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Under the Patronage and Honoring of HRH Prince Saud bin Nayef bin Abdulaziz, Governor of the Eastern Province: Saudi Cancer Foundation Successfully Launches "AlSharqia Wardiyah 18"

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Case Report

Tubular Ileal Duplication Associated with Diffuse Polyposis and Metastatic Adenocarcinoma Presenting as Acute Small Bowel Obstruction in an Adult: First Reported Case and Review of the Literature

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KEYWORDS : Tubular ileal duplication, Intestinal polyposis and malignant transformation, intestinal obstruction, Adenocarcinoma, Adult.

ABSTRACT : Tubular duplication of the alimentary tract is rare during adulthood. Most of duplications are cystic in nature. Malignant changes in cystic duplication had been described in literature. We encountered male in his 50,s with no previous significant complaints presented with acute intestinal obstruction. On diagnostic laparoscopy found to have multiple peritoneal, omental deposits with transition zone at narrowed junction of duplicated loop with ileal loop. Resection and primary anastomosis with uneventful postoperative recovery was achieved. Postoperative histopathology examination revealed metastatic adenocarcinoma stage pT4N2M1. Interestingly tubular duplicated segment was blind at proximal end and contained multiple mucosal polyps (more than 100 in number) which were small, sessile and mostly range in size from 0.1 to 0.4 cm in diameter while the largest polyp measured 0.7 cm and was located immediately proximal to tumor. Based on available research we hypothesize that pathogenesis in our patient was driven by the unique anatomy and microenvironment of the tubular duplication segment. A milieu of blind proximal end of the duplicated loop combined with heterotropic gastric mucosa, delayed transit times, accumulation of abnormal non intestinal secretions and consequent bacterial overgrowth lead to state of chronic persistent mucosal inflammation. This combined insult resulted in mucosal hyperplasia forming polyps and subsequent dysplastic changes that ultimately transitioned into adenocarcinoma suggesting adenoma carcinoma sequence. Whenever encountered, complete surgical resection of the duplicated segment should be undertaken due to high probability of dysplasia and malignancy transformation of mucosal lesions as highlighted in this case report.

Introduction

Alimentary tract duplications are rare congenital anomalies, accounting for approximately 0.1% to 0.3% of all congenital malformations. Roughly 80% of these cases are diagnosed in children under two years of age, making an adult presentation exceptionally rare. Although duplications can occur anywhere along the gastrointestinal tract, the ileum represents the most common site. (1)

While malignancy arising within an enteric duplication cyst has been documented as a rare complication in adults, a tubular duplication of the small bowel presenting with concurrent multiple polyposis and adenocarcinoma has not yet been reported in the literature. Notably, the histopathological identification of multiple polyps composed of hyperplastic heterotropic gastric mucosa with focal high grade dysplasia in our case provides strong evidence supporting the adenocarcinoma – carcinoma sequence as the oncogenic mechanism in small intestinal duplication loop adenocarcinoma supported by mutations in KRAS: c.35G>A, p (G12D) and TP53:c.742C>T, p. (248W) genes and amplification in MYC gene using molecular assays.

Case Report

An African male in his 50s with no known comorbidities presented to the emergency department with a 10–day history of progressive abdominal pain and distension. The pain was described as sharp, colicky, initially localized to the upper abdomen, and subsequently becoming generalized. He reported associated episodes of nausea and projectile, bilious vomiting over the preceding 2 days. The patient also noted a significant unintentional weight

loss of approximately 10 kg over the past 4 to 6 weeks, accompanied by a several–month history of anorexia and generalized malaise for which he had not sought medical attention. He reported no recent changes in bowel habits or hematochezia.

