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EosinophilicAscitesDuetoEosinophilicGastroenteritis:ACaseReportand Review

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1. Abstract

Eosinophilic gastroenteritis (EGE) is a rare disease characterized by the presence of gastrointestinal symptoms with an abnormal eosinophilic infiltrate of the intestinal wall and exclusion of other causes of secondary eosinophilic infiltration. EGE has various gastrointestinalsymptomsdependingonthedepthofeosinophilic infiltration of the bowel wall: mucosal, muscular, or subserosal.In particular, EGE with ascites is uncommon among them, and mainlyappearedinsubserosaltype.Wehaveexperiencedacaseof diagnosingandeffectivelytreatingEGEaccompaniedwithascites in a 40-year-old woman.

A 40-year-old woman visited the emergency room presenting withabdominalpainanddiarrheawithadurationofapproximate- ly three weeks. Laboratory test results showed leukocytosis of 11,900/mm3witheosinophilia(5,378/mm3,45.2%),andhighserumlevelofIgE(1,484IU/mL).OnabdominopelvicCT,longsegment edematous wall thickening of stomach to colon and a large amountofascitesinpelviccavityweredemonstrated.Paracentesis was performed, and eosinophilia was observed in ascites. On endoscopy, there was diffuse mucosal edema in the entire stomach, duodenum,andcolon.Randombiopsieswereperformed,andsignificant eosinophil infiltration was identified. Parasitic infection wasexcludedthroughstoolexamination,andtherewerenomeaningfulresultsinbloodtestsincludingfoodallergentestsandANA, ANCA, C3 and C4.

The patient had a history of eosinophilic gastroenteritis, and she wasthoughttohaverecurrenceofeosinophilicgastroenteritiswith ascites, therefore steroid administration was begun (prednisolone 40mg/d).Withinoneweek,symptomsandeosinophilcountimproved,andimprovementofintestinaledemaandasciteswerealso observed in follow-up CT.

2. Introduction

Eosinophilic Gastroenteritis (EGE) is a rare disorder of unknown etiology with an abnormal eosinophilic infiltrate of the intestine wall [1]. It can present with various gastrointestinal symptoms depending on the depth of eosinophilic infiltration of the intestinal wall, and is classified into 3 subtypes as follows: mucosal, muscular,andsubserosal[2,3].Itsmainsymptomsareabdominal pain, nausea, vomiting, diarrhea, weight loss, etc, [4]. EGE with ascites appears in the subserosal type, which is known to be the rarest form, representing approximately 10% of the total eosinophilicgastroenteritis[5].Confirmationofeosinophilicinfiltration in gastrointestinal wall through biopsy is essential of diagnosis. Peripheral eosinophilia appeared in most cases, but it is not an essentialfactorfordiagnosis,andotherdiseasesthatcausesecond- ary eosinophilic increases should be excluded [1].

Wehaveexperiencedacaseofdiagnosingandeffectivelytreating eosinophilic gastroenteritis with ascites in a 40-year-old female patient who complaining of abdominal pain

3. Case Report

A40-year-oldwomanvisitedtheemergencyroompresentingwith abdominal pain and diarrhea that lasted about three weeks. She also complained of nausea and intermittent vomiting, accompanied by mild abdominal distention. Blood pressure was 112/71 mmHg, heart rate was 103 beats per min, respiratory rate was 20 breaths per min, and oxygen saturation was 97% in room air. On physical examination, there was diffuse tenderness of the whole abdomen,butrigiditywasnotobserved.Shewastaking methima-

zole for hyperthyroidism and had a history of hospitalization for eosinophilicgastroenteritis6yearsprior.Shedidnothaveallergic conditions such as asthma, rhinitis, or food intolerance.

Initial laboratory test results showed leukocytosis of 11,900/mm3 with eosinophilia (5,378/mm3, 45.2%), high serum level of IgE (1,484 IU/mL), eosinophil count (9,340/mm3, normal range 50~500/mm3) and eosinophilic cationic protein (ECP >200 ng/mL). Other blood tests showed no significant abnormalities. Abdominopelvic CT showed long segment edematous wall thickeningofstomachtocolon.Additionally,therewasalargeamountof fluidcollectioninsubhepaticspaceandpelviccavitywithmesentericinfiltration(Figure1).Diagnosticparacentesiswasperformed and approximately 1Lwas drained.Ascitic fluid was translucent, andWBCwas5600/mm3(eosinophilcell91%,mononuclearcell 7%, and lymphocyte 1%). No malignant cells were detected and culture of the ascetic fluid was negative.

