Volume 2(4), pages 413-418, April 2026
DOI: 10.65221/0194
eISSN: 3115-5898

https://reports.afjur.com

AFJUR
African Journals, Universities and Research

African Research Reports

Case Report

Too late to thrive: Delayed diagnosis of congenital intestinal obstruction in infants presenting with failure to thrive, a case series

Emem Imo Akpanudo[1,2]*, Monday Akpabio Ituen[1,2], Iniofon Clement Akpaette[2], Eti-Inyene Michael Emmanuel[2] and Aniekpeno Effiong Eyo[2]

[1]Pediatric Surgery Unit, Department of Surgery, Faculty of Clinical Sciences, University of Uyo, Uyo, Nigeria.
[2]Pediatric Surgery Unit, Department of Surgery, University of Uyo Teaching Hospital, Uyo, Nigeria.

Received 13 February, 2026; Accepted 20 April, 2026

Failure to thrive in infancy refers to inadequate weight gain or growth and may result from a range of nutritional, medical, or psychosocial conditions. Although intestinal obstruction is a recognised neonatal surgical emergency, it is rarely considered a cause of failure to thrive beyond the newborn period. Delayed or incorrect diagnosis in such cases may result in prolonged malnutrition and increased perioperative risk. Three infants presented with failure to thrive secondary to delayed or missed diagnosis of congenital intestinal obstruction. The cases included a 17-week-old infant with ascending colonic stenosis, an 8-month-old infant with a fenestrated Type I ileal atresia, and a 13-month-old child with partial duodenal obstruction due to malrotation with Ladd's bands. All patients had persistent gastrointestinal symptoms from early infancy and presented with severe growth failure. These cases highlight the potential for congenital intestinal obstruction to present atypically with feeding intolerance and poor weight gain. A high index of suspicion and early referral for surgical evaluation are essential to improve outcomes in affected infants.

Key words: Congenital abnormalities; delayed diagnosis; intestinal atresia, intestinal obstruction; failure to thrive; malnutrition.

INTRODUCTION

Failure to thrive (FTT), also referred to as weight faltering or growth faltering, is defined as weight-for-age below the fifth percentile on standardised growth charts, a drop-in weight percentile of more than two major centile lines, or less than 80% of the median weight-for-height/length ratio. The World Health Organization describes growth faltering as a fall in weight-for-age z-score of ≥1.0 (Franceschi et al., 2021; Cooke et al., 2023; Smith et al., 2025). It is a descriptive term rather than a diagnosis, reflecting underlying nutritional, medical, or psychosocial conditions that impair normal growth and development. Although most commonly seen in children aged 1 to 2 years, it can occur at any time during childhood.

Persistent malnutrition in children with FTT is associated with several short- and long-term complications, including impaired immune function, delayed neurodevelopment, stunting, and increased susceptibility to infection and hospitalisation (Franceschi et al, 2021; Emmanuel et al,

*Corresponding author. E-mail: ememakpanudoh@uniuyo.edu.ng.

2023). In infants, the consequences may be more severe due to limited physiological reserves, high metabolic demands, and the rapid pace of growth in early life (De Sanctis et al, 2021). Early identification and treatment of the underlying cause are essential to reversing growth failure and preventing long-term sequelae. While FTT is most often attributed to nutritional or medical causes, surgically correctable conditions should also be considered, particularly in infants with persistent gastrointestinal symptoms. Intestinal obstruction is a well-recognised neonatal surgical emergency but is not commonly considered a cause of FTT beyond the newborn period (Seltz, 2008). In some cases, congenital anomalies such as intestinal atresia or malrotation may present with chronic gastrointestinal symptoms, including recurrent vomiting and feeding intolerance. This atypical presentation may lead to delayed diagnosis and prolonged growth failure (Sharma et al., 2024). This case series describes three infants who presented with FTT secondary to missed or delayed diagnosis of congenital intestinal obstruction, highlighting the diagnostic challenges, nutritional consequences, and surgical outcomes.

