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The Discovery of Familia Adenomatous Polyposis in Patients with Intestinal Malrotations: A Rare Case

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Abstract. Familial Adenomatous Polyposis (FAP) is a rare hereditary condition characterized by numerous colorectal adenomas with a high risk of malignancy. Intestinal malrotation is a congenital anomaly that can lead to midgut volvulus, a life-threatening complication. The co-occurrence of these two distinct entities is exceptionally rare. This case report aims to describe the clinical presentation, diagnostic challenges, and management of a rare case where FAP was discovered incidentally during surgery for a late-diagnosed midgut volvulus. We present the case of an 8-year-old boy with a prolonged history of recurrent abdominal pain, vomiting, and diarrhea. The diagnosis was confirmed via abdominal CT scan and subsequent exploratory laparotomy. Surgical intervention for midgut volvulus revealed unexpected multiple polyps in the small intestine and colon, leading to the diagnosis of *FAP*. The patient recovered well postoperatively, but the FAP finding necessitates long-term surveillance. This case highlights the importance of considering anatomical anomalies in children with chronic gastrointestinal symptoms and underscores that a surgical emergency can unexpectedly reveal an underlying hereditary syndrome, drastically altering long-term management and family screening.

Keywords: Adenomatous polyps; familial adenomatous polyposis; gastrointestinal polyps; intestinal malrotation; midgut volvulus; pediatric surgery

INTRODUCTION

Polyps are masses that project into the lumen of the gastrointestinal tract. The pathophysiology of polyp formation involves complex molecular and genetic mechanisms. In normal intestinal epithelium, there is a delicate balance between cell proliferation in the crypts and programmed cell death (apoptosis) at the villus tips. Polyp formation occurs when this homeostatic balance is disrupted, leading to abnormal cellular proliferation and accumulation (De Souza Maia et al., 2024; Jacobson et al., 2023; Kowdley et al., 2019, 2020; Syngal et al., 2015). The development of adenomatous polyps specifically involves mutations in tumor suppressor genes, most notably the adenomatous polyposis coli (APC) gene located on chromosome 5q21-22. Loss of APC function disrupts the Wnt signaling pathway, resulting in accumulation of β-catenin in the nucleus, which promotes transcription of genes involved in cell proliferation and inhibition of apoptosis. This molecular cascade ultimately leads to the formation of adenomatous polyps, which, if left untreated, have the potential for malignant transformation into colorectal carcinoma (Van Nieuwenhuysen et al., 2015; Wang et al., 2019; Yamaguchi et al., 2018).

Epidemiologically, polyps are common during childhood, occurring in approximately 1% of preschool and school-age children globally (Durno et al., 2019). According to data from the World Health Organization (WHO), gastrointestinal polyps represent a significant pediatric

health concern, with incidence rates varying across geographical regions. In North America and Europe, the prevalence of juvenile polyps—the most common type in children—ranges from 0.5% to 2% in the general pediatric population (Hyer et al., 2020). The National Institutes of Health (NIH) estimates that approximately 1 in 8,000 to 1 in 14,000 individuals are affected by Familial Adenomatous Polyposis (FAP), an autosomal dominant condition characterized by the development of hundreds to thousands of adenomatous polyps (National Cancer Institute, 2021).

Due to their high incidence, gastrointestinal polyps are the most common cause of rectal bleeding in toddlers and children aged 2 to 5 years, accounting for approximately 60–90% of cases of painless rectal bleeding in this age group (Durno et al., 2019; Poddar & Thapa, 2021). However, it is crucial to consider differential diagnoses when evaluating pediatric rectal bleeding. Other important causes include anal fissures (which are the most common cause of painful rectal bleeding in infants and toddlers, often associated with constipation), Meckel's diverticulum (a congenital anomaly present in approximately 2% of the population that can cause painless rectal bleeding due to ectopic gastric mucosa), inflammatory bowel disease (including Crohn's disease and ulcerative colitis, which typically present with bloody diarrhea, abdominal pain, and systemic symptoms), infectious colitis (caused by bacterial pathogens such as Campylobacter, Salmonella, or Shigella species), and vascular malformations such as arteriovenous malformations or hemangiomas (Thakkar & Gilger, 2013; Fox et al., 2020). A comprehensive diagnostic approach including detailed history, physical examination, laboratory investigations, and appropriate imaging or endoscopic procedures is essential to differentiate among these conditions.