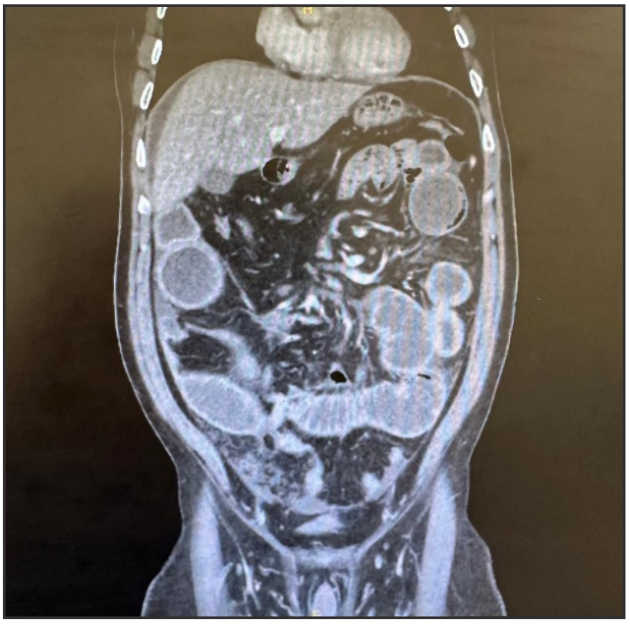
On physical examination, the patient had stable vital signs. The abdomen was distended with generalized tenderness, but without guarding or rigidity. Hyperactive bowel sounds were noted, suggestive of a mechanical bowel obstruction.

Initial laboratory evaluations revealed a total white blood cell count of 7.8 × 10^9/L, hemoglobin of 13.9 g/dL, and marked thrombocytosis with a platelet count of 605 × 10^9/L. Serum creatinine was 73 µmol/L, potassium was 3.7 mmol/L, bicarbonate was 29 mmol/L, and serum lactate was normal at 0.9 mmol/L.

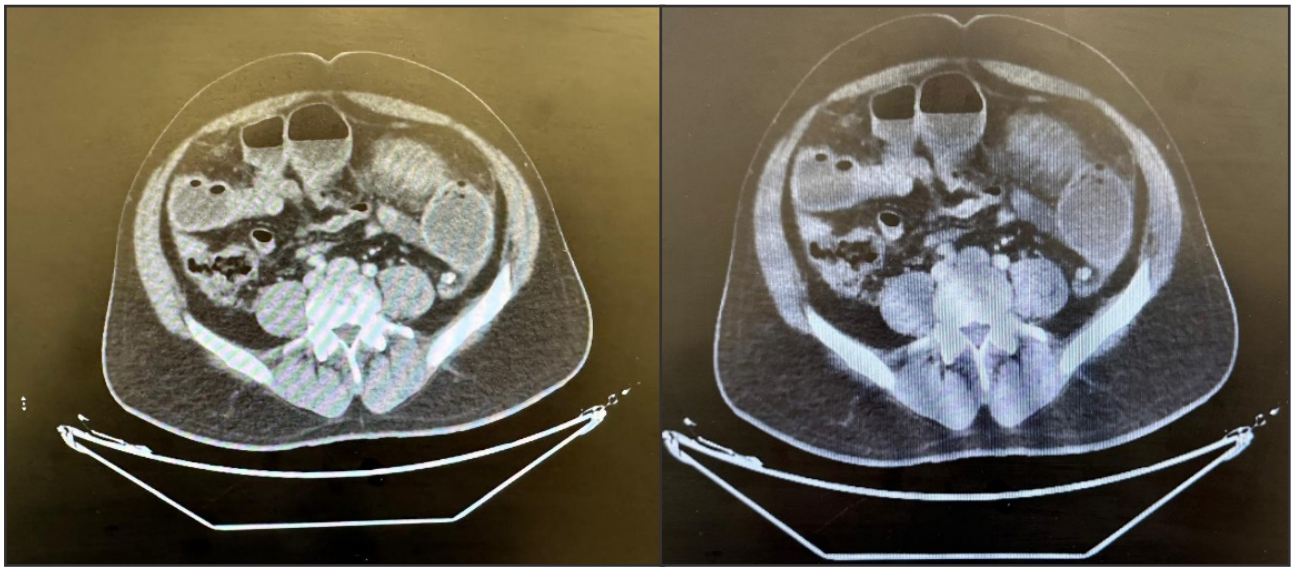
Abdominal radiography demonstrated multiple air–fluid levels and dilated small bowel loops. A computed tomography (tomography) scan of the abdomen and pelvis revealed multi–lobed, dilated small bowel loops extending to the mid–ileum, reaching a maximum diameter of 5 cm. A distinct transition point with an abrupt caliber change and distal collapsed loops was identified. The scan also noted a few ill–defined, focal areas of increased attenuation within the omental fat and mesentery, interpreted as inflammatory changes versus mesenteric nodules. No obstructing mass lesions, pneumoperitoneum, or solid organ abnormalities were visualized. The primary radiologic impression was a high–grade small bowel obstruction with an abrupt transition point, with inflammatory bowel disease considered in the differential diagnosis.

