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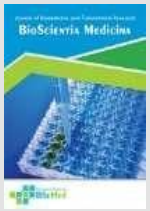

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The Anicteric Giant: A Rare Case of Giant Choledocholithiasis and Multiple Cholelithiasis Presenting with Paradoxically Normal Bilirubin Profiles

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A B S T R A C T

Background: Giant choledocholithiasis, defined as common bile duct (CBD) calculi exceeding 15 mm, typically presents with Charcot's triad or marked biochemical cholestasis. The phenomenon of silent or anicteric giant stones remains a dangerous diagnostic blind spot. We present a rare case of a massive biliary stone burden presenting with paradoxically normal bilirubin and liver enzyme profiles, challenging standard screening algorithms. **Case presentation:** A 61-year-old female presented with a 12-month history of intermittent epigastric pain and nausea, initially misdiagnosed as gastritis. Despite the chronicity, she denied jaundice or fever. Biochemical analysis revealed a Total Bilirubin of 0.39 mg/dL (Reference: 0.1–1.2 mg/dL) and normal gamma-glutamyl transferase (GGT) levels, indicating an absence of biochemical obstruction. Magnetic resonance cholangiopancreatography (MRCP) identified multiple cholelithiasis and a solitary giant CBD stone. Intraoperative exploration confirmed a CBD dilated to 22 mm containing a calculus measuring 28 mm × 22 mm. Due to the massive ductal dilation and risk of recurrent stasis, the patient underwent a retrograde cholecystectomy followed by biliary reconstruction via Roux-en-Y choledochojejunostomy. **Conclusion:** Giant choledocholithiasis can exist in a silent phase due to ductal compliance and the ball-valve mechanism, rendering bilirubin an unreliable screening tool. This case underscores the necessity of cross-sectional imaging in chronic abdominal pain, even when biochemical markers are normal. Roux-en-Y reconstruction remains the definitive management for giant stones in significantly dilated ducts to prevent recurrence and sump syndrome.

1. Introduction

Choledocholithiasis, defined as the presence of calculi within the common bile duct (CBD), represents one of the most significant and potentially hazardous complications of gallstone disease. It is a prevalent clinical entity, affecting approximately 10–20% of symptomatic patients with cholelithiasis.[1] Under normal physiological conditions, the biliary tree functions as a conduit, and the majority of stones that migrate from the gallbladder into the common bile duct are small, often measuring less than 5 mm. These smaller calculi typically pass spontaneously through the ampulla of Vater into the duodenum without necessitating intervention. However, a distinct and surgically complex subset of patients develops giant choledocholithiasis.[2]

The nomenclature surrounding giant stones has evolved. While historical literature and older surgical texts often cited varying thresholds—ranging from 20 mm to as large as 50 mm—to categorize a stone as giant, the contemporary clinical consensus has become more rigorous. Current guidelines, including those utilized for endoscopic decision-making and risk stratification, now define giant choledocholithiasis as

calculi measuring 15 mm or greater in transverse diameter.[3] This specific threshold of 15 mm is not arbitrary; it represents a critical anatomical tipping point where the diameter of the stone exceeds the maximal safe dilation diameter of the distal biliary sphincter. Consequently, these macro-calculi represent a distinct pathological entity associated with significantly higher rates of procedural failure for standard endoscopic retrograde cholangiopancreatography (ERCP) and a marked increase in morbidity resulting from complications such as suppurative cholangitis and biliary pancreatitis.[4]

For over a century, the clinical recognition of common bile duct obstruction has been anchored in the teachings of Jean-Martin Charcot.[5] The classic presentation, widely taught in medical curricula, is Charcot's triad: the simultaneous manifestation of cholestatic jaundice, biliary colic, and fever. This triad is a clinical reflection of acute pathophysiology: a stone impacts the distal duct, causing a rapid rise in intrabiliary pressure.[6] When this pressure exceeds the hepatic secretory pressure, the flow of bile arrests, leading to the regurgitation of conjugated bilirubin into the systemic circulation (jaundice) and creating a stagnant pool of bile liable to bacterial colonization (fever).

