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RESEARCH ARTICLE

EOSINOPHILIC ESOPHAGITIS IN ADULTS: A CASE SERIES AND LITERATURE REVIW

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Abstract

Eosinophilic esophagitis (EO) is an inflammation of the esophagus due to abnormal immune response to exogenous allergens.Currently there is an increase in diagnostic cases thanks to a better current recognition of symptoms. The esophago-gastro-duodenal fibroscopy must be carried out to make the diagnosis, confirmed by esophageal biopsies (more than 15 eosinophils per high magnification field). Our series includes 5 patients whose histological diagnosis is confirmed,3 men and 2 women with an average age of 47,4 .The clinical presentation is very variedwhich3 patients suffer from dysphagia, 2 patients with gastroesophageal reflux syndrome and 1 patient has dyspepsia, and chest pain. The endoscopic aspect is not pathognomonic, we repport a normal appearance, pseudotracheal aspect of the esophageal mucosa,a cottony appearance of the esophagus,an impassable stenosis of the esophagus, and an ulcerated esophagitis.All our patients responded well to the therapeutic protocol received with a good evolution.

Introduction:-

Eosinophilic esophagitis (EO) is the most common cause of chronic inflammation of the esophagus after peptic esophagitis, first described in the early 1990s.It is defined by an abnormal immune response of the esophageal mucosa to exogenous allergens resulting in chronic infiltration of this mucosa by eosinophils.[1]It is a disease probably under-diagnosed in France [2]and even in Europe with an increasing prevalence, estimated on average in 40–56 cases per 100,000 inhabitants, [3]the increase in the number of diagnosed cases is due to better current recognition of symptoms [4]. The clinical presentation is very varied, mainly dominated by gastroesophageal reflux and dysphagia. [5]The oeso-gastroduodenal fibroscopy is essential in the diagnosis. We can observe normal mucosa, whitish deposits, a pseudo-tracheal appearance or even a stenosis, but esophageal biopsies are essential to confirm the diagnosis [6, 7].We report five clinical cases with a variety of symptoms and endoscopic presentations. As well as the histological images which confirm the diagnosis of an EO, with the evaluation of the therapeutic response.

Methods:-

This study was carried in the Gastroenterology department, Military hospital Moulay Ismail, Meknes, Morocco. Prospective review of five cases whose diagnosis of eosinophilic esophagitis is confirmed on esophageal biopsies. The clinical characteristics, endoscopic appearances, and outcomesaftertreatmentofpatientswill be analyzed and compared with the literature .All patients received helicobacter pylori eradication Treatment (if it is positive) with a protocol based on PPI and inhaled corticosteroid therapy.

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Results:-

Case 1:

A 52-year-old man, with no medical history, consulted for intermittent solids dysphagia with weight loss andasthenia. The clinical examination was normal. The standard biological assessment did not revealed any anomalies. An abdominal a CT scan were performed showing no anomalies. The esogastroduodenalfibroscopyshows a normal-looking esophagus andcongestiveantritis.

Esophageal biopsies showed a regular mucosawith a chorion, which houses a discreet inflammatory infiltrate and the surface epithelium harboring numerous eosinophilic polymorphonuclear cells, and gastric biopsiesshowshelicobacterpylorigastritis

The patient received helicobacter pylori eradication treatment with a protocol based on PPI and inhaled corticosteroid therapy with good clinical outcome.

Case 2:

A 43-year-old man, with no medical history, consulted for intermittent stomachachewith gastroesophageal reflux syndrome. The clinical examination was normal. The standard biological assessment did not revealed any anomalies. The esogastroduodenalfibroscopyshows a cottony appearance of the esophagus (figure 1) erythematouspangastritis.