Upper endoscopy revealed diffuse mucosal edema in the entire stomachandduodenum.Randombiopsieswereperformedateach segmentofstomachandduodenum.Colonoscopyshoweddiffuse mildmucosaledemaintheentirecolon,andrandombiopsieswere performed (Figure 2). Biopsy results showed significant eosinophilinfiltrationwasobservedintheentireintestine,especiallycolon(morethan30/highpowerfield)(Figure3).Parasiticinfection was excluded through stool test and specific antibody tests, and therewerenospecificfindingsinthefoodallergenskinpricktest. In addition, blood test including antinuclear antibody,ANCA, C3 and C4, and peripheral blood smear were performed, and results were not significant.

The patient was diagnosed with recurrent eosinophilic gastroenteritis with ascites, therefore oral corticosteroid administration was begun (prednisolone 40 mg/day). Blood testing after 3 days showed eosinophil cell count decrease 2.7% (initial 45.2%), and after5days,patient'snauseaandabdominalpainsymptomswere also improved. The eosinophil count was also decreased to 150/ mm3 (50~500 mm3), within normal range.

Abdominopelvic CT was performed on the 14th day of hospitalization, and diffuse edematous wall thickening from stomach to colon was significantly decreased. The substantial amount of ascites seen in subhepatic space and pelvic cavity were no longer visible(Figure4).Afteraweekofsteroidadministration,thedose was reduced 30 mg/day, and it was planned to be tapered every 7 daysforthe2-monthfollow-upperiod.Aweekafterdischarge,the eosinophil count was in the normal range in outpatient care, and the patient complained of no symptoms.

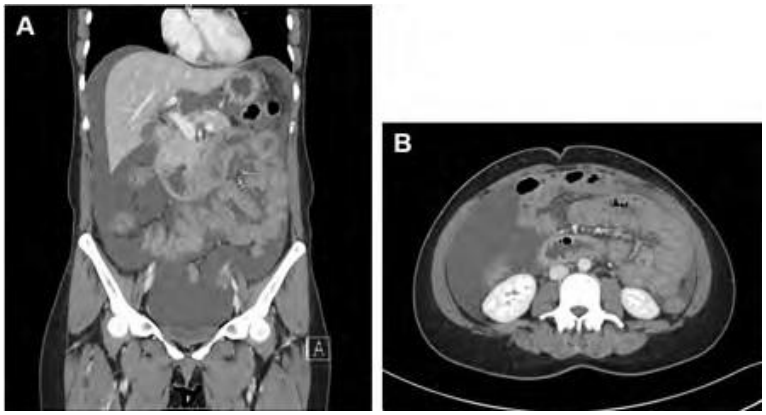

Figure 1: Abdominopelvic CT(A, transverseview; B, coronal view) Long segmental edematouswall thickening of stomach~colon. Large amountof fluidcollectioninsubhepatic&pelviccavitywithmesentericinfiltration.

