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Case Report

Jejunoileal atresia and multiple ileal diverticulae in a twelve-day-old infant: A Case Report

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Abstract

A twelve-day-old neonate presented with persistent bilious vomiting and dehydration. A transverse supra-umbilical laparotomy was done. Later, he died and autopsy was done. A jejunoileal segment and several other ileal segments were collected. The jejunoileal segment was dilated. It measured 11 cm in length and had a blind end. One of the ileal segments measured 16 cm in length. Ileum showed multiple small diverticulae at the mesenteric border and each diverticula measured 3 to 4 mm in length. Lumen of ileum appeared collapsed or strictured. Further, the lumen of diverticulae contained soft eosinophilic material. He was finally diagnosed as a case of jejunoileal atresia associated with multiple terminal ileal diverticulae.

Keywords: *Jejunoileal dilatation multiple ileal type IV diverticulosis*.

Introduction

Jejunal atresia is defined as a complete closure of the jejunal lumen due to one of the following reasons, e.g. persistence of thin intraluminal diaphragm or occlusion of proximal end may persist. Moreover, seventy-five percent of cases were aged <2 years[1]. On the contrary, stenosis is an incomplete occlusion of the ileum. Further, diverticulum may lead to various complications. In addition, diverticulae may also be detected during laparotomy[1]. Herewith, we present a case of congenital intestinal obstruction associated with multiple diverticulae.

Case Report

A 12-day-old infant presented with frequent vomiting and dehydration. Laparotomy was done. He was found to have a congenital jejunal atresia. In addition, the present patient had multiple diverticulae in the mesenteric side of ileum with nodules. True diverticulum possessed all the coats of intestine and mucosa was similar to ileum.

Present patient had a dilated bowel segment, measuring 11 cm in length. Moreover, its distal end was closed (figure 1A). Ileum showed several segments, biggest segment measured 16 cm in length(figure 1B). Each diverticulum measured 3 to 4 mm in length. Further, mesenteric ileal border showed outpouchings, suggesting formation of diverticulae (figures 1&2). Lumen of bowel appeared collapsed or strictured. The lumen of diverticulum contained eosinophilic material. It was composed of proteinaceous substance, necrosed tissue debris and hemolyzed material (figure 2). In addition, most of the diverticulae communicated with the lumen of bowel.

