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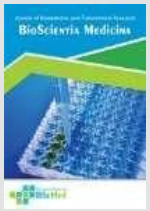

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From Antenatal Clue to Postnatal Cure: Surgical Management of a Symptomatic Jejunal Duplication Cyst in an Infant

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A B S T R A C T

Background: Alimentary tract duplication cysts represent a rare and challenging subset of congenital anomalies. Their non-specific symptomatology often leads to their misidentification as more common pediatric conditions, making a clear diagnostic pathway essential. With jejunal localization being particularly uncommon, these lesions can range from being asymptomatic to causing life-threatening abdominal emergencies. The evolution of high-resolution prenatal imaging, however, has fundamentally altered their management, enabling proactive postnatal intervention. **Case presentation:** A three-month-old female infant, with a history of a non-specific cystic intra-abdominal mass identified on a second-trimester antenatal ultrasound, was referred for progressive abdominal distension and non-bilious vomiting. Postnatal examination revealed a palpable right upper quadrant mass. A contrast-enhanced computed tomography (CT) scan confirmed a 3.9 x 3.9 x 3.3 cm thick-walled jejunal duplication cyst causing partial obstruction. The patient underwent a successful exploratory laparotomy with segmental jejunal resection and primary end-to-end anastomosis. The postoperative course was uneventful, with complete resolution of symptoms. Histopathology confirmed a benign jejunal duplication cyst without heterotopic mucosa. **Conclusion:** Jejunal duplication cysts are a critical, albeit rare, consideration in the differential diagnosis of an infant with an abdominal mass or intestinal obstruction. This case serves as a paradigm of modern perinatal care, where an antenatal clue facilitates a planned, definitive postnatal cure. Complete surgical resection remains the gold standard, preventing severe complications and ensuring an excellent long-term prognosis.

1. Introduction

Congenital malformations of the gastrointestinal (GI) tract represent a diverse spectrum of developmental anomalies that can manifest at any point from the neonatal period through adulthood. Among these, alimentary tract duplications are a particularly infrequent entity, with an estimated incidence of 1 in 4,500 to 1 in 10,000 live births.[1,2] These anomalies are defined by a classic triad of histological and anatomical features: (i) the structure must be in intimate anatomical contact with, and often shares a common wall and blood supply with, a segment of the native alimentary canal; (ii) it must be lined by a recognizable gastrointestinal-type mucosa; and (iii) it must possess a distinct layer of smooth muscle in its wall.[3,4] These duplications can occur at any location along the digestive tube, from the esophagus to the rectum. The small intestine is the most frequently affected segment, accounting for over half of all cases, with the ileum being the most

common site of involvement.[5] Duplications of the jejunum are comparatively less common, representing approximately 8-15% of all GI duplications.[6] Anatomically, these lesions are classified into two main types: cystic, which is the more common form (approximately 80% of cases) and does not communicate with the adjacent intestinal lumen, and tubular, which may have one or more connections to the native bowel.[7]

The clinical presentation of a jejunal duplication cyst is notoriously variable and is largely dictated by its size, location, and the presence of ectopic mucosa.[8,9] This variability poses a significant diagnostic dilemma. For the pediatrician or emergency physician, a duplication cyst can present as simple feeding intolerance, a surgical abdomen with acute obstruction, or unexplained anemia, making it a critical, albeit rare, consideration in a wide range of clinical scenarios.[10] The non-specific nature of these symptoms can often lead to delayed diagnosis or misdiagnosis, underscoring the importance of a high index of suspicion. The diagnostic journey often begins with clinical suspicion, followed by targeted imaging.[11] Ultrasonography is typically the initial modality of choice in infants, often revealing the pathognomonic "gut signature" or "double wall" sign.[12] For more detailed anatomical delineation, cross-sectional imaging with computed tomography (CT) or magnetic resonance imaging (MRI) is invaluable. The universally accepted standard of care is complete surgical resection to alleviate symptoms and, crucially, to preempt potential life-threatening complications, including volvulus, perforation, and the rare but documented risk of malignant transformation in adulthood.[13,14]

This case report presents a comprehensive illustration of the ideal management timeline for a congenital anomaly, serving as a paradigm of modern perinatal care. Just a few decades ago, such a diagnosis would have been impossible until the onset of severe, life-threatening symptoms. The evolution of perinatal medicine, particularly high-resolution fetal imaging, has transformed this paradigm from one of reactive emergency surgery to one of proactive, planned intervention. The novelty of this manuscript, therefore, is not in the rarity of the condition alone, but in its detailed narrative, which seamlessly bridges the gap between a non-specific prenatal suspicion and a definitive postnatal surgical cure. This narrative reinforces a clinical pathway that optimizes patient outcomes and serves as a powerful educational tool. Therefore, the aim of this manuscript is to present a detailed, evidence-based account of a symptomatic jejunal duplication cyst in an infant first identified antenatally. We aim to highlight the nuanced diagnostic process, provide a granular description of the surgical technique, and conduct an extensive, analytical review of the current literature. Through this, we offer a comprehensive overview of the embryology, pathophysiology, and state-of-the-art management of this important surgical condition, providing a high-yield educational resource for clinicians.