Because of this well-understood mechanism, modern diagnostic workflows have been designed to detect this specific sequence of events. Risk stratification algorithms, such as those rigorously proposed and updated by the American Society for Gastrointestinal Endoscopy (ASGE), heavily weight biochemical markers as the primary predictors of choledocholithiasis. In these algorithms, total bilirubin and liver enzymes (specifically Alkaline Phosphatase and GGT) serve as the gatekeepers for further imaging or intervention. Clinicians are trained to harbor a high index of suspicion for CBD stones only when total bilirubin levels exceed thresholds of 1.8 to 4.0 mg/dL. In this algorithmic worldview, the magnitude of the biochemical derangement is presumed to correlate linearly with the likelihood and severity of the obstruction.[7]

However, clinical practice frequently defies algorithmic rigidity. A paradox exists that poses a substantial diagnostic hazard to the uninitiated clinician: the anicteric giant stone. Emerging clinical evidence and case series suggest that the classic presentation is far from universal.[8] A significant minority of patients harboring choledocholithiasis do not manifest classic jaundice. Retrospective analyses indicate that up to 5-10% of patients with confirmed CBD stones may present with normal or near-normal bilirubin levels.

This dissociation between the physical burden of the stone and its biochemical expression creates a dangerous diagnostic blind spot. In these silent or anicteric cases, the absence of jaundice can act as a cognitive bias, leading primary care physicians and gastroenterologists to rule out biliary obstruction prematurely. Symptoms of vague epigastric pain or nausea are frequently misattributed to more common benign conditions such as functional dyspepsia, gastritis, or peptic ulcer disease. This misdiagnosis initiates a cascade of delay; while the patient is treated with acid-suppression therapy, the underlying pathology progresses. This diagnostic lag allows the stone to grow further, often leading to massive ductal dilation (megaduct) and potentially irreversible secondary biliary cirrhosis before the true etiology is identified.[9]

Once identified, the management of giant stones presents a formidable technical challenge. While ERCP is indisputably the gold standard for clearing stones smaller than 10 mm, stones larger than 15 mm introduce significant complexity. These macro-calculi often defy extraction via standard biliary sphincterotomy and balloon trawling, as the stone is simply too large to pass through the incised papilla. To manage these cases endoscopically, clinicians must employ advanced, resource-intensive, and technically demanding adjuncts. These include mechanical lithotripsy (crushing the stone with a basket), electrohydraulic lithotripsy (EHL) performed under direct visualization with single-operator

cholangioscopy (SOC), or laser fragmentation.

However, these advanced endoscopic modalities are not without risk or limitation. They carry higher rates of complications, such as basket impaction, and may not be available in all centers. Furthermore, they address the *stone* but not the duct. In cases where the bile duct has become irreversibly dilated and atonic due to chronic obstruction, clearing the stone endoscopically may leave the patient with a non-functioning sump duct that is prone to stasis and recurrent stone formation. In such scenarios, or when endoscopic attempts fail, surgical exploration becomes the definitive treatment. The choice of surgical reconstruction is equally nuanced and depends heavily on the diameter of the duct and the patient's physiological reserve. Options range from primary closure over a T-tube to biliary-enteric bypasses such as Choledochoduodenostomy (CDD) or Roux-en-Y choledochojejunostomy. Each carries specific risks regarding leakage, recurrence, and long-term reflux cholangitis.[10]

This study presents a detailed analysis of a rare case involving a 61-year-old female with giant choledocholithiasis (28 mm) and multiple cholelithiasis who maintained a completely normal bilirubin profile and normal liver enzymes. We aim to discuss the pathophysiology of this silent obstruction, specifically focusing on the biomechanical interaction between the ball-valve mechanism and biliary compliance (The Law of Laplace) that allows such massive pathology to evade biochemical detection. Furthermore, we aim to delineate the decision-making framework favoring Roux-en-Y reconstruction over other surgical options in the context of a megaduct. The novelty of this study lies in the granular analysis of the profound dissociation between extreme morphological pathology (a giant 28 mm stone within a 22 mm duct) and the complete absence of biochemical markers. This case provides a cautionary evidentiary basis for revising clinical suspicion thresholds, suggesting that in chronic presentations, normal labs should not preclude advanced imaging.

2. Case Presentation

In adherence to ethical standards for clinical research and publication, written informed consent was rigorously obtained from the patient regarding the documentation and dissemination of this case report, including all accompanying diagnostic imagery. The subject of this study, a 61-year-old female, presented to the surgical outpatient clinic at Dr. Moewardi Regional General Hospital with a chronic and diagnostic-challenging profile. Her primary clinical complaint was a recurrent, somewhat nebulous epigastric pain that had persisted for a duration of 12 months prior to her surgical consultation. The patient's symptom profile was characterized by pain described as oscillating between a dull, gnawing discomfort and acute colicky episodes. This discomfort was intermittent in nature, with the patient rating the severity as moderate—specifically falling between 4 and 6 on the Visual Analog Scale (VAS). Clinically significant was the trajectory of the pain; while centered in the epigastrium, it occasionally radiated to the right scapular region, a classic albeit often overlooked sign of biliary pathology.

