# Demographic features and diagnostic criteria of Primary Eosinophilic Colitis among adult patients with emphasis on clinicopathological correlation

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**Abstract**

**Objective**

Primary Eosinophilic Colitis (PEC) is the rarest and least understood entity in the spectrum of eosinophilic gastrointestinal diseases. This study aims at describing the clinicopathological characteristics of this disease and to explore histological criteria that can improve its diagnostic accuracy. **Methods**: In this retrospective clinico-pathological study, we describe the clinical and histological features of 22 cases of PEC. The computerized system of the Histopathology Department at Jordan University Hospital was searched for cases diagnosed as tissue eosinophilia (130 cases). The medical records were reviewed to check the final diagnosis and only cases with PEC were included. The demographic features were documented, and all the histopathological slides were reviewed and the main histological features, mainly the number of eosinophils per high power field were recorded. **Results**: 16.9% of cases of tissue eosinophilia turned out to be PEC. These were distributed equally between genders with the median age being 49.5 years. The most common presentation was diarrhea and/or abdominal pain. Colonoscopy was normal in 72.7 % of cases. 40.9% of Patients had associated allergic conditions, mostly milk intolerance, and 18.2 % had peripheral eosinophilia. Histologically there was dense eosinophilic inflammation with features of eosinophil activation including degranulation and eosinophilic cryptitis and crypt abscesses. There was no correlation between the density of the eosinophilic infiltrate and these histological features. **Conclusion**: Gastroenterologists and pathologists should keep a high index of suspicion when faced with cases of unexplained diarrhea and/ or abdominal pain. Pathologists reporting colonic biopsies should actively search for eosinophils in the colonic biopsies when faced with such history. However, diagnosis cannot be made on histological grounds alone, and clinico-pathological correlation is essential to diagnose PEC after excluding all possible secondary causes.

**Keywords**: Colon; Eosinophil; Eosinophilic Colitis; Histologic Diagnosis.

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## 1. Introduction

Primary Eosinophilic Colitis (PEC) is a rare, poorly understood disease of the colon characterized by inflammatory infiltrate rich in eosinophils. It can occur in isolation or as part of a generalized eosinophilic gastrointestinal disease (EGID) which can affect any part of the gastrointestinal tract (GIT).[1] [2].

PEC is the rarest and least understood entity in the spectrum of EGID. PEC is better described among children, especially infants. Research addressing its clinico-pathological features, pathogenesis and management among adults is scanty with only 3 case series, and 59 case reports.

There is no consensus on the criteria for diagnosing PEC, however most researchers define PEC as suggested by Tally et al [3] in1990 as the presence of tissue eosinophilia (TE) (increased eosinophils in colonic biopsies), in symptomatic patients, which cannot be explained by secondary causes of eosinophilia, such as infections, drug reactions, inflammatory bowel disease, autoimmune diseases, cancer and several other conditions [4] [5] [6] [7].

Although tissue eosinophilia is a prerequisite for diagnosis, there is no agreed quantitative definition of TE. The published literature contains several suggested thresholds ranging from as low as 20 eosinophils per high power field (HPF) [8] [9] [10], to as high as 100 eosinophils per HPF in the caecum and 64 in the rectosigmoid [11] [12]. The issue is complicated by the fact that normal numbers of eosinophils in the GIT are not known and, in the colon, there is evidence that their numbers vary according to the exact site and that their density decreases from the proximal to the distal colon [13] [14].

The etiology of PEC is also poorly understood. There is an association with allergic conditions, and it is thought that T helper 2 cells play a major role in its pathogenesis [1] [15] [16], but this wasn't adequately investigated in the literature.

The exact prevalence of PEC is not known and there are only two population-based studies, both conducted in the USA, that estimated the prevalence of this disease [17] [18]. However, incidence of PEC is thought to be increasing. Alfadda et al reviewed the number of published research papers about EC and noticed an increased number of publications since 2000 [19]. In 2013, two case series about PEC in adults were published, one as a letter to the editor describing 5 cases [10], and the other included 7 cases [9], whereas in 2017 a case series from Spain reported 22 cases [8].