2. Case Presentation

A three-month-old female infant was referred to the pediatric surgery outpatient clinic with a two-month history of progressive abdominal distension and a one-week history of non-bilious vomiting. The mother's obstetric history was significant for a cystic mass noted in the fetal abdomen during a routine second-trimester ultrasound. The infant was born at term and had been feeding well until the recent onset of postprandial vomiting. The parents denied any history of fever, diarrhea, or changes in stool. A detailed summary of the history and objective physical findings, which collectively pointed toward a subacute obstructive process secondary to a palpable mass, is presented in Figure 1. Figure 1 is designed to narrate the patient's journey, integrating historical data with objective physical examination findings to build a cohesive diagnostic picture. The journey begins with the Patient Profile, establishing the demographic context: a well-nourished infant in her first few months of life. The central clinical puzzle is introduced in the Presenting Complaint and Symptom Timeline,

which details the insidious onset of progressive abdominal distension over two months, a chronic symptom that acutely escalated with the recent development of non-bilious vomiting. This chronology is critical, suggesting a gradually worsening obstructive process rather than an acute inflammatory event. The physical examination findings, graphically represented in the central schematic, are the cornerstone of the clinical diagnosis. The Inspection panel notes the visible asymmetry of the abdomen, a direct external manifestation of the underlying pathology. This finding is precisely localized in the Palpation section, which documents a discrete, firm, and mobile 4x4 cm mass in the Right Upper Quadrant (RUQ). The anatomical diagram provides a clear visual anchor for this finding, demarcating the abdominal quadrants and highlighting the exact location of the palpable mass. This tangible evidence transitions the diagnosis from a vague complaint of "distension" to a specific concern for a structural anomaly. Contextualizing these findings are the Vitals & General Exam and Associated Symptoms panels, which collectively paint a picture of a hemodynamically stable infant without signs of systemic illness or peritonitis, thereby narrowing the differential diagnosis away from infectious or acutely ischemic etiologies. Finally, the most crucial piece of the historical puzzle is highlighted in the Antenatal Clue panel. The knowledge of a prenatally detected cystic mass transforms the entire clinical encounter. It provides a powerful diagnostic anchor, allowing the postnatal findings to be interpreted not as new, isolated phenomena, but as the expected evolution of a known congenital anomaly. This integration of prenatal and postnatal data is the hallmark of modern perinatal care and is the central narrative theme of this clinical presentation.

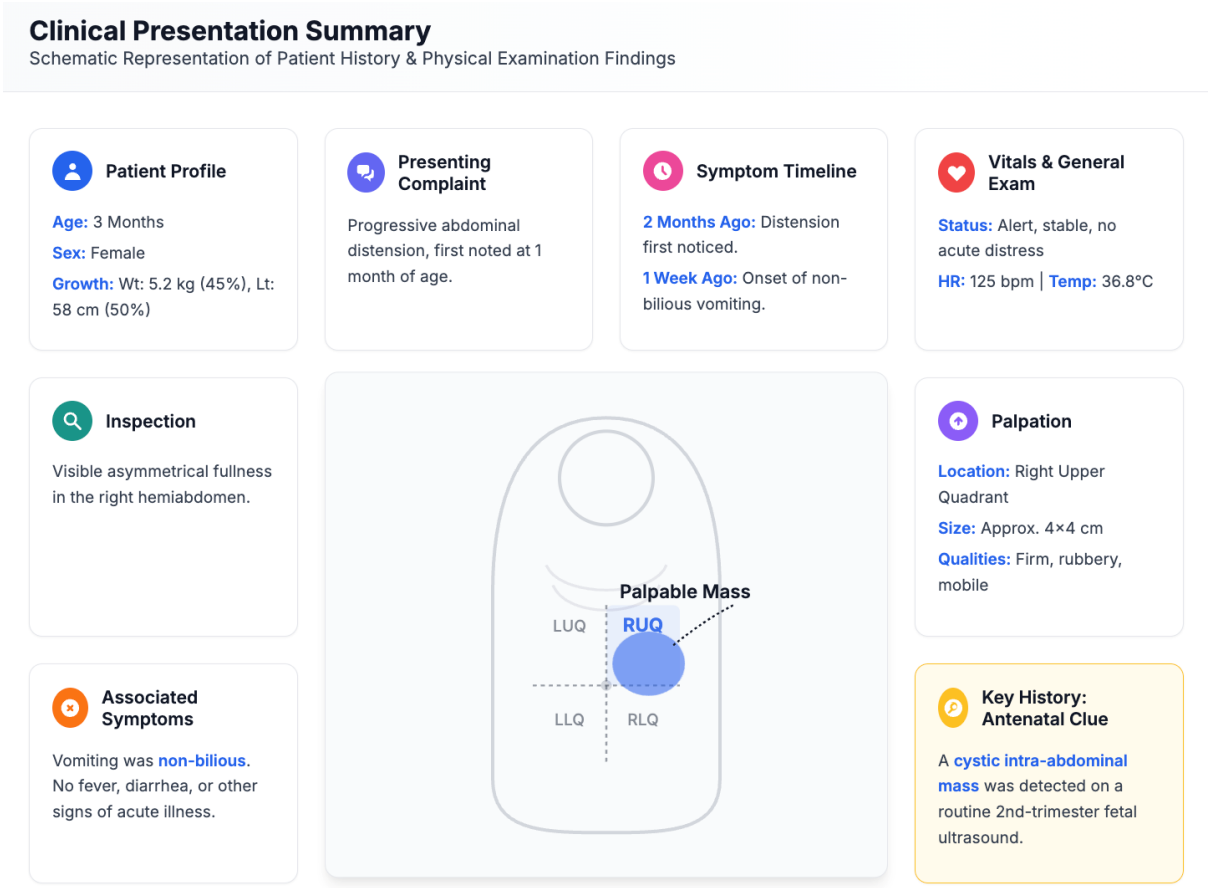

Figure 1. Patient history and physical examination findings.