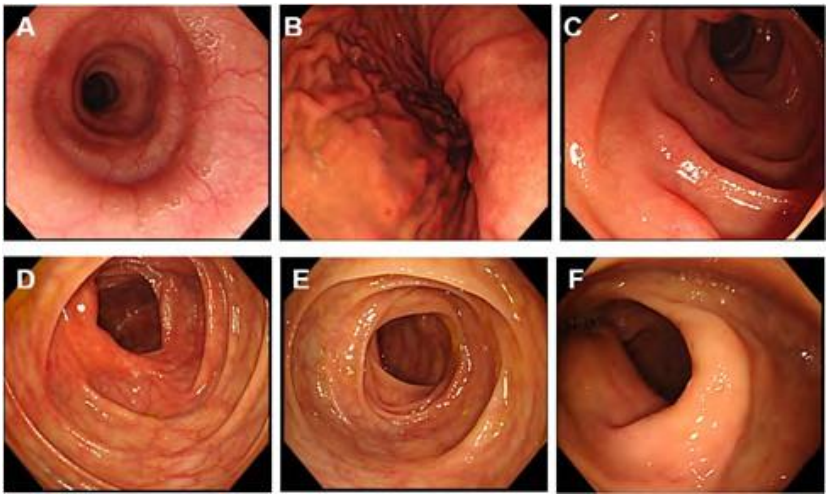

Figure2: Endoscopicfindings(A,esophagus;B,stomachbody;C,duodenum;D,A-colon;B,T-colon;C,Rectum).Diffusemucosaledemainthe entire esophagus, stomach, duodenum and colon

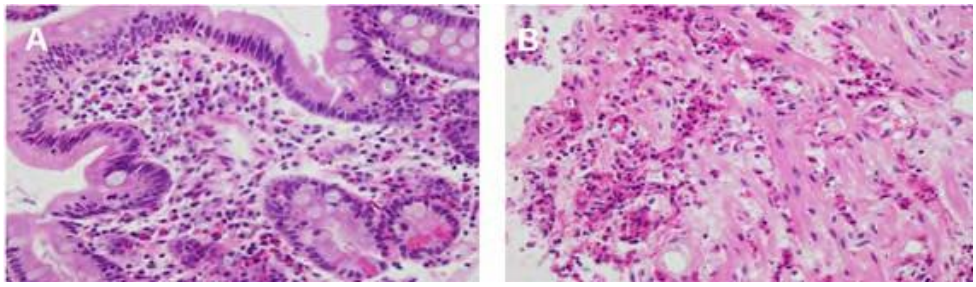

Figure 3: Histopathologic findings (A, duodenum; B, colon). Eosinophil infiltration was observed in the entire intestine, especially colon (more than 30/high power field)

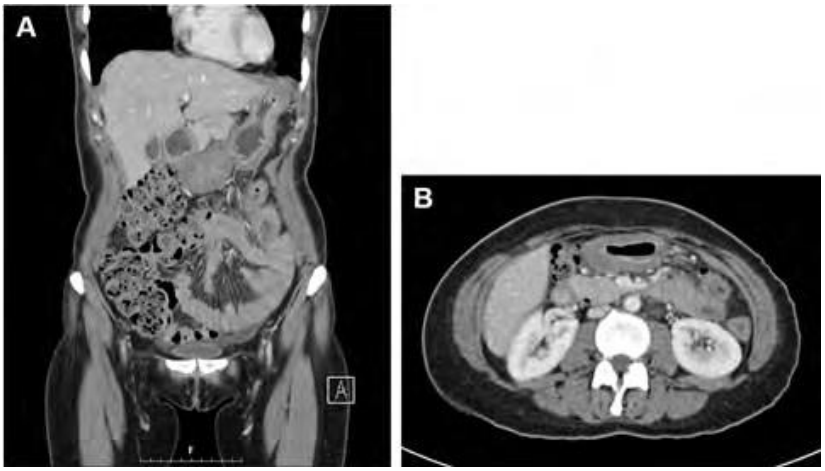

Figure4: AbdominopelvicCT(A,transverseview;B,coronalview).Decreasedintheextentofedematouswallthickeningofstomach~colon. Disappearanceoflargeamountoffluidcollectioninsubhepaticspace&pelviccavity

4. Discussion

EGE is a rare disease characterized by the presence of gastrointestinal symptoms with an abnormal eosinophilic infiltrate of the intestinal wall and exclusion of other causes of secondary eosinophilic infiltration [3]. In particular, EGE with ascites is uncommon, so we are reporting this case in comparison with previously reported20similarcases(Table1).6-20EGE'sprevalenceisfrom 8 to 24 per 100,000 and most patients are diagnosed between 30-50yearsofage[6].Thepatientinthiscasewasalsoa40-year-old woman, and 13 of the 20 cases compared were in this age range.

The etiology remains obscure, but there are many reports that cytokines such as IL-4 and IL-5 which activate eosinophil are related [4]. In addition, the fact that EGE patients often accompanied allergic diseases such as asthma, rhinitis, or food intolerance also provides clues to the etiology [1]. Of the 20 cases we compared,7 patients had allergic diseases. Although patient in our case did not have a history of allergic disease, the possibility of allergy to methimazole is being evaluated.