The patient's 12-month clinical odyssey illustrates the diagnostic difficulties inherent in anicteric biliary disease (Figure 1). The initial phase (months 1-4) of the disease was insidious. The patient experienced intermittent epigastric discomfort, which she attributed to minor gastrointestinal upset, managing the symptoms independently with over-the-counter antacids. As symptoms failed to resolve, in month 5, the patient sought professional medical advice from a primary care physician. Due to the conspicuous absence of specific biliary signs—such as jaundice or palpable RUQ mass—the clinical picture mimicked functional gastric disorders. Consequently, a presumptive diagnosis of dyspepsia and gastritis was established, and a therapeutic trial of Proton Pump Inhibitors (PPIs) was prescribed. Despite adherence to the PPI regimen, the patient's symptoms proved refractory to acid-suppression therapy. This period (months 6-10) was marked by a silent progression; the patient remained afebrile and denied any history of

jaundice, dark urine (choluria), or pale stools (acholia), effectively masking the underlying biliary obstruction. The clinical turning point occurred in the eleventh month when the intensity of the pain escalated, necessitating a referral to the Hepatobiliary Surgery unit (months 11-12). This culminated in her admission in the twelfth month for comprehensive imaging and definitive surgical intervention.

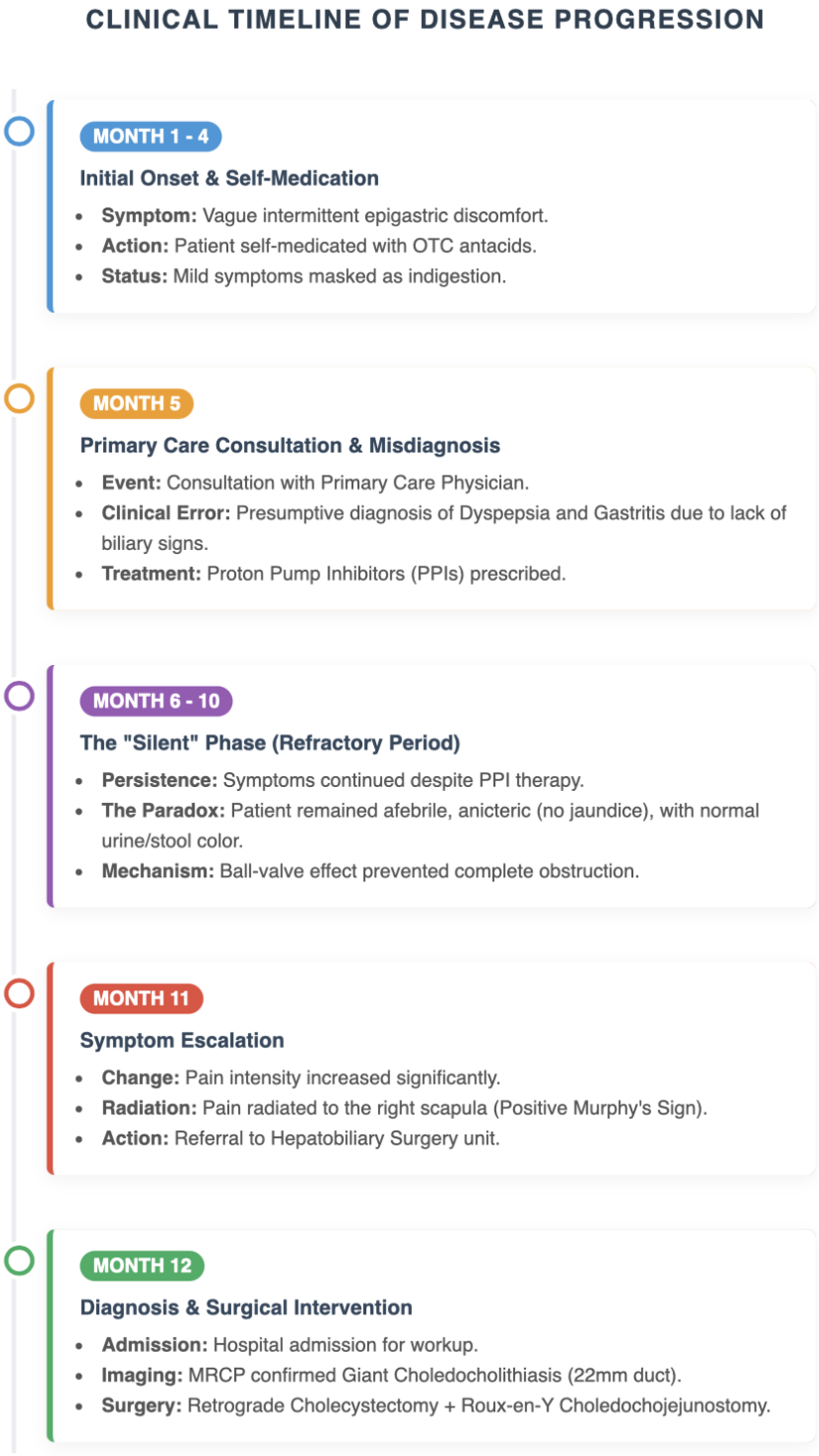

Figure 1. Clinical timeline of disease progression.

