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A case report of midgut atresia presenting with gastroschisis

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***Abstract*---**We describe a case of a newborn who presented at birth with midgut atresia and mummified midgut tissue protruding from abdominal wall defect that had closed spontaneously. This case highlights an instance of spontaneous closure of an abdominal wall defect, associated with an in-utero midgut vascular incident. It offers unique insight into the potential mechanisms underlying gastroschisis pathophysiology.

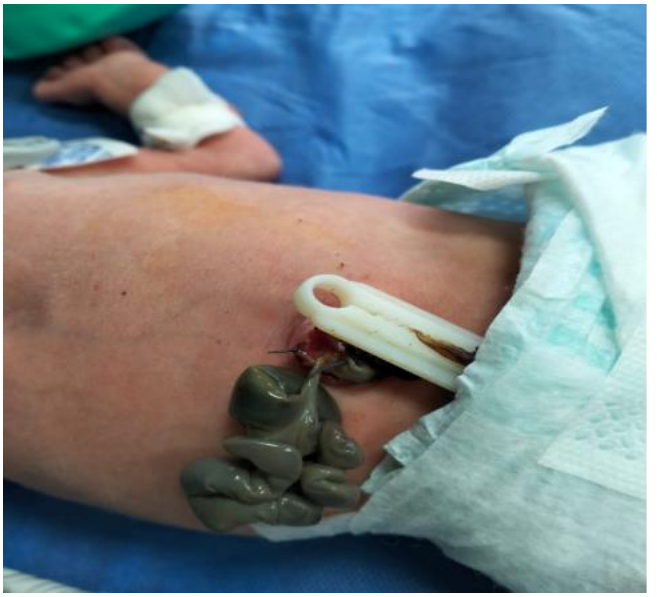
***Keywords*---**newborn, gastroschisis pathophysiology, midgut atresia.

The case

A preterm female 34 weeks gestation age was referred to our unit on second day of birth due to gastroschisis and lack of surgical facilities in the hospital she was born in. The baby had normal vaginal delivery with birth weight of 2.1 kg and APGAR score was 5,8.

The mother is 19 years old, primigravida, with O +ve blood group.
Mother reported that 3D U/S done at 5th month of gestation showed a congenital anomaly in the abdomen, however there was no documented report. On examination, the baby was not pale nor cyanosed or jaundiced. Her temperature 36.7 c, pulse rate 128 bpm, respiratory rate 48 breath/min. saturation 96 % on room air.

Abdominal examination showed that her abdomen was not distended, protruded greenish-brown small bowel remnant arising from the right side of umbilicus with no abdominal wall defect as shown in fig 1. The digital rectal examination was normal and the child had normal female external genitalia.



Her labs on admission were as follow: TLC 15.5 X 10^9 /L, Hb 19.1 gm /dl, hematocrite 48 %, platelet 298 X 10^9/L, RBS was 9.2 mmol/L, CRP 2.9, creatinine 0.7 mg/dl, PT 16.3 sec., INR 1.2. Abdominal X ray showed double bubble sign suggestive of duodenal atresia, Echocardiography and transcranial U/S showed no abnormality.

The infant underwent an exploratory laparotomy on second day of admission and was found to have malrotation with a midgut atresia resulting in a markedly dilated foregut (18-19 cm), which was connected by a fibrous cord to the mummified tissue arising from the abdominal wall, and a hindgut microcolon. No abdominal wall defect was noted. Both Duodenostomy and colostomy were done with no anastomosis. in addition to placing a central line and baby was dependent on total parenteral nutrition. She subsequently developed complications of short bowel syndrome. Become cachectic with faltering of growth, and sepsis developed with multiple electrolyte disturbance and then the child succumbed.

Discussion

There is a well established association between gastroschisis and intestinal atresia, with intestinal atresia occurring in approximately 10-15 % of gastroschisis cases. Nearly all cases of gastroschisis involve abnormal intestinal rotation and fixation, research, including experimental studies (1), has suggested that intestinal atresia may result from an in – utero vascular accident, potentially caused by volvulus, intussusception, or incarceration. In this case it seems that mid gut atresia resulted from an in-utero vascular event involving the superior mesenteric artery as indicated by mummified remnants of mid gut. However it is uncertain whether this vascular accident was caused by a midgut volvulus or by incarceration and strangulation of the midgut at the site of abdominal wall defect before it spontaneously closed.

An intriguing aspect of this case is the spontaneous closure of the abdominal wall defect. The exact cause and development of gastroschisis remain only partially understood. The most widely accepted theory suggests that gastroschisis originates from abnormal involution of the right umbilical vein (2), leading to a mesenchymal defect where the body stalk meets the body wall. Spontaneous closure of gastroschisis is rare, with only a few cases reported in the literature. All reported cases of spontaneous gastroschisis closure involved significant bowel length loss and the formation of atresia in utero, leading to congenital short bowel syndrome (3-6). This case, along with others, suggests an association between spontaneous gastroschisis closure and congenital short bowel syndrome.

A possible explanation for this association is that spontaneous closure of gastroschisis may lead to congenital short bowel syndrome by incarcerating the intestine, causing atresia during the closure process. Alternatively, it is possible that the intestines contribute to spontaneous gastroschisis closure; removal of a significant portion of the intestine could allow the remaining bowel to reduce, restoring abdominal domain and enabling the abdominal wall to close spontaneously. However, it remains unclear whether there is a cause-and-effect relationship or simply an association between spontaneous gastroschisis closure and congenital short bowel syndrome.

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