Adenomatous polyps occur in less than 3% of all children with polyps and are often discovered incidentally in association with other complaints (Durno et al., 2019). In this case, they were found together with midgut malrotation volvulus, which was diagnosed late. This case report presents a rare clinical scenario where two distinct pathological conditions—familial adenomatous polyposis and intestinal malrotation with volvulus—coexist in a single pediatric patient. The delayed diagnosis of malrotation, spanning several years despite recurrent symptoms, highlights critical challenges in pediatric surgical diagnosis and underscores the importance of maintaining high clinical suspicion for anatomical anomalies in children presenting with chronic gastrointestinal symptoms (Nehra & Goldstein, 2011; Durno et al., 2019). The objective of this case report is to describe the clinical presentation, diagnostic workup, and management of this rare combination of conditions, while discussing the implications for early detection and long-term follow-up.

MATERIALS AND METHOD

This study employed a qualitative research type, specifically a descriptive case report design, to provide an in-depth analysis of a unique clinical presentation. The data population and data sample were intrinsically linked, consisting of a single pediatric patient who presented with intestinal malrotation and was subsequently diagnosed with Familial Adenomatous Polyposis (FAP). The sampling technique was purposive, as the case was selected specifically due to its rarity and the valuable clinical insights it provides regarding diagnostic challenges and coincident pathologies.

The primary research instrument was the patient's medical record, which served as the

source for retrospective data collection on clinical history, physical examination findings, laboratory results, and radiological imaging. Data were supplemented by operative notes and histopathology reports. The data analysis technique involved a comprehensive descriptive analysis and thematic synthesis of the collected clinical data. This included a chronological reconstruction of the patient's medical journey, an evaluation of the diagnostic process, and a discussion of the management outcomes in the context of existing literature on both malrotation and FAP.

RESULTS AND DISCUSSION

Clinical History

An 8-year-8-month-old boy presented with abdominal pain that had been persistent for a long time but had worsened over the past month. The pain was intermittent and severe enough to cause crying during episodes. The pain was affected by position, with the most comfortable being lying prone with knees flexed since symptom onset. The abdominal pain was accompanied by diarrhea occurring one to two times daily, described as spraying in character. The abdomen gradually became more distended. The patient also experienced persistent nausea and a constant feeling of fullness. Vomiting occurred daily, containing food and water. Appetite remained normal, but the parents hesitated to provide food, fearing it would trigger vomiting. There were no complaints of fever, cough, runny nose, shortness of breath, or urinary problems.

Further history revealed that the patient had exhibited symptoms since around one year of age. At that time, he often vomited once or twice per episode, with episodes occurring nearly monthly. The vomiting included food and fluids and was sometimes projectile. Each episode was usually accompanied by abdominal bloating. The patient was frequently taken to a pediatrician, but the condition was always considered normal gastrointestinal problems. As he approached two years of age, similar symptoms recurred and further investigations were performed, including insertion of a nasogastric tube with contrast study. According to the parents, results showed intestinal narrowing, but it was deemed safe and no intervention was recommended.

Symptoms persisted, and by six years of age, complaints became more frequent. Vomiting occurred three to four times weekly, and watery stools sometimes mixed with stool particles occurred up to twice daily almost every week. However, symptoms improved with antiemetics and antibiotics, which reassured the parents. Since they were repeatedly told the condition was safe, they paid little attention to it. Between ages one and six, the patient was hospitalized five to six times.

By eight years old, in February 2025, the patient began complaining of abdominal pain not previously reported. The pain was described as stabbing and relieved by lying prone with knees flexed. The abdomen again became distended, although abdominal bloating had accompanied previous complaints. The sensation was described as if the abdomen were filled with water, shifting to the lowest point whenever the patient changed position. The patient belched more frequently, with the belching developing an acidic smell, which became bothersome. Vomiting became more frequent, nearly daily and up to twice per day, with food intake declining. Diarrhea occurred more than three times daily, characterized by an initial explosive watery stool that relieved discomfort and then ceased. After each episode of vomiting

or defecation, the patient felt more comfortable. Appetite decreased significantly. Laboratory tests and stool examinations were normal, and a colon-in-loop study was performed, but according to the parents, there was no recommendation for surgery since symptoms improved and further evaluation was not pursued. The mother reported the patient's growth and development were good and externally normal.

