NSAID induced perforated peptic ulcer in a pediatric sickle cell patient

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Peptic ulcer disease is a relatively rare entity in the pediatric population. Given the trend toward multimodal pain control for pain crises in Sickle Cell Disease patients, they are at an increased risk of developing complications secondary to peptic ulcer disease. We discuss a case of a Sickle Cell Disease patient on multimodal therapy that presented with a perforated peptic ulcer requiring emergent surgery. While multimodal therapy helps ease the dependency on narcotic pain medication, it does present other potential problems like potential bleeding or perforation. For those that can be categorized in this select group of patients, routine surveillance with esophagogastroduodenoscopy should be considered for those at highest risk to prevent devastating complications.

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Peptic ulcer disease in children is relatively rare compared to the adult population with an estimated prevalence of 8.1% in Europe and 17.4% in the US. It is more common in the second decade of life and is frequently attributed to H. pylori infection and the use of nonsteroidal anti-inflammatory drugs (NSAIDs) [1]. Patients with Sickle Cell Disease are at increased risk for developing gastroduodenal ulcers secondary to intrinsic disease mechanisms in addition to medication related side effects. Pain crises result from episodic sickling events leading to vaso-occlusion with ischemia and subsequent infarction of tissues, which further weakens host defenses [2]. These pain crises are a common cause of hospitalization in this patient population, and adequate pain control can be difficult to achieve. Multimodal medications are utilized in this group of patients including opioids, salicylates, and NSAIDs, which places them at a significant risk of developing ulcerations relative to the general population [3]. Our case report discusses one such patient with a perforated peptic ulcer in the setting of Sickle Cell Disease and NSAID use.

1. Case report

A 13-year old African American male with a past medical history significant for sickle cell anemia (hemoglobin SS disease) and constipation presented to the Medical University of South Carolina with a one week history of abdominal pain. The character of his pain was consistent with that of prior pain crises until the evening of presentation when he experienced a sudden onset of worsening very sharp abdominal pain located in the epigastric region. He was taken to a nearby hospital where a CT scan of the abdomen demonstrated a moderate amount of pneumoperitoneum. At that point the patient was transferred to our hospital for further care. Of note, the patient reported that he has been taking naproxen (220 mg tablets) twice daily in combination with his narcotics for general pain relief. He was also taking stool softeners and Miralax for management of chronic constipation.

At the time of initial examination at our institution, the patient appeared critically ill, was febrile (39 °C), tachycardic (heart rate of 122), respiratory rate of 24, blood pressure of 120/74, and oxygen saturation of 95% with supplemental oxygen at 2 L/m. His abdominal exam was remarkable for generalized distention but soft. No clear evidence of acute peritonitis. Labarotary evaluation was significant for WBC 12.7, hemoglobin 7.2, and platelet count 724. Basic metabolic panel was unremarkable.

Initial management included fluid resuscitated with intravenous fluids and packed red blood cells (20 cc/kg). Due to the presence of pneumoperitoneum, patient was taken to the operating room for...
diagnostic laparoscopy. Operative findings were significant for major inflammatory changes in the area of the pylorus. The proximal duodenum and antrum of the stomach were adherent to the undersurface of the liver with omentum around it. There was a large amount of purulent fluid through the entire abdomen. The colon was very dilated but no areas of colonic or small bowel perforation were identified. Intra-operative Esophagoduodenoscopy was performed while the laparoscope was focused on the duodenum and pyloric region which allowed us to confirm the presence of a perforation at that site with air extravasation during insufflation. On the intraluminal examination, there was a large friable ulceration of the gastric mucosa in the pre-pyloric location. The inflammatory phlegmon around the pylorus was so significant that we could not place laparoscopic sutures to close the area of perforation. For that reason, the procedure was converted to an exploratory laparotomy via limited upper midline incision. Careful inspection of the stomach, pylorus, and duodenum revealed the presence of an inflammatory phlegmon 5 cm in diameter at the pre-pyloric region with a 2 mm pin-hole perforation in the center with active bile extravasation, consistent with perforated pre-pyloric ulcer (Fig. 1). A couple of non-absorbable closing sutures were placed at the perforation site and a Graham patch was performed with placement of the great omentum over the perforated ulcer site. The peritoneal cavity was copiously irrigated. A nasogastric tube was positioned in the stomach just proximal to the perforation, and a blake drain was placed next to the repair. Cultures were obtained of the peritoneal fluid.