Initial laboratory investigations were performed to assess for underlying infection, anemia, or metabolic disturbance. An abdominal ultrasound was performed as the first-line imaging study, which was highly suggestive of an enteric duplication cyst. To provide a definitive anatomical roadmap for surgical planning, a contrast-enhanced abdominal CT scan was subsequently performed. The comprehensive results of these investigations, which solidified the preoperative diagnosis, are detailed in Figure 2. Figure 2 is logically structured to represent the clinical workflow, beginning with foundational laboratory assessments and culminating in advanced, high-resolution cross-sectional imaging, thereby illustrating a comprehensive and modern diagnostic pathway. The left panel summarizes the Laboratory Findings, which serve a crucial exclusionary role. The normal hematological and biochemical parameters—including a stable hemoglobin level, a non-elevated white blood cell count, and a negligible C-reactive protein level—effectively ruled out acute infectious, significant inflammatory, or hemorrhagic processes. This allowed the clinical focus to sharpen decisively on a congenital structural anomaly as the most probable etiology for the patient's symptoms. The right panel, dedicated to Imaging Findings, forms the core of the diagnostic argument. It depicts a multimodal imaging approach that provides increasingly specific information. The initial investigation, an Abdominal CT scan, revealed the pathognomonic "double wall" sign. This finding is virtually diagnostic of an enteric duplication. CT scan represents the distinct hyperechoic mucosal and hypoechoic muscular layers of a bowel wall, unequivocally confirming the lesion's gastrointestinal origin. To achieve superior anatomical delineation for surgical planning, a Contrast-Enhanced Computed Tomography (CT) scan was subsequently performed. The CT scan not only confirmed the ultrasound findings but also provided a precise three-dimensional characterization of the lesion, defining its exact dimensions (3.9 x 3.9 x 3.3 cm), its thick, enhancing wall, and its critical anatomical relationship—arising from a jejunal loop and causing extrinsic luminal compression. The identification of associated mesenteric lymphadenopathy further completed the radiological picture. Ultimately, Figure 2 illustrates a powerful diagnostic synergy: laboratory tests provided a stable clinical baseline, the ultrasound offered a highly specific diagnosis, and the CT scan delivered the essential anatomical roadmap. This cascade of evidence converged to establish the confident Preoperative Diagnosis of a symptomatic jejunal duplication cyst, enabling a well-planned and successful surgical intervention.

Following preoperative optimization and informed consent, the patient underwent an exploratory laparotomy. The intraoperative findings confirmed the radiological diagnosis, revealing a classic jejunal duplication cyst causing extrinsic compression. A definitive segmental resection with primary anastomosis was performed. The postoperative course was uncomplicated, with a rapid return to normal gastrointestinal function. Figure 3 is structured to present a clear, chronological narrative, beginning with the critical intraoperative details and concluding with the key milestones that marked a successful recovery. The left panel, Surgical Intervention Details, encapsulates the core components of the operative procedure. It confirms the definitive treatment was an exploratory laparotomy, which allowed for a segmental resection of the affected portion of the jejunum. The intraoperative findings detail the precise nature of the lesion—a 4x4 cm tense, fluid-filled cyst causing extrinsic compression—which perfectly aligns with the preoperative imaging. The reconstruction method, a meticulous single-layer, hand-sewn end-to-end anastomosis, is highlighted as the crucial step in restoring the continuity of the gastrointestinal tract, a standard and time-tested technique in pediatric surgery. The Postoperative Recovery timeline, presented on the right, offers a concise yet powerful testament to the success of the intervention. The progression from the return of bowel function on day two to the initiation of enteral feeding on day three, and advancement to full feeds by day four, illustrates

a rapid and textbook recovery. The complete resolution of the preoperative vomiting and the patient's discharge home in excellent condition on the sixth postoperative day signifies a complete and successful surgical cure. Complementing this data are the Intraoperative Images, which provide direct visual evidence of the pathology and its resolution. The first image clearly shows the isolated duplication cyst, its characteristic appearance, and its intimate anatomical relationship with the native jejunum. The second image, showing the completed anastomosis, serves as a visual confirmation of the successful surgical reconstruction.