A thorough review of the patient's history was conducted to rule out confounding etiological factors. The patient reported no history of alcohol consumption or smoking, reducing the likelihood of alcoholic liver disease or malignancy associated with tobacco use. Furthermore, her surgical history was naive, with no previous abdominal operations that could suggest adhesive disease. Her medical background was remarkably free of common metabolic comorbidities; she explicitly denied any history of hypertension or diabetes mellitus, presenting as an otherwise healthy candidate for surgical reconstruction.

On physical examination, the patient was anicteric with clear sclerae. Her BMI was 26.5 kg/m². Vital signs were stable (BP 120/80 mmHg, HR 78 bpm, Temp 36.7°C). Abdominal examination revealed a soft, non-distended abdomen. However, deep palpation elicited mild tenderness in the right upper quadrant (RUQ) with a positive Murphy's sign. No palpable mass was identified in the RUQ, though the gallbladder was suspected to be distended based on percussion. Biochemical investigations were performed upon admission. Crucially, to verify the paradoxical findings, liver function tests were repeated 24 hours later, confirming the results. The data presented below represents the confirmed values.

TABLE 1. COMPREHENSIVE CLINICAL FINDINGS

Summary of Patient Status upon Admission

Parameter / Finding	Patient Value / Description	Reference / Interpretation
1. PATIENT PROFILE & HISTORY		
Demographics	61-Year-Old Female	Elderly
Chief Complaint	Intermittent Epigastric Pain (12 Months)	Chronic Presentation
Pain Characteristics	Dull to Colicky; Radiating to R Scapula	Typical Biliary Colic
Pain Severity	VAS Score 4-6 / 10	Moderate Intensity
2. VITAL SIGNS & ANTHROPOMETRY		
Blood Pressure	120/80 mmHg	Stable
Heart Rate	78 bpm	Normal
Temperature	36.7°C	Afebrile
BMI	26.5 kg/m²	Overweight
3. PHYSICAL EXAMINATION		
General Appearance	Clear Sclerae (Anicteric)	Paradoxical Finding
Abdominal Palpation	Soft, Non-distended; No palpable mass	No Peritonitis
Specific Signs	Positive Murphy's Sign; RUQ Tenderness	Suggests Cholecystitis
4. LABORATORY ANALYSIS (24HR CONFIRMATION)		
Total Bilirubin	0.39 mg/dL	Ref: 0.1 – 1.2 (Normal)
Direct Bilirubin	0.15 mg/dL	Ref: 0.0 – 0.3 (Normal)
Liver Enzymes (AST/ALT)	AST: 28 U/L I ALT: 32 U/L	Ref: 0 – 35 (Normal)
GGT	35 U/L	Ref: 8 – 61 (Normal)
Alkaline Phosphatase	110 U/L	Ref: 40 – 129 (High-Normal)
WBC Count	7,800 /µL	Ref: 4,500 – 11,000 (Normal)
5. IMAGING STUDIES (ULTRASOUND & MRCP)		
Gallbladder Findings	Distended; Multiple hyperechoic structures	Cholelithiasis
Common Bile Duct (CBD)	Gross dilation; Diameter: 22 mm	Megaduct
CBD Pathology	Large, ovoid, signal-void filling defect	Giant Choledocholithiasis
Intrahepatic Ducts	No Intrahepatic Biliary Dilation (IHBD)	Incomplete Obstruction

The diagnostic trajectory began with non-invasive imaging to corroborate the clinical suspicion of biliary pathology. The initial investigation utilized transabdominal ultrasonography, the first-line modality for biliary complaints. This study provided the first objective confirmation of pathology, revealing a distended gallbladder containing multiple hyperechoic structures that demonstrated the classic acoustic shadowing characteristic of cholelithiasis. While the ultrasound successfully identified the proximal common bile duct (CBD) and noted a significant dilation exceeding 10 mm, the exam was limited by a common artifact: bowel gas obscuration. This gas interference prevented visualization of the distal CBD, leaving the precise cause of the obstruction undefined.

