ECRONICON OPEN ACCESS

EC GASTROENTEROLOGY AND DIGESTIVE SYSTEM

Case Study

Primary (Isolated) Eosinophilic Colitis. Clinical Case

Saifutdinov RG[1]*, Dvorjankina TV[2], Saifutdinov RR[1,2], Trefilova YS[1] and Saifutdinova TV[1]

[1]KSMA - Branch Campus of the FSBEI FPE RMACPE MOH Russia, Kazan, Russia

[2]City Clinical Hospital № 12, Kazan, Russia

***Corresponding Author:** Saifutdinov RG, Doctor of Medical Sciences, Head of the Department of Hospital and Polyclinic Therapy, KSMA - Branch Campus of the FSBEI FPE RMACPE MOH Russia, Kazan, Russia.

Received: January 16, 2024; **Published:** March 04, 2024

Abstract

A clinical case of a man with the most common form (mucosal variant) is presented primary (isolated) eosinophilic colitis with damage to the mucous membrane of the colon. The disease was manifested by diarrhea, malabsorption, and enteropathy with loss of protein. The diagnosis was made on the basis of intestinal symptoms, eosinophilia in peripheral blood, a normal picture of the colon mucosa, the results of histological examination of the biopsy - eosinophilic infiltration of the colon mucosa, with careful exclusion of other causes of eosinophilia.

Keyword: *Eosinophilic Colitis; Primary; Isolated; Clinical Case; A Man*

Abbreviations

AAD: Antibiotic-Associated Diarrhea; ALT: Alanine Aminotransferase; AO: Abdominal Organs; AST: Aspartate Aminotransferase; ARVI: Acute Respiratory Viral Infection; AVHC: Antibodies to Viral Hepatitis C; Bas: Basophils; BBA: Biochemical Blood Analysis; BlCr: Blood Creatinine; BMI: Body Mass Index; BP: Blood Pressure; CCH: City Clinical Hospital; Cin: Color Indicator; CRP: C-Reactive Protein; DU: Duodenum; EC: Eosinophilic Colitis; ECG: Electrocardiography; EGID: Eosinophilic Gastrointestinal Disorders; EIA: Enzyme Immunoassay; Eo: Eosinophils; FCS: Fibrocolonoscopy; FGDS: Fibrogastrduodenoscopy; FoV: In the Field of View; FRM: Frequency of Respiratory Movements; GBT: General Blood Test; GIT: Gastrointestinal Tract; GUA: General Urine Analysis; Hb: Hemoglobin; Hbs: Antigen; HD: Hypertension Disease; HIDV: Human Immunodeficiency Virus; HR: Heart Rate; Ht: Hematocrit; IBD: Inflammatory Bowel Diseases; LEKT: Leukotrienes; Lym: Lymphocytes; MC: Microscopic Colitis; Mon: Monocytes; MRS: Micro-Reaction to Syphilis; NSAID: Nonsteroidal Anti-Inflammatory Drugs; PPI: Proton Pump Inhibitors; PT: Platelets; RBC: Red Blood Cells; RES: The Rate of Erythrocyte Sedimentation; RHMA: Recombinant Human Monoclonal Antibodies; Seg: Segments; ST: Sticks; Ur: Urea; USE: Ultrasound Examination; WBC: White Blood Cells

Introduction and Case Study

Patient I., 58 years old, was hospitalized in the gastroenterological department of the city clinical hospital (CCH) No. 12 on 05/15/2023 with complaints of frequent, up to 5 times a day, unformed watery stools without pathological impurities, occurring 15 - 20 minutes after any meal. He denies nighttime urge to defecate. Weight loss of 15 kg during the month (from mid-April 2023) due to a decrease in the number of meals and the volume of food.

Anamnesis morbi: Until December 2022, complaints and symptoms from the intestine did not bother. In mid-December 2022, weakness appeared, fever increased to subfebrile values, chest congestion, which is why he started taking azithromycin for 3 days - 1 capsule (500 mg). After taking azithromycin, the nature of the stool changed, watery diarrhea appeared up to 3 times a day, then a few days after stopping taking the antibiotic, the stool returned to normal. In the middle of April 2023. again, according to the words, he suffered an acute respiratory viral infection (ARVI): there was abundant catarrhal discharge from the nose. He took teraflu and antiviral drugs as prescribed by the therapist. A week after the start of taking the drugs, the stool again became frequent, watery, up to 3 - 4 times a day. He took antidiarrheal medications - without effect. He was sent to the CCH No. 12 for further examination and treatment.

