Barrett's Esophagus and Treatment of Gastroesophageal Reflux Disease (GERD)

Dr. Vedat KIRIMLIOĞLU, Dr. Fuat ATALAY, Dr. Orhan ELBİR, Dr. Metin ŞAVKILIOĞLU, Dr. Canbek SEVEN, Dr. Alpaslan GENCER

Özet: *BARRETT ÖSEFAGUS VE GASTROÖSEFAGEAL REFLÜ HASTALIĞININ (GÖRH) TEDAVİSİ*

Barret ösefagus mide metaplastik columnar epitelinin ösefagusun çok katlı yassı epitelinin yerini almasıdır. Normann Barret tarafından 1950 yılında tarif edilmiştir. Gastroösefageal reflü'den şikayet edenlerin % 10'da geç komplikasyon olarak görülür. En tehlikeli komplikasyonu ösofageal adenokarsinom olup teşhis edildiğinde küratif reseksiyonu mümkün değildir.

Anahtar kelimeler : Barrett ösofagus, gastroösefageal reflü hastalığı ösefageal adenokarsinom

Summary: *Barrett's esophagus is a condition in which the normal stratified squamous epithelium of the esophagus is replaced by a metaplastik columnar epithelium of the stomach. It is first described by Normann Barrett in 1950. It develops as a late complication in 10 % of patients who suffer from gastroesophageal reflux disease (GERD). The most serious complication is the esophageal adenocarcinom which is unresectable for cure at the time it is datected.*

Key Words: Barrett's esophagus, gastroesophageal reflux disease (GERD) esophageal adeno carcinoma.

Barrett's esophagus is a condition in which the normal stratified squamous epithelium of the esophagus is replaced by a metaplastic columnar epithelium of the stomach. It is first described by Normann Barrett in 1950 (1). It develops as a late complication in 10 % of patients who suffer from gastrosophageal reflux disease (GERD). The most serious complication is the esophageal adenocarcinoma which is unresectable for cure at the time it is detected.

DEFINITION

Barrett's esophagus is defined as the presence of columnar epithelium 2-3 cm proximal to the lower esophageal spincter (LES) (2). The normal esophagus may contain normal gastric mucosa by a substantial length of squamose epithelium. There are three types of Barrett's mucosa, junctional (cardiac), fundic, specialised columnar (intestinal metaplasie) (1-4). Some authers advocate an "unclassified" type which is a variant of specialised columnar epithelium with a high grade dysplasie (3). Acid is also produced by metaplastic columnar epithelium but the volume is not enough to produce peptic ulser disease. Pepsinogen and gastrin are also found in specialised columnar epithelium.

CLINICAL BACKGROUND

The avarage age at the time of the diagnosis of Barrett's esophağus is about 55 years (1).

Fellow, Gastrointestinal Surgery Department, Attending surgeon, Gastrointestinal Surgery Department.

The male to female ratio is 4:1. There is a bimodal age distribution: 0 to 15 years and 40 to 80 years. Though some patients with Barrett's esophagus may be asymptomatic heartburn is the most common presenting symptom. Dysphagia, regurgitation, gastrointestinal bleeding, nocturnal aspiration, pyrosis chronic obstructive pulmonary disease (COPD) are other presenting symptoms. Complications of Barrett's esophagus are same with those of (GERD) which are esophagitis, esophageal ulser, stricture being found 50 % at or below the squamo-columnar junction (6), esophageal bleeding, iron deficiency anemia, aspiration pneumonia, dysplasia and adenocarcinoma of the esophagus. The increased risk of adenocarcinoma is between 30 and 125 times greater than that in the healty population (7).

DIAGNOSIS

The usefulness of the radiographic findings are limited because they are neither spesific nor sensitive enough to distinguish squamous epithelium from columnar epithelium. Monometric studies and 24 hour esophageal pH studies are useful to localise the lower esophageal sphincter (LES) and to obtain the biopsies above the region when doubt exists. Patients with Barrett's metaplasia show a prolonged exposure to acidic GER (pH 4) than the patients whith reflux esophagitis because of the delayed acid clearance evident in Barrett's esophagitis (1). Endoscopic examination is the cornerstone of the diagnosis. Metaplastic mucosa can easly be recognized by endoscopy by sharp demarcation between the pale squamos esophageal mucosa and velvet pink gastric columnar mucosa extending to the proximal esophagus rather than its normal localisation at the diaphragmatic hiatus. Endosonography is another diagnostic tool which shows that the esophageal mucosa is thicker than normal in columnar lined region of the Barrett's esophagus. Radionuclide imaging is used to diagnose the metaplastic columnar mucosa which concantrates 99 m Tc pertechnetate. Endoscopic staining method using Lugol's solution in the diagnosis of Barrett's esophagus in patients who have nonspecific mucosal abnormalities is another diagnostic method (8).