The aim of this study was to describe our experience in Jordan University Hospital (JUH) of PEC among adolescent and adult patients (15 years and above) with emphasis on its demographic features, clinical and histological diagnostic criteria as well as association with peripheral eosinophilia and other allergic conditions. Such studies are required to define this rare entity and improve our understanding of its clinio-pathological features. We also compared our results with those of similar studies in the literature and highlighted the main similarities and differences.

## PATIENTS AND METHODS.

### Cases

This is a retrospective analysis of PEC cases among adolescent and adult individuals (15 years old and above) that were diagnosed at JUH between the first of January 2016 and the first of August 2018.

The computerized system in the Histopathology Department at JUH was searched for colonic biopsies diagnosed with tissue eosinophilia (130 cases). Patients' electronic clinical records were then checked to find out the final diagnosis of these patients. Tissue eosinophilia in the majority was due to secondary causes, leaving 22 patients qualifying for the diagnosis of primary EC, defined as tissue eosinophilia of at least 20 eosinophils/ HPF in the colonic biopsies of symptomatic individuals who underwent full clinico-pathological assessment to rule out secondary causes of TE. This assessment included discussion at the regular clinico-pathological meetings led by two of the authors (Awad and Abusneineh) held in the gastrointestinal unit at JUH. Because parasitic infections are relatively common in Jordan, wet

mount stool analysis is part of the routine investigation of patients with diarrhea; all the 22 cases had negative stool analysis for ova and parasites.

We excluded cases where tissue eosinophilia was due to inflammatory bowel disease, drug induced or caused by other causes of eosinophilia. Three cases were in children between 3 and 6 years and these also were excluded.

For the included 22 cases, we documented the main demographic features including age, gender, clinical symptoms, endoscopic and histological findings, stool analysis results, number of eosinophils in peripheral blood samples (peripheral eosinophilia), patients' allergic history and family history of any allergic diseases in first degree relatives, treatment and follow up information regarding improvement of symptoms and any relapses. For any missing information in patients' data, we phoned the patients directly to complete the records.

**Histological assessment.**

The Hematoxylin and Eosin-stained slides of these 22 cases were retrieved from the histopathology lab archives and reviewed by a gastrointestinal histopathologist, who counted the number of eosinophils per high power field with a 40x objective lens, resulting in a 400-fold magnification with a field diameter of 0.24 $mm^2$ using Olympus BX51 microscope. The highest and lowest counts of eosinophils per HPF were recorded as well as those of three other consecutive fields to calculate the mean of eosinophils per HPF in the 5 fields. Other histological criteria including eosinophilic cryptitis, crypt abscesses, surface involvement, eosinophil degranulation and the presence of lymphoid aggregates were also recorded.

**Statistical analysis**

The data was presented on Numbers 1.0 of apple iOS. Categorical data was presented as numbers and percentages. For the continuous data, the mean, median and standard deviation were calculated. Two tailed t test was used to compare the means of variables between genders and the correlation between number of eosinophils per HPF and several clinical and histological variables. A significant p value was considered to be $< 0.05$

**Ethical considerations**

This study was approved by the University of Jordan ethical committee and the JUH Institutional Review Board (IRB)

## RESULTS

**Demographic and clinical features**

During the study period, 130 colonic biopsies were diagnosed with TE in the histopathology department at JUH. Of these 22 cases, (16.9%) were diagnosed as PEC after full clinico-pathological evaluation. Table 1 summarizes the final diagnoses/ reasons of exclusion from the study of the rest of the cases.

The 22 PEC cases were distributed equally between males and females (male to female ratio 1:1). The age range was 15-87 years (standard deviation= 20.0) with the mean age being 46.8 and the median 49.5

The age range among the 11 male patients was 15-67 (SD= 18.7) with the mean being 42.1 and the median 50 whereas in females, the age range was 16-87 (SD =20.9) with a mean of 51.5 and a median of 49. However, there is no significant statistical difference of the age distribution between both genders as measured by the 2 tailed t test ($p = 0.28$)

Table 2 summarizes the clinical features, treatment and follow up of the 22 PEC cases. Regarding the clinical history, 7 patients presented with diarrhea (two of which were bloody), 5 with abdominal pain and 10 with both of these complaints.

Colonoscopy was normal in 16 patients (72.7%). Four patients (18.2) had signs of non-specific inflammation, namely: edema, hyperemia and loss of vascular pattern (cases 10, 13, 14, 16 in table 2). Two patients' colonoscopy studies showed cecal ulcerations (case no 12 and 21)

16 (72.7%) patients received steroids treatment, one patient refused treatment, 2 patients reported improvement of symptoms without treatment. For the remaining 3 patients,

we don't have records about their treatment.

