Cancer Like Eosinophilic Gastritis

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Özet: *KANSERE BENZEYEN EOZİNOFİLİK GASTRİTİS*

Epigastralji, bulantı, halmsizlik şikayetleri mevcut olan 40 yaşındaki erkek hastanın müracatı üzerine yapılan andoskopik muayenede, yaygın ülserovejetan tümoral yapı tesbit edildi. Israrla alınan biyopsilerde maliğnite tesbit edilemedi. Oldukça yaygın ve tıbbi tedaviye dirençli lezyon nediniyle Billroth I operasyonu uygulandı. Histopatolojik tanı eozinofilik gastritis idi. Üç aylık kkontrollerdeki endoskopik muayenelerde midenin geri kalan mukozasında eritematöz değişiklikler ve ödem husule geldi. Altıncı ayda yaklaşık 3 cm çapında küçük kurvaturada tümör kitlesi oluştu. Alınan biyopsiler eozinofilik gastritis ile uyumlu idi. Hastaya iaki ay süreyle yaklaşık 40 mg/gün metil prednizolon verildi. Tedavinin birinci ayında mümör kitlesinin tabanında, kitlenin küçülmesiyle aşıkar hale gelen sütür ipeği göründü. Endoskopik biyopsi pensi ile sütür ipeği çıkarıldı. İki buçuk ay sonunda lezyon tümüyle kaybolduğu için steroid kesildi ve iki yıllık takıp sonrasında nüks görülmedi. Sonuç olarak eozinofilik gastritis, endoskopik görünümü mide kanseri ile uyumlu olmasına rağmen histopatolojik olarak tanı konulamayan olgularda, ayırıcı tanıda önemli ölçüde düşünülmelidir. Eozinofilik gastritisin gelişimi önce mukozal hiperemi ve ödem, daha sonra da ülserovejetan kitle şeklinde kendini göstermektedir. Steroid tedavi dramatik iyilik sağladığı içintedavide ilk tercih olarak kullanılmalıdır.

Anahtar Kelimeler: Eozinofilik gastroenteritis, gastritis, kortikoterapi, mide kanseri.

Summary: *A 40 year old man with complaints of epigastralgia, nausea and weakness was initially diagnosed as stomach cancer by endoscopic examination due to a large ulcero-vegetative tumor mass. Histopathology of the biopsy specimen consistently failed to prove malignancy. A Billroth I resection was performed for the giant, intractable lesion without any complications. Histopathological diagnosis was eosinophilic gastroenteritis (EG). At 3 months follow-up; endoscopy revealed erythematous changes and edema in mucosa of the remnant stomach and at 6 months follow-up; a tumor mass of 3 cm in diameter which was located at the lesser curvature was detected. Serial biopsies were all consistent with EG and the patient was given methyl prednisolone 40 mg per day orally for two months. A suture thread which became apparent at the base of the lesion after 1 month of steroid therapy was succesfully removed endoscopically. Steroid therapy provided excellent resolution in about 2.5 months and no recurrence was encountered in two years follow-up. In conclusion; EG should be seriously considered in patients with an endoscopical diagnosis of gastric carcinoma in whom histopathology consistently fails to confirm malignancy. Evolution of gastric EG seems to follow a sequence of mucosal hyperemia-protrusion-ulceration. Steroid therapy may offer dramatic improvement and should be the first line therapeutic approach.*

Key Words: Eosinophilic gastroenteritis, gastritis, corticotherapy, stomach cancer.

Eosinophilic gastroenteritis (EG) is an uncommon disease of the gastrointestinal tract which was discribed by Kaijser in 1937 (1). From what was initially a nonspesific and confusing picture, a clearer entity has begun to emerge in recent years. Up to now, about 200 cases of EG have been reported and the diagnostic criteria such as; the presence of gastrointestinal symptoms, biopsies showing eosinophilic infiltration of one or more areas of the gastro intestinal tract from esophagus to colon and no evidence of any parasitic or extraintestinal disease were established (2).

