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Case Report
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Motility Disorder, Meckel's Diverticulum and Mass in a Toddler Girl-Mere Coincidence or Causation Link

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Abstract

We present unique and an interesting case of a toddler girl who had all three in one lesion with a known case of hypoganglionosis. Patient presented with recurrent right sided abdominal colicky pain, vomiting and a right lower abdominal mass. During abdominal ultrasound, an ileoileal intussusception was found. Patient underwent minimal invasive approach using laparoscopy at which the ileoileal intussusception was reduced and an inverted Meckel's diverticulum with ectopic mucosal nodule was discovered. Limited segmental ileal resection containing diverticulum and primary anastomosis resulted in resolution with good recovery.

Keywords: Abdominal Pain; Appendicular Mass; Congenital Colorectal Motility Disorder; Gut Hypomotility; Intermittent Hydronephrosis; Intussusception; Kidney Sign; Laparoscopy; Meckel's Diverticulum; Pseudo Hirschsprung's Disease; Variant Hirschsprung's Disease; Target Sign

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Introduction

Congenital colorectal motility disorder can cause partial functional obstruction of the distal large bowel by growing to block the flow of gas and fecal matter from the gut. This could be in the form of Hirschsprung's disease occasionally but most importantly a variety of variants are more common than previously thought [1-3]. Meckel's diverticulum is the commonest of the anomaly of the umbilical anomalies in general and vitello-intestinal duct in particular [4-9]. It may be associated with other anomalies including congenital colorectal motility disorders. The Meckel's diverticulum when gets inverted into the ileal lumen can cause the ileoileal or ileocolic intussusception. Intussusception is most common in infants between 5 to 9 months during weaning period and its occurrence outside the age of 2 years is rare and may be associated with other anomalies, battery ingestion, trichobezoar, etc [10-13]. Each of these conditions usually present as a single entity in most instances dual lesions may be occasionally combined but the combination of the triad in one patient is very rare benign lesions originating from the gut.

Case Report

A 4-year-old girl presented with recurrent episodes of right sided intermittent abdominal colicky pain, vomiting, abdominal fullness and a mass felt in the right lower abdomen since last 4 days. Patient had no fever or rigors and was passing flatus and opening small amounts of stool but felt bloated. Girl was born at term weighing 3200 gm after an uneventful pregnancy and normal anomaly scan by spontaneous vaginal delivery and passed urine and meconium in first 24 hours following birth.

Patient developed chronic constipation and hypomotility of distal colon since weaning in late infancy with few minor episodes of enterocolitis treated conservatively as gastroenteritis due to teething troubles. At the age of 8 months, patient had severe constipation and abdominal distention and lower gastrointestinal water-soluble contrast showed a transition zone at rectosigmoid junction suggestive of Hirschsprung's disease (Figure 1A-C). However, rectal suction biopsy demonstrated few ganglion cells with no hypertrophied nerve fibers and no enhance acetyl cholinesterase activity suggestive of hypoganglionosis. Parents wanted conservative treatment.

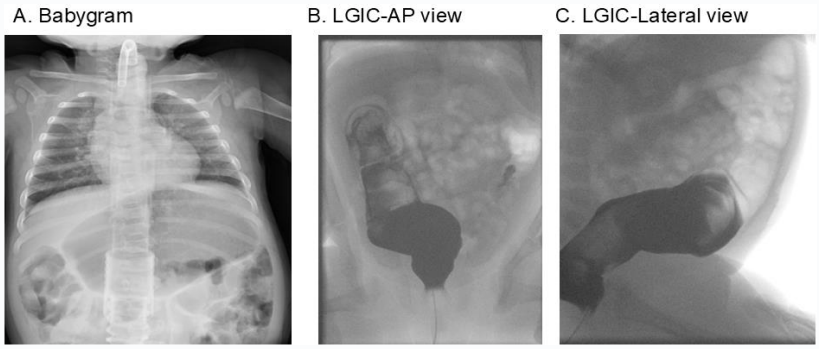

Figure 1: A. Babygram showing sigmoid and left colonic fecal loading with dilated transverse and right colon, B. Lower gastrointestinal water-soluble contrast with transition zone at rectosigmoid junction on anteroposterior view, C. Lateral view with collapsed rectum with tube, transition zone at rectosigmoid with dilated loaded sigmoid colon.