The follow up period of these patients was between 1-24 months (average FU period 8.8 months). Patients are asymptomatic except four patients who still have gastrointestinal symptoms. One of these is still on treatment with only 1 month follow up, the second patient who didn't improve was prescribed steroids but refused treatment as she was concerned about steroids side effects (case 3). The third patient reported recurrence of symptoms with certain foods. The fourth case (case 2 in table 2) reported having symptoms when she was approached by the telephone conversation; however, her initial treatment is not recorded, and she didn't attend the GI clinic afterwards.

Nine of the 22 patients (40.9%) reported history of allergy or food intolerance, with milk intolerance being the most common (4 cases). Three patients (13.6%) reported family history of allergic conditions including Eczema, Asthma and egg allergy.

Four cases (18.2 %) had peripheral eosinophilia (table 2). It's notable that all those with peripheral eosinophilia had positive allergic history.

Immunoglobulin E (IgE) was examined for two patients only, it was normal in one case (case 8, IgE =18 IU/ ml) but elevated in the other case (case 18, 339 IU/ml). The normal range in our lab is less than 150 IU/ml.

**Histopathological findings**

The histopathological slides of these cases were reviewed. Table 3 details the main histological features of each case.

In all but 5 cases, the biopsies were labelled as random colonic without specifying the exact site. For statistical purposes, we included the site of the highest eosinophilic count per HPF for the 5 cases where sites were specified.

The number of colonic mucosal fragments examined in each biopsy ranged from 5-17. They all showed a dense eosinophilic inflammatory infiltrate within the lamina propria (figure 1). The mean eosinophilic count in the most densely inflamed HPF was 55.3, range: 30-104 with the median being 49.5 and the standard deviation 23.8.

There is no statistically significant difference between the number of eosinophils per HPF and peripheral eosinophilia (p= 0.09), history of associated allergic diseases (p= 0.30) or positive colonoscopy (p= 0.67)

It was noticed that the number of eosinophils varied between different fields examined, and in many cases the minimum number and the mean of counting 5 HPF resulted in a figure less than the proposed threshold. Only 4 cases (18.2%) exhibited 30 or more eosinophils per HPF when the field with the lowest number was considered. 11 cases (50%) showed a mean of 5 fields less than the threshold.

All cases exhibited features of eosinophil degranulation and eosinophilic cryptitis, defined as the presence of at least one eosinophil invading a crypt wall. Crypt abscess (eosinophils within the crypt lumen) was seen in only one case. Surface involvement by eosinophilic infiltrate was recognized in 11 cases (50%) and was mild in all of them.

Lymphoid aggregates were present in 12 cases (54.5%).

In 13 cases, ileal biopsies were sent for the patients and in 10 of these ileal tissue eosinophilia was documented (76.9%)

There was no statistically significant difference between number of eosinophils per HPF and ileal involvement (p=0.76), the presence of surface involvement (p= 0.76) or lymphoid aggregates (P= 0.84)

There is also no correlation between the number of eosinophils per HPF and absence of relapses (p=0.57).

**DISCUSSION.**

**Clinical presentation**

In this study, we describe 22 cases of PEC diagnosed at JUH over a three-year period. PEC in our experience affects men and women equally with the median age being 49.5 years. It presents with diarrhea and/or abdominal pain. Colonoscopy is normal in the majority of patients (73%), but some might have edema, hyperemia and loss of vascular pattern; ulceration might be seen rarely. 40.9 % of patients have associated allergic conditions, mostly milk intolerance, and 18% have

peripheral eosinophilia. Associated ileal involvement is documented in 77% of our patients.

There is no correlation between the density of the eosinophilic infiltrate as judged by the numbers eosinophils per HPF in the most densely infiltrated area and history of peripheral eosinophilia or allergic conditions. This observation was reported by other researchers as well [14].

Histologically, cases of PEC exhibit a dense eosinophilic inflammatory infiltrate along with surface involvement, eosinophilic cryptitis and crypt abscesses, eosinophil degranulation and lymphoid hyperplasia. There is no correlation between the density of the eosinophilic infiltrate and these histological features.

