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Case Report

A blind end and a tiny colon: an unusual case of acquired microcolon in a child

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ABSTRACT

Long-segment acquired microcolon following neonatal colonic diversion without documented ischemic insult is uncommon. We report a case of 6-year-old boy, underwent neonatal colostomy, was brought for definitive reconstruction. Diagnosis of acquired microcolon was made intraoperatively with operative findings and frozen section biopsies for ganglion cells. Restoration of bowel continuity was done by excision of long segment microcolon and Duhamel pull-through with martin modification. Absence of any surgical details of the first surgery and blind distal stoma with microcolon in contrast enema make this case interesting and worth reporting.

Keywords: Acquired, Colostomy, Contrast enema, Microcolon, Necrotizing enterocolitis

INTRODUCTION

A microcolon is an abnormal variant of colon with a smaller width, which may be due to disuse; but the length usually remains normal.[1] Microcolon may be either congenital or acquired. The fetus often consumes amniotic fluid during the second and third trimesters. This fluid, when combined with digestive juices, forms meconium, which travels via the gut and expands the colon before birth.[2] Children with bowel atresia or an obstruction close to the colon have a congenital microcolon, which causes the colon to be underutilized during intrauterine life.

An acquired microcolon may develop in children who have had long-term enterostomies, but it has also been documented in adults with long-term ileostomies or transverse colostomies.[3,4] Typical radiologic characteristics of a microcolon on contrast enema include shortening of the bowel, and absence of haustrations.[3,4] Patients seeking stomal closure may experience obstructive symptoms and anastomotic leaking if they have an acquired microcolon because of the significant disparity between the proximal and distal bowel limbs.

CASE REPORT

A 6-year-old boy, who underwent emergency colostomy on day 3 of life due to feed intolerance and progressive abdominal distension, presented to us for definitive management. The operative details from the neonatal procedure were unavailable. Following colonic diversion, the child remained clinically stable and passed stools regularly through the stoma.

Clinical examination revealed a healthy child with a functioning left-sided colostomy. The anal opening was normally positioned with satisfactory sphincter tone. Digital rectal examination demonstrated a patent anal canal and rectum. The distal stoma, however, could not be cannulated. Only proximal stoma opening was visible. Contrast enema demonstrated free passage of contrast

through the anal canal and rectum. Beyond the rectosigmoid region, contrast progressed only through a thin thread-like channel before terminating proximally, suggestive of severe long-segment narrowing of the descending and sigmoid colon (Figure 1). Because the distal stoma was not visible, a conventional distal cologram could not be performed. The radiological findings raised the possibility of Hirschsprung disease, congenital colonic stenosis, or an acquired long-segment fibro stenotic process.



Figure 1: Contrast enema showing rectum of normal calibre and narrow lumen sigmoid and descending colon.

Intraoperatively, dense inter-bowel adhesions were found. The functioning stoma was located at the transverse colon near the splenic flexure. Proximal end of distal bowel was blind ended. The descending and sigmoid colon distal to the stoma were markedly narrowed throughout their entire length till the rectosigmoid junction (Figure 2). The transverse colon proximal to the stoma was dilated and loaded with fecal matter. The possibility of Hirschsprung disease was excluded by presence of ganglion cells on serial frozen section biopsies from multiple levels of the colon (stoma opening, proximal end of distal bowel and at the level of peritoneal reflection). The narrowed descending and sigmoid colon, unsuitable for reconstruction, were resected up to the level of the peritoneal reflection and sent for histopathological examination. The splenic flexure was completely mobilized and transverse colon was brought down through the retro rectal plane, and a Duhamel pull-through with martin modification was performed with 75 mm linear GIA stapler.

The postoperative recovery was uneventful. Oral feeds were initiated after return of bowel function on post operative day 4, and the child recovered satisfactorily and discharged on day 6.

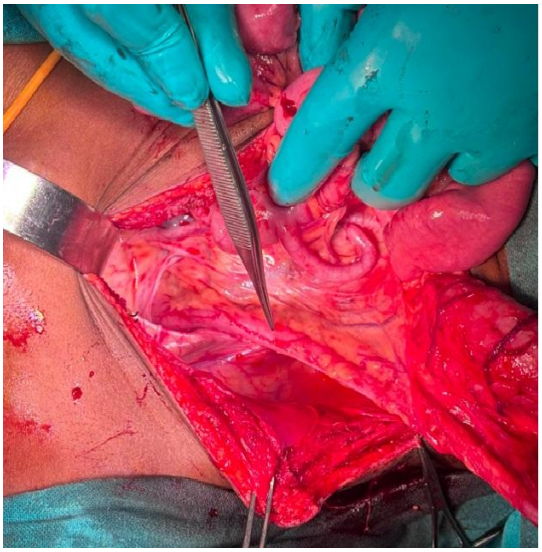


Figure 2: Intraoperative image showing microcolon.