Given the high–grade mechanical intestinal obstruction in a virgin abdomen, the patient was taken for an emergency diagnostic laparoscopy. Intraoperative findings revealed ascites along with multiple omental and peritoneal nodules distributed across the subhepatic, pelvic, and paracolic gutters. Marked dilation of the small intestine was observed, with a transition zone in the mid–ileum at the distal site of a 75–cm small intestinal duplication segment. Segmental resection of the involved bowel and a primary end–to–end anastomosis were performed. The patient's postoperative recovery was uneventful, and he was subsequently referred to the oncology center for evaluation regarding adjuvant chemotherapy versus hyperthermic intraperitoneal chemotherapy (HIPEC).

Gross examination of the resected specimen revealed a parent loop of small bowel measuring 74 × 6 cm, sharing a common mesenteric fat pad with an attached,



CT IMAGES.1



2



OPERATIVE IMAGE..3



MACROSCOPIC IMAGES NO.4 – 5

duplicated small bowel loop measuring 60 × 5 cm. The duplicated loop was blindly ending at its proximal margin and communicated distally with the parent loop. The outer surface of junction demonstrated relative narrowing and a 2–cm long constriction margin.

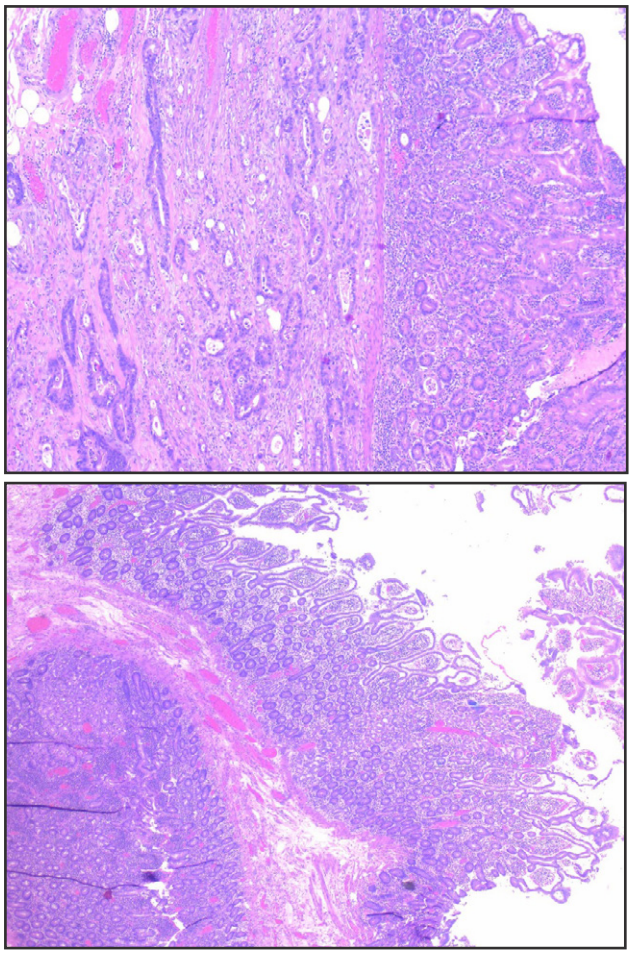
Upon opening the specimen along the anti–mesenteric border, a firm, infiltrative, grey–white to tan tumor measuring 2.5 × 2.5 × 2.0 cm was identified within the constricted region, involving both the parent and duplicated loops. The mucosa of the parent loop was otherwise unremarkable. However, the mucosa of the duplicated loop revealed multiple mucosal polyps (more than 100 in number) which were small, sessile that mostly range in size from 0.1 to 0.4 cm in diameter; the largest polyp measured 0.7 cm and was located immediately proximal to the tumor **.** Regional lymph nodes and several irregular tumor–like deposits were isolated from the peri–intestinal mesenteric fat.