By July 2025, symptoms increased in frequency, and the child appeared wasted, unable to tolerate food, with a constantly full abdomen. From February to July, the patient's weight decreased by approximately 6–7 kg, with a body weight of 14 kg on hospital admission. An abdominal CT scan with contrast was performed, revealing the current condition. After a prolonged illness history, the patient was referred to our hospital for further evaluation and management.

Regarding other history, there was no history of febrile seizures or asthma. The patient had typhoid fever at age 4–5 years. The mother reported this was her second pregnancy, with no miscarriages. The interval between births was five years, and the patient has one sibling. Delivery was by cesarean section due to term pregnancy with premature rupture of membranes (PROM). The mother had regular antenatal visits, and ultrasounds showed no abnormalities. The mother had no medical problems before or during pregnancy. There was a family history of type 2 diabetes mellitus in the maternal grandmother. The mother had multiple drug allergies, including nearly all antibiotics (oral and injectable) and mefenamic acid. She reported increased emotional stress during this pregnancy due to family issues. There was no history of trauma or traditional abdominal massage. Before this pregnancy, the mother used an intrauterine device (IUD) for five years and became pregnant two months after removal. Regarding parental background, the mother is a university graduate (bachelor's degree) and works as a civil servant, while the father is a high school graduate working in the private sector.

Overview of Gastrointestinal Polyps in Children

Polyps are masses that project into the gastrointestinal tract lumen. From a histological perspective, polyps can be classified based on their cellular origin and growth pattern. Some polyp-like masses are subepithelial in origin, arising from the lamina propria or submucosa, but most true polyps originate from the epithelial lining of the gastrointestinal tract (Brosens et al., 2011; Calderwood et al., 2020). In children, polyps are less common than in adults and often present as isolated lesions known as juvenile polyps. Juvenile polyps are typically hamartomatous in nature, characterized by cystically dilated glands, abundant lamina propria, and an inflammatory infiltrate (Latchford & Phillips, 2011). These polyps are generally considered benign with minimal malignant potential when solitary, although multiple juvenile polyps (juvenile polyposis syndrome) carry an increased cancer risk (Agarwal et al., 2020).

Polyps may also be part of genetically related diseases characterized by multiple polyps (polyposis), familial inheritance, and increased lifetime risk of gastrointestinal or extraintestinal cancers (Syngal et al., 2015). Polyposis syndromes in children are classified into adenomatous and hamartomatous types based on their histopathological characteristics. Adenomatous polyposis syndromes are characterized by polyps with dysplastic epithelium and significant malignant potential, while hamartomatous polyps show overgrowth of normal tissue components with variable malignancy risk depending on the specific syndrome (Valle et al.,

2019).

As mentioned previously, polyps occur in approximately 1% of preschool and school-aged children globally, with juvenile polyps representing the majority (approximately 90%) of pediatric polyps (Durno et al., 2019). The peak incidence occurs between ages 2 and 10 years, with a median age of presentation around 5 years (Poddar & Thapa, 2021). Due to their high incidence, gastrointestinal polyps are the most common cause of rectal bleeding in toddlers and children aged two to five, accounting for 60-90% of cases of painless hematochezia in this age group (Thakkar & Gilger, 2013; Fox et al., 2020).

Adenomatous polyps occur in fewer than 3% of children with polyps. Adenomatous polyposis syndromes include Familial Adenomatous Polyposis (FAP), which represents the classic form caused by germline mutations in the APC gene; Gardner syndrome, a variant of FAP characterized by additional extraintestinal manifestations including osteomas, dental abnormalities, desmoid tumors, and various soft tissue tumors; and Turcot syndrome, another FAP variant associated with central nervous system tumors, particularly medulloblastomas and glioblastomas (Valle et al., 2019). Recent molecular genetic studies have identified genotype-phenotype correlations, with mutation location in the APC gene influencing disease severity and extraintestinal manifestations.

Hamartomatous polyposis syndromes include Juvenile polyposis syndrome (JPS), characterized by multiple juvenile polyps throughout the gastrointestinal tract with increased lifetime cancer risk due to mutations in SMAD4 or BMPR1A genes (Agarwal et al., 2020); Cowden disease (also known as PTEN hamartoma tumor syndrome), caused by mutations in the PTEN gene and associated with multiple hamartomas in various organs, increased breast, thyroid, and endometrial cancer risks, and characteristic mucocutaneous lesions (Tan et al., 2012); and Peutz-Jeghers syndrome (PJS), an autosomal dominant condition caused by STK11/LKB1 gene mutations, characterized by hamartomatous polyps throughout the gastrointestinal tract, distinctive perioral and buccal pigmentation, and significantly elevated risks of gastrointestinal and extraintestinal malignancies (Beggs et al., 2010; Giardiello et al., 2014).