Case 1

A 17-week-old female infant was admitted with failure to gain weight, recurrent abdominal swelling, non-bilious vomiting, and constipation. These symptoms had been recurrent since the first week of life. She was the second of a set of monozygotic twins born at 35 weeks gestation via spontaneous vaginal delivery. There was no history of maternal illness or polyhydramnios. Her birth weight was 2.05 kg, and she passed meconium within 24 h of life. She had been previously managed in a peripheral hospital, where her symptoms were attributed to “excess gas” from poor feeding practices. Each time, she was kept nil per os and treated with bisacodyl suppositories. Despite regular feeding at home, she continued to lose weight. On examination, she appeared wasted, pale, and dehydrated. Her weight was 1.0 kg and length 50 cm, both well below the 5th percentile for corrected age and consistent with severe underweight (weight-for-age z-score < -3 SD based on World Health Organisation (WHO) growth standards). Her abdomen was distended, with visible peristalsis, tympanitic percussion notes, and hyperactive bowel sounds. Laboratory investigations showed a haemoglobin concentration of 8.7 g/dL and mild hyponatraemia (serum sodium: 132 mmol/L). Other electrolytes were within normal limits. HIV serology was negative. A plain abdominal radiograph showed markedly dilated small bowel loops throughout the abdomen, with rectal gas (Figure 1a). Following resuscitation, an exploratory laparotomy was performed. Intraoperative findings included a 2 cm segment of stenosis in the ascending colon approximately 8 cm from the ileocaecal valve, with a dilated caecum and small intestine (Figure 1b). A right hemicolectomy was carried out. However, due to her severe malnutrition, a primary anastomosis was deferred, and an end ileostomy was fashioned. Histopathological examination of the resected specimen demonstrated the presence of ganglion cells, excluding Hirschsprung’s disease. She commenced graded enteral feeds on the second postoperative day and was discharged when her weight had appreciated to 2.0 kg, with plans for continued outpatient nutritional rehabilitation with the paediatric gastroenterologists and nutritionists. Ileostomy reversal was scheduled once she attained a weight corresponding to at least the 15th percentile for her corrected age.

Case 2

An 8-month-old male infant presented to the paediatric emergency unit with a 4-day history of constipation, abdominal swelling, and bilious vomiting. He was born at 38 weeks by spontaneous vaginal delivery in a peripheral hospital. Antenatal care was routine and uneventful. Birth weight was 3.0 kg, and his length was 50 cm. He passed meconium on the second day of life and by the end of the first week, he began to develop progressive abdominal distension, constipation, and vomiting. An abdominal radiograph performed at that time reportedly showed dilated small bowel loops with gas in the rectum. A contrast enema revealed a contrast-filled colon with a narrowed rectum and rectosigmoid segment. A presumptive diagnosis of Hirschsprung’s disease was made at the peripheral hospital, and he was managed with saline enemas. He was re-admitted on four separate occasions with similar symptoms and treated conservatively. The mother was instructed to continue enemas and bisacodyl suppositories at home. Despite these measures, his symptoms persisted. There was no fever or features of enterocolitis. On examination, he appeared chronically ill-looking and wasted. He was afebrile, pale, anicteric, and moderately dehydrated. He weighed 4.8 kg, well below the 5th percentile for age (weight-for-age z-score < -3 SD). His abdomen was grossly distended, but there were no signs of peritonitis, no palpable masses or organomegaly. Bowel sounds were hyperactive. Digital rectal examination revealed an empty rectum with good sphincter tone. Other systems were unremarkable. Laboratory findings included haemoglobin of 9.3 g/dL, mild hyponatraemia (serum sodium of 133 mmol/L), and hypokalaemia (serum potassium of 3.4 mmol/L). He was resuscitated and scheduled for exploratory laparotomy with planned colostomy and colonic biopsies. Intraoperatively, a fenestrated Type I ileal atresia (mucosal web with a central opening) was found approximately 8 cm proximal to the ileocaecal valve (Figure 2). The atretic segment was excised, but due to the infant’s malnourished state, a temporary end ileostomy was fashioned. No additional biopsies were taken. Postoperative recovery was uneventful. Ileostomy closure and ileocolic anastomosis