To resolve this anatomical ambiguity, the patient underwent magnetic resonance cholangiopancreatography (MRCP), which served as the definitive diagnostic modality. The MRCP findings were dramatic, revealing gross morphological changes to the extrahepatic biliary tree. The common bile duct was measured at a maximum diameter of 22 mm, a finding indicative of a megaduct resulting from chronic, high-grade obstruction. Within this dilated channel, the scan identified a large, ovoid, signal-void filling defect in the mid-to-distal CBD, consistent with a giant calculus. Crucially, the imaging noted a distinct absence of intrahepatic biliary duct (IHBD) dilation. This specific finding was pivotal, as it suggested that the obstruction was not complete; rather, the ductal compliance and the stone's mobility likely allowed for intermittent biliary flow, explaining the patient's anicteric status despite the massive stone burden.

Following the anatomical mapping, a multidisciplinary team convened to determine the optimal therapeutic approach. The decision-making framework required balancing procedural invasiveness against the risk of recurrence and long-term morbidity. Endoscopic retrograde cholangiopancreatography (ERCP), often the standard of care for choledocholithiasis, was carefully considered but ultimately rejected for two primary reasons based on the specific pathology of this case: (1) Stone dimensions and extraction risk: The stone, estimated at greater than 20 mm on MRI, significantly exceeded the extraction capacity of a standard endoscopic sphincterotomy. Attempting to remove a stone of this magnitude endoscopically would likely necessitate advanced fragmentation techniques, such as mechanical lithotripsy. The team recognized that such maneuvers carry a heightened risk of complications, specifically basket impaction, where the retrieval device becomes trapped within the duct, necessitating emergency surgical salvage; (2) Ductal atony and recurrence: Perhaps more critical was the consideration of the ductal pathology itself. The CBD diameter of 22 mm indicated that the duct had become a megaduct—a structure that had lost its elasticity and tone. The team reasoned that even if the stone could be successfully cleared endoscopically, the duct would likely remain atonic and aperistaltic. This baggy, non-functional duct would serve as a reservoir for bile stasis, creating an environment perfect for the formation of primary CBD stones (recurrent choledocholithiasis).

Consequently, the team concluded that open surgical exploration with definitive biliary drainage was indicated. The choice of reconstruction was equally nuanced. The team selected a Roux-en-Y Choledochojejunostomy over the simpler Choledochoduodenostomy (CDD). While CDD is technically faster, performing a side-to-side anastomosis in a patient with a massive duct creates a blind pouch in the distal CBD. This anatomical configuration carries a high risk of sump syndrome, where food particles and debris accumulate in the distal pouch, leading to recurrent infection and cholangitis. The Roux-en-Y approach was chosen to eliminate this risk and prevent reflux.

The patient underwent a laparotomy under general anesthesia to execute this plan. Upon entering the abdominal cavity, the surgeons encountered a thickened, chronically inflamed gallbladder, confirming the chronicity of the disease. Tactile

exploration of the biliary tree revealed a CBD that was palpably dilated to approximately 2.5 cm and contained a large, mobile, hard mass, confirming the preoperative imaging. The procedure began with a cholecystectomy. Due to the dense chronic inflammation obscuring the critical anatomy of Calot's triangle, the surgeons employed a retrograde or fundus-down technique. This safety maneuver involves dissecting the gallbladder from the liver bed first, moving toward the cystic duct, thereby minimizing the risk of inadvertent injury to the biliary or vascular structures. Following the removal of the gallbladder, a longitudinal choledochotomy was performed on the supraduodenal portion of the CBD. Through this incision, the surgeons extracted a giant, solitary calculus. Morphometric analysis of the explanted specimen revealed impressive dimensions of 28 mm × 22 mm × 15 mm. Visually, the stone was dark-pigmented, contrasting with the multiple smaller mixed stones removed from the gallbladder, suggesting a stasis-driven etiology for the CBD stone.

The final phase was the biliary reconstruction. The distal CBD was ligated to prevent reflux into the blind end. A Roux limb of the jejunum, measuring 60 cm in length, was meticulously prepared. This specific length is critical to creating a sufficient physical distance between the bowel continuity and the biliary tree to prevent entero-biliary reflux. An end-to-side choledochojejunostomy was constructed using single-layer interrupted absorbable sutures, ensuring a watertight and patent connection. Bowel continuity was then restored via a jejunojejunostomy.

The validity of this aggressive yet physiological surgical approach was borne out by the patient's postoperative course. The recovery was uncomplicated, and the patient was discharged on the seventh postoperative day. At the 3-month follow-up, the clinical picture had completely normalized; the patient reported a complete resolution of the epigastric pain that had plagued her for a year. Furthermore, repeat biochemical analysis showed that liver function tests remained within normal limits, confirming the successful restoration of biliary drainage and the absence of stasis or stricture.