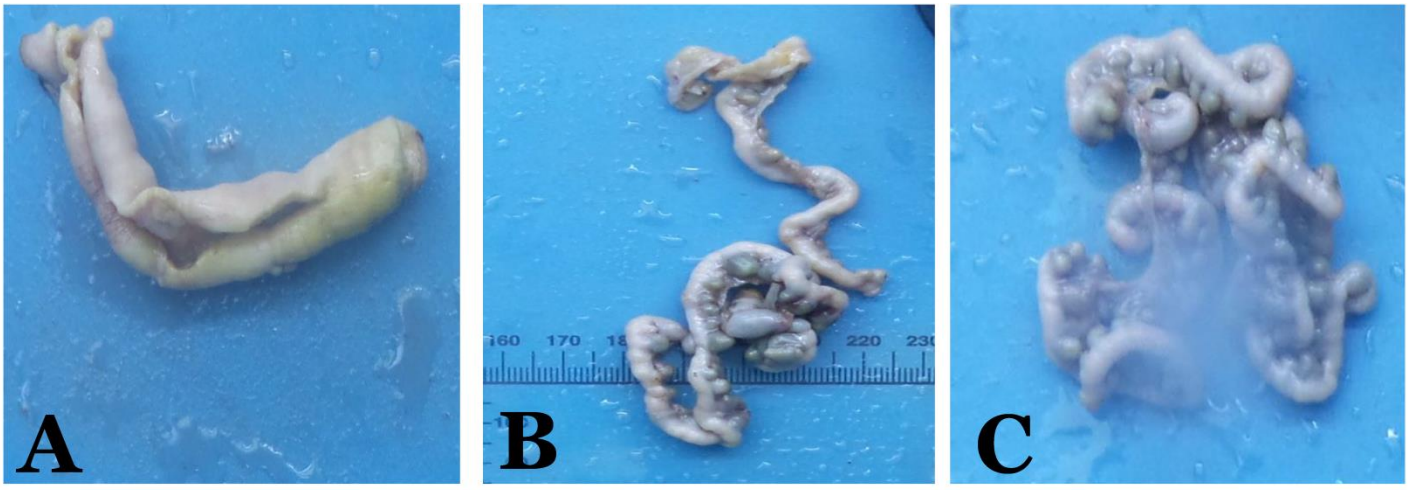

Figure 1:(A) Photomicrograph shows a diverticulum distal to jejunoileal atresia. Diverticulum had thicker wall as compared to normal wall. **(B)** Shows several diverticulae at the mesenteric border of ileum (Arrow points the diverticulae). **(C)** Shows multiple diverticulae at the mesenteric border of ileal segment(Arrow points the diverticulae).

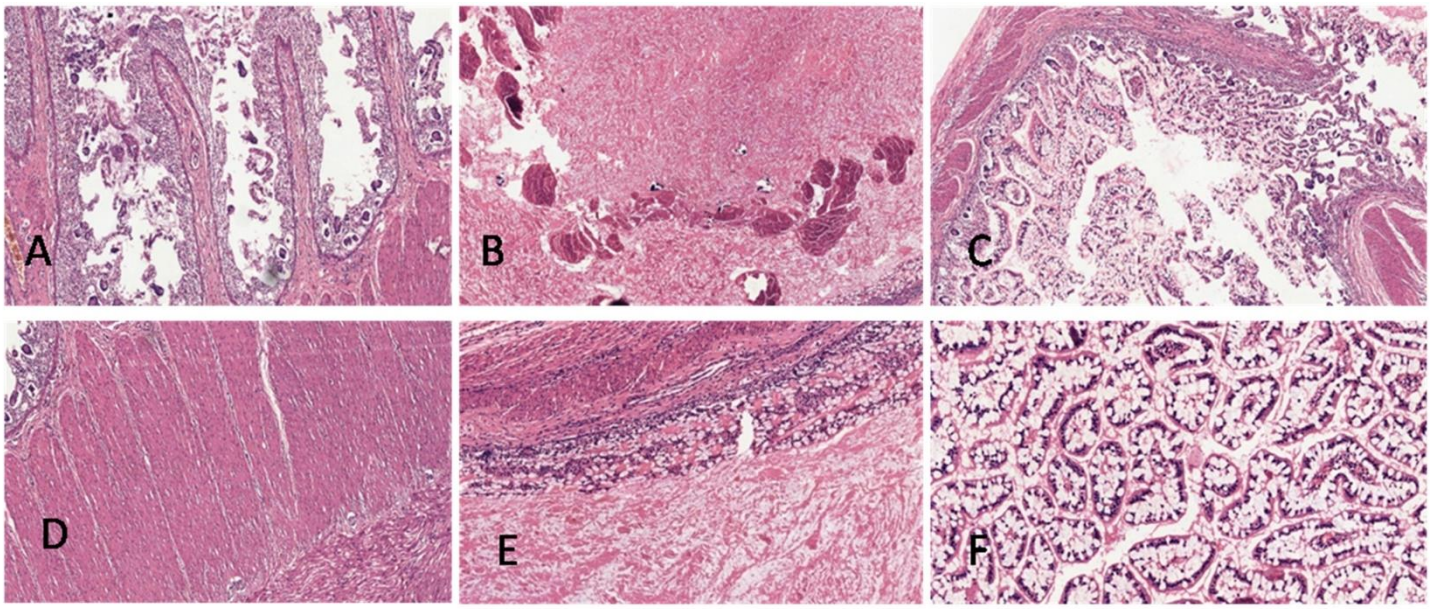

Figure 2: (A) Photomicrograph shows outpouching of mucosa (HE×400) **(B)** Shows necrosed tissue debris, calcification, meconium and hemolyzed red cells giving a eosinophilic appearance. **(C)**Shows normal muscle thickness (HE×400). **(D)**Shows hypertrophy and hyperplasia of smooth muscle cells (HE×400). **(E)**Shows hyperplasia and hypertrophy of muscle cells (HE×400).**(F)** Shows hyperplasia of mucosal glands (HE×400).