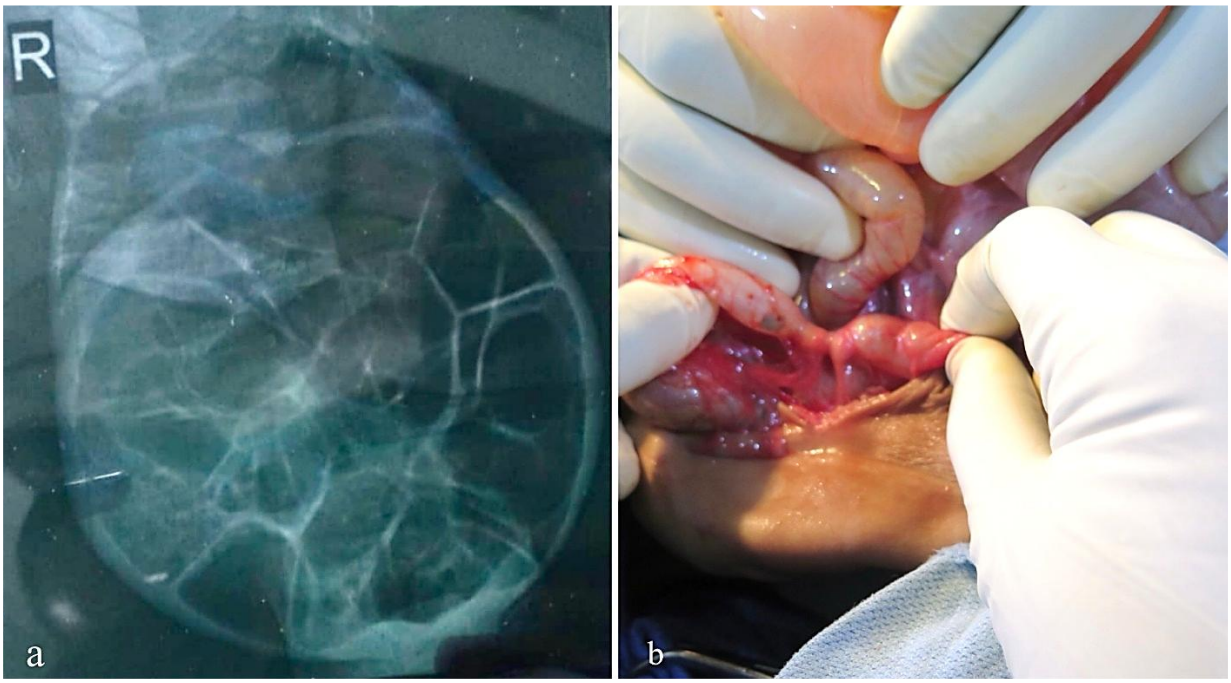

Figure 1. (a) Plain abdominal radiograph of showing markedly dilated loops of small bowel occupying the entire abdominal cavity. (b) Intraoperative findings demonstrating stenosis of the ascending colon.

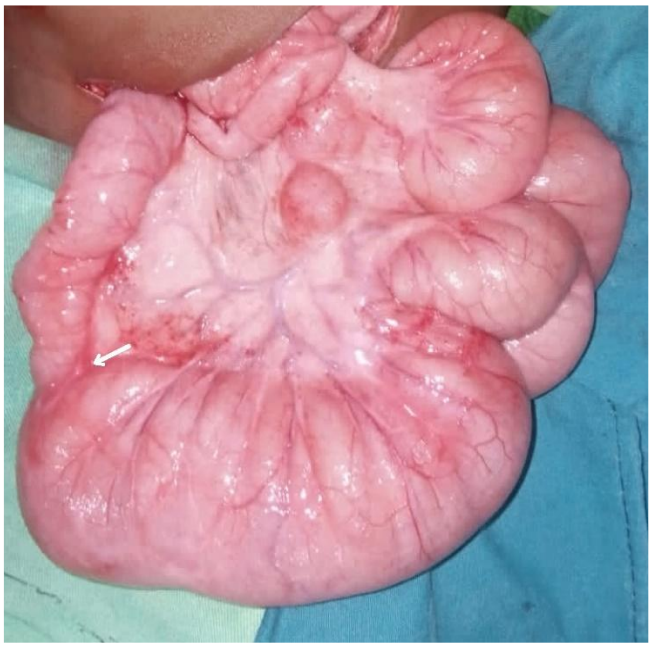

Figure 2. Intraoperative photograph showing a bulbous, dilated ileal loop with a narrowed distal segment. The white arrow indicates the site of atresia, which was confirmed intraoperatively as a fenestrated Type I ileal atresia.

were performed 14 weeks later, when he had gained weight to 9 kg. He remained well on follow-up.