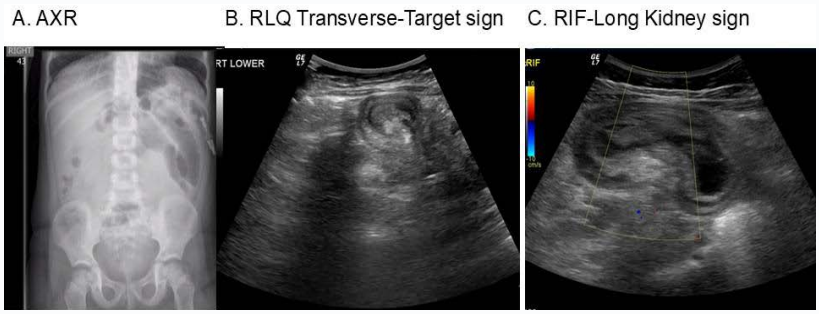

Figure 2: A. Abdominal radiograph showing dilated jejunal ileal loops with scanty gas in the distal bowel and soft tissue mass in the right side, B. USS transverse view of right lower quadrant showing typical target sign with layers of bowel, C. USS longitudinal view of right iliac fosaa demonstrating clear kidney sign.

Patient has been seen by his general practitioner with recurrent short lived right sided abdominal pain for last few months who initially thought it to be grumbling chronic appendicitis and did not refer to us. However, this time he suspected an appendicular mass and referred to us for further management.

Physical examination showed stable vital signs and abdomen was full, soft, non-tender with no signs of peritonism and mildly tender partially mobile mass was palpable in right lower abdomen.

Urinalysis was normal. Complete blood count and biochemical profile including C-reactive protein were all normal. Abdominal radiograph revealed dilated small bowel loops in the left upper quadrant with paucity of bowel gas distally suggestive of partial bowel obstruction (Figure 2A), Abdominal Ultra Sound Scan (USS) showed right sided abdominal mass to be an ileoileal intussusception with a clear target sign on the transverse view (Figure 2B) and a bean like kidney sign on the longitudinal view of the scan (Figure 2C) confirming the diagnosis. The bowel vascularity was good but air enema was not possible as it did not extend beyond the ileocecal valve into the large bowel.

At the viable reducible ileoileal intussusception was confirmed and reduced easily. The lead point was formed by the inverted narrow neck Meckel's diverticulum containing a nodule of an ectopic tissue near its tip. A decision was taken to resect it despite being viable due to presence of an ectopic tissue and narrow base. A limited segmental ileal resection containing Meckel's diverticulum and primary end to end anastomosis were carried out uneventfully.

The post operative recovery was good and patient was discharged home after 72 hours. The histopathological examination confirmed Meckel's diverticulum containing gastric mucosal tissue in the distal nodule near its tip. At 15-year long term follow-up visit, patient is asymptomatic, well and thriving with normal USS of the abdomen and the underlying bowel hypomotility and dysbiosis which are being aggressively regular and persistent conservative management of underlying congenital bowel motility disorder as patient and parents were not willing to treat it *via* endosurgical intervention, diagnosis and possible correction.

Discussion

Colorectal motility disorders can exist simultaneously but are not directly caused by the diverticulum itself that is understandable but the reverse could be possible. However, at first sight the motility disorder is likely an unrelated coincidence but recent experimental, translational, clinical, investigative,

and therapeutic evidence has revealed the secret pathways in which it could be possible as explained later [14-16].

A link between the diverticulum and intussusception has been established as cause-and-effect phenomenon for a long time. Based on medical literature, a Meckel's diverticulum can cause a mass and intussusception, especially in a toddler. Causation link between Meckel's diverticulum and intussusception definitely exist in which the diverticulum can act as a "lead point." In a small percentage of children, a Meckel's diverticulum contains ectopic (displaced) tissue, such as gastric or pancreatic mucosa as seen in our case. This ectopic tissue can become a fixed "lead point" that can be pulled into the next section of the bowel, initiating a process called intussusception thus creating a "mass." The telescoping of the intestine during intussusception creates a palpable mass, which is a

classic finding on physical examination especially as a complication in toddlers. Symptomatic Meckel's diverticulum, particularly leading to complications like intussusception, most often presents in children under two years old. This age group has a higher incidence of complications from Meckel's than any other. Our case being 4 years old was an exception.