There is also no correlation between the number of eosinophils per HPF and absence of relapses. Previous studied reported no correlation between eosinophil density and severity of symptoms [6].

This series is the fourth of its kind in published literature in the English language. In our series, EC was equally distribution between genders, this is in line with Alfadda experience [9], but differs from the other two case series that show female predominance. Mansoor also reported that 66% of patients in his population based study were females [18]. The mean age in all four series is in the fourth decade. Table 4 compares our results with those from the previously published cases series.

**Histological considerations**

Histological diagnosis of PEC is challenging as there is no agreed cutoff point regarding the minimum number of eosinophils required to make a diagnosis. Research is needed to establish the number of normal eosinophils in various segments of the colon and to evaluate the minimum number of eosinophils to make a diagnosis of PEC. The suggestion of putting several thresholds according to the exact site in the colon is theoretically the best way to avoid over-diagnosis, but in clinical practice, biopsies are usually sent in one container labeled as random colonic mucosa. This is particularly done when colonoscopy is normal, which is the case in the majority of EC patients. Other histological criteria detailed in this study and other studies can aid the diagnosis but no

single histopathological feature is diagnostic of PEC, as such thorough clinico-pathological correlation and exclusion of all possible causes of TE is mandatory before diagnosing PEC. This approach was recommended by other investigators as well [7].

We suggest that patients with diarrhea and/ or abdominal pain who have histological evidence of TE need to undergo a full review of their clinical history with emphasis on drug and allergic history. Eosinophilic blood count and stool analysis to rule out infections are recommended as routine workup.

For colonic biopsies that show TE we recommend reporting the number of eosinophils per HPF in the most densely infiltrated area, as well as evaluating cryptitis, crypt abscesses, lymphoid aggregates and surface epithelial involvement. Studies are needed to investigate which of these histological features, if any, have a better predictive value for diagnosing PEC.

Most authors rely on the highest count of eosinophils. However, Diaz Del Arco et al suggested combining the mean number of eosinophils in 5 fields and the highest number of eosinophils and used 40 eosinophils per HPF as the cutoff point; they found that this will increase the sensitivity and specificity of diagnosing PEC [22]. In our experience, taking the mean can result in under diagnosis, however we cannot compare our results with theirs because they counted the most densely infiltrated five HPF, a method different from ours.

We agree with Shifflet's recommendation of taking multiple biopsies to evaluate PEC because the disease is patchy and the eosinophilic infiltrate is not distributed equally between the colonic biopsies [16]

**Limitations**

This is a single institution study and the number of cases studied is low. However, PEC is a rare disease, this study along with Del Arco's are the largest two case series of PEC in adults.

Another limitation is that we do not have information about the exact site of biopsies as the majority were sent in one container labeled as random colonic biopsies. Given that there is variation in the normal number of eosinophils among different segments of the colon, using one cutoff point to diagnose TE is inaccurate.

**Conclusion**

PEC is rare but seems to be increasing in incidence. Gastroenterologists and pathologists need to be aware of this entity and keep a high index of suspicion when faced with cases of unexplained diarrhea and/ or abdominal pain. We recommend that gastroenterologists take several biopsies as the disease can be patchy and to send the biopsies from the right and left colon in separate containers. Pathologists reporting colonic biopsies should actively search for eosinophils in the colonic biopsies when faced with such history. However, diagnosis cannot be made on histological grounds alone, and clinico-pathological correlation is essential to diagnose PEC after excluding all possible secondary causes.

In our practice, we hold regular histopathological meetings fortnightly to discuss all problematic, non-neoplastic gastric cases, this proved to be very helpful in reaching final diagnoses in several inflammatory conditions including PEC.

Counting the number of eosinophils per HPF in the most densely infiltrated area seems to be sufficient to assess TE especially if combined with other histological features of eosinophil activation such as degranulation, surface involvement and eosinophilic cryptitis and crypt abscesses.

More research is needed to establish the number of normal eosinophils in colonic mucosa and to compare the histological features of PEC with those of secondary causes of TE.