3. Discussion

This case report serves as a critical interrogation of the conventional heuristic used in hepatobiliary surgery: that the severity of biliary obstruction correlates linearly with the derangement of biochemical markers. The clinical presentation of a patient harboring a giant calculus, measuring 28 mm in diameter, within a massively dilated common bile duct (CBD) of 22 mm, represents a significant anatomical burden. Yet, this gross morphological pathology existed in a state of biochemical silence, evidenced by a total bilirubin of 0.39 mg/dL and, perhaps more surprisingly, a normal gamma-glutamyl transferase (GGT) level.[11]

In standard clinical teaching, choledocholithiasis typically manifests through the prism of Charcot's triad or, at a minimum, cholestatic jaundice. The pathophysiology of jaundice is mechanical: an obstruction raises the intrabiliary pressure.[12] When this pressure exceeds the hepatic secretory pressure—physiologically estimated at approximately 30 cmH_2O—the flow of bile is arrested, and conjugated bilirubin regurgitates into the systemic circulation. The complete absence of this phenomenon in the presented case compels a detailed pathophysiological dissection. We propose that two synergistic biomechanical phenomena—the ball-valve mechanism and the principle of biliary compliance governed by the Law of Laplace—conspired to mask the disease.

The first mechanism explaining the anicteric presentation is the mobility of the stone itself.[13] Unlike small stones that frequently become impacted in the distal ampulla, causing acute, unremitting obstruction, giant stones often float within the biliary column. In the context of a dilated duct, the calculus is not fixed in a single position. Instead, it functions as a ball-valve. Under this mechanic, the stone intermittently occludes the ampulla of Vater, often precipitated by meals or changes in posture, causing a transient rise in biliary pressure that manifests

clinically as biliary colic. This correlates with the patient's history of intermittent, moderate epigastric pain. However, because the duct is dilated, the stone is capable of disimpacting and floating proximally when the pressure equilibrates. This creates periods of intermittent patency.[14]

Crucially, the pharmacokinetics of bilirubin elimination play a vital role here. The half-life of bilirubin in the bloodstream is approximately 4 hours. Because the obstruction is not continuous, these windows of patency allow the liver sufficient time to drain the accumulated bile and clear bilirubin from the serum.[15] Consequently, the serum bilirubin levels never reach the threshold required to manifest as clinical jaundice (typically >2.5 mg/dL) or even biochemical hyperbilirubinemia. The patient's intermittent pain profile supports this theory of a mobile, floating stone rather than a fixed, high-grade impaction.

The second, and perhaps more profound, physiological factor is the compliance of the biliary tree. The common bile duct is not a rigid pipe; it is a biological tube capable of remodeling. The mechanics of this system are governed by the Law of Laplace, which states that Wall Tension (T) is proportional to the product of Pressure (P) and Radius (r) (T= P x r).

In cases of acute obstruction (a small stone in a non-dilated duct), the radius is small. Therefore, a rapid rise in pressure (P) leads to a rapid increase in wall tension (T), triggering pain and immediate backflow of bile. However, in chronic obstruction, the biliary tree undergoes significant remodeling, characterized by the loss of elastic fibers and atrophy of the smooth muscle within the ductal wall. This remodeling results in a highly compliant, baggy, or atonic duct.

In our patient, the CBD had dilated to a massive 22 mm. According to the Law of Laplace, as the radius (r) increases, the wall can accommodate a larger volume of fluid without a corresponding linear increase in pressure (P). The dilated duct essentially acts as a low-pressure reservoir or buffer. Even if the distal end is partially obstructed by the stone, the immense volume of the megaduct absorbs the pressure rise. This buffering capacity prevents the intrabiliary pressure from reaching the critical secretory threshold necessary to reverse hepatocyte secretion. This physiological compliance explains the discordance seen on imaging: gross dilation of the extrahepatic duct without any upstream intrahepatic biliary duct (IHBD) dilation or enzymatic elevation. The pressure was dissipated within the extrahepatic reservoir, sparing the liver parenchyma.[16]

The case highlights a significant limitation in contemporary risk stratification algorithms. The current guidelines, such as those promulgated by the American Society for Gastrointestinal Endoscopy (ASGE), rely heavily on biochemical markers to triage patients. Under these protocols, bilirubin is a primary determinant. Patients presenting with normal bilirubin and normal liver enzymes are typically categorized as low risk (<10%) or intermediate risk (10-50%) for choledocholithiasis.[17] The standard recommendation for these low-risk groups is often expectant management or less invasive testing, rather than immediate visualization.

