

# 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.

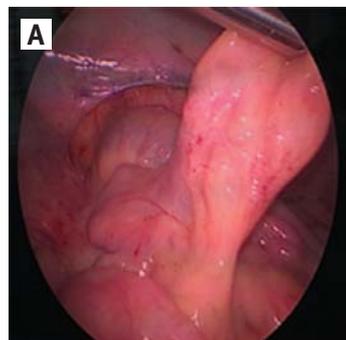
During the first 24 hours of admission, his hemoglobin level dropped to 7.9 g/dl, requiring a blood transfusion. Bleeding from a Meckel's diverticulum was suspected and a radionuclide scanning with technetium-99m pertechnetate suggested ectopic gastric mucosa in the pelvic inlet [Figure 1].

Explorative laparoscopy revealed a Meckel's diverticulum, which was excised [Figure 2A, 2B]. Histopathologic assessment confirmed that the Meckel's diver-

ticulum 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.

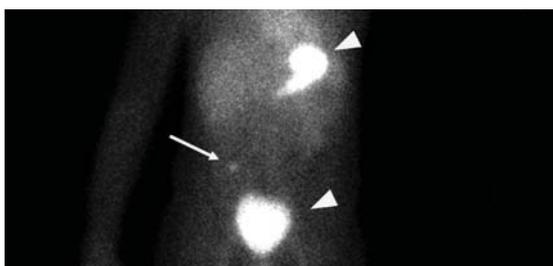
**Figure 2. [A]** Endoscopic view of an anti-mesenteric Meckel's diverticula



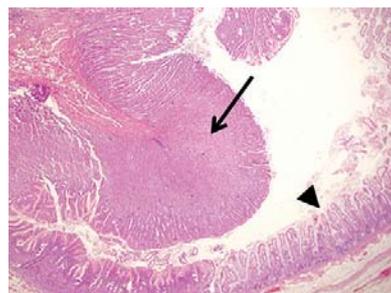
**[B]** A 1 × 1 × 2 cm Meckel's diverticula excised from the intestine



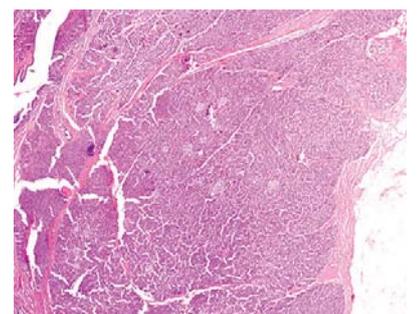
**Figure 1.** Technetium-99m (Tc-99m) pertechnetate scan in a healthy 11 year old male presented with bloody stools, indicating increased uptake in the right lower quadrant (arrow). Normal uptake of the Tc-99m nuclide is seen in the stomach and bladder (arrowheads)



**Figure 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 diverticula 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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### Capsule

#### 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, **Beutier** 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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### Capsule

#### 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 (hβ2m)-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-B7/hβ2m-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 disulfide-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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