**Table 1: Causes of tissue eosinophilia in 130 cases diagnosed at JUH between 1/January/2016 and 1/August /2018.**

| Final diagnosis/ reason of exclusion | Number (percentage) |
|---|---|
| PEC | 22 (16.92%) |
| Inflammatory Bowel disease | 60 (46.15%) |
| Number of eosinophils less than the threshold of this study (20/HPF) * | 10 (7.69) |
| Lost follow up before reaching a diagnosis | 13 (10%) |
| Drug induced | 7 (5.38%)<br>[3 colchicine 4 NSAIDS] |
| Post-surgical | 7 (5.38%) |
| Nonspecific symptoms, improved on meternidazole | 5 (3.85%) |
| Children (3-6 years) | 3 (2.31%) |
| miscellaneous | 3 (2.31%)<br>[1 ischemic colitis<br>1 persistent ameba<br>1 SLE] |
| Total | 130 |

* These cases were diagnosed as mild tissue eosinophilia in the initial histopathology reports with no quantitative assessment of the number of eosinophils. None of them was regarded clinically as PEC.

**Table 2: Summarizes the clinical features, treatment and follow up of the 22 PEC cases.**

| case | age | | gender | symptoms | Allergy | Blood eosinophils% | steroid | F.U months |
|---|---|---|---|---|---|---|---|---|
| 1 | 54 | | F | D | none | 3.0 | yes | 11 |
| 2 | 44 | | F | D, P | none | 1.5 | NK | 12 symptomatic |
| 3 | 47 | | F | D, P | milk | 1.5 | refused | 13 symptomatic |
| 4 | 71 | | F | D, P | Milk | 1.5 | yes | 2 |
| 5 | 31 | | M | D | none | 0.5 | yes | 3 |
| 6 | 46 | | F | D, P | none | 0.9 | yes | 3 |
| 7 | 49 | | F | D | **Eczema** | **17.2** | yes | 20 |
| 8 | 67 | | F | D | none | 3.8 | yes | 5 |
| 9 | 35 | | M | D, P | Milk Asthma | **7.4** | imp | 8 |
| 10 | 15 | | M | P | none | 2.90% | yes | 6 |
| 11 | 65 | | F | D | none | 0.9 | imp | 6 |
| 12 | 67 | | M | P | Drug | 2.1 | yes | 7 |
| 13 | 50 | | M | D (bloody) | milk | 0.4 | yes | 5 |
| 14 | 50 | | M | D, P | none | 1.4 | yes | 7 |
| 15 | 15 | | M | D ( bloody),P | none | 2.6 | yes | 4 |
| 16 | 55 | | M | D,P | **egg** | **5.2** | yes | 6 |
| 17 | 58 | | M | P | none | 3.8 | yes | 1 symptomatic |
| 18 | 25 | | M | D, P | Fish, drug | **13.9** | NK | 6 Symp/ diet |
| 19 | 62 | | M | D, P | none | 2.1 | NK | 6 |
| 20 | 16 | | F | P | atopy | 1.3 | yes | 14 |
| 21 | 87 | | F | P | none | 5.0 | yes | 24 |
| 22 | 21 | | F | D | none | 3.6 | yes | 23 |

Table 2: clinical features, treatment and follow up period of the PEC cases.
F= female, M= male, D= diarrhoea, P (pain; abdominal), NK= not known. imp= improved without treatment.
Normal eosinophils in blood 5%.