Microscopic evaluation on routine Hematoxylin & Eosin stain (H&E) demonstrated a moderately–to–poorly differentiated invasive adenocarcinoma arising within and involving both the parent and duplicated small bowel loops. Proximal and distal resection margins were free of carcinoma. The tumor extended through the muscularis propria to involve the serosal surface. Lymphovascular invasion was confirmed, with metastasis identified in 3 out of 17 isolated lymph nodes alongside separate mesenteric tumor deposits. Peritoneal and omental nodules were histologically confirmed as metastatic adenocarcinoma. pTNM stage (AJCC 8th edition): pT4N2M1 .

On immunohistochemistry, the tumor showed variable expression for Keratin 7, Keratin 19, and CDX2 (that likely indicates embryonic/stem cell nature of mucosa in which it arose). Molecular studies done later on tumor tissue blocks showed mutations in KRAS: c.35G>A, p (G12D) and TP53:c.742C>T, p. (248W) genes and amplification in MYC gene while the tumor was micro–satellite stable (MS–S).

Discussion

Enteric duplications are a group of rare congenital lesions characterized by a smooth muscle wall and an enteric mucosal lining, typically located along the mesenteric border of the intestines and sharing a common blood supply with the native bowel [1]. Enteric duplications can occur anywhere along the gastrointestinal tract, though they are most frequently located in the ileocecal region.



MICROSCOPIC IMAGES NO.6 – 7

Several theories regarding their etiology have been postulated, including abnormal recanalization following the epithelial stage of embryonic bowel development [2]. Other theories propose the persistence of embryonic diverticula or aborted gemini [3]. However, the most widely accepted mechanism is the intrauterine vascular accident theory [4]. Despite these hypotheses, no single theory completely explains all known variations of duplications[5]. The majority of enteric duplications are isolated and cystic in nature. Although long tubular duplications of the small bowel are a well–recognized entity, they remain highly rare[6].The vast majority of enteric duplications are diagnosed in the pediatric population, they are frequently associated with other congenital intestinal, genitourinary, or spinal malformations [7]. Approximately 80% of patients manifest symptoms before two years of age, while the remainder remain asymptomatic and undiagnosed until adulthood. Ultrasonography, barium studies, computed tomography (CT) scans, small intestine contrast ultrasonography (SICUS), magnetic resonance imaging (MRI), and technetium–99m scanning can assist in the preoperative assessment of enteric duplications [8]

Because of their rare occurrence and non–specific presentation, preoperative diagnosis in adult patients remains exceptionally challenging. In series of twenty–seven cases of ileal duplications in adults had been described in the world literature, with a correct preoperative diagnosis achieved in only a single case. [9] In adults, enteric duplications are most commonly diagnosed only after becoming symptomatic due to complications such as volvulus, intussusception, obstruction, perforation, or hemorrhage.

However, in our case, the emergency presentation with acute intestinal obstruction and severely dilated bowel loops significantly hindered preoperative diagnostic assessment.

Enteric duplications have been documented to harbor heterotopic mucosa, typically of gastric or pancreatic origin [10]. This heterotopic mucosa frequently serves as a nidus for bleeding and malignant degeneration. Carcinomas arising in duplication cysts are extremely rare and only few cases have been reported in literature including carcinoid tumors, squamous call carcinomas and commonly adenocarcinomas. [11, 12]. Malignant change in small bowel duplications is described most frequently.[11,13,14,15,16] followed by colonic [17,18] and rectal duplications[19]. Presence of multiple hyperplastic polyposis has been described but with high–grade dysplasia and transformation into adenocarcinoma has not been previously reported [20].