Basedonthedominantlayeroftheintestinalwallinfiltratedbyeosinophil,EGEisdividedintothreetypes:mucosal,muscular, and subserosal (Klein classification) [3, 7]. The most common form (70%) is the mucosal infiltration type, with symptoms related to dysfunction of the mucosa with malabsorption and protein-losing enteropathy (diarrhea, steatorrhea, weight loss, hypoalbuminemia).Themusclelayerinfiltrationtypeaccountsforabout20% ofthetotalEGE.Thistypeisassociatedwithlocalizedordiffused bowelwallthickening,withfeaturesofpyloricnarrowingandobstructivesymptoms(colickypain,nausea,orvomiting).Thethird type is subserosal infiltration type, characterized by ascites richin eosinophils. This type is reported to be about 10% of the to-tal eosinophilic gastroenteritis, ant it is known to occur mainly in women of childbearing age, such as the patient in our case [3, 8].

Verifyingeosinophilicinfiltrationingastrointestinalwallbybiop- sy is essential for diagnosing EGE, and peripheral eosinophilia is notnecessarilyobserved.But,peripheraleosinophiliaisobserved in most EGE patients, and it was found in all 20 cases compared, including our case. In this regard, one of the things to be careful aboutinthediagnosisofEGEistheneedtodifferentiatedoth- er diseases that cause secondary peripheral eosinophilia, such as parasiticinfection(Stronglyloidessteroralis,Toxocaracanis),vasculitis (Churg-Strauss syndrome), inflammatory bowel disease, malignancy (eosinophilic leukemia, lymphoma), and hypereosinophilic syndrome [3]. In this case, we excluded these diseases throughtestssuchasstooltest,specificantibodytest,foodallergen test,bloodtestincludingantinuclearantibody,ANCA,C3,C4and peripheral blood smear.

Although the increase in eosinophils is important factor in EGE, eosinophils are also present in the normal mucosa of the gastrointestinal tract. There is no clear standard for how many eosinophilsshouldbeinfiltratedtobeconsideredabnormalyet,many

researchers have suggested that at least 20 eosinophils/HPF infiltration is necessary [5]. In our case, more than 30 cells/HPF were observed,andinall20casescompared,morethan20eosinophils/ HPF infiltration was observed.

EGEproducesmuchlessmarkedmacroscopicchanges,andsohistopathological diagnosis is essential.As in the 20 cases we compared, endoscopic exam demonstrates nonspecific findings such as thickened mucosal folds, erythema, edema, erosion, and ulcer. Multiple samplings should be performed in endoscopic biopsies, because lesions can be distributed locally [1].

MajortreatmentofEGEiscorticosteroid,whichisgenerallyeffec- tive in most patients. In most of the 20 cases, including our case, ithasalsoimprovedthroughsteroidtherapy.Therecommenddosageisdaily20~40mgprednisolonefor7~14daysandthentapered [7].Approximately80%ofpatientsimprovetheirsymptomswithin one week and eosinophil count are normalized within 2 weeks [3].Theprognosisisgoodinmostpatients,butanestimated50% of patient experience recurrence after discontinuation of steroid therapy,sometimesrequiringlong-termadministration.Apatient in our case, also had a history of EGE before, and this event was secondEGE.Onecautionbeforesteroidtreatmentisthatparasitic infections should be completely excluded, because steroid treatment in the presence of occult parasitic infection may result in catastrophic disseminated disease. Second-line therapies include antihistamines(ketotifen),mastcellstabilizers(sodiumcromoglycate),leukotrieneantagonists(montelukast),andimmunomodulators (azathioprine). As well, it has been reported in recent years that monoclonal antibody (omalizumab) developed against IgE and IL-5 is beginning to be used successfully in treatment [7-9].

