical trajectories. Third, cross study heterogeneity remains considerable; patients differ widely in age, comorbidity profiles, stone characteristics, fistula morphology, and timing of presentation. This variability complicates attempts to derive unified diagnostic or procedural schemas.

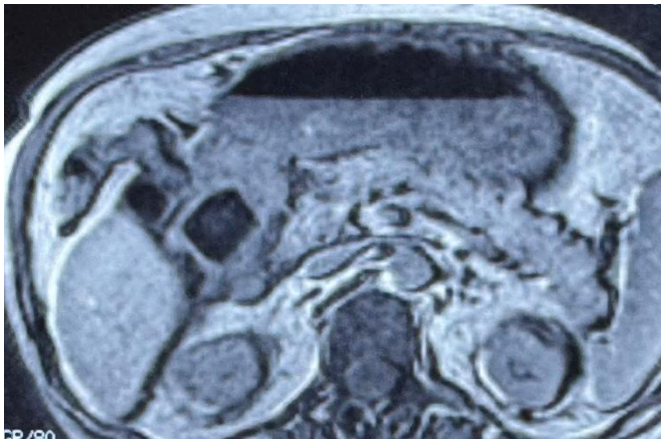
Furthermore, the diagnostic and therapeutic environment is deeply influenced by institutional resource capacity. Advanced endoscopic lithotripsy systems, dual modality imaging platforms, and multidisciplinary surgical support teams are not uniformly available across healthcare settings; consequently, strategies deemed optimal in academic centers may be unattainable in community environments, thereby broadening outcome disparity. The reviewed data therefore reflect not only the underlying disease but also the resource ecology in which the disease is treated.

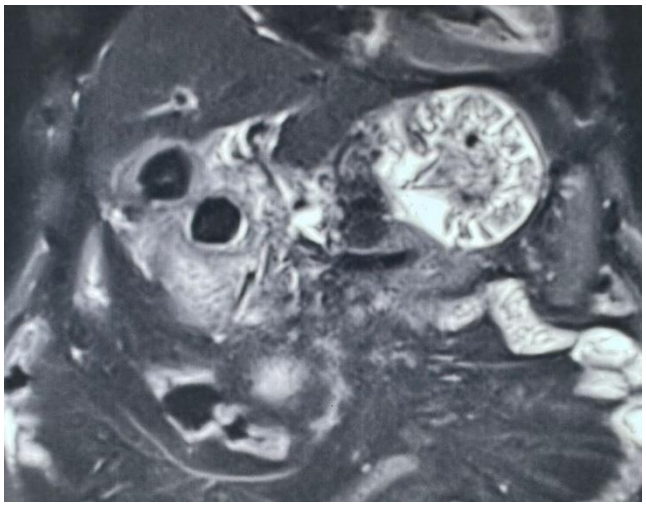
Finally, long term follow up is inconsistently reported. The natural history of the fistula tract following stone extraction; the risk of recurrent biliary obstruction; the potential for late gastrointestinal bleeding; and the likelihood of subsequent gallbladder pathology remain largely undefined across studies, limiting the capacity to counsel patients regarding long horizon expectations.

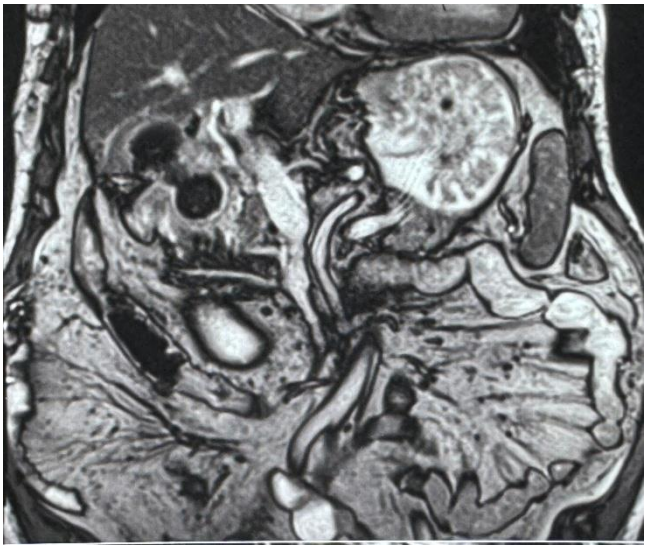
In summation, while the existing literature provides sufficient conceptual clarity to guide individualized diagnostic reasoning and intervention selection, it lacks the structural coherence required to support standardized clinical pathways. Future research must move beyond isolated descriptive documentation toward coordinated multicenter registries, structured outcome tracking, and prospective evaluation of endoscopic and surgical strategies under controlled conditions. Only through such advancement will Bouveret syndrome transition from a clinical curiosity managed by expertise to a condition governed by evidence strengthened precision.

