Ammonia Enema-Induced Recto-Colonic Necrosis, A Case Report
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Citation

Abstract
Chemical burn injuries of the gastrointestinal tract are devastating injuries requiring prompt identification and intervention. These injuries are most commonly due to ingestion of liquid alkali corrosives resulting in liquefaction necrosis of the alimentary tract. Chemical burn injuries to the rectum and colon due to corrosive enemas are quite rare. The determination of the extent of the injury and initiating the appropriate surgical treatment can be challenging. This case study illustrates the necessity of early surgical intervention when a caustic agent is introduced via enema to the rectum and colon.

CASE REPORT
A 23-year-old woman, who recently emigrated from West Africa, was brought to the emergency department by her boyfriend complaining of rectal pain and bleeding. Her boyfriend stated that two hours earlier she had used a turkey baster to “clean herself” with an ammonia enema. The patient was evaluated a few days earlier in a psychiatric crisis unit and initiated on mirtazapine, an antidepressant. She had a history of drug abuse with crack cocaine and marijuana, and of alcohol abuse. She denied any previous suicide attempts.

On physical examination, a thin individual expressing mild physical and emotional distress was encountered. The patient had a low-grade fever and stable vital signs. The abdominal examination was remarkable for left lower quadrant pain, localized and continuous in character, without signs of peritonitis. Rectal examination was significant for bright red blood. There was no evidence of ammonia injection into her vagina.

The white blood count was 10,100 cells/µL/cu mm with 81.8% segmented neutrophils and 9.6% lymphocytes. A computed axial tomography scan of her abdomen and pelvis demonstrated a thick-walled fluid-filled descending colon, sigmoid and rectum compatible with distal colonic ileus.

A small amount of ascites was noted. There was no evidence of free intra-peritoneal air to suggest perforation. A flexible colonoscopy was urgently performed and the endoscope advanced without difficulty to 30cm. There was diffuse erythema, friability, and hemorrhagic areas consistent with inflammation. The procedure was terminated at 30cm due to the severity of the inflammation and patient discomfort.

The following morning the patient was noted to have increasing abdominal pain, fever of 102°F, and an increase in the leukocytosis. She underwent a limited proctoscopy that showed obvious necrosis starting at the dentate line. The patient underwent an emergent exploratory laparotomy. Gross examination of the bowel clearly demonstrated signs of inflammatory changes in the wall of the sigmoid, descending and distal one-half of the transverse colon involving the pericolic fat and its mesentery. The affected portion of the colon was transected using a gastrointestinal stapler at the mid-transverse colon proximally and distally leaving a small rectal stump. A colostomy was created with the remaining portion of the transverse colon.

Figures 1, 2 and 3. Gross examination of sigmoid and distal transverse colon showing mucosal and submucosal hemorrhage and ischemic changes.
Histological examination showed mucosal and submucosal hemorrhage and necrosis covered with an acute inflammatory exudate. Dilated capillaries and thrombosis of blood vessels in the submucosa, transmural acute inflammation and acute serositis were also observed. These findings are consistent with hemorrhagic infarction and coagulation necrosis. The proximal margin of the specimen was healthy without any histological evidence of necrosis.

Figures 4 and 5. Histological examination illustrating transition zone between injured and normal mucosa. H&E Stain, 100x.
Her postoperative course was uneventful. She was discharged to her home tolerating a diet on postoperative day five. The patient was seen in the office twice without any complaints. Her stoma is functioning well without any evidence of stricture at the colostomy site. She is scheduled for colonoscopy to rule out any proximal strictures at a 6-month interval.

DISCUSSION

Multiple agents have been implicated in chemical burn injuries of rectum and colon, these range from drugs, radiographic contrast material and alcohol to more common household items like soaps, detergents and hydrogen peroxide. The history from the patient or family is paramount in determining what type of chemical was ingested. The treatment varies depending on the agent or chemical. Identification of alkali burns warrants prompt identification due to the mechanism of injury to the tissues. The burning process triggered by alkali corrosives causes dermal protein coagulation by a variety of mechanisms [3]. These noxious mechanisms include reduction, oxidation, salt formation, corrosion, protoplasmic poisoning, metabolic competition, competition inhibition, and finally desiccation. The liquefaction of the tissues allows further penetration of the alkali deeper into the tissues expanding the depth of tissue destruction. The tissues become saponified and alkali-soluble proteinates are formed facilitating further penetration of the agent. The concentration of the hydroxyl ion and the duration of the exposure determine the severity of the injury [4].

Chemical burns due to acids, as opposed to alkalis, may be limited by coagulation and eschar formation. A quantitative and qualitative measurement of the degree of exposure and damage are the key to making the correct treatment decision. Early recognition and aggressive surgical treatment are critical for patient salvage as illustrated in our recent experience. Management of these patients depends on the severity of damage ranging from patients that require emergency laparotomy to patients that can be treated medically. For medically treated patients closed surveillance based on imaging and repeated clinical exam is of paramount importance to allow diagnosis of complications requiring surgical treatment.

Chemical burn injuries of the rectum and colon due to corrosive enemas are quite rare, and therefore infrequently studied. There are a few cases from Africa involving alkali enemas and few retrospective studies reported in the literature. First known case report in US was published in 1987.

A retrospective study performed at the University Hospital of Fort-de-France evaluated caustic burns of the rectum and colon. This study concluded that caustic burns to the colon have a high mortality due to the absence of peritoneal inflammatory signs [1].

References

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