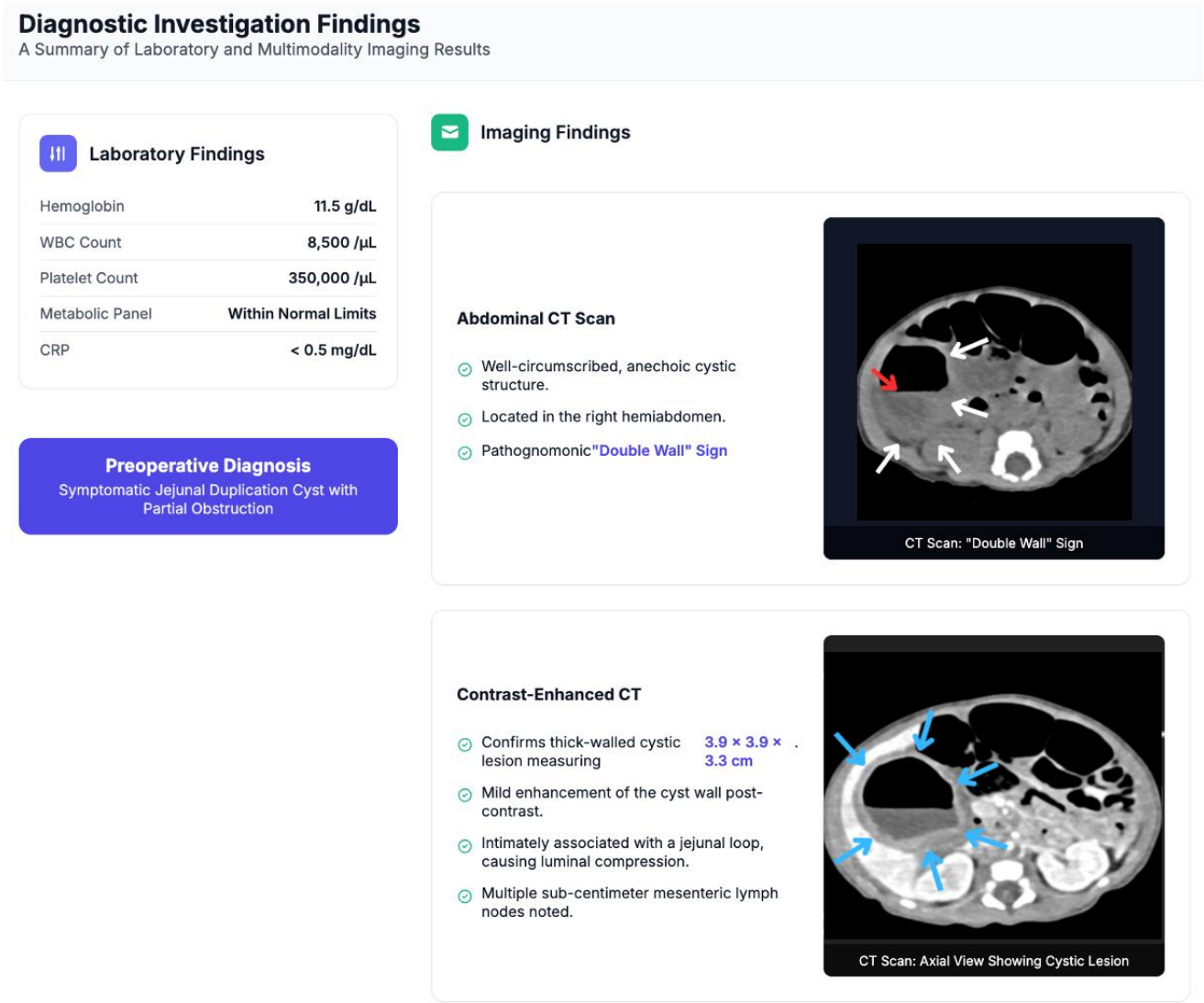

Figure 2. Diagnostic investigation findings.

The resected specimen was sent for histopathological analysis. This final step in the diagnostic journey provided the definitive diagnosis, confirming the lesion's benign nature and ruling out the presence of ectopic tissue or malignancy. The detailed gross and microscopic findings are presented in Figure 4.