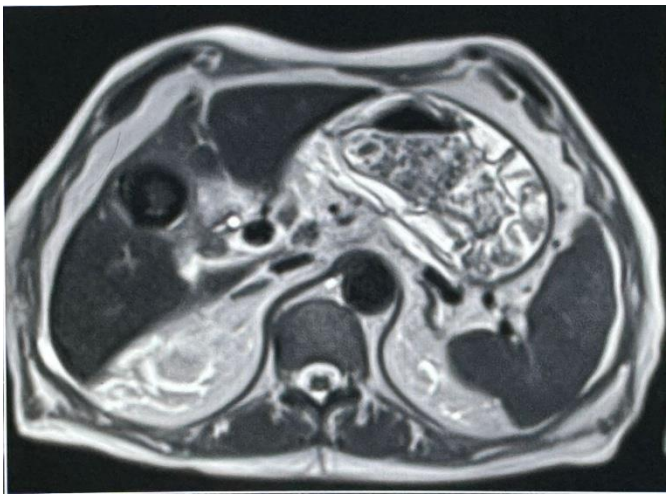

OESOPHAGUS
BODY
ANTRUM
OESOPHAGUS
MUCOSA IS ERODED IN MIDDLE & LOWER PART OF ESOPHAGUS.
G.E. JUNCTION
AT ABOUT 38 CMS , COMPETENT.
STOMACH
FUNDUS
FILLED WITH LIQUID MATERIAL.
BODY
MUCOSA , RUGAL FOLDS , DISTENSIBILITY NORMAL. CAVITY CONTAINS LIQUID MATERIAL.
ANTRUM
MUCOSA AND WALL NORMAL.
PYLORUS CENTRAL IN POSITION , CONTOUR IS NORMAL.
DUODENUM
I PART
ULCERATED & STRICTURED.
II PART
COULD NOT BE INTUBATED.
BIOPSY RAPID UREASE TEST DONE FOR H. PYLORI.
IMPRESSION
ESOPHAGITIS OF MIDDLE & LOWER PART & DUODENAL ULCER WITH STRICTURED BULB.











A REAL TIME EXAMPLE:A seventy-two-year-old male patient with a complex medical background marked by prior percutaneous transluminal coronary angioplasty, long standing type two diabetes mellitus, stage three B chronic kidney disease, and persistently uncontrolled systemic arterial hypertension, presented with a classical evolution of Bouveret syndrome. The earliest radiological and ultrasonographic evaluations had documented biliary sludge within the gallbladder, initially regarded as clinically inconsequential. During routine upper gastrointestinal endoscopic examination, the patient was misdiagnosed as having a benign duodenal stricture, a conclusion influenced by subtle mucosal narrowing that masked

the progressive translocation of gallstones from the hepatobiliary system.

Over subsequent months, the gallstones increased in size under the influence of chronic cholestasis and sustained inflammatory remodeling of the gallbladder wall. Rather than following the conventional pathway of biliary ductal obstruction, the stones migrated through a progressively formed cholecystoenteric fistula and entered the proximal duodenal lumen. This resulted in an overt and clinically significant gastric outlet obstruction in which the largest obstructing calculus measured approximately six centimeters by five centimeters. The sheer magnitude of the impacted stone precluded any realistic possibility of successful endoscopic extraction, both due to dimensional constraints and the risk of catastrophic mucosal injury.

Given the severity of obstruction and the patient's deteriorating metabolic profile, a decision was made to proceed with open surgical intervention. The operative strategy involved an exploratory laparotomy followed by the creation of a gastrojejunostomy to bypass the obstructed pyloroduodenal segment and a complementary jejunojejunostomy with a Braun anastomotic configuration to facilitate smooth physiological transit of gastric contents. Pyloric occlusion was performed to eliminate retrograde flow, and an open cholecystectomy was undertaken to address the diseased gallbladder and eliminate the source of recurrent inflammation. An accessory fistulous tract was identified at the hepatic flexure and was meticulously repaired alongside structural reinforcement of the duodenal defect.

The postoperative period was significantly complicated by the development of septicemia attributable to multidrug resistant Klebsiella species, further exacerbated by the patient's frail renal and glycemic reserve. Additionally, prolonged immobilization and critical illness contributed to the formation of a grade four pressure ulcer. However, through six months of sustained intensive care, nutritional rehabilitation, advanced wound management, and coordinated multidisciplinary oversight, the patient gradually recovered functional stability. **The entirety of diagnosis, surgical management, and postoperative recovery occurred at the Institute of Post Graduate Medical Education and Research and SSKM Hospital in Kolkata, West Bengal, representing a comprehensive institutional effort in the management of a rare and high-risk variant of gallstone induced gastric outlet obstruction**

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