**Table 3: The histopathological features of PEC cases.**

| Case | Fragments number | Eos /HPF highest | Eos/HPF Lowest | Eos/HPF Mean of 5 fields | Lymphoid aggregates | Surface involvement | Ileal eosinophilia (eos/HPF) |
|---|---|---|---|---|---|---|---|
| **1** | 7 | 60 | 13 | 32.8 | NO | YES | YES (110) |
| **2** | 6 | 66 | 19 | 43.6 | NO | YES | YES (73) |
| **3** | 6 | 47 | 30 | 39.4 | YES | YES | YES (47) |
| **4** | 13 | 50 | 10 | 21 | NO | NO | NO |
| **5** | 11 | 44 | 12 | 23.4 | NO | NO | Yes (75) |
| **6** | 10 | 49 | 5 | 25.8 | YES | NO | NK |
| **7** | 8 | 97 | 25 | 66.6 | NO | YES | NK |
| **8** | 10 | 52 | 9 | 26 | YES | NO | NK |
| **9** | 14 | 66 | 24 | 40 | YES | YES | NK |
| **10** | 7 | 66 | 24 | 23.4 | YES | YES | YES (93) |
| **11** | 10 | 104 | 30 | 55.2 | YES | NO | NK |
| **12** | 9 | 31 | 13 | 19 | NO | YES | Yes (45) |
| **13** | 17 | 92 | 10 | 48 | NO | NO | NK |
| **14** | 11 | 34 | 10 | 23.8 | YES | YES | NO |
| **15** | 7 | 40 | 12 | 20 | YES | YES | NO |
| **16** | 8 | 100 | 31 | 54.2 | YES | NO | NK |
| **17** | 8 | 53 | 35 | 43.3 | NO | YES | Yes (58) |
| **18** | 11 | 30 | 5 | 18.2 | YES | NO | YES (53) |
| **19** | 5 | 31 | 12 | 25.6 | YES | NO | NK |
| **20** | 6 | 33 | 16 | 23.8 | YES | NO | NK |
| **21** | 12 | 31 | 12 | 22.4 | NO | YES | YES (53) |
| **22** | 7 | 41 | 5 | 18 | NO | NO | YEs (80) |
| | **Total Yes (%)** | | | | 12 (54.5) | 11 (50) | 10 of 13 known (76.9% ) |
| | **Max** | 104 | 35 | 66.6 | | | |
| | **Min** | 30 | 5 | 18 | | | |
| | **Median** | 49.5 | 12.5 | 25.7 | | | |

| Case | Fragments number | Eos /HPF highest | Eos/HPF Lowest | Eos/HPF Mean of 5 fields | Lymphoid aggregates | Surface involvement | Ileal eosinophilia (eos/HPF) |
|---|---|---|---|---|---|---|---|
| | **Mean** | 55.3 | 16.5 | 32.4 | | | |
| | **SD** | 23.8 | 9.2 | 14.1 | | | |

**Table 4: Comparison between published cases series of PEC.**

| Study | Number of cases | % PEC of TE cases | M:F ratio | Mean age | Allergy | PE | Positive endoscopy | Mean eos/HPF |
|---|---|---|---|---|---|---|---|---|
| **Current study Awad et al. 2018** | 22 | 17% | 1 to 1 | 46 | 40.9% | 18% | 27% | 55 |
| **Diaz del Arco et al 2017** | 22 | 21% | 1:3.4 | 41 | 18% | 18% | 14% | 70 |
| **Al Fadda et al 2013** | 7 | - | 1:1.3 | 45 | 71% | 14% | 57% | - |
| **Salazar et al 2013** | 5 | 42% | 1:4 | 46 | 40% | 20% | 40% | - |