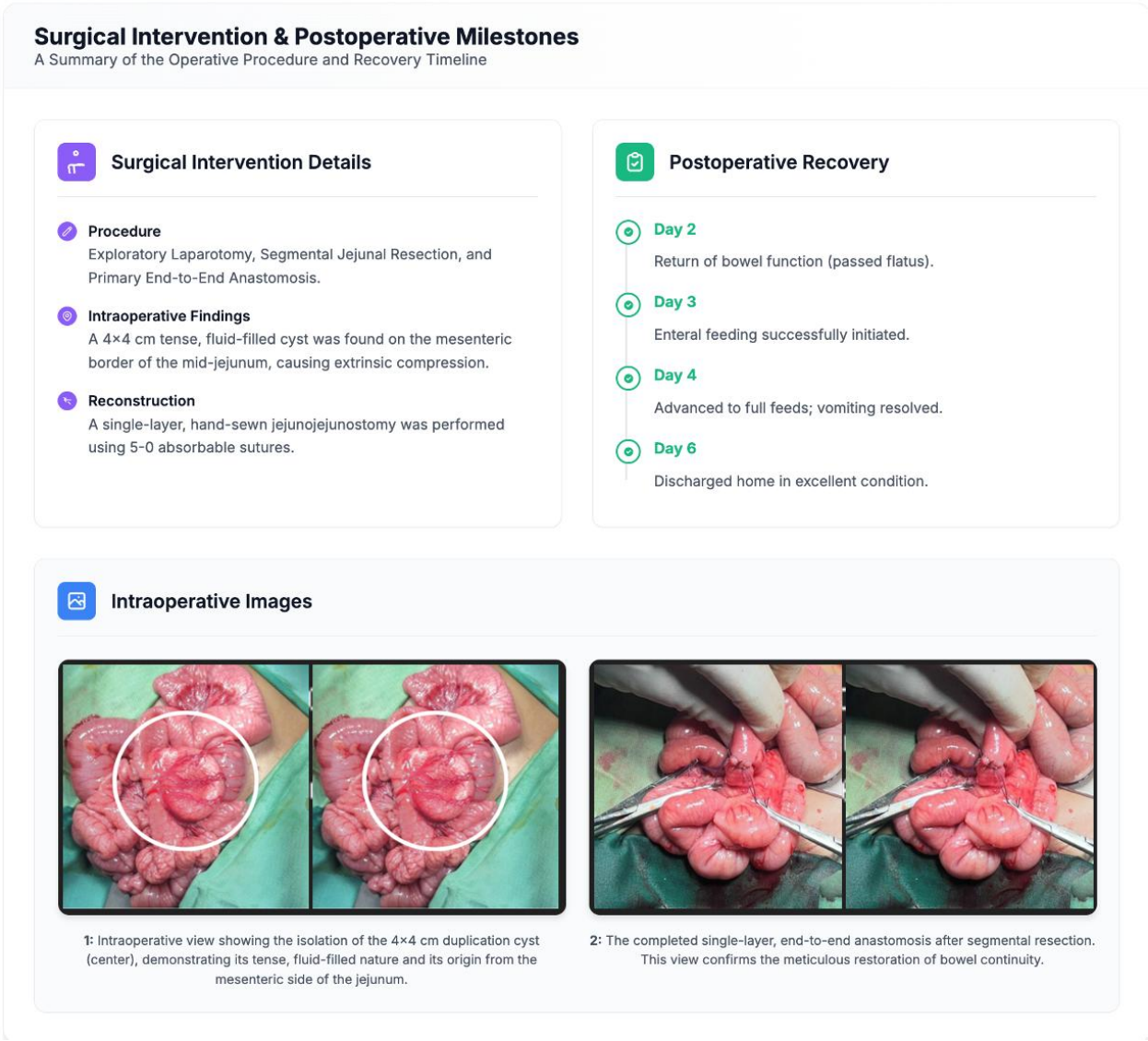

Figure 3. Surgical intervention and postoperative milestones.

3. Discussion

This case report presents a paradigmatic journey through the diagnosis and management of a symptomatic jejunal duplication cyst, a rare congenital anomaly whose clinical importance far outweighs its statistical frequency. The narrative, from a subtle shadow on a prenatal scan to a tangible surgical cure, encapsulates the triumphs of modern perinatal and pediatric surgical care. The ensuing discussion aims to weave the threads of this individual experience into the broader tapestry of scientific understanding, delving into the profound complexities of embryological development, the diverse and often dramatic clinical presentations, the sophisticated diagnostic armamentarium available to the modern clinician, and the nuanced principles of surgical intervention that define the standard of care for this fascinating condition.[15] The genesis of an alimentary tract duplication is a story of exquisite developmental choreography gone awry. The formation of the primitive gut tube is one of the earliest and most fundamental events in embryogenesis, a process of such complexity that it is vulnerable to a variety of subtle insults.[16]

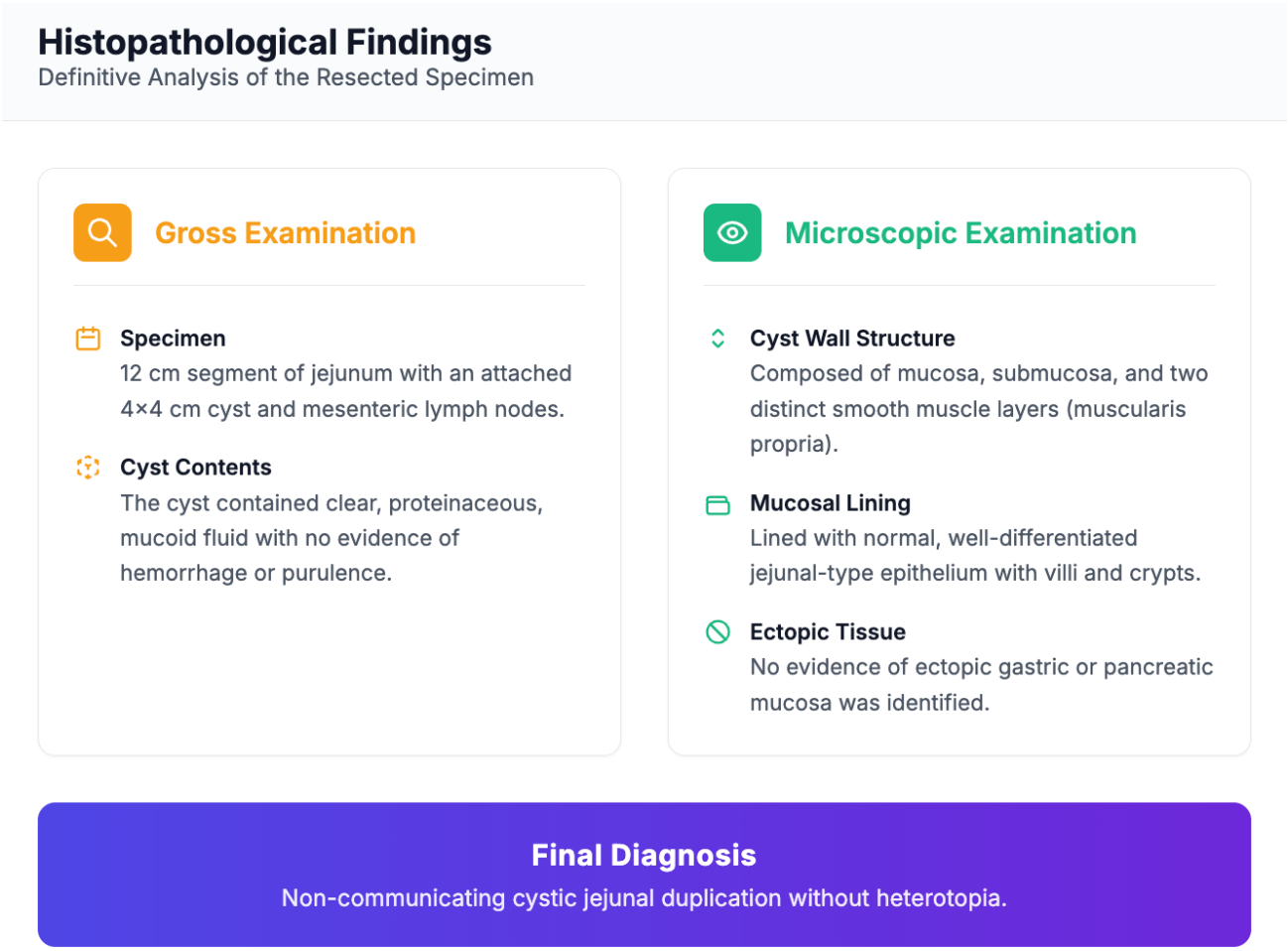

Figure 4. Histopathological findings.