Ascites in EGE is rare. There are no clear diagnostic criteria yet, andendoscopicfindingsarealsonon-specific,soitisadiseasethat canbemissedinclinicalpractice.Inparticular,ifeosinophilcount increase and ascites are observed in a patient complaining of gastrointestinal symptoms, it is necessary to distinguish this disease, and the exclusion of other diseases that cause eosinophil increase isalsoimportant.Sincemostsuchpatientsrespondwelltosteroid treatment,prognosisisfavorable.Therefore,wehopethiscasereportwillbeanopportunitytoadvancethediagnosisandtreatment of eosinophilic gastroenteritis.

Table1: Casereportsofeosinophilicgastroenteritispresentingwithascites

Reference	Age/ Sex	Symptoms	Peripheral eosinophilia	Involvedorgan	Endoscopic findings	Allergic disease	Treatment
FenogolioLM, (2003)[8]	29/F	Abdominaldistension, Diarrhea,Vomiting	+	stomach, duodenum	Hyperemia	-	PD 25mg/d
HepburnIS, (2010)[9]	20/F	Abdominal distension/ pain, vomiting, diarrhea	+	-	erythema	-	PD 40mg/d
BleibelF,(2012)[10]	55/M	Abdominaldistension/ pain	+	duodenum	hyperemia	-	PD 20mg/d
LiaoWH,(2012)[11]	43/M	Abdominal pain	+	colon	erythema	-	Spontaneous resolution
DeMatosBrasil AA, (2013)[12]	49/M	Abdominaldistension, bloating	+	Peritoneum,mesenteric lymph node	erythema	+ (rhinitis,lactose intolerance)	PD 30mg/d, Restricted diet
CukoL, (2014)[13]	37/F	Abdominaldistension/ pain, diarrhea	+	Esophagus, stomach	hyperemia	-	PD 40mg/d
BaekMS,(2014)[14]	68/F	Abdominalpain,nausea, diarrhea	+	duodenum	Hyperemia, edema,erythema	-	PD 30mg, Montelukast 10mg
CaglerE,(2015)[1]	35/M	Abdominaldistension, Nausea	+	esophagus, duodenum	edema	+ (asthma, rhinitis)	PD 40mg/d
AlsulaimanRM, (2015)[15]	28/F	Abdominaldistension/ pain	+	Stomach,duodenum, ileum, colon	edema	-	PD 40mg/d
Khalil H,(2016)[16]	25/F	Abdominaldistension/ pain, nausea, skin rash	+	Esophagus,stomach, duodenum, rectum	erythema	+ (asthma,rhinitis, dermatitis	PD 30mg/d, Restricted diet
LourencoLC, (2017)[2]	27/M	Abdominaldistension/ pain	+	stomach	none	+ (asthma, rhinitis)	PD 40mg/d
FerreiraAA, (2017)[17]	41/F	Abdominaldistension, diarrhea, anorexia	+	-	none	-	PD 40mg/d
SantosC, (2018)[18]	32/F	Abdominalpain,nausea, diarrhea	+	Ileum, colon	hyperemia	-	PD 40mg/d

ShiL,(2018)[19]	57/F	Abdominalpain/ distension	+	duodenum	Erosion, hyperemia	-	Ketotifen
SabanJ,(2019)[20]	30/F	Abdominalpain/ distension,	+	Esophagus	normal	-	PD,Restricted diet
WanFeng, (2020)[8]	26/M	Abdominaldistension/ pain, diarrhea	+	Stomach, colon	Erythema, edema	-	PD 40mg/d
El RayA,(2020)[3]	30/F	Abdominal distension, vomiting,epigastricpain	+	Esophagus,stomach, duodenum	hyperemia	+ (iron intolerance)	PD 40mg/d
Devi S,(2020)[6]	44/M	Abdominalpain/ distension	+	Esophagus, stomach	erythema	+ (asthma)	PD 40mg/d
TianXQ,(2021)[5]	34/M	Abdominalpain, diarrhea, nausea	+	Stomach, ileum, colon	Hyperemia, edema	-	PD 40mg/d
SequeiraM, (2022)[4]	24/F	Abdominal pain, diarrhea,vomiting	+	colon	Hyperemia	+ (iron intolerance)	Suspend iron supplement
Present case	40/F	Abdominal pain/ distension,diarreha	+	Esophagus,Stomach, colon	edema	-	PD 40mg/d

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