Familial Adenomatous Polyposis

Among adenomatous polyposis syndromes, FAP is the most common in children, with an estimated incidence of 1 in 8,000 to 1 in 14,000 individuals worldwide (National Cancer Institute, 2021). FAP is characterized by progressive development of hundreds to thousands of adenomatous polyps in the colon, typically beginning in adolescence but potentially developing in childhood (Syngal et al., 2015). It is an autosomal dominant inherited disease defined by the presence of 100 or more adenomatous polyps in the colon and rectum, with a very high risk of progression to colorectal carcinoma (CRC) if left untreated (Valle et al., 2019). The genetic basis involves germline mutations in the APC gene, which functions as a tumor suppressor gene critical for regulating cell adhesion, migration, and apoptosis through its role in the Wnt signaling pathway.

The natural history of FAP follows a predictable progression. By age 15, approximately 50% of gene carriers develop detectable polyps on colonoscopy, with nearly 95% developing polyps by age 35 (Vasen et al., 2008; Stoffel et al., 2015). The lifetime risk of colorectal cancer in untreated patients approaches 100%, with an average progression time from adenoma

formation to CRC of approximately 10 to 15 years, though this can range from as early as the teenage years to the fourth decade of life. The median age of colorectal cancer diagnosis in untreated FAP patients is approximately 39 years, significantly younger than sporadic colorectal cancer (Giardiello et al., 2014).

FAP is often asymptomatic in early stages, particularly during childhood and early adolescence when polyp burden is lower (Durno et al., 2019). Clinical manifestations typically emerge during the teenage years or early adulthood as polyp numbers increase. Common presenting symptoms include rectal bleeding (hematochezia), which may be occult or visible; changes in bowel habits including diarrhea or constipation; abdominal pain or cramping; mucus discharge per rectum; and in advanced cases with significant polyp burden, symptoms of anemia such as fatigue, pallor, and tachycardia (Vasen et al., 2008; Syngal et al., 2015). In some cases, FAP is discovered incidentally during evaluations for other symptoms or through family screening programs when a relative is diagnosed with the condition (Stoffel et al., 2015; Monahan et al., 2020).

Case Analysis

In this case, suspicion of FAP arose incidentally due to the patient's presenting complaints rather than through systematic screening. The suspicion was identified earlier than would typically be expected in an asymptomatic FAP carrier because of accompanying symptoms eventually diagnosed as midgut volvulus malrotation—a congenital anomaly of intestinal rotation and fixation that can lead to intestinal obstruction and vascular compromise (Nehra & Goldstein, 2011). Notably, the malrotation itself was diagnosed late despite a prolonged history of symptoms, representing a significant diagnostic delay with potential implications for patient morbidity.

The patient had symptoms since early childhood, primarily recurrent abdominal bloating followed by vomiting and diarrhea, consistent with early features of malrotation with intermittent volvulus—a condition characterized by abnormal twisting of the intestinal mesentery that can cause intermittent obstruction and potentially life-threatening ischemia if complete vascular occlusion occurs (Lampl et al., 2009; Nehra & Goldstein, 2011). The pattern of intermittent symptoms improving with conservative management, followed by recurrence, is characteristic of partial or intermittent volvulus, where the intestinal twist may spontaneously reduce before causing irreversible damage. This intermittent presentation can lead to diagnostic delays, as occurred in this case, because symptoms may temporarily resolve, giving false reassurance to both parents and healthcare providers (McVay et al., 2007; Hagendoorn et al., 2011). It remains uncertain whether delayed detection was due to parental unawareness, limitations in access to advanced diagnostic facilities, or failure to recognize the significance of symptoms by medical professionals across multiple encounters over several years.

Malrotation with volvulus occurs in approximately 1 in 6,000 live births based on clinical series, with radiological barium studies showing a lower apparent incidence of 0.2% due to many cases remaining asymptomatic, and autopsy studies estimating the true anatomical incidence of malrotation at approximately 0.5% to 1% of the general population (Lampl et al., 2009; Pickhardt & Bhalla, 2002). The discrepancy between anatomical incidence and clinical presentation suggests that many individuals with malrotation remain asymptomatic throughout life, never coming to clinical attention (Applegate et al., 2009).