Anamnesis vitae: He works as a welder in metrostroy. He lives in Kazan with his wife. She has two healthy daughters. His father died at the age of 59 from lung cancer, his mother - at the age of 80, suffered from hypertension (HD) and she was obese. There are no allergic reactions. Previous illnesses: Hepatitis A at the age of 17, chronic gastritis, colds - no more than 2 times a year. Bad habits: does not smoke, drinks alcohol no more than 1 time in 3 months, no more than 200 ml of vodka. He served in the army in Ukraine in the missile forces.

Status praesens objectives: Height: 176 cm, weight: 69 kg, body mass index (BMI) = 22 kg/m^2. The condition is satisfactory, the consciousness is clear. The skin and visible mucous membranes are physiologically colored. The peripheral lymph nodes are not enlarged, there is no swelling.

Respiratory system: No special features, frequency of respiratory movements (FRM) - 18 per minute. Breathing is vesicular, there is no wheezing.

Cardiovascular system: The area of the heart does not appear to be changed. The boundaries of the heart are within normal limits. The heart tones are of normal sonority. The heart rate (HR) is 72 per minute. Pulse of satisfactory filling, blood pressure (BP): 100/60 mmHg.

Digestive system: The tongue is overlaid with a thick white coating on the back. The belly is soft, painless. The size of the liver according to M.G. Kurlov is normal (9x8x7 cm), the spleen is not enlarged.

The urinary system is without features. F.I. Pasternatsky's symptom is negative on both sides. Urination is painless, diuresis is normal.

The following laboratory and instrumental studies were carried out (normal clinic indicators are indicated in parentheses).

General blood test (GBT) from 05/15/23: Erythrocytes (Er): 5.2x10^{12}/l (3.7-4.7x10^{12}/l), hemoglobin (Hb): 151 g/L (130 - 160 g/L), color index (Cin): 0.87 (0.85 - 1.05), platelets (Pt): 286x10^9/L (200-400x10^9/L); Hematocrit (Ht): 47% (35 - 50%); white blood cells (WBC): 11x10^9/L (4.0-9.0x10^9/L, eosinophils (EO): 12% (0 - 5%), sticks (St): 1% (1 - 6%), segments (Ceg): 58% (47 - 72%), lymphocytes (Lym) - 21% (18 - 38%), monocytes (Mon): 8% (3 - 11%), the rate of erythrocyte sedimentation (RES): 6 mm/h (2 - 15 mm/h).

GBT from 05/18/2013: Er: 4.7x10^{12}/L, Hb: 141 g/L, Cin: 0.89, Pt: 239x10^9/L; Ht: 42.5%; WBC: 8.5 x 10^9/L, basophil (Bas): 1%, Eo: 23%, St: 11%, Ceg: 38%, Lym: 21%, Mon: 6%, RES: 7 mm/h.

GBT from 05/26/23: Er: 5.1x10^{12}/L, Hb: 149 g/L, Cin: 0.87, Pt: 253x10^9/L; Ht: 45.8%; WBC: 12.5 x 10^9/L, Bas: 1%, Eo: 23%, St: 3%, Ceg: 48%, Lym: 22%, Mon: 3%, RES: 3 mm/h.

General urine analysis (GUA) from 05/15/23: quantity - 100 ml. specific gravity - 1030, color - straw yellow, acidic reaction, slight sediment, protein - 0, bilirubin +, Leu 1-2 in the field of view (FoV), Er. 0 in FoV, flat epithelium 2-4 in FoV.

GUA from 05/26/23: quantity - 100 ml. specific gravity 1020, light turbidity, color - straw yellow, acidic reaction, slight sediment, protein - insignificant traces, Leu 1-2 in the field of view (FoV), Er. 1-2 in FoV, flat epithelium 1-2 in FoV.

Biochemical blood analysis (BBA) from 05/15/23: total bilirubin: 7.5 mmol/l (5 - 21 mmol/l), direct bilirubin: 3 mmol/L (3.4 mmol/L), indirect bilirubin: 4.5 mmol/L (1.7 - 17.0 mol/L), urea (Ur): 4,7,7 - 7,8 mm (7.2 mmol)/L), total protein: 61 g/L (66 - 83 g/L), alanine aminotransferase (ALT): 27 Units/L (< 40), aspartate aminotransferase (AST): 21 (< 40), blood creatinine (BlCr): 9 mmol/L (59 - 104 mmol/L).