While the exact cause of these duplications remains elusive, several compelling theories offer windows into the potential mechanisms, each rooted in a different critical moment of embryonic life. Understanding these theories is not merely an academic exercise; it provides a conceptual framework for appreciating the vast anatomical diversity of these lesions, from tiny cysts to long, complex tubular structures, and their intimate, often perilous, relationship with the native gastrointestinal tract. The "split notochord" theory is perhaps the most elegant and historically significant of these hypotheses, particularly in its power to explain the association between certain duplications and vertebral anomalies.[17] Proposed in the mid-20th century, it focuses on the critical interplay during the third week of gestation between the developing notochord (the precursor to the vertebral column) and the endoderm (the precursor to the gut lining). In normal development, these two structures separate cleanly. The theory posits that a focal, abnormal adhesion between the endoderm and the overlying ectoderm can act as a tether. As the notochord forms and ascends, this tether forces it to split, creating a temporary fistula. Through this transient channel, a diverticulum of the primitive foregut can herniate dorsally, becoming entrapped and forming a duplication cyst that is often located in the posterior mediastinum. This explains why thoracic duplications are sometimes connected via a fibrous cord to spinal

anomalies like hemivertebrae or spina bifida, a constellation of findings known as the split notochord syndrome. While powerful for explaining these specific cases, its relevance diminishes for the more common, isolated duplications found within the abdomen, far from the developing spine.

For these isolated intra-abdominal lesions, such as the jejunal cyst in our patient, the "aberrant luminal recanalization" theory offers a more direct and widely accepted explanation. This hypothesis centers on a fascinating, transient stage of gut development. Around the sixth week of gestation, the primitive intestinal tube, which was initially hollow, undergoes a period of rapid epithelial proliferation that completely obliterates its lumen, transforming it into a solid cord. The lumen is then re-established by a process of programmed cell death (apoptosis) and vacuolization, where multiple small cavities form within the solid cord and subsequently coalesce to form a single, continuous channel.[18] The theory proposes that duplication cysts arise from a failure of this process. If one or more of these vacuoles fail to merge with the main developing lumen, they can persist as isolated, epithelium-lined cysts trapped within the bowel wall. If a series of adjacent vacuoles fails to connect properly, they could form a long, parallel tubular structure. This theory beautifully accounts for the fact that duplications share a common muscular wall and blood supply with the native intestine, as they both arise from the same primordial structure. It elegantly explains why the mucosal lining of the cyst is typically of the same type as the adjacent bowel, as it is derived from the same endodermal cells.

A third, mechanistically distinct hypothesis is the "intrauterine vascular accident" theory. This concept moves away from primary developmental errors and instead suggests a secondary insult. It posits that a localized vascular event, such as a transient thrombosis, embolism, or kinking of a mesenteric vessel during fetal life, could lead to a segmental infarction of the developing bowel wall. The ischemic segment could then undergo a complex process of resorption and healing that results in the formation of a separated, cystic structure. This theory is supported by experimental models and by the occasional clinical finding of duplications that seem more detached from the native bowel than classic theory would suggest. It may be particularly relevant for duplications that are found at some distance from the mesenteric border or those that have a more attenuated connection to the native bowel's blood supply. It is entirely plausible that these proposed mechanisms are not mutually exclusive. The clinical and anatomical heterogeneity of duplication anomalies suggests that they may indeed represent a final common pathway for a variety of different embryological insults occurring at different times and locations along the developing gut tube. The specific morphology and location of a given duplication may well be a reflection of the specific developmental process that was disrupted.[19]

The clinical presentation of a jejunal duplication cyst is a study in variability. These lesions are true chameleons of the abdominal cavity, capable of mimicking a wide range of more common pediatric pathologies. The specific signs and symptoms are determined by a dynamic interplay of factors: the cyst's size, its precise location, the rate of its expansion, and, most critically, the nature of its mucosal lining. The most common clinical scenario, exemplified by our patient, is that of intestinal obstruction. This is a simple yet effective mechanism of disease. As the cystic duplication slowly enlarges from accumulating secretions, it exerts a progressive extrinsic pressure on the pliable lumen of the adjacent jejunum. Initially, this may cause intermittent, crampy abdominal pain and partial obstruction, leading to postprandial fullness and occasional vomiting. As the compression worsens, the symptoms become more constant and severe, with progressive abdominal distension and more frequent, eventually bilious, vomiting, signaling a high-grade obstruction that constitutes a surgical emergency.

Beyond this insidious compressive effect, duplications can precipitate acute abdominal catastrophes through more dynamic mechanisms.

They can serve as a pathological lead point for intussusception, a process where a segment of the intestine telescopes into the segment immediately distal to it. The duplication cyst, acting as a foreign body, is grasped by the peristaltic waves of the intestine and dragged forward, pulling the bowel wall along with it. This process not only obstructs the bowel lumen but, more dangerously, drags the mesentery along with it, compressing the mesenteric vessels and leading to venous congestion, edema, and eventual arterial ischemia and intestinal gangrene. The classic presentation of intussusception—intermittent colicky pain, a sausage-shaped abdominal mass, and "currant jelly" stools (stool mixed with blood and mucus from the ischemic bowel)—can be precisely mimicked by a duplication cyst. Similarly, a segment of jejunum burdened by a heavy, fluid-filled duplication cyst can become unstable, creating a predisposition to volvulus. In this scenario, the entire loop of bowel, including the cyst and its mesentery, twists on its vascular pedicle. This leads to a closed-loop obstruction and, more critically, a complete cut-off of the blood supply, resulting in rapid hemorrhagic infarction of the bowel. The presentation of a volvulus is often sudden and dramatic, with the acute onset of severe, unremitting abdominal pain, vomiting, and signs of shock.