In this paper, we present a case of EG which imitated stomach cancer and recurred after sur-

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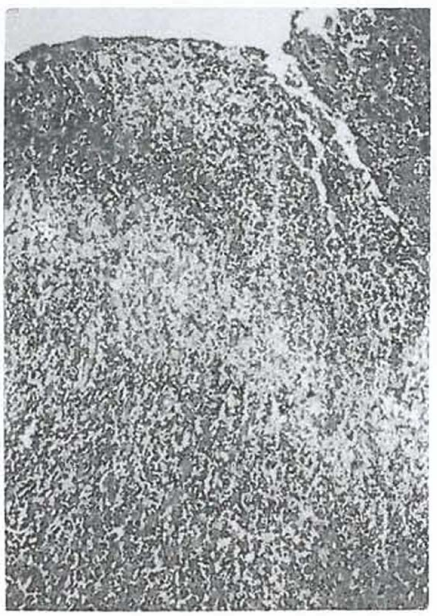

Figure 1: Fissuring ulceration of the gastric wall. Diffuse infiltration by inflammatory cells without any organized pattern (HE, x40).

gical therapy. Recurrence of EG was successfully controlled with steroid therapy and endoscopic removal of a suture thread which might had a partial role in the recurrence.

Case Report:

A 40 year old man was referred to our hospital with complaints of epigastric pain-fullness, nausea and weakness after being initially diagnosed as having peptic ulcer in an outpatient clinic. His past medical history was unremarkable except epigastralgia of several years duration and a course of H_2 blokage therapy which was successful. His physical examination was normal besides a tender, hard mass in the epigastrium.

Laboratory examinations, including serum immuno-electrophoresis and peripheral eosinophilia were in the normal range except mild anemia and a fasting blood glucose level of 175 mg/dl. His stool was positive for occult blood and negative for paraside eggs. Upper GI series revealed an ulcero-vegetative tumor located at the postero-inferior aspect of the distal stomach with marked rigidity at this area. X-Ray examination of the small and large intestine were normal.

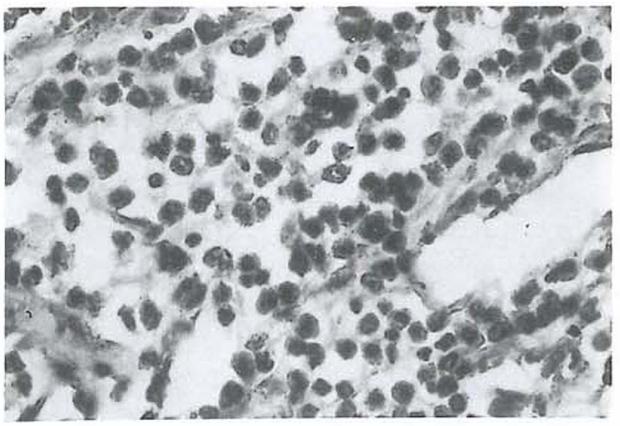

Figure 2: Close up view of the infiltration rich in leukocytes, particularly eosinophile and some plasmocytes, lymphocytes (HE, x250).

Esophagogastroduodenoscopy showed an ulcerovegatative, borman IV like tumor mass between the distal corpus and prepyloric antrum located on the posterior side of the great curvature. Insufflation failed to open the stomach. Endoscopic diagnosis of an advanced gastric carcinoma was not confirmed with multiple biopsies which were all unequivocally and consistently interpreted as non-malignant. Ultrasonography revealed target sign with thickness of the stomach wall, 12x8x6 cm in diameter. At surgery, a Billroth I gastrectomy was performed for the intractable lesion. Intraoperative findings such as a large tumor with serosal and lymphatic involvement were also highly suggestive of malignancy. Macroscopically, the lesion was 14x12 cm in size with elevated borders and central ulceration of 5 cm in diameter. Cut surface of the lesion was appeared indurated and infiltrated, with an irregular serosal surface in this area with a wall thickness of 5 mm. Gastric rugae, besides proximal parts, were atrophic. Submucosa, distal to the lesion was shiny, edematous and widened. Innumerable lymph nodes, mostly packed together and the largest one with a diameter of 3.5 cm in size were dissected from both curvatures.