Postoperatively the patient did not experience any complications. He underwent an upper GI contrast study on postoperative day five that demonstrated mucosal inflammatory changes in the pre-pyloric region, no contrast extravasation, and no obstruction of the gastric outlet. His NGT was removed at that point and his diet was advanced as tolerated. The patient was discharged from the hospital on postoperative day seven. The patient underwent repeat esophagoduodenoscopy on postoperative day twenty-five that demonstrated a well-healed pre-pyloric ulcer (Fig. 2).

Fig. 1. Intraoperative EGD image demonstrating the pre-pyloric ulcer.

Fig. 2. Postoperative EGD image demonstrating a healed pre-pyloric ulcer 4 weeks after the acute event.

2. Discussion

Peptic ulcer disease is very rare in the pediatric population. Pediatric patients that develop this problem are at risk for the type of complications like perforation and bleeding seen in adult patients. Comprehensive review of the medical literature demonstrated multiple series looking at peptic ulcer disease in children, mostly attributable to H. pylori infections, medications, and stress related ulcerations [3–7]. There were only 2 reports, one case report and a case control study that specifically examined peptic ulcer disease in sickle cell patients [8,9]. Oftentimes when children do present with complications like perforation, they are in extremis. A high index of suspicion is required to promptly treat these children to prevent deleterious consequences. Fortunately, extensive operations are usually not required in this patient population and laparoscopic and/or open patch repair has been demonstrated to be safe and effective [4,7,10]. Patients at high risk for peptic ulcer disease include the critically ill, steroid users, those unknowingly infected with H. pylori, NSAID users, and sickle cell patients. Our patient tested negative for H. pylori and had risk factors of sickle cell anemia in addition to heavy NSAID use as a multimodal agent during his pain crises. He tested negative again for H. pylori during his follow up endoscopy. The delay in diagnosis of our patient was due to his chronic pain, as his pain crises often began with abdominal pain. In addition, given the history of severe constipation the patient received more laxatives and the primary care providers believed that the constipation was contributing to his abdominal pain. Interestingly, he did not demonstrate peritonitis on physical exam which correlates with the intraoperative findings of a fairly well contained perforation, with minimal bile and enteric contents spillage. The intraoperative appearance of his pre-pyloric ulcer was one of a chronic ulcer with an acute perforation, and he had not previously been treated with a proton-pump inhibitor or histamine blocker. It is likely that our patient had developed a chronic ulcer which subsequently developed an acute perforation.

Non-narcotic, multimodal agents are an integral part of a well-designed pain regimen for sickle cell patients to prevent narcotic dependence. Review of the literature fails to yield novel therapies for prevention of perforated peptic ulcers other than routine use of proton pump inhibitor or histamine blocker therapy in people with known ulcers and preventing ulcer formation with smoking.
cessation and minimal use of NSAIDs. For this particular patient population where NSAID therapy is a mainstay of decreasing their reliance on narcotic pain medication, minimizing adverse effects with a mandatory proton pump inhibitor regimen as well as scheduled surveillance endoscopy is the best method to reduce this risk. Another option is incorporating the use of Carafate which is routinely used in our adult bariatric population for anastomotic ulcers when identified. NSAID therapy needs to be discontinued once ulcers are identified and other strategies for appropriate pain control need to be instituted at the discretion of the primary physician managing the pain regimen. A low threshold for perforated peptic ulcer disease should be present in this population to prevent their acute pain crisis from masking a serious underlying condition as in our patient in this case report.

References