Discussion

John Hunter failed to identify the diverticular disease, but two of his specimens showed colonic diverticulae in his museum[2]. Keith recognized two types of diverticular disease, e.g. diverticula without muscular lesion and diverticula with muscle disease[3]. It was suggested that patients without fever and raised leucocyte counts might be classified as acute diverticular disease or painful diverticular disease[4]. Further, in Europeans, intestinal muscles become weak during old age, resulting in development of numerous diverticulae. Jejunoileal disease is another rare disease due to mucosal protrusion through muscularis mucosae. It may lead to obstruction, malabsorption, diverticulitis, bleeding and perforation. Further, obstruction may lead to volvulus and intussusception[5]. Jejunal diverticula was first reported in the year 1794[6]. Most of the patients with diverticulae may not have symptoms; asymptomatic disease is known as diverticulosis. Further, 2 types of diverticular disease may develop. One of the types may be associated with stricture and another may be associated with bleeding. Symptomatic uncomplicated diverticular disease is similar to irritable bowel syndrome[7]. Ilial atresia develops when blood supply is impaired infoetus. In type IV atresia multiple obstructions develop in the ileum[8]. Most important feature of the present case was dilatation of a jejunoileal segment. Occlution distal to dilated segment might have resulted in more open jejunoileal segment. Moreover, jejunoileal atresias were relatively less frequent than duodenal atresias (~1 in 6000 live births)[9]. Another feature of present patient was presence of multiple small diverticulae at the mesenteric border of the ileum. Weakness of intestinal smooth muscle might have contributed to development of type IV diverticulosis. Moreover, obstruction in the upper gastrointestinal tract might have resulted in bilious vomiting. Initially, it was suggested that congenital intestinal atresia might be due to a defect in growth[10]. Later, two other theories were proposed, e.g. Tandler's theory of imperfect canalization and theory of vascular insufficiency[11].

Conclusion(S)

Present patient relates to a twelve-day-old infant who had multiple congenital outpouchingsat the mesenteric border of jejunoileal diverticulum. In addition, the patient had a large dilated jejunoileal segment. Obstruction distal to the segment might have resulted in its dilatation. Moreover, imperfect canalisation and vascular deficiency in the ilium might have contributed to development of multiple diverticulae.

References

1. Hamilton SR, Faber JL, Rubin E. Congenital disorders atresia and stenosis, Pathology, 3rd ed 1999; 703-704.
2. Pathological series in the Hunterian museum. Descriptive catalogue part II, Edinburgh and London: E.S. Livingstone, 1972:1049,1050.
3. Keith SA. A demonstration on diverticula of the alimentary tract of congenital or obscure origin. Br Med J 1910;1:376-80.
4. Painter NS. The aetiology of diverticulosis of the colon with special reference to the action of certain drugs on the behavior of the colon. Ann R CollSurgEngl 1964;34:98-119.
5. Ghandour R, Khalifeh G. OrmNB, Rakka M. Jejunal diverticular disease: a report of three Cases, J SurgCas Rep 2020;11; 1-4.
6. Painter NS, Burkitt DP. Diverticular diseaseof colon: A deficiency disease of western civilization. B Med J. 1971;2:450-454.
7. Rezapour M, Ali S, StollmanN. Diverticular disease: An update on pathogenesis and management. Gut Liver 2018; 12 (2):125-132.

8. Muacevic A, Adler J. Type 4 ileal atresia and anorectal malformation in a neonate: A rare association. Cureus 2022;14 (4):e24295.
9. Hayek J, Goldman H, Kozakewich BW. Embryology and developmental disorders, In 'Pathology of the gastrointestinal tract', second edition, Si-Chun Ming and Goldman Harvey (editors) 1998, Þ169-193.
10. Meckel J. Handbuch der pathologischen Anatomie. 1 Leipzig, Reclam 1812.
11. Tandler J. Morpholjb 1902;29:187-216.