The clinical plot thickens considerably with the presence of heterotopic mucosa within the duplication cyst, a finding in up to 35% of cases. The most common type of ectopic tissue is gastric mucosa. This misplaced tissue is not merely a histological curiosity; it is fully functional. The parietal cells within the ectopic gastric mucosa produce hydrochloric acid, and the chief cells produce pepsin, creating a highly acidic and proteolytic environment within the closed space of the cyst. The native jejunal mucosa lining the rest of the cyst, or the adjacent native small bowel, is not equipped to handle this acid exposure. The result is predictable: peptic ulceration. This ulceration can lead to a variety of clinical presentations. It may cause chronic, occult blood loss, leading to an otherwise unexplained iron-deficiency anemia in a child. Alternatively, it can erode into a submucosal blood vessel, causing acute, massive, and painless lower gastrointestinal bleeding, presenting as melena or bright red blood per rectum. If the ulcer penetrates the full thickness of the cyst wall, it can perforate. Perforation into the free peritoneal cavity results in chemical and bacterial peritonitis, a life-threatening condition presenting with a rigid, board-like abdomen and systemic sepsis. Perforation can also occur into an adjacent organ, such as the native bowel, creating a fistula, or into the pancreas, causing acute pancreatitis. Ectopic pancreatic tissue is the second most common type of heterotopia. This tissue can become inflamed, leading to pancreatitis within the cyst, which can present with severe epigastric pain and elevated serum amylase and lipase, further muddying the diagnostic waters. It is this unpredictable potential for catastrophic complications arising from ectopic mucosa that forms one of the most compelling arguments for the surgical resection of all duplication cysts, even those that are found incidentally and are completely asymptomatic. The risk of waiting is simply too great.

In the contemporary surgical era, the diagnosis of a jejunal duplication cyst is rarely a complete surprise at laparotomy. The sophisticated arsenal of imaging modalities available allows for a highly accurate preoperative diagnosis in the majority of cases, enabling meticulous surgical planning and informed counseling of the family. The diagnostic algorithm is a stepwise process, typically beginning with the least invasive techniques and progressing as needed to provide complete anatomical definition. As our case vividly illustrates, the diagnostic journey can now begin even before birth. The routine use of high-resolution antenatal ultrasonography for fetal anatomical surveys has led to an increase in the incidental detection of fetal intra-abdominal cysts. While many of these are benign and transient, the sonographer may identify features suggestive of an enteric origin. A cyst that appears to have a discernible wall with different echogenic layers, or one that demonstrates peristalsis, should raise the suspicion of

a duplication cyst. A prenatal diagnosis, even if non-specific, is of immense value. It allows for a multidisciplinary team to be assembled, including pediatric surgeons and neonatologists, and for a plan to be made for postnatal evaluation and monitoring. It transforms an unexpected postnatal emergency into an anticipated clinical scenario.

Postnatally, abdominal CT scan is the undisputed first-line imaging modality in any infant or child with a suspected abdominal mass or obstructive symptoms. It is safe, portable, readily available, and provides excellent spatial resolution without the use of ionizing radiation. In the context of a duplication cyst, CT scan can reveal a pathognomonic finding: the "double wall" or "gut signature" sign. This sign is the direct visualization of the different layers of the bowel wall. The inner layer, comprising the mucosa and submucosa, appears hyperechoic (bright), while the outer layer, the muscularis propria, appears hypoechoic (dark). The presence of this two-layered wall surrounding a cystic structure is virtually diagnostic of an enteric duplication. CT scan can also assess the contents of the cyst (typically anechoic fluid), its size, its relationship to the adjacent bowel, and the presence of any complications such as inflammation or internal debris. A contrast-enhanced CT scan, as was performed in our patient, provides exquisite anatomical detail. It can precisely delineate the cyst's dimensions, wall thickness, and its exact relationship to the native bowel, the mesenteric vessels, and other abdominal organs. The administration of intravenous contrast helps to define the vascularity of the cyst wall and can highlight inflammatory changes. CT is also excellent at identifying associated findings, such as the mesenteric lymphadenopathy seen in our case, or complications like perforation or abscess formation. MRI serves as an excellent alternative to CT, with the major advantage of avoiding ionizing radiation, a particularly important consideration in the pediatric population. MRI can be superior in differentiating the fluid contents of the cyst and in characterizing its relationship with solid organs and the biliary system, making it especially useful for duplications in the duodenum or near the pancreatic head.

In the specific clinical scenario of a child presenting with lower gastrointestinal bleeding and a suspected duplication cyst, a specialized nuclear medicine scan can be the key to diagnosis. A Technetium-99m pertechnetate scan, commonly known as a Meckel's scan, is designed to detect ectopic gastric mucosa. The pertechnetate ion is selectively taken up and secreted by the mucoid cells of the gastric mucosa. When injected intravenously, the radiotracer accumulates in the stomach, but also in any other location in the body that contains ectopic gastric tissue, such as a Meckel's diverticulum or, pertinently, a duplication cyst. A focus of abnormal radiotracer uptake in the abdomen that appears concurrently with the stomach is a highly specific indicator of ectopic gastric mucosa and can pinpoint the source of bleeding, guiding the subsequent surgical exploration. The judicious use of this multimodal imaging approach allows the pediatric surgeon to enter the operating room not with a question, but with a clear anatomical roadmap and a well-defined surgical plan. The definitive and universally accepted treatment for a jejunal duplication cyst is complete surgical resection. The rationale for surgery is twofold and compelling: first, to relieve the symptoms of obstruction, pain, or bleeding that the cyst is causing; and second, and arguably more importantly, to prevent the future development of potentially lethal complications, such as volvulus, perforation, or malignant degeneration. The principle of surgery is simple—remove the anomaly—but the execution requires a nuanced understanding of the unique anatomy and a meticulous surgical technique to preserve the function of the remaining intestine.