BBA from 05/16/2013: calcium: 1.14 mmol/L (1.05 - 1.3 mmol/L), potassium: 4.53 mmol/L (3.5 - 5.5 mmol/L), sodium: 143 mmol/L (135 - 155 mmol/L), blood urea: 4.2 mmol/L (2.8 - 7.2 mmol/l), CRP: 6.7 mg/l (up to 5 mg/l).

Micro-reaction to syphilis (MRS) from 05/15/23: negative. Antibodies to HIDV from 05/18/2013: not detected. Enzyme immunoassay (EIA) dated 05/16/23: Hbs antigen and antibodies to viral hepatitis C were not detected.

General analysis of feces (coprogram 05/16/23: Shape and density - liquid; Color brown, Mucus - no, Blood - no, Pus - no, Worm eggs were not detected, Fat +, Starch +, Residues of vegetable food 0-1, Residues of digested vegetable fiber 1-4, Bacteria +, Yeast cells ++, Blood (Gregersen reaction) is negative.

General analysis of feces (coprogram) 05/22/23: Shape and density - liquid; Color light brown, Mucus - no, Blood - no, Pus - no, Worm eggs were not detected, Fat +++, Starch +++, Residues of vegetable food 0-2, Residues of digested vegetable fiber 2-3, Bacteria ++, Mucus ++, Yeast cells ++, Blood (Gergersen reaction) is negative.

Analysis for fecal dysbiosis 05/16/23: the number of bifidobacteria was reduced to 10^5 (10^9-10^{10}) and lactobacilli to 10^6 (10^7-10^8), the growth of conditionally pathogenic microflora to 10^5 (<10^4).

Ultrasound examination (USE) of the abdominal organs (AO) 05/19/2013: Liver: The dimensions of the right lobe are 147 mm (140 mm), the diameter of the left lobe is 74 mm (70 mm). The contours are smooth. The structure is homogeneous, the echogenicity is slightly increased. The intrahepatic bile ducts are not dilated. The vascular pattern has not been changed. Gallbladder: Dimensions: 55x27 mm (length - from 5 to 12 cm; width - from 2 to 3.5 cm; wall thickness in the uncut state - 2 mm, shortened - 3.5 mm). The walls are thickened. The contents are inhomogeneous. Concretions - single up to 12mm; Holedoh 5 mm (up to 5 mm). The portal vein is 10 mm (< 12 mm). Pancreas: The size is not enlarged. The contours are smooth. The echogenicity is hyperechoic. The echostructure is homogeneous (the head of the pancreas is 18 - 30 mm, the body is 7 - 20 mm, the tail is 7 - 25 mm. The diameter of the Wirsung duct is 1 mm). Spleen: 109x49 mm (square no more than 55 mm^2). The contours are smooth. The structure is homogeneous. Focal changes: not detected. The splenic vein is 5 mm (up to 6 mm). No free liquid has been detected.

USE AO 29/05/23: Liver: The size of the right lobe is 141 mm, the diameter of the left lobe is 70 mm. The contours are smooth. The structure is homogeneous, the echogenicity is slightly increased. The intrahepatic bile ducts are not dilated. The vascular pattern has not been changed. Gallbladder: Dimensions: 63x27 mm. The walls are thickened. The contents are inhomogeneous. Concretions - single up to 12mm; Choledoch 5 mm. The portal vein is 10 mm. Pancreas: the size is not enlarged. The contours are smooth. The echogenicity is hyperechoic. The echostructure is homogeneous. Spleen: 112x52 mm. The contours are smooth. The structure is homogeneous. Focal changes: not detected. The splenic vein is 5 mm. No free liquid has been detected.

FGDS 07/04/23: The pear-shaped sinuses are free, the act of swallowing is not violated. The esophagus is freely traversed throughout. The mucous membrane is without features, the cardiac pulp closes completely. A jagged line at the level of the anatomical cardia. Stomach: the lumen contains a meager amount of light liquid, mucus. During inversion, the endoscope is completely covered by the cardia. The mucous membrane is hyperemic. The folds of the mucous membrane are elastic. The gatekeeper is not deformed, it closes, we pass through. The bulb of the duodenum (DU): pink mucous membrane, folds of medium size. The postbulbar department is passable, the mucous membrane is without features.