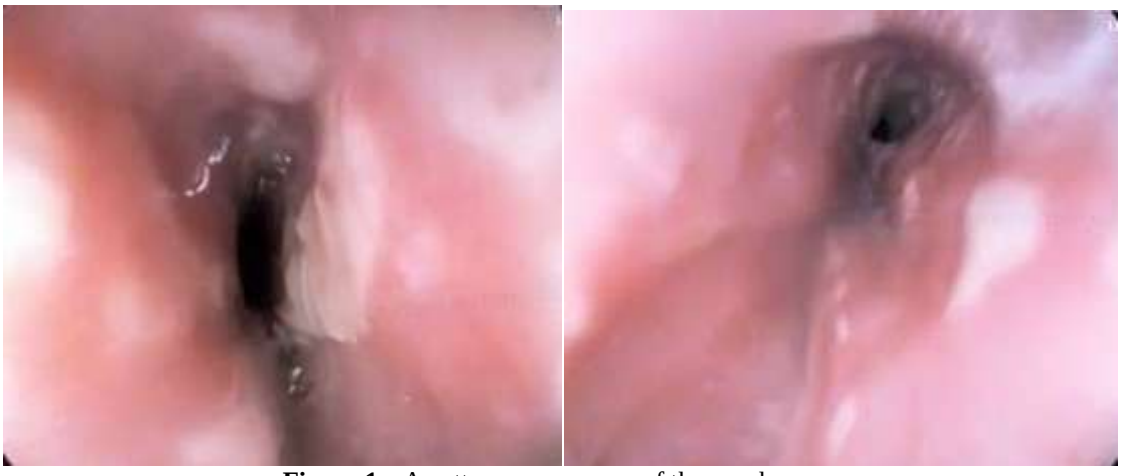

Figure 1:- A cottony appearance of the esophagus.

Esophageal biopsies showed achorion, which houses a discreet inflammatory infiltrate and the surface epithelium harboring numerous eosinophilic polymorphonuclear cells

The patient received helicobacter pylori eradication treatment with a protocol based on PPI and inhaled corticosteroid therapy with good clinical outcome.

Case 3:

A 38-year-old woman, with no medical history, consulted for esophagogastric reflux syndromewith weight loss and dyspepsia. The clinical examination was normal. The standard biologicalassessmentdid not revealedanyanomaliesapartfromaslightincrease in the rate of polynucleareosinophils. An abdominal ultrasoundwereperformedshowing no anomalies. The esogastroduodenalfibroscopyshows a pseudotracheal aspect of the esophageal mucosa (figure 2).

Figure 2:- A pseudotracheal aspect of the esophageal mucosa.

Esophageal biopsies showed the surface epithelium harboring numerous eosinophilic polymorphonuclear cells andgastricbiopsiesshows helicobacter pylori gastritis

The patient received helicobacter pylori eradication treatment with a protocol based on PPI and inhaled corticosteroid therapy with good clinical outcome.

Case 4:

A 46-year-old woman, followed for diabetes, consulted for intermittent solids and fluid dysphagia with weight loss, atypical chest pain, andasthenia. The clinical examination was normalapart from a dehydrated patient. The standard biological assessment did not revealed any anomalies. An abdominal a CT scan were performed showing no anomaliesoutside of a dilated esophagus. The esogastroduodenalfibroscopyshows an impassable stenosis of the esophagusthat is balloon dilated. (Figure 1)

