Eosinophilic Gastroenteritis (EG) was originally described by Kaijser in 1937, as a disorder characterized by tissue eosinophilia that can affect different layers of the bowel wall, anywhere from mouth to anus. Primary eosinophilic gastroenteritis is defined as disorders that primarily affect the gastrointestinal tract with eosinophil-rich inflammation in the absence of known causes for eosinophilia such as parasitic and bacterial infections (including Helicobacter pylori), inflammatory bowel disease, hypereosinophilic syndrome, myeloproliferative disorders, chronic granulomatous disease, collagen vascular disease, drug injury, and drug hypersensivity.


The GE diagnosis necessarily requires histological (biopsy or surgical specimen) or cytological (on ascites) confirmation of abnormal eosinophilic infiltrate.

The majority of the authors have considered as cut-off for the diagnosis a minimum number of eosinophils exceeding 20 x high-power field. The diagnosis is difficult due to the presence of patchy distribution and to the absence of characteristic endoscopic pictures.

In clinical suspicion is therefore recommended multiple mucosa biopsies also seemingly unscathed.


In 1970 Klein classified EG into three categories: a mucosal, muscular and serosal form.

The mucosal form (the most common variant) is characterized by vomiting, abdominal pain, diarrhea, blood loss in stools, iron-deficiency anemia, malabsorption and protein-losing enteropathy.

The muscularis form is characterized by infiltration of eosinophils predominantly in the muscularis layer, leading to thickening of the bowel wall, which might result in gastrointestinal obstructive symptoms mimicking pyloric stenosis or other causes of gastric outlet obstruction.

In these cases, the presence of allergy or food intolerance is rare.


The serosal form occurs in a minority of patients with eosinophilic gastroenteritis, and it is characterized by eosinophilic ascites.

We report a case of a 34 year old man with diffuse abdominal pain, not painful on palpation deep and shallow, with a negative sign Blumberg and Murphy. There was no remarkable feature in the physical examination. The abdomen ultrasound examination showed an abundant Douglas effusion. Red blood cells: 4.350.000/mm3, hematocrit: 41,70%, leukocyte: 6.900/mm3 (neutrophil: 78,90%, eosinophil: 2,80%, lymphocyte: 13,10%), platelet: 244.000/mm3 was counted in the blood examination. Liver and renal functions were in normal range. Parasitologic examination and bacterial culture of stool were normal. The abdomen CT scan without contrast confirmed the pelvic cavity effusion. The omental adipose thickening plans was evident, mostly in the epigastric and left subfrenica sites, and in gastro-hepatic ligament. Esophagogastroduodenoscopy (EGD) revealed distal esophagitis. In the body and in the antrum, the stomach showed hypertrophic plicae and poorly distensible. The plicae mucosa was normal, as well as duodenal mucosa. Antrum and duodenum biopsies were completed. The laparoscopic investigation confirmed the presence of ascites and omentum edema. The gastric wall appeared edematous and congested. At last, biopsy of the gastric wall and omentum, ascites fluid withdrawal and peritoneal washing were performed. The antrum, the duodenum, the gastric wall and the omentum anatomic specimens were sampled and stained with haematoxylin-eosin. The ascites fluid and the peritoneal washing were stained with haematoxylin-eosin and papanicolau.

**MATERIAL AND METHODS**

Gastric mucosa H&E 10X

Duodenal mucosa H&E 10X

Peritoneal washing PAP 20X

Ascites fluid H&E 20X
RESULTS

Gastric mucosa biopsies showed a moderate chronic gastritis, slightly active, not atrophic. Research of Helicobacter Pylori was negative. Duodenal mucosa was regular. The cytological ascites examination and peritoneal washing showed mesothelial cells, some lymphocytes and numerous eosinophils. Gastric wall biopsy showed an intense muscular and serosal inflammatory infiltration composed of eosinophils. The fatty tissue fragment corresponding to the omentum showed focal mesothelial cells hyperplasia and nonspecific chronic inflammation with numerous eosinophils marginalized also into the vessels.

CONCLUSION

Eosinophilic Gastroenteritis is a rare disease, its symptoms are related to the organ or to the organ affected area. The EG diagnosis necessarily requires histological and cytological confirmation of abnormal eosinophilic infiltrate. The majority of the authors have considered as cut-off for the diagnosis a minimum number of eosinophils exceeding 20 per high-power field. Our case presents in gastric muscle layer and in peritoneal serous intense eosinophilic infiltration in accordance with the criteria described in literature.