The standard surgical procedure for a cystic duplication of the jejunum is a segmental resection with a primary end-to-end anastomosis.[20] This was the approach employed successfully in our patient. The choice of incision, typically a transverse supraumbilical laparotomy in an infant, is made to provide adequate exposure of the abdominal cavity.

Upon entering the abdomen, the surgeon performs a systematic exploration to confirm the diagnosis and identify the exact location of the duplication. The lesion is characteristically found on the mesenteric border of the jejunum. The surgeon must then carefully assess the vascular anatomy. The duplication and the adjacent segment of the native jejunum are supplied by the same terminal branches of the superior mesenteric artery, which travel through their shared mesentery. This shared blood supply is the anatomical feature that makes simple cyst enucleation a dangerous and generally unfeasible option. Attempting to dissect the cyst off the native bowel would inevitably disrupt these shared vessels, leading to devascularization and ischemia of the segment of jejunum that is left behind. This would place an anastomosis at high risk of leaking or forming a stricture. Therefore, the surgeon must resect the entire segment of jejunum that harbors the duplication. The points of transection, proximally and distally, are chosen in an area of healthy, pliable, and unquestionably well-vascularized bowel, typically several centimeters away from the visible extent of the duplication. The mesentery corresponding to the segment to be resected is carefully divided and ligated, preserving the main vascular arcades that will supply the remaining bowel. Once the segment is isolated, it is resected. The reconstruction of intestinal continuity is the most critical step of the procedure. An anastomosis, the surgical connection between the two cut ends of the bowel, is performed. In an infant, this is often done with a hand-sewn technique, using fine, absorbable sutures in a single, interrupted layer. The goal is to create a tension-free, well-vascularized, and watertight connection that will heal without leaking or narrowing. The surgeon must ensure that the two ends of the bowel are of similar diameter and that there is no twisting or angulation of the bowel at the anastomotic site. Once the anastomosis is complete, the defect in the mesentery is closed with sutures to prevent the future risk of an internal hernia.

While segmental resection is the workhorse procedure, the surgeon must be prepared to adapt the technique for unusual or complex duplications. For exceedingly long tubular duplications, where a massive resection would result in the devastating consequences of short bowel syndrome, a bowel-preserving technique is necessary. The most established of these is mucosal stripping. In this procedure, the surgeon makes a long incision on the antimesenteric border of the duplicated segment, opening it like a book. Then, using delicate dissection, the entire mucosal lining of the duplication is meticulously stripped away from the underlying common muscular wall. This removes the source of secretions and any potential ectopic mucosa, effectively de-functionalizing the duplication while preserving the muscular tube and its blood supply, which can then be closed or trimmed. For large cystic duplications in surgically challenging locations, such as those embedded in the head of the pancreas or the C-loop of the duodenum, where resection would endanger the bile duct or pancreatic duct, a less radical approach may be the safest option. In these cases, internal marsupialization can be performed. This involves creating a large, permanent window between the lumen of the duplication cyst and the lumen of the adjacent native duodenum or stomach. This allows the cyst to drain continuously, decompressing it and preventing the accumulation of secretions, thereby alleviating its obstructive symptoms without the need for a high-risk resection. In the 21st century, the field of pediatric surgery has been revolutionized by the adoption of minimally invasive surgery (MIS). Laparoscopy, using a camera and long, thin instruments inserted through several small keyhole incisions, is now a well-established and often preferred approach for resecting duplication cysts in appropriately selected patients. The laparoscopic approach offers the potential for significantly reduced postoperative pain, shorter hospital stays, a faster return to feeding, and superior cosmetic results. The fundamental principles of the surgery remain the same—complete resection of the duplication with a safe anastomosis—but they are accomplished with magnified vision and enhanced

dexterity. The feasibility of a laparoscopic resection depends on the size and location of the cyst, the age and size of the patient, and the experience of the surgical team.

4. Conclusion

This case of a prenatally suspected and postnatally managed symptomatic jejunal duplication cyst serves as a powerful affirmation of the principles of modern pediatric surgery. It underscores the critical importance of maintaining a high index of suspicion for this rare anomaly when faced with an infant presenting with an abdominal mass or signs of intestinal obstruction. The journey from a shadowy finding on a fetal ultrasound to a definitive histopathological diagnosis illustrates a seamless continuum of care, highlighting the synergy between advanced diagnostic imaging and decisive surgical intervention. The classic presentation, the pathognomonic radiological signs, and the successful outcome following segmental resection and primary anastomosis represent a model of effective management for this condition. The uneventful and complete recovery of our patient reinforces the established doctrine that complete surgical excision is not only therapeutic but also prophylactic, providing a permanent cure and eliminating the lifelong risk of unpredictable and severe complications. This case contributes to the collective clinical experience, reminding us that even in the face of rare and unusual pathology, a systematic approach grounded in a deep understanding of embryology, pathophysiology, and surgical technique can consistently lead to excellent, life-affirming outcomes for our youngest patients.

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