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Phlegmonous gastritis associated with invasive *Pseudomonas aeruginosa* infection

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Dear Editor,

We report the case of a 49-year-old female that was hospitalized due to a recent diagnosis of acute lymphoblastic leukemia. As a consequence of induction chemotherapy (CALGB 10403 scheme), she developed severe neutropenia (0.04 10^3/ul) and infectious prophylaxis was started (acyclovir and anidulafungin). On day 6 of chemotherapy, she complained of epigastric pain, fever, coffee ground emesis and melena. Physical exam showed gingival and soft palate necrosis. Auxiliary examinations showed a high C reactive protein (14.5 mg/dl, ULN 1 mg/dl) and an abdominal computed tomography (CT) scan revealed generalized gastric thickening and generalized contrast enhancement (Fig. 1A and B). An upper endoscopy showed pangastritis with friable mucosa, superficial and deep ulcers and pus (Fig. 1C and D). Gastric biopsies cultures were negative for fungi and mycobacteria. Broad-spectrum antibiotics (meropenem and vancomycin) were initiated and surgery was consulted, but she rapidly developed multiple organ failure and died.

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Necropsy was performed that revealed hyperemic and swollen edematous gastric folds, and multiple erosions. Microscopic evaluation of gastric mucosa is shown in figure 1E and F. Tissue cultures of numerous organs and tissues (liver, spleen, lung, tongue, and palate) were positive for *Pseudomonas aeruginosa (P. aeruginosa),* therefore the etiology of phlegmonous gastritis was considered.

Discussion

Phlegmonous gastritis consists of purulent inflammation of the gastric wall. Immunosuppression is a risk factor and is frequent in these patients (1). It has been reported in patients with neoplasia and those receiving induction chemotherapy (2,3). Overall mortality is up to 27 % (1). The most commonly involved pathogen is *Streptococcus* spp., with few reported cases of *P. aeruginosa* (2,4). The systemic compromise of *P. aeruginosa* infection and the different mechanisms of resistance to broad-spectrum antibiotics that it possesses could have contributed to the patient's outcome (5).

Phlegmonous gastritis should be suspected in cases of sudden onset epigastric pain, vomiting, upper gastrointestinal bleeding, and significant wall thickening of the stomach on imaging. Antibiotic therapy should be started early, which contributes to improving outcomes (1).

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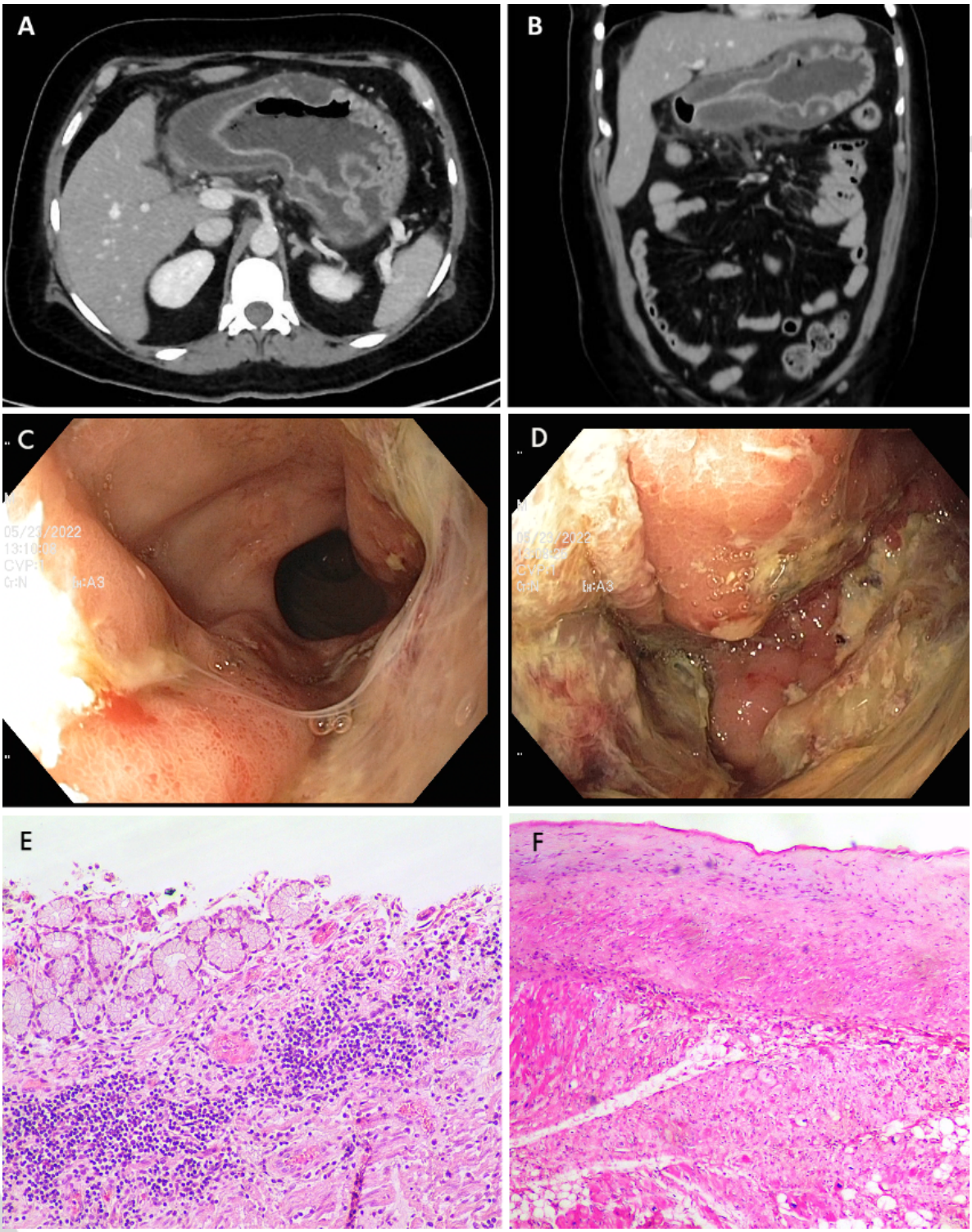

Fig. 1. A (axial) and B (coronal). Abdominal computed tomography with intravenous contrast showed generalized gastric wall thickening, submucosal edema and diffuse

mucosal enhancement. C and D. Upper endoscopy images (Olympus 180, Evis Exera II) with white light imaging. C. Frontal view at the antrum showing congestive antral mucosa, friability and yellow exudates resembling pus. D. Frontal view of the mid-gastric body, showing congestive mucosa and deep ulcers with necrosis and yellow exudates. E and F. Microscopic evaluation of the gastric mucosa (hematoxylin and eosin, 20x). E. Loss of the oxyntic epithelium with reactive epithelial changes, erosions, ulceration and a dense inflammatory cell infiltrate. F. The epithelium has been replaced by necrosis and fibrin deposition. There is an inflammatory cell infiltrate with scattered neutrophils.