DISCUSSION

Acquired microcolon is an uncommon condition in children, the underlying mechanism of which may differ among patients. It has most frequently been described as a late complication of neonatal intestinal injury, particularly necrotizing enterocolitis (NEC) and ischemic insult to bowel.[5-7] Fouad et al reported three infants who developed acquired colonic atresia in association with significant cardiovascular compromise despite the absence of documented NEC, suggesting that intestinal hypoperfusion may contribute to the development of acquired colonic obstruction.[8] Rattan et al also reported acquired colonic atresia in a term infant without a documented history of NEC.[9] These reports suggest that the absence of recognized NEC does not exclude an acquired colonic lesion. In our case, the child underwent neonatal colostomy on third day of life for feed intolerance and progressive abdominal distension. There was no documented history of prematurity, NEC or cardiovascular compromise as well as, the original neonatal operative records were not available. Although, an unrecognized neonatal ischemic or inflammatory insult cannot be excluded but the precise mechanism responsible for the colonic abnormality remains uncertain.

Microcolon is a recognized radiological finding in neonatal intestinal obstruction and may occur when the colon remains unutilized because of a proximal obstruction.[1,2,10] Laya et al described imaging patterns of microcolon and their relevance to the diagnosis of neonatal lower intestinal obstruction.[3] In our case, contrast enema demonstrated a patent anal canal and rectum, followed by a thin, thread-like lumen extending from descending colon till the rectosigmoid junction. The involvement of the descending and sigmoid colon, with preservation of the rectum, was particularly notable in our case. The distal stoma opening was also not visible

and could not be cannulated, preventing a conventional distal cologram. These findings highlight the importance of careful contrast evaluation of the distal bowel before definitive reconstruction when the previous operative anatomy is not certain.

The intraoperative findings further characterized the extent of the abnormality. The proximal end of distal bowel was blind-ended, and the descending and sigmoid colon were markedly narrowed, whereas the proximal transverse colon was dilated and loaded with fecal matter. Rattan et al similarly described a blind-ended distal colon, dense interloop adhesions and proximal bowel dilatation in an infant with acquired colonic atresia.[9] However, the extent of the abnormality in our case was distinctive because severe narrowing involved a long segment of the distal colon. Whereas acquired colonic atresia is generally described as a localized interruption of bowel continuity, the present findings demonstrated extensive distal colonic hypoplasia and narrowing. This distinction is clinically very important because the length and quality of the affected bowel determine whether a conventional anastomosis is feasible or whether resection and a more extensive reconstructive procedures are required. Nevertheless, as the original neonatal anatomy is unknown, it is not possible to determine whether the entire abnormality developed after a diversion or whether an underlying lesion was already present during the first surgery.

Another important differential diagnosis is Hirschsprung disease. In our case, frozen-section biopsies from multiple colonic levels demonstrated ganglion cells, arguing against long-segment Hirschsprung disease as the explanation for the observed narrowing. Histological assessment was therefore important in this patient because the anatomical findings were atypical and the original diagnosis was unavailable. However, the presence of ganglion cells does not establish the cause of the stenosis or independently distinguish all congenital from acquired structural abnormalities.

The definitive management was guided by the extent of the abnormal bowel and the quality of the remaining colon. Because the descending and sigmoid colon were markedly narrowed and unsuitable for reconstruction, the affected segment was resected and the healthy transverse colon was mobilized through the retrorectal plane for a Duhamel pull-through with Martin modification. This approach avoided an end-to-end anastomosis involving the abnormal distal colon and allowed reconstruction using healthier proximal bowel while preserving the rectum.

Although the published literature on acquired colonic atresia describes the need for individualized surgical management according to the extent of the lesion.[5,8,9] But the evidence specifically evaluating the Duhamel-pull-through with Martin procedure for acquired microcolon is limited. The uneventful postoperative recovery in our patient demonstrates the technical feasibility of this approach in the individual case. Longer-term follow-up would be necessary to assess bowel function, continence, growth and late complications.

The principal clinical contribution of this case is the description of extensive distal colonic narrowing and a blind-ended distal bowel in a child presenting for definitive reconstruction several years after neonatal diversion, without an available record of the original diagnosis or operation. The combination of contrast-enema findings, intraoperative anatomy and ganglion cells on frozen-section biopsy illustrates the importance of defining the distal bowel anatomy and excluding relevant alternative diagnoses before reconstruction. Prolonged exclusion from the fecal stream may have contributed to the observed hypoplasia; however, an unrecognized neonatal insult or pre-existing structural abnormality remains possible. Therefore, this case expands the clinical description of unusual acquired colonic abnormalities encountered after prolonged diversion while emphasizing that the operative plan should be individualized according to the extent and quality of the remaining bowel.

We are reporting this case as this case provides three practical learning points: Longstanding diversion can result in severe distal colonic hypoplasia and stenosis, and acquired microcolon may progress beyond simple calibre reduction; Inadequately documented neonatal colostomy should prompt careful assessment of the distal colon before definitive reconstruction. The original operative record is an important component for subsequent surgical planning; and When the distal colon is unusable, reconstruction should be based on the length and quality of healthy proximal bowel. In selected patients, extensive mobilization and a Duhamel pull-through can provide successful definitive reconstruction.

Due to absence of the original neonatal operative records, preoperative imaging, and histopathology report of first surgery, the exact initiating event cannot be proven. Therefore, the most appropriate interpretation is that the child developed acquired microcolon, probably related to prolonged distal diversion and disuse, with an unrecognized neonatal insult remaining a possible alternative explanation.

CONCLUSION

This case expands the clinical description of unusual acquired colonic abnormalities encountered after prolonged diversion while emphasizing that the operative plan should be individualized according to the extent and quality of the remaining bowel.

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