However, as this case demonstrates, low risk by biochemical standards is not synonymous with no pathology. The reliance on these algorithms created a diagnostic blind spot for the referring physicians. The patient's symptoms were attributed to gastritis and dyspepsia for months because her lab work did not scream biliary obstruction. This delay is not benign; it allowed the stone to grow and the duct to dilate further, transforming a potentially endoscopic case into a complex surgical one.

This evidence supports a revision of clinical suspicion thresholds. In patients with chronic, unexplained epigastric pain—specifically those in the geriatric population (>60 years) where ductal compliance is naturally higher—biochemistry is an unreliable screen.[18] In such cases, cross-sectional imaging, such as magnetic resonance cholangiopancreatography (MRCP), should be considered mandatory regardless of liver function test results. Continued reliance on labs alone in this

patient could have led to catastrophic sequelae, such as acute suppurative cholangitis or biliary pancreatitis, once the compensation mechanisms finally failed (Figure 2).

The management of a 28 mm stone encased within a 22 mm duct presents a complex surgical dilemma. The treatment must address not only the obstructing agent (the stone) but also the underlying anatomical failure (the dilated duct). The multidisciplinary team evaluated four potential modalities: Endoscopic retrograde cholangiopancreatography (ERCP), open choledochotomy with T-tube drainage, Choledochoduodenostomy (CDD), and Roux-en-Y Choledochojejunostomy (RYCJ).

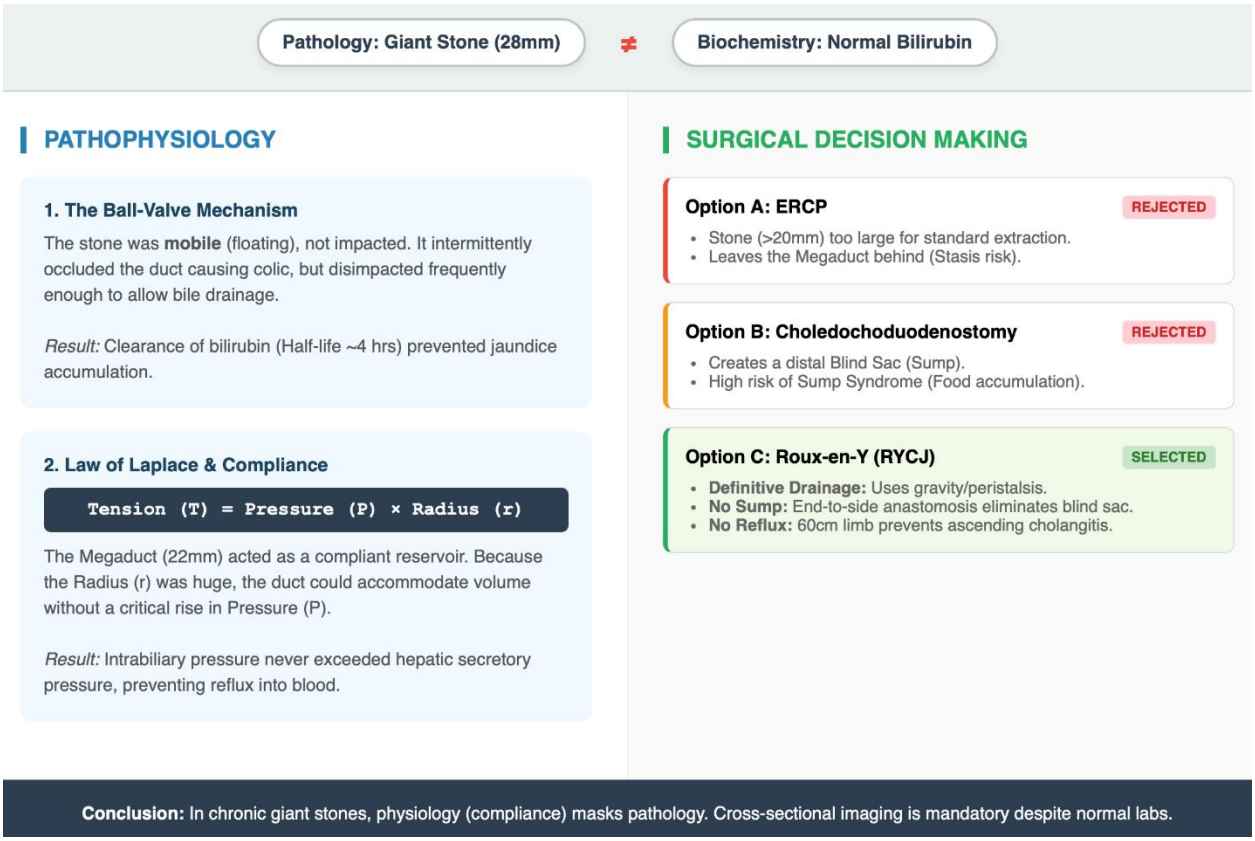

Figure 2. Pathophysiology and surgical decision-making of this case.