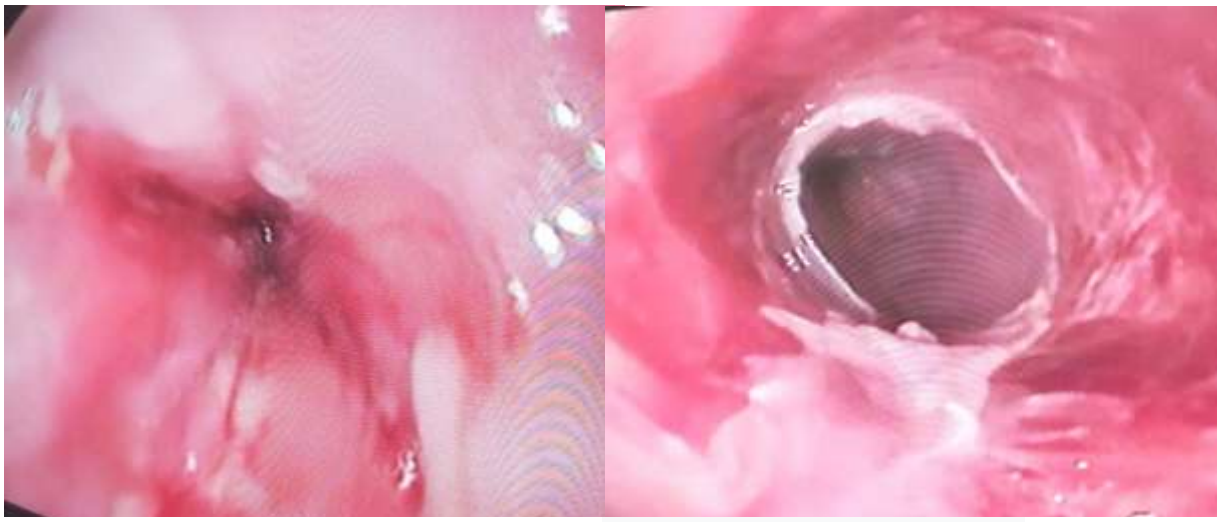

Figure 3:- An impassable stenosis of the esophagus B: ballon dilated of stenosis.

Esophageal biopsies confirmedconfirmed the diagnosis of eosinophilicesophagitis

The patient received helicobacter pylori eradication treatment with a protocol based on PPI and inhaled corticosteroid therapy more than several balloon dilation sessions with good clinical outcome.

Case 5:
A 58-year-old man, with no medical history, consulted for intermittent dysphagiawithstomachache. The clinical examination was normal. The standard biological assessment did not revealed any anomalies. An abdominal a CT scan were performed showing no anomalies. The esogastroduodenalfibroscopyshows an ulcerated esophagitis class 3according to the classification of Savary and Miller (figure 4) and micronodularantritis.

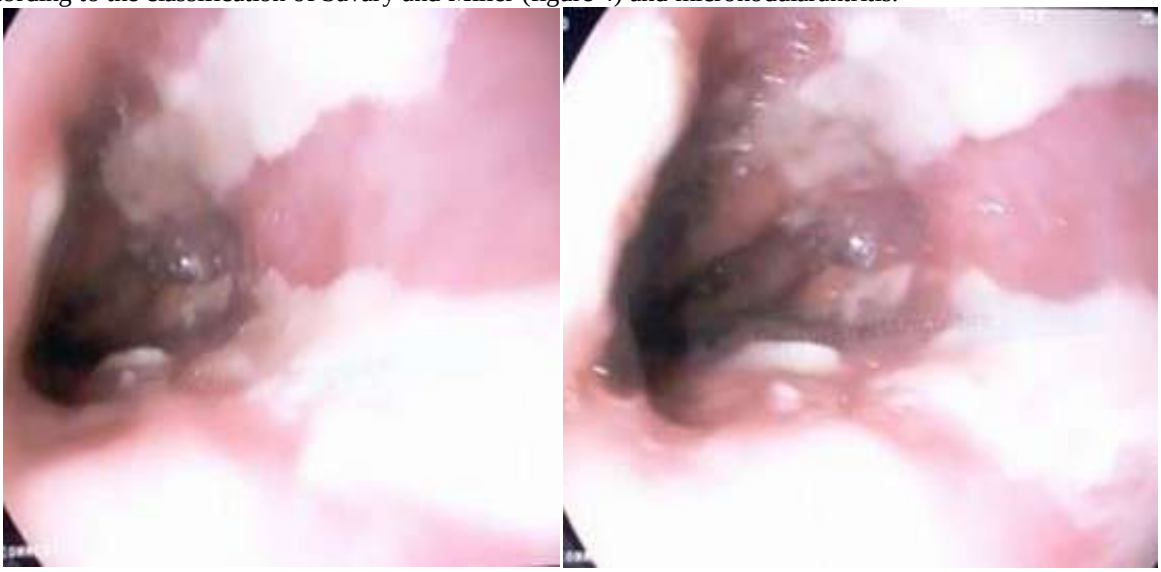

Figure 4:-Anulcerated esophagitis class 3according to the classification of Savary and Miller.

