Acute segmentary ulcerative duodenitis induced by Streptococcus pyogenes mimicking Inflammatory Bowel Disease

Dear Sir,

Infection with Streptococcus pyogenes is common and worldwide in distribution. This pathogen is responsible for a variety of severe diseases (i.e., invasive infections and nonpurulent sequelae) which originate approximately 500,000 deaths each year as a whole. We report, to our knowledge, the first case of acute ulcerative duodenitis as a nonsuppurative complication of S. pyogenes infection.

A 65-year-old woman was seen in the emergency department for severe abdominal pain, vomiting, and fever over the last 24 h. Ten hours before appearing with these complaints she had noticed malaise and sore throat. On admission, her temperature was 38.7 °C, her blood pressure was 112/75 mm Hg, and her heart rate was 88 beats/min. Physical examination was remarkable for pharyngeal redness, enlarged hyperemic tonsils, and increased epigastric pain upon palpation, although there was neither parietal muscular spasm nor rebound tenderness. Laboratory results were relevant for white blood cell count of 17.3 × 10⁹/L (90% neutrophils), C-reactive protein of 42.4 mg/dL (0.5 mg/L), and creatinine serum level of 1.19 mg/dL. Findings from chest radiography were normal. Abdominal ultrasonography showed an epigastric, lengthened mass-like formation. Computed tomography (CT) scan of the abdomen demonstrated severe thickening and marked luminal stenosis of the third duodenal segment (Fig. 1, Panels A and B); small and large bowel had a normal appearance. Esophagogastroduodenoscopy revealed an abnormal third duodenal portion, which showed wall edema, erythema, spontaneous hemorrhage, and multiple superficial and deep ulcerations (Fig. 1, Panel C); several duodenal biopsies were taken. Blood samples for culture were also obtained. She was started on intravenous piperacillin/tazobactam (4 g every 8 h) and an initial “watchful waiting” attitude was adopted. Three days after admission, surprisingly, S. pyogenes was isolated from blood cultures. A subsequent echocardiogram showed no abnormalities. Afterwards, duodenal wall biopsies revealed ulcerations and the presence of a mucosal and submucosal inflammatory infiltrate predominantly lymphocytic, which was mainly made up of CD3+ cells (T-lymphocytes) (Fig. 1, Panel D). Additionally, CD8+ T-lymphocyte (suppressor/cytotoxic T cell) appeared as the predominant phenotype of intraepithelial lymphocytes (Fig. 1, Panel E). Granuloma, vasculitic signs, or neoplastic cells were not seen. Gram staining, culture for Helicobacter pylori, and molecular tests for cytomegalovirus, enterovirus, Epstein–Barr virus, herpes simplex virus 1 and 2, herpes virus 6, 7 and 8, and Mycobacterium tuberculosis were all negative. Biopsied material was also subjected to polymerase chain reaction amplification of the 16S rRNA gene to investigate the possible presence of bacteria and the result was negative. Autoantibodies, anti-Saccharomyces cerevisiae antibodies, and serology for HIV infection were negative. Outcome was favorable under antibiotic treatment as the sole treatment and she was discharged asymptomatic on the seventh hospital day. Three days later, a control endoscopy revealed mild duodenal edema. Six months later, abdominal CT, gastroduodenoscopy, colonoscopy, and laboratory results were normal and she remained well. She goes on symptom free after 24 months of follow-up.

This clinical observation is a reminder that S. pyogenes bacteremia, an invasive streptococcal infection, can present with acute abdominal pain as the main symptom. We are unaware of any other report of acute ulcerative duodenitis caused by this microorganism. In our patient, bacteremia was probably a rare complication of her acute pharyngitis. As there were no predominant neutrophilic infiltration, abscesses, or other purulent collections and the duodenal microbiologic studies were negative, we hypothesize that duodenitis could have been caused not by the microorganism itself, but rather by suppressor/cytotoxic T-lymphocytes activated in response to certain streptococcal components (exotoxin and others). A similar mechanism has been proposed to explain a case of protein-losing gastroenteropathy associated with S. pyogenes-induced toxic shock-like syndrome. Given that acute ulcerative duodenitis can occur as a complication of S. pyogenes disease, clinicians should consider this possibility when attending patients with acute abdominal pain.

Conflict of interest

None.

References

Figure 1  Diagnostic procedures performed in a patient with acute ulcerative duodenitis induced by *Streptococcus pyogenes*. Panels A and B. Abdominal computed tomography (CT) scan images. Coronal (A) and sagittal (B) CT views showing a thick-walled, stenotic, third duodenal segment (arrows). Panel C. Upper gastrointestinal endoscopy revealing wall edema, white-yellowish exudate, luminal stenosis, and multiple ulcerations depicting a "cobblestone" appearance on the third duodenal portion. Panels D and E. Immunohistochemical study carried out on duodenal biopsy samples. The inflammatory infiltrate, a predominantly lymphocytic one, is mainly made up of CD3+ cells (T-lymphocytes) (D). CD8+ T-lymphocyte (suppressor/cytotoxic T cell) appears as the predominant phenotype of intraepithelial lymphocytes (E) (de, duodenal epithelium; du, duodenal ulcer).


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