Case 3

A 13-month-old male was brought to the outpatient clinic with a history of recurrent vomiting and feeding intolerance from birth, which had worsened over the preceding two weeks. The vomiting was initially non-bilious but later became bilious, occurring up to six to eight times daily. It was neither copious, projectile, nor bloodstained. The mother also noted intermittent upper abdominal swelling after feeds. There was no associated constipation, but growth had been poor since birth. The child had been repeatedly managed conservatively at a private hospital with nil per os, intravenous fluids, and antibiotics, with discharge after transient improvement on each occasion. He was delivered at term via spontaneous vaginal delivery, with a birth weight of 3.2 kg. There was no history of delayed passage of meconium. He was predominantly breastfed, with complementary infant formula and cereals introduced at five months. Feed volumes varied due to poor tolerance. At presentation, he was cachectic, weighing 3.0 kg and measuring 56 cm in length, both below the 5th percentile for age (weight-for-age z-score < -3 SD). He was afebrile, with unremarkable cardiorespiratory findings. His abdomen was scaphoid and digital rectal examination was normal. Laboratory investigations revealed a haematocrit of 31%, hyponatraemia (serum sodium of 131 mmol/L), hypoalbuminaemia (serum albumin of 3.0 g/dL) and hypokalaemia (serum potassium of 3.0 mmol/L). A barium meal showed a distended stomach and proximal

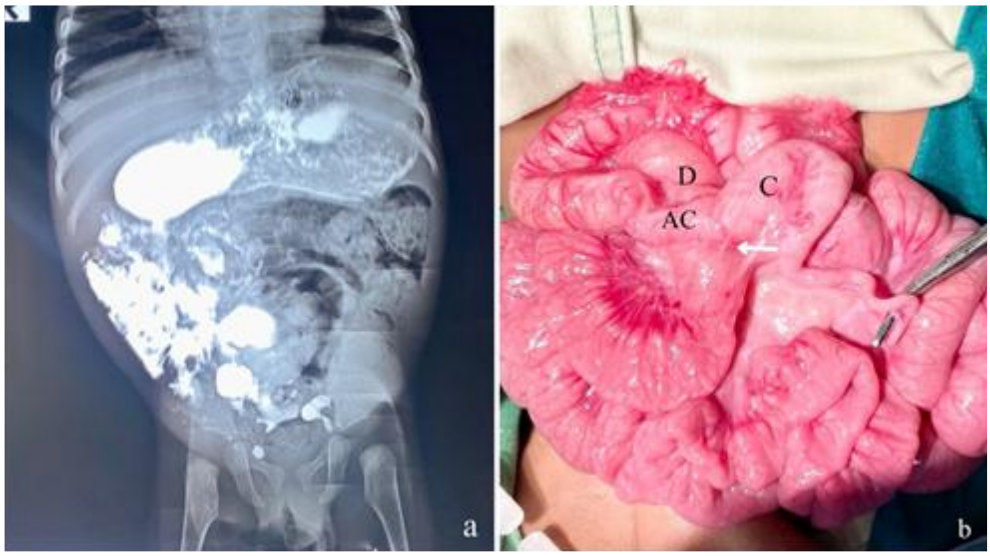

Figure 3. (a) Barium meal showing a distended stomach and duodenum with contrast passing distally, suggestive of partial duodenal obstruction. (b) Intraoperative findings demonstrating abnormal intestinal positioning. The duodenum (D) is compressed by the ascending colon (AC) and Ladd's bands (white arrow) extending from the caecum (C).