Histopathologically; the surface of the lesion was undergone almost complete loss of mucosa besides the edges in which several mucosal structures can still be seen (Figure 1). The extensive ulceration at the center was found to be eventuated in fistulization, characterized by attached omental and hepatic tissues to the sero-

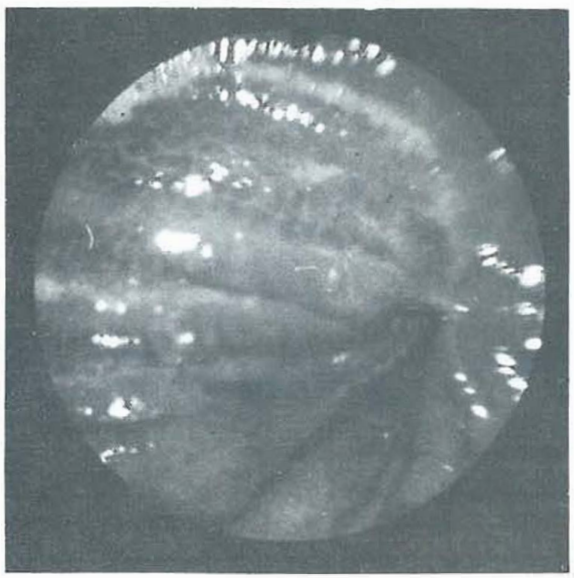

Figure 3: Endoscopic picture showing prominent hyperemia-edema of the gastric mucosa.

Figure 4: Endoscopic picture showing broad-based tumor with eroded mucosa located at the posterior wall, near the angulus.

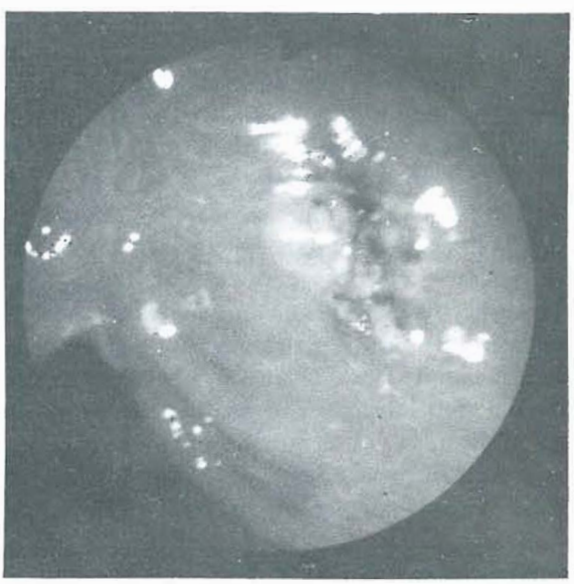

Figure 5: Endoscopic picture 1 month after streoid therapy, Note the presence of a silk suture thread at the base of the tumor.

sa. Prominent infiltration of the wall by leukocytes, mainly eosinophile was observed along the lesion (Figure 2). Plasma cells and lymphocytes were less pronounced.

There were no giant cells accompanying the polymorphic infiltration making the differentiation from Hodgkin's disease easier. Cytologic atypia; neither in epithelial nor in the other components was observed. The changes in other areas of the stomach were non-specific. Twenty three lymph nodes appeared highly hyperplastic with non-specific reactive changes. Diagnosis was; giant non-neoplastic ulcer, compatible with EG and benign reactive hyperplastic lymph nodes. Although endoscopic biopsies were taken randomly. The biopsies did not reveal any eosinophilic infiltration.

The patient was discharged after an uneventful and endoscopy at 3 months follow-up revealed edema and hyperemia of the remnant stomach mucosa. Follow up endoscopy at 6 moths showed a sessile, hard, eroded tumour mass of 3 cm in diameter with a broad base and a fragile mucosa located at the lesser curvature (Figure 3,4). In the light of serial biopsies, the patient was diagnosed as recurrent EG and was given methyl prednisolone 40 mg per day orally. Endoscopic examination was repeated in every 15 days. Tumor size was decreased and the suture thread at the base of the lesion became apparent after 1 month of steriod therapy (Figure 5).