Malignancies of the small intestine remain uncommon, despite the small bowel constituting the longest mucosal segment of the gastrointestinal tract. Carcinoma of the small intestine is substantially less prevalent than esophageal, gastric, or colorectal cancers. Small bowel adenocarcinoma (SBA) presents at a median age of 62 years (the sixth decade of life) and exhibits a male predominance of 53–62% [21]. Although SBA can arise sporadically, it is frequently found in patients with specific genetic syndromes or those with preexisting gastrointestinal inflammatory and immunological disorders.

Several physiological mechanisms have been proposed to explain the relative resistance of the small bowel to carcinogenesis. These include the aggregation of lymphoid tissue with abundant immunoglobulin A (IgA) production, a high concentration of protective digestive enzymes, rapid epithelial cell turnover, and swift transit times that minimize mucosal exposure to bacteria and dietary carcinogens.

Consequently, the precise pathogenesis of SBA is not fully understood. However, several critical molecular pathways leading to invasive SBA have been identified. These include *APC* gene mutations causing protein dysfunction and subsequent beta–catenin over activation in up to 40%

of cases, mismatch repair (MMR) protein dysregulation in 7–35% of cases, *STK11* mutations in Peutz–Jeghers syndrome, and *SMAD4* or *BMPR1A* mutations in juvenile polyposis syndrome. By comparing proliferation markers across normal mucosa, adenomatous polyps, and invasive adenocarcinoma, investigators have postulated that the classic adenoma–to–carcinoma sequence applies to SBA. Patients with underlying genetic syndromes are likely further along this adenoma–to–carcinoma continuum [22, 23].

Chronic inflammation also appears to play a critical role in small bowel carcinogenesis. This hypothesis explains why specific gastrointestinal disorders and environmental risk factors predispose patients to malignancy. In inflammatory bowel disease, particularly crohn's disease, an abnormal immune response to intestinal micro flora and environmental exposures triggers chronic trans mural inflammation [24]. This inflammatory state releases pro–inflammatory cytokines that promote cell proliferation while simultaneously damaging the epithelial barrier. This structural damage can directly induce dysplasia and allow luminal carcinogens to pass through disrupted intercellular junctions into the cellular environment. Dysplastic cells are subsequently thought to accumulate somatic mutations in *KRAS*, *p53*, and MMR proteins, which are established drivers in the adenoma–to–carcinoma sequence [25, 26].

Similarly, in celiac disease, a chronic inflammatory process drives the development of dysplasia from flattened mucosa as a result of repetitive exposure to dietary gluten [27]. Consequently, patients with celiac disease exhibit higher odds of developing extraintestinal lymphomas, pancreatobiliary cancers, and esophageal adenocarcinomas [27].

Due to nonspecific symptoms these tumors are commonly diagnosed at an advanced stage with metastatic disease. (11,16,28). If malignant change is found in duplicated bowel loops, the high rates of lymph node metastaseses should be considered(11). The mode of metastasis is similar to that of primary small bowel cancer(11, 29).Curative resections could hardly be performed (11, 28).Thus the prognosis is generally poor once malignant change has occurred.

To the best of our knowledge, our case represents the first reported instance of a tubular small intestinal duplication presenting concurrently with adenocarcinoma, intestinal obstruction, and diffuse polyposis. Based on these available research data, we hypothesize that the pathogenesis in our patient was driven by the unique anatomy and microenvironment of the tubular duplication segment. The blind proximal end, combined with ectopic gastric mucosa, delayed transit times, and the accumulation of secretions, created an environment ripe for bacterial overgrowth. This induced a state of chronic persistent mucosal inflammation that resulted in mucosal hyperplasia forming polyps and subsequent dysplastic changes that ultimately transitioned into adenocarcinoma.In our case clinical presentation was delayed further until the adenocarcinoma within the duplicated loop extended locally into the adjacent native bowel loop, becoming large enough to cause mechanical obstruction. The advanced nature of the disease was further evidenced by the presence of multiple peritoneal and omental nodules.In this case, primary cytoreductive surgery or hyperthermic intraperitoneal chemotherapy (HIPEC) was not initially performed. The immediate priority of emergency clinical management was the relief of the mechanical intestinal obstruction and stabilization of the patient rather than achieving an oncological cure. Following recovery from the emergency surgery and subsequent histopathological confirmation, the patient was referred to a specialized oncological center for further evaluation regarding HIPEC and adjuvant systemic chemotherapy.