Fibrocolonoscopy (FCSC) 30/05/23: Bowel preparation according to the Boston School 5 points (1-2-2). The rectum, sigmoid, descending, transverse colon, ascending were examined. There is a large amount of cloudy liquid contents in the lumen. The mucous membrane is of normal color, the vascular pattern is erased in places, the gaustration is preserved, corresponds to the anatomical localization. A biopsy was taken from different sites: ascending - 1, transversum - 2, descending - 4, sigmoid - 4. Endoscopic conclusion: Chronic (Eosinophilic?) colitis.

The result of cytological examination: Ascending intestinal mucosa: Reactive changes in the intestinal epithelium. By background: fibrin. Mucosa of the transverse colon: Proliferation of intestinal epithelium. By background: fibrin, eosinophilic leukocytes. The mucosa of the descending intestine: Reactive changes in the intestinal epithelium. By background: fibrin, eosinophilic leukocytes. Sigmoid colon mucosa: Proliferation of goblet cells. By background: fibrin, eosinophilic leukocytes. There are no violations of the architectonics of crypts, focal damage to the surface epithelium, pronounced diffuse inflammatory, lymphoplasmocytic with a large number of eosinophils, neutrophils. Conclusion: exacerbation of chronic colitis. More data for eosinophilic colitis.

ECG from 05/16/23: sinus rhythm with a heart rate of 65 beats per minute. The electrical axis of the heart is horizontal. Low-amplitude ECG from the extremities.

Differential diagnosis

Initially, doctors suspected antibiotic-associated diarrhea (AAD). Because, for the first time, it appeared in the patient against the background of the use of the antibiotic azithromycin. In addition, this diagnosis was supported by the fact that the patient did not have diarrhea at night and there were no “red flags” of this disease (inflammatory reaction in the blood (leukocytosis, shift of the blood formula to the left, acceleration of RES).

The next diagnosis was a rotavirus infection occurring with diarrhea. But this disease was rejected by doctors, because the viral infection was in doubt, no one in the family was sick at the same time with him, and the symptoms of diarrhea appeared against the background of an improvement in the condition. Diarrhea on the background of COVID-19 was also rejected. The SARS-CoV-2 virus was not detected.

Diseases such as celiac disease, Whipple’s disease, Gordon’s disease, ulcerative colitis, Crohn’s disease, and especially diarrhea caused by infectious diseases (salmonellosis, dysentery, etc.) were excluded, because there were no corresponding “key” symptoms.

Since the patient had liquid feces and weight loss of 14 kg in 1 month, there were no visual changes in the large intestine at the FCS, microscopic colitis was excluded. However, women are more likely to suffer from this disease, in old age, their stools are more often and more liquid, almost watery.

As we can see from the laboratory tests presented above, eosinophilia of 12% was detected in the clinic in the first GBT (norm: 0 - 5%). Subsequently, it reached 23%. A parasitic infection has been ruled out. This made it possible to suspect eosinophilic colitis, which was confirmed by the morphology of the biopsy material, where eosinophils were found in almost all parts of the colon (Figure 1).

On 06/14/23 a consultation was conducted on this patient by employees of the Department of Hospital and Polyclinic Therapy of the KSMU - branch of the Federal State Budgetary Educational Institution of the Russian Ministry of Health with doctors of the gastroenterological department of the City clinical hospital No. 12. The diagnosis was made: primary (isolated) eosinophilic colitis.

The administration of metronidazole (250 mg x 3 times a day) and 5-ASA (sulfasalazine 1 g/day) quickly improved the patient’s condition: the stool returned to normal the next day.