Suture thread was removed endoscopically with biopsy forceps and the lesion was disappeared quickly. Steroid was decreased by 10 mg in every week, and stopped after 2 months. The pa-

tient was symptom and recurrence free in 2 years follow-up.

Discussion

EG is an uncommon disease, characterized by infiltration of eosinophilic leukocytes in the mucosa(predominant type), muscularis propria, and/or serosa of the gastrointestinal tract (3). The cause of most cases of EG is unknown although an allergic mechanism seems to be likely (4-7). The clinical manifestations of the disease are related to the location of the gastrointestinal part involved and infiltrated with the eosinophile and the depth of the infiltration (3,8).

The most commonly involved segments of the gut are stomach, small bowel, large bowel and esophagus respectively (2).

Patchy involvement of lesions sometimes makes the diagnosis a real challenge because the degree of eosinophilic infiltration may vary considerably with in the same lesion (2). Talley et al showed that eosinophilic infiltration was found only in 21 out 26 cases with EG (2). High blood IgE levels and peripheral eosinophilia can be found more often in cases with allergic basis and were absent in our case (5).

However, presence of a silk suture thread precisely at the bottom of the recurrent EG; may raise the question whether a local hypersensitivity reaction was responsible for the recurrence in our patient since its removal with steroid therapy resulted in complete abolition of symptoms and quick disappearance of the tumor.

Radiologic and endoscopic examinations are very important tools for diagnostic purposes but both of them are frequently inconclusive as in the case reported here in. Nodular or diffuse thickness of the gastric folds, polyp and/or erosions can be found by the radiologic examinations (9). Discrete, circular ulcers less than 0.5 cm in diameter scattered through out the mucosa of the stomach, nodularity, hyperemia and edema of the mucosa, thickness of the mucosal folds and deformity of the antrum were also cited (10). To our knowledge, the ulcer in our case represents the largest ulceration resulting from an EG reported up to date and differentiation of the giant lesion from a Borman IV type of gastric cancer by.X-ray examinations, endoscopy and even intraoperatively was impossible. All of the information obtained from X-ray examinations and endoscopy including rigidity, tumor with a large central ulcer and intraoperative findings were consistent with malignancy.

Frequent endoscopic follow-up after surgery had enabled us to observe the evolution of EG in otherwise normal remnant gastric mucosa. Although it may vary from case to case, a mucosal hyperemia-edema followed by a tumor like lesion may be a typical pattern of evolution in many patients with gastric EG. Ulceration seems to be later stage as we did not see it during the recurrence.

Differential diagnosis of EG must also include polyarteritis nodosa, Crohn's disease, parasitic infestations, lymphoma, systemic mastocytosis, hypereosinophilic syndrome and histiocytosis-X (8,11,12).

The place of surgery in the treatment of gastric EG should ideally be restricted only to complications and this can be achieved by better diagnosis with endoscopy and endoscopic biopsy in experienced endoscopists hands who are aware of the condition. Short course steroid therapy was reported to be beneficial in the treatment of EG and was successful in our patient in the treatment of the reccurence (2,8). No clinical or endoscopical reccurence occured in our patient after a 2 months 40 mg/day steroid therapy during the 2 years follow-up period. The value of sodium chromoglycate in the treatment of EG is controversial and needs further confirmation (2).

In conclusion; EG should be seriously considered in patients with a X-ray and endoscopical diagnosis carcinoma in whom histopathology consistently fails to confirm malignancy, There are two important points which deserves emphasis: 1) Natural evolution of gastric EG seems to follow a sequential pattern which is; mucosal hyperemia-edema followed by tumor like protrusion and eventually ulceration if left untreated. 2) Steroid therapy may offer dramatic improvement and should be the first line therapeutic approach.

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