PE: peripheral eosinophilia

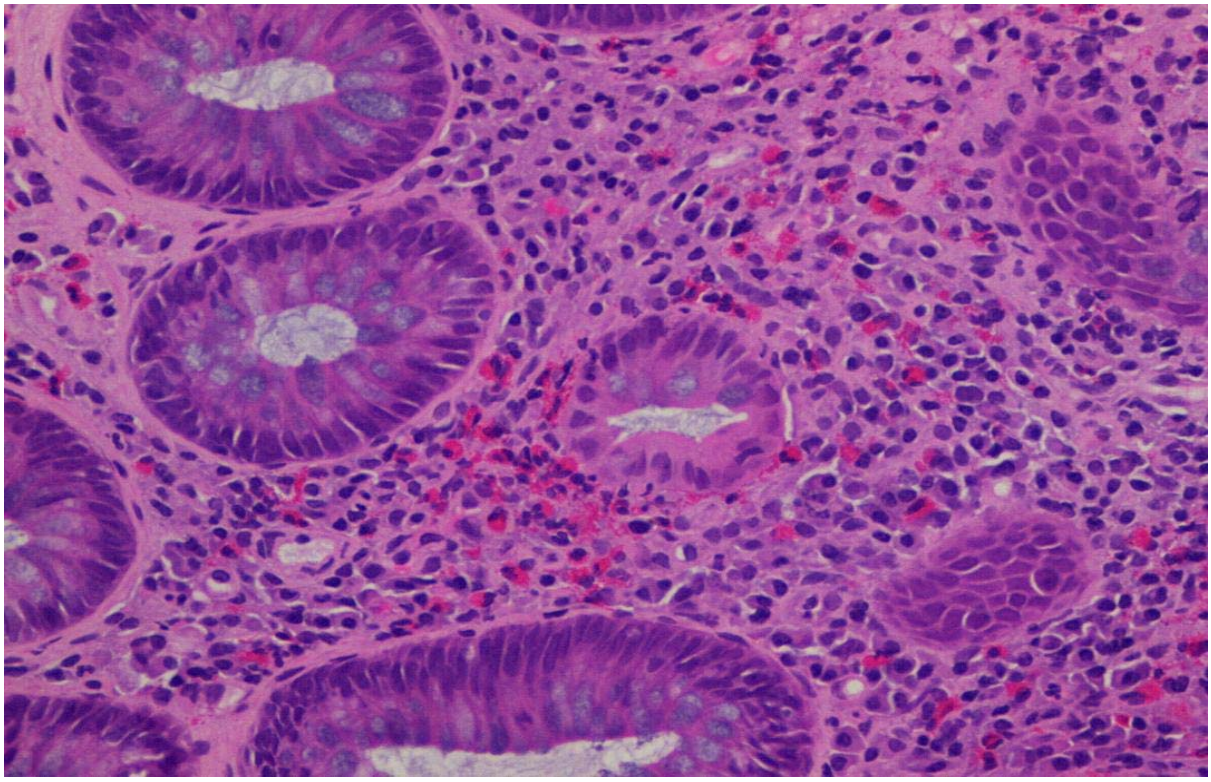

**Figure 1: Eosinophils within lamina propria, H &E 40X.**

**References**


1 Uppal V, Kreiger P, Kutsch E. Eosinophilic Gastroenteritis and Colitis: a Comprehensive Review. Clin Rev Allergy Immunol 2016; 50 (2), 175-188

2 Egan M, Furuta GT. Eosinophilic gastrointestinal diseases beyond eosinophilic esophagitis. Ann Allergy Asthma Immunol 2018; 121 (2), 162-167

3 Talley NJ, Shorter RG, Phillips SF, Zinsmeister AR. Eosinophilic gastroenteritis: a clinicopathological study of patients with disease of the mucosa, muscle layer, and subserosal tissues. Gut 1990; 31, 54-58.

4 Lucendo AJ. Eosinophilic diseases of the gastrointestinal tract. Scand J Gastroenterol 2010; 45 (9) , 1013-1021.

5 Gaertner WB, Macdonald JE, Kwaan MR, Shepela C, Madoff R, Jessurun J, Melton GB. Eosinophilic colitis: university of Minnesota experience and literature review. Gastroenterol Res Pract 2011; 2011, 857508

6 Bates AW. Diagnosing eosinophilic colitis: histopathological pattern or nosological entity? Scientifica (Cairo) 2012; 2012, 9 pages ID 682576.

7 Conner JR, Kirsch R. The pathology and causes of tissue eosinophilia in the gastrointestinal tract. Histopathology 2017; 71, 177-199

8 Diaz Del Arco C, Taxonera C, Olivares D, Fernandez Acenero MJ. Eosinophilic colitis: Case series and literature review. Pathol Res Pract 2018; 214, 100-104

9 Alfadda AA, Shaffer EA, Urbanski SJ, Storr MA. Eosinophilic colitis is a sporadic self-limited disease of middle-aged people: a population-based study. Colorectal Dis 2014; 16, 123-129.

10 Fernandez Salazar LI, Borrego Pintado H, Velayos Jimenez B, Gonzalez Hernandez JM. Differential diagnosis and management of histologic eosinophilic colitis. J Crohns Colitis 2013; 7, e20-21

11 Collins MH, Capocelli K, Yang GY. Eosinophilic Gastrointestinal Disorders Pathology. Front Med (Lausanne) 2017; 4, 261

12 Collins MH. Histopathologic features of eosinophilic esophagitis and eosinophilic gastrointestinal diseases. Gastroenterol Clin North Am 2014; 43, 257-268

13 Turner KO, Sinkre RA, Neumann WL, Genta RM. Primary Colonic Eosinophilia and Eosinophilic Colitis in Adults. Am J Surg Pathol 2017; 41, 225-233

14 Behjati S, Zilbauer M, Heuschkel R, Phillips A, Salvestrini C, Torrente F, Bates AW. Defining eosinophilic colitis in children: insights from a retrospective case series. J Pediatr Gastroenterol Nutr 2009; 49, 208-215.