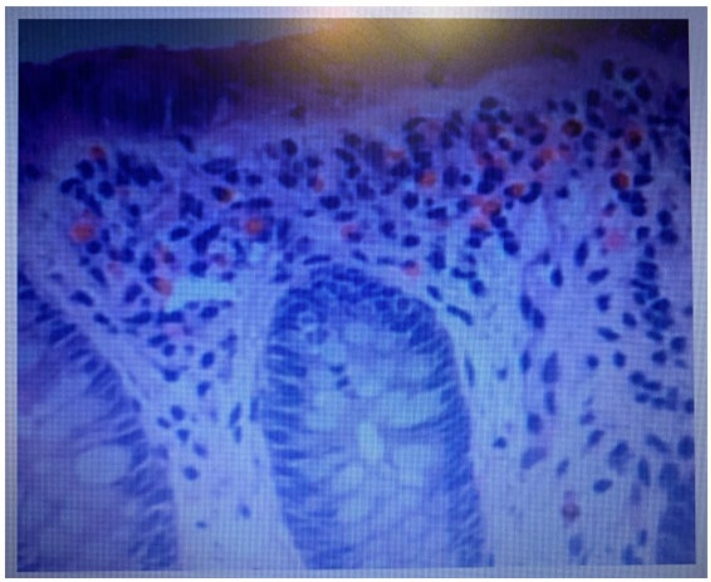

Figure 1: *Eosinophilic colitis. Cytology (hematoxylin-eosin staining, ×400).*

Discussion

Eosinophilic colitis (EC) is an inflammatory bowel disease caused by eosinophilic infiltration of the colon wall [14]. The latter develops in various disorders, including food allergies, parasitic infestations and inflammatory bowel diseases. However, cases of the primary form of EC are presented only in a few reports [5,16,21].

The EC was first described in 1937 by R. Keeper [3]. The etiology of primary EC is unknown. The interaction between genetic and environmental factors is assumed [16,21]. Intestinal eosinophilia can develop a second time in celiac disease and inflammatory bowel diseases [15] autoimmune diseases and systemic connective tissue diseases [(scleroderma, vasculitis, Charg-Strauss syndrome, Tolosa-Hunt syndrome)] [4].

Secondary causes of EC are: helminthiasis (trichocephalosis, enterobiosis and strongyloidosis) [15], medications (clozapine, carbamazepine, rifampicin, gold preparations, naproxen, tacrolimus, etc. [4,18].

Close attention is paid to drugs that cause various pathophysiological processes. In this regard, an interesting analogy can be drawn with microscopic colitis (MC). Thus, Beaugerie and Pardi proposed a score scale with varying degrees of confidence in the participation of a particular drug in the development of microscopic colitis [8]. Drugs that are highly likely to "trigger" MC: acarbose, aspirin, cyclo-3-fort, lansoprazole, NSAIDs, ranitidine, sertraline, ticlopidine, PPIs. Some scientists are puzzled by the fact that there is still no such thing as drug-associated MC. In most drugs considered as trigger factors of MC, one of the side effects is the development of diarrhea. The more often colonoscopy with biopsy is performed in patients with drug-associated diarrhea, the more often MC is established [9].

It should be noted that drug EC has been described in response to clozapine, carbamazepine, rifampicin, NSAIDs, cyclosporine and gold preparations [16,18,20].

Our patient also took various medications to relieve his painful condition (azithromycin, NSAIDs, antiviral drugs).

EC has a different frequency by age and clinical features in children and young adults (20 - 25 years old) [1,2]. According to the prevalence of eosinophilic infiltration, EC can occur with damage to the entire large intestine or in individual segments of the colon. Our patient had a lesion of the entire large intestine.

There are no clear clinical manifestations that distinguish isolated colon disease from widespread eosinophilia involving the esophagus, stomach and small intestine. Possible clinical manifestations of eosinophilic colitis: abdominal pain; weight loss; malabsorption; bloody and non-bloody diarrhea; eosinophilia (in a general blood test); eosinophilic ascites; intestinal obstruction (inversion, invagination); exudative enteropathy (enteropathy with loss of protein); infiltration and perforation of the colon wall; prolonged fever. However, the clinical manifestations of EC depend mainly on damage to the layers of the colon wall by eosinophilic infiltration [5].

The most common form of EC is the mucous variant. It is associated with damage to the mucous membrane and is manifested by diarrhea, malabsorption and enteropathy with loss of protein. What our patient had. The transmural variant is less common, characterized by thickening of the colon wall and manifests itself acutely, sometimes with acute intestinal obstruction (intussusception or inversion) or by perforation [12]. A very rare form of serous EC is accompanied by ascites, eosinophils predominate (95%) in ascitic fluid [17].

Endoscopic changes in EC, such as focal erythema, depletion of the vascular pattern and superficial ulceration, are nonspecific. In some cases, the mucous membrane looks completely normal (as in our patient). A biopsy is essential for the diagnosis, which makes it possible to detect eosinophilic infiltration of the colon mucosa [7].