duodenum with narrowing of the second and third parts, as seen in Figure 3a. Contrast was seen passing into the distal bowel, suggestive of a partial duodenal obstruction. He was resuscitated and optimised for surgery. At laparotomy, the caecum and small bowel were found in the left upper quadrant. The duodenum was compressed by Ladd's bands extending from the caecum, as well as by the malpositioned ascending colon, as shown in Figure 3b. The mesenteric base was narrowed, but there was no bowel ischaemia. A Ladd's procedure was performed. He commenced feeds within 48 h, which he tolerated. However, on the sixth postoperative day, he became dyspnoeic and febrile. His temperature was 38.9°C, pulse rate 128 beats per minute, respiratory rate 56 cycles per minute, and oxygen saturation was 68% in room air and 90% with intranasal oxygen. Chest examination revealed intercostal recessions, wheezing, and widespread coarse crepitations. A diagnosis of aspiration pneumonitis was made, and despite supportive care, he died 24 h later.

DISCUSSION

This case series highlights the consequences of delayed or missed diagnosis of intestinal obstruction in infancy, demonstrating a pattern of diagnostic dilemma, prolonged undernutrition, and late surgical intervention. Failure to thrive in infancy, although a common clinical concern in paediatrics, is not a typical presentation of congenital intestinal obstruction, which is usually diagnosed and treated during the neonatal period (Seltz, 2008; Sharma et al., 2024). All three children in this report presented beyond the neonatal period, and this pattern reflects both atypical clinical presentation and gaps in clinical suspicion. As noted by Ssentongo et al. (2018), the intermittent and apparently self-limiting nature of symptoms may contribute to diagnostic oversight; however, this also highlights the need for consistent diagnostic follow-through when symptoms persist. The differential diagnoses of FTT are broad, and include gastro-oesophageal reflux disease, cow's milk protein allergy, metabolic and endocrine disorders, and non-organic causes such as inadequate milk supply or poor feeding practices (Smith et al., 2023). In our cases, these possibilities were considered but were excluded during surgical evaluation based on the pattern of gastrointestinal symptoms and imaging findings. Gastro-oesophageal reflux disease and cow's milk protein allergy typically present with non-bilious vomiting, regurgitation, feeding intolerance, and poor weight gain (Franceschi et al., 2021). In contrast, our patients presented with symptoms characteristic of intestinal obstruction, including bilious vomiting, abdominal distension, and constipation. The absence of atopic features and the lack of response to conservative or dietary measures further argued against an allergic or functional aetiology. In addition, the presence of dilated bowel loops on plain radiography in each case supported a surgically correctable obstructive cause.

All three children demonstrated severe growth failure, with anthropometric indices far below expected values for age, reflecting chronic undernutrition. In partial intestinal obstruction, nutritional compromise results from reduced intake due to vomiting and anorexia, as well as ongoing fluid and electrolyte losses (Krasaelap et al., 2019), and

Table 1. Summary of published reports of delayed diagnosis of congenital intestinal obstruction beyond the neonatal period.

Author/Year	*Age at diagnosis*	*Delay from symptom onset*	*Initial diagnosis*	*Final diagnosis/Anomaly*	*Surgery performed*	*Outcome*
Sarkar et al. (2011)	2 years	Since 4-5 weeks of age	No clear organic diagnosis.	Duodenal web with central aperture	Duodenotomy with excision of web	Uneventful recovery, weight gain
Ekenze et al. (2019) (Case 1)	11 weeks	Since neonatal period	Chronic constipation, inconclusive investigations	Solitary ascending colonic stenosis	Right hemicolectomy with ileo-transverse anastomosis	Uneventful recovery, weight gain
Ekenze et al. (2019) (Case 2)	9 weeks	7 weeks	Suspected Colonic stenosis	Multiple colonic stenoses	Resection of stenotic segments with ileo-sigmoid anastomosis	Uneventful recovery, weight gain
Jumbi and Mwika (2020)	1 year	Since neonatal period	Hirschsprung's disease	Colonic web/colonic stenosis at the splenic flexure	Resection of stenotic area with colostomy; delayed colostomy reversal	Uneventful recovery, weight gain, normal stooling
Lamichhane et al. (2020)	5 years	Since 45 days of life	Suspected Pyloric stenosis	Malrotation with Ladd's bands	Ladd's procedure and appendicectomy	Uneventful recovery; weight gain
Dahal et al. (2024)	2 years	Since first week of life	Hirschsprung's disease	Sigmoid colonic stenosis	Resection of stenotic segment with colostomy; delayed anastomosis	Uneventful recovery; weight gain
Garcia et al. (2024)	16 weeks	From 2 weeks of age	Gastrointestinal infection, cow's milk allergy	Transverse colonic stenosis	Extended right hemicolectomy with ileocolic anastomosis	Uneventful recovery; weight gain
Wong et al. (2024)	9 months	1 month	Overfeeding, gastroenteritis	Type 1 jejunal atresia with fenestrated web	Resection and jejuno-jejunal anastomosis	Uneventful recovery, weight gain
Nassiri et al. (2025)	14 months	Since 40 days of life	Cow's milk allergy, gastritis	Type I jejunal atresia with mucosal diaphragm and bands	Resection and end-to-end anastomosis	Uneventful recovery; catch-up growth