15 Yan BM, Shaffer EA. Primary eosinophilic disorders of the gastrointestinal tract. Gut 2009; 58, 721-732.

16 Shifflet A, Forouhar F, Wu GY. Eosinophilic digestive diseases: eosinophilic esophagitis, gastroenteritis, and colitis. J Formos Med Assoc 2009; 108, 834-843

17 Jensen ET, Martin CF, Kappelman MD, Dellon ES. Prevalence of Eosinophilic Gastritis, Gastroenteritis, and Colitis: Estimates From a National Administrative Database. J Pediatr Gastroenterol Nutr 2016; 62, 36-42.

18 Mansoor E, Saleh MA, Cooper GS. Prevalence of Eosinophilic Gastroenteritis and Colitis in a Population-Based Study, From 2012 to 2017. Clin Gastroenterol Hepatol 2017; 15, 1733-1741.

19 Alfadda AA, Storr MA, Shaffer EA. Eosinophilic colitis: an update on pathophysiology and treatment. Br Med Bull 2011; 100, 59-72.

20 Okpara N, Aswad B, Baffy G. Eosinophilic

colitis. World J Gastroenterol 2009; 15, 2975-2979.

21 Pascal RR, Gramlich TL, Parker KM, Gansler TS. Geographic variations in eosinophil concentration in normal colonic mucosa. Mod Pathol 1997; 10, 363-365.

22 Diaz Del Arco C, Taxonera C, Munoz LE, Olivares D, Fernandez Acenero MJ. Eosinophilic colitis: experience in a large tertiary hospital. Rom J Morphol Embryol 2017; 58, 783-789.

# السمات الديموغرافية والمعايير التشخيصية لالتهاب القولون اليوزيني بين المرضى البالغين مع التركيز على الارتباط المرضي النسيجي

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**الملخص**

**هدف:** التهاب القولون الأولي اليوزيني (PEC) هو المرض الأكثر ندرة والأقل فهماً في طيف أمراض الجهاز الهضمي اليوزيني. تهدف هذه الدراسة إلى وصف الخصائص الإكلينيكية المرضية لهذا المرض واستكشاف المعايير النسيجية التي يمكن أن تحسن الدقة التشخيصية لهذا المرض.

**أساليب**: في هذه الدراسة السريرية المرضية بأثر رجعي ، نصف السمات السريرية والنسيجية لـ 22 حالة من .PEC تم البحث في النظام المحوسب لقسم التشريح المرضي في مستشفى الجامعة الأردنية عن الحالات التي تم تشخيصها على أنها كثرة اليوزينيات في الأنسجة (130 حالة). تمت مراجعة السجلات الطبية للتحقق من التشخيص النهائي وتم تضمين الحالات المصابة بـ PEC فقط. تم توثيق الخصائص الديموغرافية ، وتم مراجعة جميع الشرائح النسيجية المرضية وتم تسجيل السمات النسيجية الرئيسية ، وبشكل رئيسي عدد الحمضات.

**نتائج**: تبين أن 16.9٪ من حالات فرط الحمضات في الأنسجة هي .PEC تم توزيعها بالتساوي بين الجنسين بمتوسط عمر 49.5 سنة. كان العرض الأكثر شيوعًا هو الإسهال و / أو آلام البطن. كان تنظير القولون طبيعيًا في 72.7٪ من الحالات. 27.3٪ من المرضى يعانون من حالات حساسية مصاحبة ، معظمهم من عدم تحمل الحليب ، و 18.2٪ لديهم فرط الحمضات المحيطي. من الناحية النسيجية كان هناك التهاب كثيف اليوزيني مع خصائص تنشيط الحمضات. لم يكن هناك ارتباط بين كثافة الارتشاح اليوزيني وهذه السمات النسيجية.

**استنتاج**: لا يمكن تشخيص PEC على أسس نسيجية فقط ؛ الارتباط السريري المرضي ضروري لتشخيص PEC بعد استبعاد جميع الأسباب الثانوية المحتملة. إن تاريخ حالات الحساسية شائع بين حالات PEC ، وهو اكتشاف قد يلقي الضوء على التسبب في هذا المرض.

**الكلمات الدالة:** القولون. الحمضات. التهاب القولون اليوزيني. التشخيص النسيجي.