Biopsy examination usually demonstrates layers of eosinophilic infiltration into the lamina propria of the mucous membrane of the colon, less often with spread to the submucosal and muscular layers. Other histological findings include eosinophilic microabscesses, eosinophilic cryptitis and intraepithelial eosinophils located mainly in the surface layers [6]. Multiple biopsies are needed (as in MC), since eosinophilic infiltration not only spreads unevenly in EC, but also the number of eosinophils normally has a wide range in various segments of the colon, demonstrating a proximal-distal distribution of 35 eosinophils in the cecum with a decrease to 8 - 10 eosinophils in the rectum (in the field of microscopy of a large magnification×200). Unlike the esophagus, eosinophils are resident cells of the mucous membrane of the small and large intestine, however, the normal number of eosinophils is not clearly defined, which makes it difficult to interpret the biopsy results [13].

Some consensus has been reached on the diagnostic criteria for eosinophilic esophagitis, namely the presence of more than 15 eosinophils (in the field of view at magnification ×200) in the squamous cell mucosa of the esophagus [11]. However, the diagnostic criteria of EC are not generally recognized, although most authors used the diagnostic threshold of 20 eosinophils during microscopy of a biopsy of the colon mucosa (×200) [13]. It should be noted that the normal values of tissue eosinophils vary greatly in the mucous membrane of various segments of the colon, increasing in the proximal direction from less than 10 eosinophils in the rectum to more than 30 eosinophils (×200) in the caecum [7]. Thus, the location of the biopsy is also important for interpreting the results.

There is limited information about the drug therapy of EC, as it is a rather rare phenomenon that makes it difficult to conduct randomized clinical trials.

In EC, prednisolone is administered orally in dosages similar to those used for inflammatory bowel diseases (IBD), with an initial dose of 40 - 60 mg/kg (1 - 2 mg/kg per day) for 8 weeks. Then the dose is gradually reduced, followed by complete withdrawal of the drug after 6 - 8 weeks. In most studies, the oral administration of prednisolone improves clinical symptoms and pathomorphological data in patients with EC [2].

Immunosuppressants are prescribed for severe, refractory or steroid-dependent EC [19]. The combination of glucocorticoids and azathioprine can reduce eosinophilic infiltration of the gastrointestinal mucosa and control diarrhea in EGID [6].

The leukotriene receptor antagonist Montelukast selectively blocks the At1 receptors of cysteinyl leukotrienes (LTC4, LTD4 and LTE4). It reduces the effect of leukotrienes, mediators of chronic inflammation, blocks the migration and chemoattraction of eosinophils in many tissues, including the gastrointestinal tract (GI tract). Montelukast is prescribed orally at a dose of 10 - 40 mg per day, the course of therapy is up to 3 - 4 months. The drug normalizes the parameters of eosinophils in peripheral blood and relieves gastroduodenal symptoms in children with eosinophilic infiltration of the duodenal mucosa [6]. However, the role of montelukast in EC has yet to be evaluated.

Stabilizers of mast cell membranes. Ketotifen blocks the calcium channels necessary for their degranulation. This stabilizes the cell membrane and prevents the release of histamine and other mediators of allergy and inflammation. It also blocks H1-histamine receptors, suppressing the accumulation of eosinophils in tissues. The course is 12 months and is an alternative to steroid therapy for EC [6]. Sodium cromoglycate may have certain prospects for induction of remission and maintenance therapy in patients with EC. But, it should be noted that it is not effective in eosinophilic esophagitis. Studies concerning the use of mast cell membrane stabilizers in EC have not been conducted [6].

Therapy with biological drugs. Omalizumab, a selective immunosuppressant, is a recombinant human monoclonal IgG1k antibodies that selectively bind to human IgE, preventing unexpected anaphylactic reactions by limiting the release of mediators [6]. Omalizumab is administered subcutaneously at a dose of 150-375 mg every 2 weeks for 8-16 weeks. Mepolizumab is a human monoclonal antibody to interleukin-5 (IL-5), a key cytokine that stimulates the growth and activity of eosinophils. It is administered intravenously 750 mg every 2 weeks for 16 weeks. Mepolizumab significantly reduces eosinophilic infiltration in eosinophilic esophagitis [10]. However, further clinical studies are needed to assess the prospects of human monoclonal antibodies in EC.

The forecast for EC. Originated in early childhood, it has a good prognosis and tends to spontaneous recovery, often within a few days. Unlike children, EC in adults usually has a chronic course with periods of moderate activity and remission [16].