Esophageal biopsies showed a chorion, which houses an inflammatory infiltrate and the surface epithelium harboring numerous eosinophilic polymorphonuclear cells and gastric biopsiesshows helicobacter pylori gastritis

The patient received helicobacter pylori eradication treatment with a protocol based on PPI and inhaled corticosteroid therapy with good clinical outcome, and control fibroscopy showed the disappearance of esophageal ulcerations.

Discussion:-

Eosinophilic esophagitis (EO) is an abnormal immune response of the esophageal mucosa to exogenous allergens resulting in chronic infiltration of this mucosa by eosinophils. [1] (more than 15 eosinophils per high magnification field) [8]It is a disease probably under-diagnosed inEurope with an increasing prevalence, estimated on average in 40–56 cases per 100,000 in habitants [3].There is a male predominance with a ratio3/1 8 and It most often affects young adults between the ages of 30 and 50.[9]In our study we report 3 men and 2 women with an average age of 47.4 .

The clinical presentation is very varied, mainly dominated by gastroesophageal reflux and dysphagia [5]. In our study,3 patients suffer from dysphagia 2 patients with gastroesophageal reflux syndrome and 1 patient has dyspepsia, andchestpain.

The standard biologicalassessmentdid not revealedany anomalies apartfromaslightincreaseofpolynuclearеosinophilsin a single patient.

An esophago-gastro-duodenal fibroscopy must be carried out to make the diagnosis of eosinophilic oesophagitis. The endoscopic aspect is not pathognomonic. It typically finds longitudinal striae of the esophagus, whitish deposits, an aspect of trachealization of the esophagus, a thickened mucosa reducing the caliber of the esophagus up to real stenoses preventing the passage of the endoscope [6, 7].
The esophago-gastro-duodenal fibroscopywas performed in all our patients revealing: a normal appearance in a single patient,apseudotracheal aspect of the esophageal mucosa in a single patient,a cottony appearance of the esophagus in a single patient,an impassable stenosis of the esophagus in a single patient, and An ulcerated esophagitis class 3according to the classification of Savary and Miller in a single patient.

Histological examination after staining with HES will make to look for infiltration of the esophageal mucosa by polymorphonuclear eosinophils (> 15 eosinophils/field at magnification 400) which can group together and form micro-abscesses, and signs of chronic esophagitis revealed by elongation of the papillary ridges, edema, and hyperplasia of the basal layer. In the normal state, there are no polymorphonucleareosinophils in the esophagealmucosa [10–11].

The esophageal biopsies of our 5 patients showedaregular mucosa with a chorion, which houses a discreet inflammatory infiltrate and the surface epithelium harboring numerous eosinophilic polymorphonuclearcells (figure 5) (> 15 eosinophils/field) .the presence of helicobacter pylori was confirmed in gastric bipsies in 4 patient.