Conclusion

As a rare entity and nonspecific symptoms, enteric duplication is rarely considered in the differential diagnosis of adult acute abdomen. Preoperative diagnosis remains exceptionally difficult due to the rarity of this pathology. We believe it is highly valuable to report new clinical presentations to review the most relevant diagnostic and therapeutic aspects of this rare disease. Chronic stasis within a tubular duplication loop may create a microenvironment of prolonged inflammation, bacterial overgrowth leading to mucosal injury and polyposis. Activation of adenocarcinoma carcinoma sequence ultimately leads to adenocarcinoma. Pathological findings in our case report provide strong support hypothesis of adenocarcinoma carcinoma sequence as small bowel adenocarcinoma etiology. Whenever encountered, complete surgical resection of the duplicated segment should be undertaken due to the high propensity for malignant transformation, as highlighted in this case

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References.

1. Ladd WE, Gross RE. Surgical treatment of duplications of the alimentary tract: enterogenous cysts, enteric cysts, or ileum duplex. Surg Gynecol Obstet. 1940;70:295–307.

2. Bremer JL. Diverticula and duplications of the intestinal tract. Arch Pathol. 1944; 38:132–140.
3. Letelier AM, Barría CM, Beltrán MA, Monacó M. Duplicación intestinal: diagnóstico y tratamiento de una condición inusual. Rev Chil Cir. 2009; 61(2):171–175.
4. Favara BE. Enteric duplications: thirty–seven cases: a vascular theory of pathogenesis. Am J Dis Child. 1971; 122(6):501–506.
5. Orr MM, Edwards AJ. Neoplastic changes in duplication of the alimentary tract. Br J Surg. 1975;62(4):269–274.
6. Wrenn EL Jr, Hollabaugh RS. Alimentary tract duplications. In: Ashcraft KW, editor. Pediatric surgery. 3rd ed. Philadelphia: W.B. Saunders; 2000. p. 527–535
7. Shah KR, Joshi A. Complete genitourinary and colonic duplication: a rare presentation in an adult patient. J Ultrasound Med. 2006; 25(3):407–411. doi:10.7863/jum.2006.25.3.407
8. Hocking M, Young DG. Duplication of the alimentary tract. Br J Surg. 1981; 68(2):92–96.
9. Fiorani c., Scaramuzzo et al.Intestinal duplication in adulthood: A rare entity, difficult to diagnose. World journal of gastrointestinal surgery, 3(8), 128–130.
10. Olajide AR, Yisau A, Abdulraseed NA, Kashim IO, Olaniyi AJ, Morohunfade AO. Gastrointestinal duplications: experience in seven children and review of the literature. Saudi J Gastroenterol. 2010; 16(2):105–109. doi:10.1038/1319–3767.61237
11. Kusunoki N, Fukuda Y, Tomita T, Shinonaga S, Tanaka T, Shirasaka C, et al. Adenocarcinoma arising in a tubular duplication of the jejunum. J Gastroenterol. 2003; 38(8):781–785. doi:10.1007/s00535–002–1146–8
12. Kuraoka K, Nakayama H, Kagawa T, Ichikawa T, Yasui W. Adenocarcinoma arising from a gastric duplication cyst with invasion to the stomach: a case report with literature review. J Clin Pathol. 2004; 57(4):428–431. doi:10.1136/jcp.2003.013953
13. Beltrán MA, Barría CM, Contreras MA, Wilson CS, Cruces KS. Adenocarcinoma en una duplicación intestinal del íleon: reporte de un caso. Rev Med Chile. 2009;137(10):1341–1345. doi:10.4067/S0034–98872009001000009
