CASE PRESENTATIONS

PARTICULARITIES OF MYCOTIC GASTRITIS IN CHILDREN

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ABSTRACT

Mycotic gastritis is still an open chapter in specialized research due to the small number of cases described in literature, to its incompletely known complications and prognosis, which creates therapeutic difficulties. The authors report the case of an immune-competent 5 years and 4 months old male child, in whom upper digestive endoscopy revealed eso-gastro-duodenitis caused by *Aspergillus*.

Keywords: mycotic gastritis, child

INTRODUCTION

Acute gastritis has numerous causes, among which medicine and alcohol abuse, duodenal-gastric bile reflux, ischemia, different bacterial, viral or fungal pathogenic agents, acute stress (shock), ionizing radiations, allergies, food poisoning, direct trauma. The common disease triggering mechanism for all these etiologies is the imbalance between the aggressive and defensive factors (1).

Fungi involvement in gastric lesions has been disregarded, as the emphasis was laid on their causing various cutaneous and pulmonary conditions. Few cases are described in literature in which fungi caused gastritis (2,3), ulcer (4,5) and even gastric perforations (6).

PURPOSE OF THE PAPER

The purpose of the paper was to describe a case of gastritis with severe evolution due to *Aspergillus* infection, which was ameliorated by antimycotic treatment.

CLINICAL CASE

A 5 years and 4 months old male child, of normal height and weight, with no significant personal

history of disease, was urgently hospitalized as he suffered from coffee ground vomitus and violent epigastralgia.

Clinical examination upon hospitalization: altered general condition, suffering face, normal looking elastic teguments, normal cardiac and pulmonary stethacoustic parameters, normally structured oral cavity, soft depressible abdomen, which moves up and down with the breathing and which becomes painfully either spontaneously or when palpating the epigastrium, normal stools, coffee ground vomitus, physiological urination, psychomotor agitation, no signs of meningeal irritation.

Anamnesis: the symptoms occurred suddenly, about 6 hours before the patient's coming to hospital, first by violent epigastralgia, followed by coffee ground vomitus. The symptoms were not correlated with the food the patient had eaten. No mycosis risk factors were identified, as the mother denied having given him antibiotics or packaged fast-food.

The results of the routine examinations – full blood count, transaminases, direct and indirect bilirubin, iron, calcium, magnesium, glycemia, urea, amylase – were within normal limits. IgG, IgA, IgM – within normal limits. AntiHIV1/HIV2 antibodies – negative.

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Digestive endoscopy revealed mycotic eso-gastro-duodenitis with considerable gastric mucosa lesions (Fig. 1 and 2).

The culture on Sabouraud agar medium revealed the presence of fungus belonging to the genus Aspergillus. We have no more advanced technical conditions to achieve the type of mycosis.

The histopathological exam detected an inflammatory infiltrate including many lymphocytes and plasma cells.

The disease had a positive course thanks to the Itraconazol and hepatoprotective medications ad-

ministered for 4 weeks, and the follow-up digestive endoscopy was within normal limits.

The administration of proton-pump inhibitors (Omeprazol) together with Itraconazol was even considered, but recent research has contraindicated the association of these medicines, as Itraconazol absorption is dependent on gastric acidity. The authors suggest that a transitional gastric pH reduction may be achieved by administering an acid solution (for instance, coke) (13).

The specificity of the case consists of the occurrence of extensive gastric mycotic lesions, which caused hemorrhage in an immune-competent host.





Figure 1. Mycotic lesions (Aspergillus) in the antral area





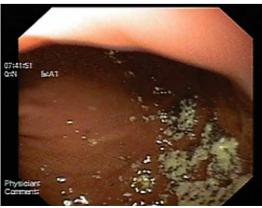


FIGURE 2. Mycotic lesions (Aspergillus) in the corporeal area

DISCUSSIONS

Fungi have been detected in the oral-pharyngeal cavities of almost 40% of the apparently healthy population (7). Fungi have high prevalence rates in food, water and air, which means that they may be carried into the stomach by food and saliva intake. In a study conducted on 30 autopsied dead bodies, fungi have been found in the stomach contents of 20 of them (8).

Fungi gastritis is mainly due to *Candida albicans* infection (which is the most commonly reported etiology). *Histoplasma, Aspergillus or Actinomyces* may rarely affect the stomach. The predisposing factor to these diseases is the patient's immunodeficiency. A case of gastric perforation due to acute *Aspergillus* gastritis was reported in a patient suffering from severe aplastic anemia (9).

Candida albicans rarely adheres to the gastric mucosa, and when it is isolated in the stomach, it most commonly appears as an ulceration or eroded mucosal fold of the stomach.

The digestive functional syndrome includes two distinct entities, namely recurrent abdominal pain and vomiting.

Abdominal pain is the most common clinical manifestation of gastritis, yet it can only be certainly diagnosed after the age of 3 years. The main symptom in infants and babies is vomiting. It may be connected to nutrition and it is accompanied by agitation, crying and it may cause anorexia and dystrophy. Vomiting is quasi constant before the age of 3 and 6 years (10). Persistent diarrhea is among the most common transit disorders.

Digestive hemorrhage may lead to anemia and it may be revealed by systematic checks for occult hemorrhage in the stool and by clinical hints such as progressive pallor, chronic fatigue, cephalalgia, dyspnea.

The clinical diagnosis cannot differentiate mycotic infection from other gastric lesions.

Upper digestive endoscopy is currently the golden standard in gastritis diagnosis. It is safe and

efficient, sensitive and specific, and it allows the detection of associated lesions and biopsy sampling (11). A hyperemic mucosa with erosions covered by cheese-like exudate or whitish pseudo-membranes should be suggestive of mycotic infection.

A positive diagnosis of mycotic infection should be set only if there are 10 colonies per plate in the first culture (12).

Fungi gastritis complications may be severe. Typical mycotic ulcer usually occurs on the small curvature of the stomach. There may be only one ulcer or there may be several lesions. The size of these ulcerations may vary from punctiform ulcerations to ulcerations that may involve the whole stomach.

Such ulceration may progressively penetrate the muscles or even the serous membrane. A case of gastric perforation causing peritonitis (6) and a case of perforation followed by fistulae formation (14) have been reported in literature.

When ulcerations cause perforation or recurrent hemorrhages, the surgical approach is the only solution.

In the few cases described in literature and solved by surgery there were no complications that may be attributed to the mycotic infection itself.

There are not enough cases of mycotic gastritis reported in literature to justify a prognosis.

CONCLUSIONS

Fungi may cause gastritis, ulcer or even gastric perforations, their clinical manifestations consisting of symptoms with no specificity. Extended mycotic lesions may also occur in an immune-competent patient. Hemorrhage is a possible complication, which may be treated either by medication or even by surgical procedures.

Itraconazol therapy was efficient in *Aspergillus* gastritis.

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