Classic malrotation with midgut volvulus typically presents in a previously healthy term neonate, with approximately 75% of symptomatic cases presenting in the first month of life, an additional 15% presenting within the first year, and the remaining 10% presenting later in childhood or even adulthood (Hagendoorn et al., 2011; Maloney & Pitt, 2014). The classic teaching emphasizes that any infant or child presenting with vomiting—especially bilious vomiting—under one year of age should be evaluated urgently for malrotation or volvulus until proven otherwise, as this represents a surgical emergency (Lampl et al., 2009; Shalaby et al., 2013). However, as this case demonstrates, late presentations beyond infancy do occur, particularly with incomplete or intermittent volvulus, and can be easily missed if clinical suspicion is not maintained (McVay et al., 2007).

Sudden bilious vomiting is a hallmark of intestinal obstruction in neonates and represents a red flag symptom requiring immediate evaluation (Applegate et al., 2009). Initial physical findings in acute volvulus may include a scaphoid or mildly distended upper abdomen in early stages, reflecting proximal intestinal obstruction (Shalaby et al., 2013). As vascular compromise progresses, leading to intestinal ischemia and eventual necrosis, clinical findings evolve to include progressive abdominal distension, peritoneal signs (guarding, rigidity, rebound tenderness), hemodynamic instability, and metabolic acidosis indicating advanced disease (Lampl et al., 2009; Nehra & Goldstein, 2011). This matches the patient's clinical course, with recurrent vomiting and intermittent bloating progressing over years to pronounced abdominal enlargement, position-dependent pain, and significant weight loss, indicating chronic partial obstruction with intermittent acute exacerbations.

Radiology plays a crucial role in diagnosing intestinal malrotation, though clinical suspicion must remain high as imaging can sometimes be falsely reassuring, particularly in cases of intermittent volvulus (Applegate et al., 2009; Pickhardt & Bhalla, 2002). Prenatal diagnosis of malrotation is rare but can be suggested by ultrasonography showing bowel dilatation, polyhydramnios (excessive amniotic fluid due to impaired fetal swallowing), meconium peritonitis, or fetal ascites (Hagendoorn et al., 2011). Postnatally, initial evaluation typically includes plain abdominal radiographs in anteroposterior and lateral or cross-table lateral views; findings may show a gasless abdomen, dilated stomach and proximal duodenum with a "double bubble" sign, or nonspecific bowel gas patterns (Shalaby et al., 2013; Maloney & Pitt, 2014).

Upper gastrointestinal contrast studies (upper GI series with small bowel follow-through) are considered the gold standard for diagnosing malrotation, with diagnostic findings including abnormal position of the duodenojejunal junction (DJ junction) to the right of midline or below the level of the duodenal bulb, a "corkscrew" or "spiral" configuration of the proximal small bowel suggestive of volvulus, and the lack of normal fixation of the small bowel mesentery (Applegate et al., 2009; Nehra & Goldstein, 2011). However, false negatives can occur, particularly in intermittent volvulus, emphasizing the need for repeat imaging if clinical suspicion remains high despite initial negative studies (Pickhardt & Bhalla, 2002). In this patient, initial plain films suggested possible obstruction, but definitive diagnosis was not made until computed tomography (CT) scan was performed years later, highlighting the diagnostic challenges inherent in chronic, intermittent presentations.

Management and Outcomes

Management of suspected or confirmed midgut volvulus includes several critical steps that must be initiated urgently (Lampl et al., 2009; Shalaby et al., 2013): First, correcting dehydration and electrolyte imbalances through aggressive intravenous fluid resuscitation, as patients often present with significant fluid deficits due to vomiting and third-spacing (Nehra & Goldstein, 2011). Second, administering broad-spectrum intravenous antibiotics to prevent or treat bacterial translocation from ischemic bowel, typically including coverage for gram-negative and anaerobic organisms (Maloney & Pitt, 2014). Third, providing gastric decompression with oro- or nasogastric tubes to reduce gastric distension and minimize aspiration risk (Applegate et al., 2009). Fourth, and most importantly, proceeding to prompt surgical exploration via laparotomy or laparoscopy, as definitive treatment requires surgical intervention (Lampl et al., 2009).