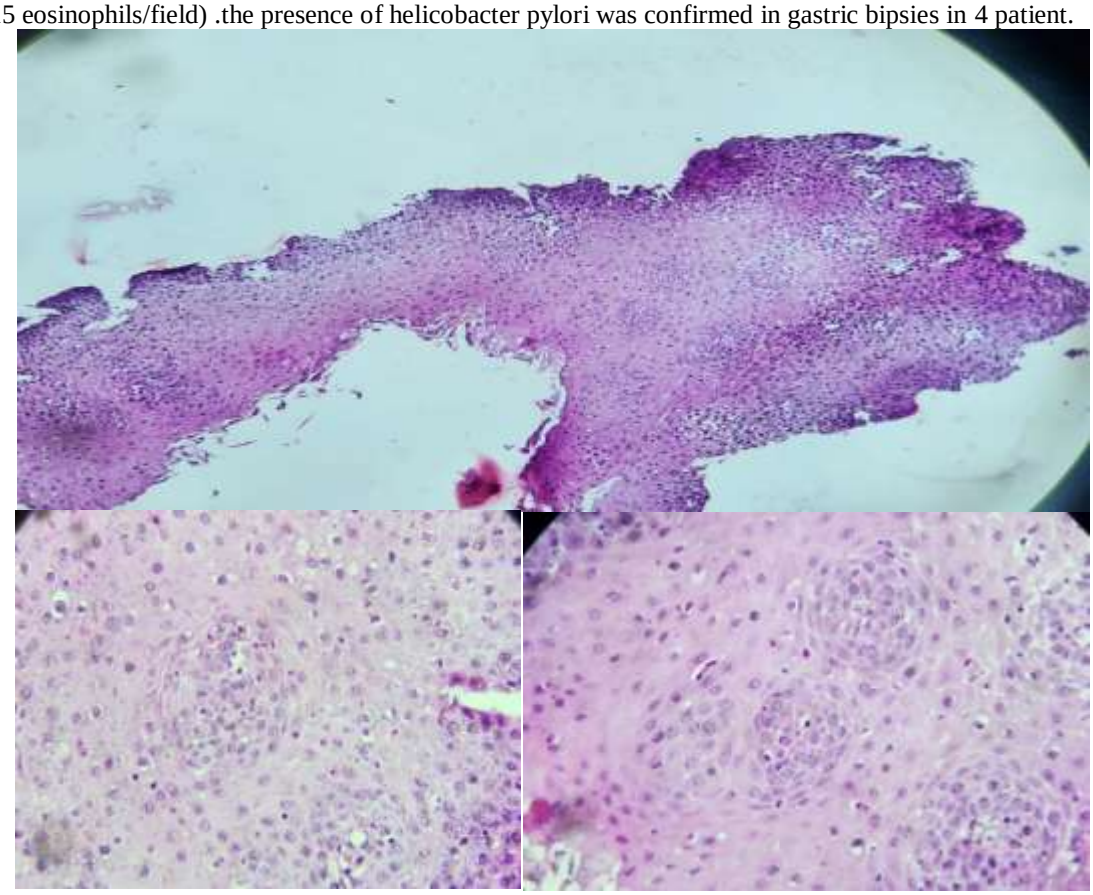

Figure 5:- A regular mucosa with the surface epithelium harboring numerous eosinophilic polymorphonuclear cells.

The new guidelines offer three treatment options based on patient preference: PPIs, topical steroids and elimination diet. In case of clinical and histological remission, it is recommended to continue long-term treatment at the minimum effective dose.

In case of histological remission with persistence of symptoms, the other causes and the initial diagnosis should be reassessed, but if there is esophageal stenosis, endoscopic dilation is recommended [12].

All patients received helicobacter pylori eradication Treatment (if it is positive) with a protocol based on: high dose proton pump inhibitor (40 mg/ day) and inhaled corticosteroid therapy par fluticasone swallowed for 8 weeks.

The patient who had an impassable esophageal stricture benefited from several sessions of balloon dilation. The 5 patients reassessedafter 8 weeks of treatment with good clinical outcomes in 4patients, the fifth patient required further sessions of balloon dilation.

Conclusion:-

EO are underestimated esophageal pathologies whose incidence is increasing.

Theirdiagnosisis made by esophageal biopsies performedendoscopicallywithprecisehistologicalcriteria.The treatments are currentlyeitherdietary or medicinal.Our series confirms a clinical and endoscopic variety of this pathological entity, and a good therapeuticresponse of associatedPPIs and swallowedcorticosteroids.

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