ERCP is generally the first-line treatment for choledocholithiasis. However, its efficacy diminishes precipitously as stone size increases.[19] For a stone exceeding 20 mm, standard sphincterotomy is insufficient for extraction. Successful removal would necessitate advanced adjuncts like mechanical lithotripsy or electrohydraulic lithotripsy. These techniques introduce significant risks, including basket impaction, ductal perforation, and hemorrhage. Furthermore, even if ERCP were successful in clearing the stone, it would leave the megaduct intact. An atonic, 22 mm duct does not regain its tone; it remains a cesspool for bile stasis, leading to a high probability of primary stone recurrence. Thus, ERCP was deemed a temporizing measure rather than a definitive cure.

Primary closure over a T-tube was historically common but is ill-suited for a duct of this magnitude.[20] Attempting to close a 22 mm duct creates tension and is prone to leakage. More

critically, like ERCP, the T-tube strategy fails to address the pathology of the dilated duct. It leaves the reservoir in place, ensuring that stasis—and therefore stone formation—continues unabated.

The decision, therefore, rested between two drainage procedures: CDD and RYCJ. Choledochoduodenostomy is technically less demanding and faster, making it an attractive option for frail, elderly patients. However, CDD involves creating a side-to-side anastomosis between the CBD and the duodenum. This geometry leaves the distal portion of the CBD (below the anastomosis) as a blind sac or sump. In a significant minority of patients (2–5%), food particles and debris accumulate in this blind sac, serving as a nidus for infection. This phenomenon, known as sump syndrome, causes recurrent cholangitis and pain. Given our patient's relatively young physiological age (61) and robust functional status, the risk of long-term Sump Syndrome was unacceptable.[17,18]

Consequently, Roux-en-Y Choledochojejunostomy (RYCJ) was selected as the superior option. RYCJ involves transecting the duct and bringing a limb of jejunum up to the biliary tree (end-to-side). This bypasses the sphincter entirely and eliminates the distal blind sac, thereby negating the risk of Sump Syndrome. Furthermore, the creation of a 60 cm alimentary limb provides a substantial physical barrier against the reflux of enteric contents into the biliary tree. While technically more complex, RYCJ offers the most durable, long-term solution with the lowest rates of reflux cholangitis and stone recurrence.

Macroscopic inspection of the extracted calculus provided insight into its etiology. The stone was dark, friable, and pigmented, characteristics consistent with a calcium bilirubinate or pigment stone. Unlike cholesterol stones, which are associated with metabolic syndrome, pigment stones are frequently associated with chronic stasis and bacterial infection (bactibilia). Bacteria produce the enzyme beta-glucuronidase, which deconjugates soluble bilirubin, causing it to precipitate with calcium. The presence of such a massive pigment stone reinforces the theory that the dilated duct acted as a stagnant swamp, promoting bacterial colonization and stone growth over a prolonged period. While definitive chemical analysis via infrared spectroscopy was not performed, the visual evidence strongly supports a stasis-infection model of pathogenesis.[19,20]

4. Conclusion

This case of the anicteric giant illustrates a profound physiological lesson: massive biliary pathology can exist in the complete absence of standard biochemical markers. The patient harbored a 28 mm stone within a 22 mm duct for over a year, yet maintained a normal bilirubin profile due to the synergistic interplay of a ball-valve mechanism and significant ductal compliance. This diagnostic silence led to a prolonged period of mismanagement where symptoms were misattributed to gastritis. Clinicians must recognize that normal bilirubin and liver enzymes—including GGT—do not reliably exclude giant choledocholithiasis. This is particularly true in chronic presentations where physiological compensation mechanisms (ductal dilation) mask the obstruction. The persistence of vague epigastric pain, especially when accompanied by a positive Murphy's sign or radiation to the scapula, warrants cross-sectional imaging (MRCP or CT). This should be the standard of care even if laboratory values are within reference ranges. The management of giant stones is not merely about extraction; it is about flow dynamics. For stones larger than 20 mm residing within significantly dilated ducts (>20 mm), simple extraction is insufficient. Roux-en-Y Choledochojejunostomy is the superior reconstructive option. It provides definitive drainage, eliminates the reservoir for stasis, and prevents the long-term complications of sump syndrome and reflux cholangitis.

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