14. Tew K, Ng KK, Lau KY. Adenocarcinoma in an ileal duplication cyst: ultrasound and computed tomography findings. Australas Radiol. 2000; 44(2):228–231. doi:10.1046/j.1440–1673.2000.00799.
15. Radich GA, Radich S, Devitt P, Thompson SK. Papillary adenocarcinoma in a small–bowel duplication in a pregnant woman. AJR Am J Roentgenol. 2006; 186(3):895–897. doi:10.2214/AJR.04.1843
16. Fletcher DJ, Sali A. Metastatic adenocarcinoma arising from a small bowel duplication cyst. Eur J Surg Oncol. 2002; 28(1):93–94. doi:10.1053/ejso.2001.1214
17. Lee J, Jeon YH, Kim MS, Choi GH, Kim MH, Joo KH, et al. Papillary adenocarcinoma arising in a duplication of the caecum. Abdom Imaging. 2008; 33(6):601–603. doi:10.1007/s00261–007–9351–4
18. Inoue Y, Nakamura H. Adenocarcinoma arising in colonic duplication cyst with calcification: CT findings of two cases. Abdom Imaging. 1998; 23(2):135–137. doi:10.1007/s002619900305
19. Downing R, Thompson H, Alexander–Williams J. Adenocarcinoma arising in a duplication of the rectum. Br J Surg. 1978; 65(8):572–574. doi:10.1002/bjs.1800650817
20. Bade R, Mahajan JK, Solanki S, Chatterjee D. Large jejunoileal duplication cyst with hyperplastic polyps–a case report with review of literature. Indian Pediatr Case Rep. 2025; 5(2):68–74. doi:10.4103/ipcares.ipcares_164_24
21. Zouaini Y, Aparicio T, Laurent–Puig P. Advanced small bowel adenocarcinoma: molecular characteristics and therapeutic perspectives. Clin Res Hepatol Gastroenterol. 2016; 40(2):154–160. doi:10.1016/j.clinre.2015.09.006
22. Sellner F. Investigations on the significance of the adenoma–carcinoma sequence in the small bowel. Cancer. 1990; 66(4):702–715. doi:10.1002/1097–0142(19900815)66:4<702::aid–cncr2820660418>3.0.co;2–z
23. Nakano Y, Chiba T, Hasegawa H, Ooka Y, Maruoka D, Shinozaki S, et al. Adenocarcinoma suggesting an adenoma–carcinoma sequence in the small bowel: a case report. Oncol Lett. 2014; 8(2):633–636. doi:10.3892/ol.2014.2215
24. Wiegand N, Menges M, Zeitz M, Tcholoste S, Stallmach A, Herrlinger KR. Prognosis of small bowel adenocarcinoma in Crohn's disease compares favorably with de novo small bowel adenocarcinoma. Colorectal Dis. 2017; 19(3):446–455. doi:10.1111/codi.13524
25. Hickey WF, Corson JM. Carcinoma arising in duplicated colon: case report. Cancer. 1967; 20(4):606–611. doi:10.1002/1097–0142(1967)20:4<606::aid–cncr2820200421>3.0.co;2–r
26. Svrcek M, Piton G, Cosnes J, Beaugerie L, Cavard C, See H, et al. Small bowel adenocarcinomas complicating Crohn's disease are associated with dysplasia: a pathological and molecular study. Inflamm Bowel Dis. 2014; 20(9):1584–1592. doi:10.1097/MIB.0000000000000114
27. Green PH, Cellier C. Celiac disease. N Engl J Med. 2007; 357(17):1731–1743. doi:10.1056/NEJMra071600
28. Devos B, Schreurs L, Van de Stadt J, de Graef J. Adenocarcinoma optredend in een cystische duplicatie van het ileum [Adenocarcinoma occurring in a cystic duplication of the ileum]. Acta Chir Belg. 1987;87(4):235–238.
29. Kim TH, Kim JK, Ryu H, Lee JS, Kim S, Myung SJ, et al. Papillary adenocarcinoma arising in a tubular duplication of the jejunum. Br J Radiol. 2010; 83(988):e61–e64. doi:10.1259/bjr/25134769