Conclusion

1. There are no recognized criteria for evaluating a diagnostically significant eosinophilic infiltrate in EC.
2. The upper range of the normal number of eosinophils in the colon mucosa is not clearly defined.
3. There is no consensus on the diagnosis and therapy of primary (isolated) EC.

Bibliography

1. Kayurova MS. "Eosinophilic colitis". *Medicine: Theory and Practice* 4.1 (2020): 112-120.
2. Nemtsov LM., *et al*. "Eosinophilic pathology of the large intestine". *Bulletin of the Volgograd State Medical University* 17.4 (2018): 7-21.
3. Sovalkin VI., *et al*. "Eosinophilic enterocolitis: clinical observation". *Therapeutic Archive* 87.2 (2015): 80-84.
4. Ahrens R., *et al*. "Eosinophilic colitis accompanied by Tolosa-Hunt syndrome: report of a case". *Journal of Gastroenterology* 38.6 (2003): 613-614.
5. Alfadda AA., *et al*. "Eosinophilic colitis: an update on pathophysiology and treatment". *British Medical Bulletin* 100 (2011): 59-72.
6. Alfadda AA., *et al*. "Eosinophilic colitis: epidemiology, clinical features, and current management". *Therapeutic Advances in Gastroenterology* 4.5 (2011): 301-309.

7. Bates AW. "Diagnosing eosinophilic colitis: histopathological pattern or nosological entity?" *Scientifica (Cairo)* (2012): 682576.

8. Beaugerie L. "Review article: drug-induced microscopic colitis - proposal for a scoring system and review of the literature". *Alimentary Pharmacology and Therapeutics* 22.4 (2005): 277-284.

9. Beaugerie L and Pardi DS. "Patients with drug-induced microscopic colitis should not be included in controlled trials assessing the efficacy of anti-inflammatory drugs in microscopic colitis". *Gastroenterology* 137.4 (2009): 1535-1539.

10. Foroughi S., *et al*. "Anti-IgE treatment of eosinophil-associated gastrointestinal disorders". *Journal of Allergy and Clinical Immunology* 120.3 (2007): 594-601.

11. Furuta GT., *et al*. "Eosinophilic esophagitis in children and adults: a systematic review and consensus recommendations for diagnosis and treatment". *Gastroenterology* 133.4 (2007): 1342-1363.

12. Jimenez-Saenz M., *et al*. "Bleeding colonic ulcer and eosinophilic colitis: a rare complication of nonsteroidal anti-inflammatory drugs". *Journal of Clinical Gastroenterology* 40.1 (2006): 84-85.

13. Lee CK and Kim HJ. "Primary eosinophilic colitis as an unusual cause of chronic diarrhea". *Endoscopy* 42.2 (2010): E279-E280.

14. Macaigne G., *et al*. "Eosinophilic colitis in adults". *Clinics and Research in Hepatology and Gastroenterology* 44.5 (2020): 630-637.

15. Mehta P and Furuta GT. "Eosinophils in gastrointestinal disorders: eosinophilic gastrointestinal diseases, celiac disease, inflammatory bowel diseases, and parasitic infections". *Immunology and Allergy Clinics of North America* 35.3 (2015): 413-437.

16. Okpara N., *et al*. "Eosinophilic colitis". *World Journal of Gastroenterology* 15.24 (2009): 2975-2979.

17. Ong GY., *et al*. "Eosinophilic gastroenteritis involving the distal small intestine and proximal colon". *Chang Gung Medical Journal* 25.1 (2002): 56-61.

18. Saeed SA., *et al*. "Tacrolimus-associated eosinophilic gastroenterocolitis in pediatric liver transplant recipients: role of potential food allergies in pathogenesis". *Pediatric Transplantation* 10.6 (2006): 730-735.

19. Rothenberg ME. "Eosinophilic gastrointestinal disorders (EGID)". *Journal of Allergy and Clinical Immunology* 113.1 (2004): 11-28 quiz 29.

20. Velchuru VR., *et al*. "Eosinophilic colitis". *Journal of Gastrointestinal Surgery* 11.10 (2007): 1373-1375.

21. Yan BM and EA Shaffer. "Primary eosinophilic disorders of the gastrointestinal tract". *Gut* 58.5 (2009): 721-732.

Volume 11 Issue 3 March 2024