Coincidence of the congenital colorectal motility disorder which is a condition where the muscles or nerves of the colon do not function properly and a vascular accident like a colonic atresia especially in water shade areas is more likely the cause [14-15]. This in turn leads to issues like slow transit chronic constipation with gas and fecal matter retention and back pressure and intestinal partial functional distal bowel pseudo-obstruction [15-16]. The pressure is transmitted to the right colon, cecum and appendix in the fetus. During situations of fetal distress, the left colon parasympathetic system enervated colorectum goes on fight or flight response, the flight mode is generally not available except for meconium staining of liquor when the fight mode fails and the increased back pressure may open up the ileocecal valve with reflux back leading to back pressure to the intestinal end of the vitello-intestinal duct making its spontaneous closure difficult resulting in the formation of Meckel's diverticulum [16].

This back pressure, similar to the pop off mechanism in posterior urethral valves and patent urachus or urachal diverticulum, is then transmitted via vitello-intestinal duct leading to its persistence either as patent, latent or its variant in the form of Meckel's diverticulum which is the commonest lesion of all others. Meckel's diverticulum and congenital colorectal motility disorder are both congenital making their association together although it apparently appears to originate from different developmental failures. Apparently, at first sight they appear to have different pathology. A Meckel's diverticulum arises from the incomplete closure of the vitelline duct during fetal development. In contrast, some motility disorders, like Hirschsprung's disease and its variants, result from a different issue: the absence of specific nerve cells in the colon.

The whole clinical picture matters. While the congenital colorectal motility disorder in special circumstances may help formation of Meckel's diverticulum which in turn can cause an acute episode of intussusception especially with ectopic tissue and inversion of Meckel's diverticulum. The presence of a co-existing motility disorder may leadto severe constipation, fecal retention, dysbiosis, small intestinal bacterial overgrowth, leaky gut and allow payers patches to get inflamed and form an intussusception in the absence of any lead point such as Meckel's diverticulum or and ectopic tissue merely as a consequence of back wash ileitis during periods of stress of weaning in the typical infant age group too.

We have observed either variants of midgut malrotation when the splenic flexure mesocolon stretches the suspensory ligament of the duodenojejunal junction down caudally and medially making the malrotation to form easily while if it pushes it cranially and laterally, it causes high insertion of the duodenojejunal junction predisposing to superior mesenteric artery syndrome with partial obstruction to third part of the duodenum leading to chronic duodenal ileus and its consequent complications. We have observed these findings in the cases of Waugh syndrome with intussusception and therefore need for the screening for associated motility disorders in these cases [17-18].

Pneumatic reduction is gold standard for an ileocolic intussusception but laparoscopy in early cases of ileoileal intussusception may be the best course of minimally invasive option [19], However, our experience tells us that when a lead point is present, an advanced intussusception with trans-anal prolapse, vascular compromise of the intussusception on ultrasound and color doppler or late diagnosed cases with complications of perforation may need open laparotomy [20]. Colon preservation techniques may be employed if feasible and possible to preserve as much of the bowel as possible especially in developing country where diarrhea in young children is common and can lead to potentially lethal outcome [21]. Meckel's diverticulum with narrow base and/or an ectopic tissue mass needs localized resection rather than preservation.

Conclusion

In summary, existence of this triad of lesions should have a definite reason for their occurrence and cannot consider mere coexistence. The chronological order in which they evolved embryo-pathogenetically and presented clinically seems to be more logical. Hypoganglionosis was the first to manifest and the chronic intermittent right lower abdominal pain was secondary to ectopic gastric tissue in the Meckel's diverticulum and finally the inversion of the Meckel's diverticulum precipitated partial obstruction and provided a lead point for the intussusception. In this case, the relationship may appear to be a combination of causation and coincidence at first sight. Causation is very clear for the inverted Meckel's diverticulum which has likely acted as the lead point that caused the acute intussusception and the resulting mass. The underlying colorectal motility disorder may seem to be a separate, coincidental medical condition that would be managed independently of the Meckel's diverticulum complications. However, recently pathogenesis of hind gut motility disorders with small left colon with partial functional obstruction has been implicated in raising the right colonic and ileocecal valve pressure prenatally leading to the ileocecal reflux and back pressure finally causing pop off mechanism on the persistence of ileal end of vitello-intestinal duct remnant as Meckel's diverticulum which may suggest its role more as causation rather than mere coincidence.

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