Current surgical guidelines emphasize that delaying surgery for confirmatory tests or prolonged resuscitation is strongly discouraged when clinical suspicion of volvulus is high, as delays in surgical intervention are associated with increased bowel necrosis rates, higher morbidity, and mortality (Nehra & Goldstein, 2011; Shalaby et al., 2013). The principle of "the sun should never set on a volvulus" underscores the urgency of surgical management (Lampl et al., 2009). Both open laparotomy and laparoscopic approaches are viable options, with choice depending on patient stability, surgeon expertise, and institutional resources. Some studies suggest laparoscopic Ladd's procedure may offer benefits including reduced postoperative pain and shorter hospital stay, though open surgery remains standard in unstable patients or when bowel viability is in question (Hagendoorn et al., 2011; Maloney & Pitt, 2014).

In this case, open surgery was performed due to diagnostic uncertainty and clinical instability. Intraoperative exploration revealed midgut malrotation as expected, but also unexpectedly identified multiple colonic and small intestinal polyps, raising immediate suspicion of FAP (Durno et al., 2019). The surgical procedure included Ladd's procedure (division of Ladd's bands, widening of the mesenteric base, counterclockwise detorsion of volvulus, appendectomy, and positioning of the small bowel on the right and colon on the left) and biopsy of representative polyps for histopathological examination (Shalaby et al., 2013). Histopathology was submitted to confirm the diagnosis of adenomatous versus other polyp types and to assess for dysplasia or malignancy; results were pending at the time of this report.

One month post-surgery, the child demonstrated excellent recovery, was physically active, and had regained 4 kg of body weight, indicating resolution of chronic malnutrition and return to normal gastrointestinal function (Durno et al., 2019). However, the incidental finding of FAP necessitates long-term surveillance and management planning. FAP requires timely screening and follow-up according to established guidelines, typically including annual colonoscopy beginning in early adolescence (ages 10-12 years) or earlier if family history suggests aggressive disease, upper endoscopy to screen for duodenal adenomas beginning in late adolescence, and periodic physical examinations to assess for extraintestinal manifestations (Vasen et al., 2008; Syngal et al., 2015).

Surgical planning in FAP patients aims at cancer risk reduction while preserving quality of life. Options include prophylactic colectomy with ileorectal anastomosis (IRA) or proctocolectomy with ileal pouch-anal anastomosis (IPAA), with choice dependent on rectal polyp burden, patient age, family preference, and colorectal cancer risk assessment (Stoffel et al., 2015; Valle et al., 2019). Timing of prophylactic surgery is typically recommended in late

adolescence or early adulthood, though decisions must be individualized based on polyp burden and dysplasia grade (Monahan et al., 2020).

Less invasive approaches may include endoscopic polypectomy for limited numbers of polyps, though this is typically temporizing rather than definitive given the progressive nature of FAP (Latchford & Phillips, 2011). Chemoprevention using nonsteroidal anti-inflammatory drugs (NSAIDs) such as sulindac or celecoxib has been investigated and shows some efficacy in reducing polyp burden, but currently lacks strong evidence for cancer prevention and is not recommended as sole therapy (Giardiello et al., 2014; Calderwood et al., 2020). These agents may be considered as adjunctive therapy in select cases, but definitive surgical management remains the standard of care for cancer prevention in FAP (Syngal et al., 2015; Durno et al., 2019).

Genetic counseling and testing are essential components of FAP management, not only for the affected patient but also for at-risk family members. First-degree relatives have a 50% chance of inheriting the mutation, and identification of at-risk individuals allows for early surveillance and intervention before cancer development (Vasen et al., 2008; Stoffel et al., 2015). Psychosocial support is also important, as diagnosis of a hereditary cancer syndrome has significant implications for quality of life, family planning, and psychological wellbeing (Monahan et al., 2020).

CONCLUSION

This case report describes the rare incidental discovery of Familial Adenomatous Polyposis (FAP) during emergency surgery for a late-diagnosed midgut volvulus caused by intestinal malrotation, highlighting diagnostic challenges due to intermittent symptoms that can obscure chronic conditions. Although the surgical intervention successfully resolved the acute volvulus, the unexpected finding of FAP drastically alters the patient's long-term care, requiring lifelong cancer surveillance and preventive measures. This underscores how a single surgical event can reveal a separate hereditary syndrome with significant implications. Future research should explore possible genetic or developmental links between intestinal malrotation and polyposis syndromes through large multi-center studies, develop standardized diagnostic protocols to minimize delays in identifying anatomical abnormalities in children with chronic abdominal symptoms, and examine the psychosocial impact on families coping with a hereditary cancer diagnosis following an emergency surgical event to improve support services.

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