may be worsened by recurrent fasting during hospital admissions and delayed initiation of nutritional rehabilitation. While surgery corrects the underlying pathology, outcomes depend on the patient's preoperative nutritional status. Malnutrition, especially with hypoalbuminaemia, increases the risk of anaesthetic and surgical complications, including poor wound healing, postoperative sepsis, and anastomotic failure (Haloho et al., 2023). For this reason, we opted for staged procedures in two cases, delaying primary anastomosis to allow nutritional rehabilitation

before ileostomy closure. Severely malnourished children also have reduced physiological reserve, which may increase vulnerability to postoperative deterioration and other adverse events, as observed in the patient who developed aspiration pneumonitis. A review of previously published reports (Table 1) shows that delayed diagnosis of congenital intestinal obstruction beyond the neonatal period is uncommon and typically associated with atypical or intermittent symptoms. Across these studies, the delay ranged from several weeks to years, although symptom onset was often traceable to early infancy. Similar to our cases, many patients were initially managed for alternative diagnoses. In several reports, including those by Ekenze et al. (2019) and Dahal et al. (2024), colonic stenosis was only identified at laparotomy following inconclusive investigations. Others described repeated hospital encounters before definitive diagnosis (Sarkar et al., 2011; Garcia et al., 2024; Nassiri et al., 2025). Despite variation in anatomical location and pathology, surgical treatment was definitive, with favourable outcomes in all cases.

In conclusion, this report highlights the need for a high index of suspicion and early surgical referral in infants with persistent gastrointestinal symptoms. Bilious vomiting at any age should be regarded as a surgical emergency until proven otherwise, and infants with persistent feeding intolerance, abdominal distension, or poor weight gain warrant early surgical evaluation. Recurrent or unexplained symptoms should not be managed expectantly without definitive assessment. Improving clinical awareness and ensuring timely access to appropriate imaging will facilitate earlier diagnosis and intervention. As demonstrated in this series, delayed recognition of surgically correctable conditions during critical periods of growth may lead to severe undernutrition, increasing the risk of adverse outcomes and potentially compromising long-term neurodevelopment.

ETHICS STATEMENT

Written Informed consent was obtained from the parents for publication of anonymised clinical information and images.

CONFLICT OF INTERESTS

The authors have not declared any conflict of interests.

REFERENCES

Cooke R, Goulet O, Huysentruyt K, Joosten K, Khadilkar AV, Mao M, Meyer R, Prentice AM, Singhal A (2023). Catch-Up growth in infants and young children with faltering growth. Journal of Pediatric Gastroenterology and Nutrition 77(1):7-15. https://doi.org/10.1097/mpg.0000000000003784.

Dahal P, Dawadi K, Parajuli S (2023). Delayed presentation of congenital sigmoid colon stenosis: a rare entity. BJR|Case Reports 10(1):uaae003. https://doi.org/10.1093/bjrcr/uaae003.

De Sanctis V, Soliman A, Alaaraj N, Ahmed S, Alyafei F, Hamed N (2021). Early and long-term consequences of nutritional stunting: from childhood to adulthood. Acta Bio-medica Dell'Ateneo Parmense/Acta Biomedica 92(1):e2021168. https://doi.org/10.23750/abm.v92i1.11346.

