Combined Gastric and Pancreatic Tissue Inside a Meckel’s Diverticulum

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CASE PRESENTATION
A previously healthy 11 year old male presented with bloody stools followed by an episode of syncope. In addition, the boy had noticed foul-smelling dark stools in the previous 2 days. At admission, his temperature was 36.8°C, blood pressure was 100/70 mmHg, and pulse was 100 beats per minute. Examination revealed a pale, well-nourished boy. The abdomen was soft, non-tender, non-distended, and without palpable masses. There were no anal fissures or hemorrhoids, and rectal exam was normal. Blood tests were significant for anemia (hemoglobin 10.6 g/dl) and elevated erythrocyte sedimentation rate (46 mm/hour). Coagulation studies were normal.

During the first 24 hours of admission, his hemoglobin level dropped to 7.9 g/dl, requiring a blood transfusion. Bleeding from a Meckel’s diverticulum was suspected and a radionucleotide scanning with technetium-99m pertechnetate suggested ectopic gastric mucosa in the pelvic inlet [Figure 1].

Explorative laparoscopy revealed a Meckel’s diverticulum, which was excised [Figure 2A, 2B]. Histopathologic assessment confirmed that the Meckel’s diverticulum contained both gastric and pancreatic heterotopic tissues [Figure 3, Figure 4].

Meckel’s diverticulum is the most common congenital abnormality in the gastrointestinal tract, affecting approximately 2% of children, and typically manifests around the age of 2 years. Gastrointestinal bleeding, bowel obstruction, diverticulitis, and intussusception are common complications of Meckel’s diverticulum, although in most cases patients are asymptomatic.

Figure 1. Technetium-99m (Tc-99m) pertechnetate scan in a healthy 11 year old male presented with bloody stools, indicating increased uptake in the right lower quadrant (arrow). Normal uptake of the Tc-99m nuclide is seen in the stomach and bladder (arrowheads)

Figure 2. [A] Endoscopic view of an anti-mesenteric Meckel’s diverticula [B] A 1 × 1 × 2 cm Meckel’s diverticula excised from the intestine

Figure 3. Microscopic view of the small bowel wall (arrowhead) partially lined by gastric type mucosa (arrow) (hematoxylin and eosin, magnification ×40)

Figure 4. Microscopic view of pancreatic tissue (hematoxylin and eosin, magnification ×40)
Most symptomatic diverticulum contain ectopic tissue, typically gastric (approximately 50%), pancreatic, or colonic. A combination of different types of mucosa occurs in only 2% of the cases. Whereas laparoscopic resection is the treatment of choice in symptomatic Meckel's diverticulum, the management of incidental Meckel's diverticulum is still controversial. A systematic review that included 2975 patients suggested that leaving an incidental Meckel's diverticulum in situ is preferred as it reduces the risk of post-operative complications with no increase in late complications, as compared to prophylactic resection [1-5].

Our case highlights the importance of high index of suspicion for Meckel's diverticulum, even in adolescence.

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References

Potent platelets in allergy
Anaphylaxis results from inappropriate immune responses to allergens. Human platelets express the immunoglobulin G (IgG) receptor FcγRIIA/CD32A and release inflammatory mediators in response to their engagement. However, the contribution of platelets to anaphylaxis is not well understood. To address this, Beutler et al. developed mouse models that express either human FcγRIIA/CD32A alone or the full human IgG receptor complexity. Anaphylaxis induced a marked decrease in platelet levels; however, preventive platelet depletion reduced anaphylaxis severity. A clinical study of patients with drug-induced anaphylaxis showed that a severe reaction was likewise associated with fewer circulating platelets. Activated platelets released serotonin, which contributed to anaphylaxis severity. Thus, platelets play a critical role in IgG-mediated anaphylaxis.

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The role of autophagy in the degradation of misfolded HLA–B27 heavy chains
Navid and co-authors tried to determine whether autophagy is involved in the degradation of misfolded human leukocyte antigen (HLA)-B27 in experimental spondyloarthritis. Bone marrow-derived macrophages from HLA-B27/human β2-microglobulin (β2m)-transgenic rats were incubated in the presence or absence of interferon-γ and proteasome or autophagy inhibitors. Immunoprecipitation, immunoblotting, and immunofluorescence analysis were used to measure HLA-B27 heavy chains and autophagy. Autophagy was induced using rapamycin. Macrophages from HLA-B27/β2m-transgenic and wild-type rats were used as controls. HLA-B27-expressing macrophages showed phosphatidylethanolamine-conjugated microtubule-associated protein 1 light chain 3B levels similar to those in both control groups, before and after manipulation of autophagy. Blocking autophagic flux with bafilomycin resulted in the accumulation of misfolded HLA-B27 dimers and oligomers as well as monomers, which was comparable to the results of blocking endoplasmic reticulum-associated degradation (ERAD) with the proteasome inhibitor bortezomib. HLA-B7 monomers also accumulated after blocking each degradation pathway. The ubiquitin-to-heavy chain ratio was twofold to threefold lower for HLA-B27 than for HLA-B7. Activation of autophagy with rapamycin rapidly eliminated to be approximately 50% of misfolded HLA-B27, while folded HLA-B27 or HLA-B7 monomeric heavy chains were minimally affected. This study is the first to demonstrate that both autophagy and ERAD play roles in the elimination of excess HLA class I heavy chains expressed in transgenic rats. The authors observed no evidence that HLA-B27 expression modulated the autophagy pathway. These results suggest that impaired ubiquitination of HLA-B27 may play a role in the accumulation of misfolded disulphide-linked dimers, the elimination of which can be enhanced by activation of autophagy. Manipulation of the autophagy pathway should be further investigated as a potential therapeutic target in spondyloarthritis.

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