Ekenze SO, Ezomike UO, Nwachukwu IE, Ariom AI, Chukwubuike KE, Nwangwu EI, Onoh US, Uwah EA. (2019). Chronic bowel obstruction from colonic stenosis in early infancy-A report of two cases. Malawi medical journal 31(1):82-85. https://doi.org/10.4314/mmj.v31i1.14

Emmanuel D, Enwani I, Ukibe N, Franco A, Duru B (2023). Prevalence and determinance of failure to thrive among children in Bayelsa State, Nigeria. International Journal of Health Sciences and Research 13(12):77-86. https://doi.org/10.52403/ijhsr.20231210.

Franceschi R, Rizzardi C, Maines E, Liguori A, Soffiati M, Tornese G (2021). Failure to thrive in infant and toddlers: a practical flowchart-based approach in a hospital setting. Italian Journal of Pediatrics 47(1). https://doi.org/10.1186/s13052-021-01017-4.

Garcia DJ, Hamade M, Lin L, Matias M, Sobhan A, Zaritsky M, Thorson C (2024). Failure to thrive in infant secondary to congenital colonic stenosis: a case report. Journal of Surgical Case Reports (12):rjae766. https://doi.org/10.1093/jscr/rjae766.

Haloho LV, Nurnaningsih N, Paryanto E (2023). Preoperative hypoalbuminemia and severe acute malnutrition as prognostic factors for postoperative complications in major abdominal surgery: A single-center retrospective study. Paediatrica Indonesiana 63(5):389-394. https://doi.org/10.14238/pi63.5.2023.389-94.

Jumbi T, Mwika P (2020). Chronic partial colonic obstruction from a congenital web. Journal of Pediatric Surgery Case Reports 57:101442. https://doi.org/10.1016/j.epsc.2020.101442.

Krasaelap A, Kovacic K, Goday PS (2019). Nutrition management in pediatric gastrointestinal motility disorders. Nutrition in Clinical Practice 35(2):265-272. https://doi.org/10.1002/ncp.10319.

Lamichhane A, Sharma R, Rajkarnikar R, Awale R, Shrestha P, Oli NC (2020). A five years old child with failure to thrive and vomiting presenting as a diagnostic dilemma: a case report. Journal of Nepal Medical Association 58(228):600-603. https://doi.org/10.31729/jnma.5134.

Nassiri H, Bouljrouf J, Ochan M, Kisra M (2025). Delayed diagnosis of type I jejunal atresia in an infant with intractable vomiting: a case report. Journal of Surgical Case Reports 2025(6):rjaf429. https://doi.org/10.1093/jscr/rjaf429.

Sarkar S, Apte A, Sarkar N, Sarkar D, Longia S (2011). Vomiting and food refusal causing failure to thrive in a 2 year old: an unusual and late manifestation of congenital duodenal web. BMJ Case Reports 2011:bcr0120113779. https://doi.org/10.1136/bcr.01.2011.3779.

Seltz LB (2008). Case 1: A green case of failure to thrive. Paediatrics & Child Health 13(8):685-687. https://doi.org/10.1093/pch/13.8.685.

Sharma C, Borkar NB, Ashwin C, Sinha C (2024). Delayed presentation of malrotation after infancy: a systematic review based on clinical presentations, associated anomalies, diagnosis, and management. Journal of Indian Association of Pediatric Surgeons 29(5):417-434. https://doi.org/10.4103/jiaps.jiaps_105_24.

Smith A, Shah M, Badireddy M (2023). Failure to thrive. In: StatPearls - NCBI Bookshelf. Treasure Island, Florida, United States of America: StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK459287/

Ssentongo P, Candela X, Amoah AKS, Opare JN, Arkorful TE, Tsai AY, Amponsah-Manu F, Oh JS (2018). Jejunal atresia causing failure to thrive: The role of camera mobile phones in aiding diagnosis in limited resource settings. Journal of Pediatric Surgery Case Reports 36:67-69. https://doi.org/10.1016/j.epsc.2018.04.015.

Wong S, Landisch R, Ruiz R, Datta S, Fuchs J (2024). Late presentation of type-I jejunal atresia in an infant: a case report. Journal of Pediatric Surgery Case Reports 110:102891. https://doi.org/10.1016/j.epsc.2024.102891.