TREATMENT OF BARRETT'S ESOPHAGUS

Medical Therapy

The milestones of the therapy are to minimise the symptoms and to hinder the development of the complications. Patients having symptoms of GERD such as heartburn, regurgitation, pyrosis are advocated to change their lifestyl which includes discontinuation of smoking, avoidence of bedtime snack, dietary modification which means the elimination of fats, chocolates, alcohol and carminatives which decrease the (LES), and percribed antacids, metaclopromide, a benzamid derivate, dopamin antagonist (9), H_2 receptor antagonists such as cimetidine, first described receptor blocker in 1976 given 800-1600 mg per day, ranitidine given 300-1200 mg per day, famotidine given 40 mg per day (10) Omeprazole H^+ $K^{+'}$ ATP' ase inhibitor is the penultimate therapy for this purpose. It is given 40 mg daily. It appears that potent acid reducing regimes provide the most effective means of healing Barrett's ulcers. Topical agents such as sucralfate may improve reflux symptoms and fascilitate healing of reflux esophagitis. Algorithm for symptomatic reflux esophagitis and management of Barrett's esophagus is shown in figure 1. (11).

Surgical Therapy

The indications for the surgical intervention in Barrett's esophagus are nonhealing deep esophageal stiructures unresponsive to bouginage or dilatation, esophageal hemmorage, perforation, severe symptomatic reflux refrac-

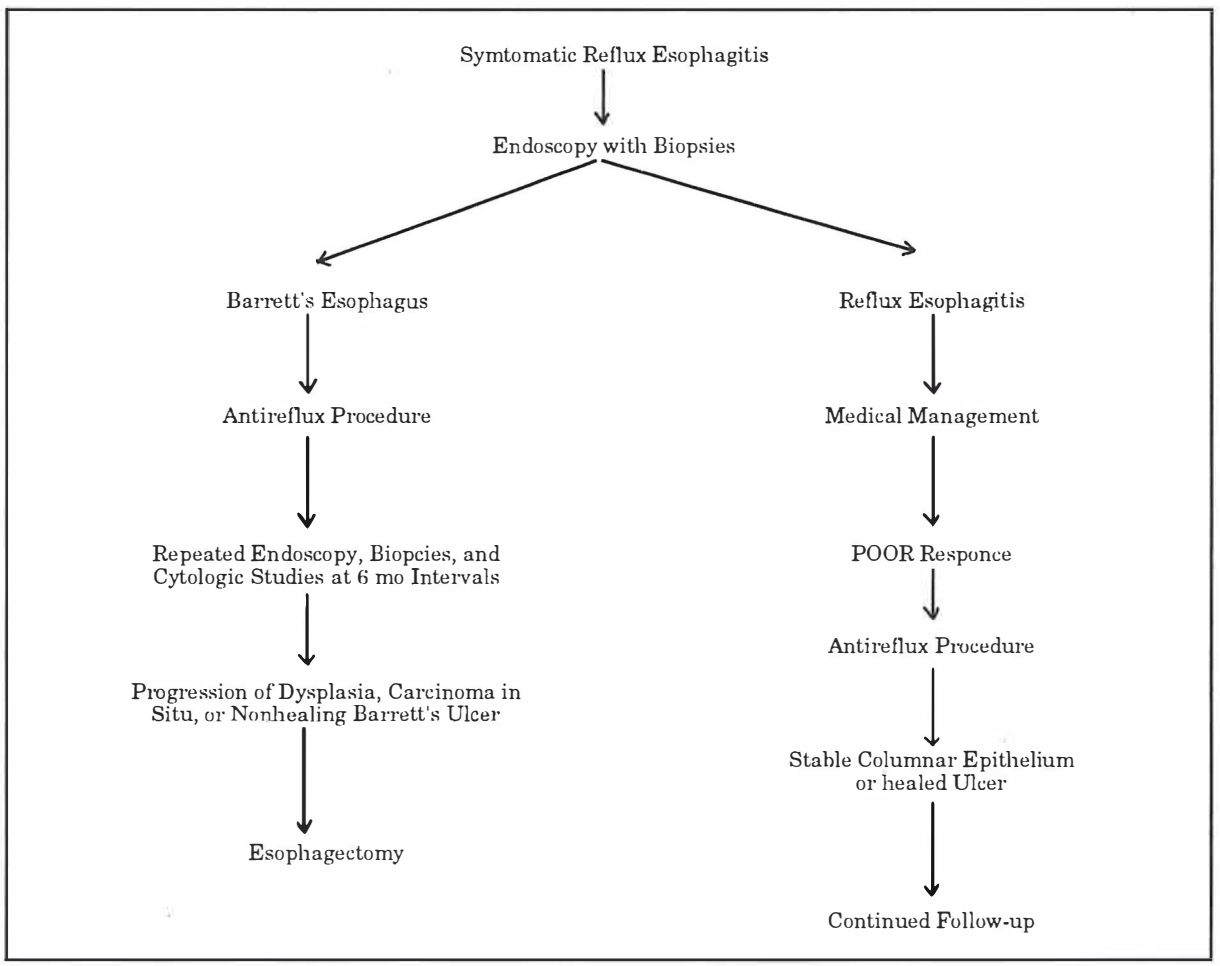

Fig 1: Algorithm for symptomatic reflux esophagitis and management of Barrett's Esophagitis

tory to medical therapy, pulmonary aspiration, moderate dysplasia, as a cancer prophylaxis with an assumption that the control of reflux will hinder the progression of high-grade dysplasia to adenocarcinoma and lastly adenocarcinoma of the esophagus.

The intervention is usually directed at constructing an effective antireflux barrier to GER. The advocated procedures are Nissen fundoplication, Hill posterior gastropexy and Belsey esophagogastroplasty (1-3).

Nissen fundoplication is in introduced by Nissen in Switzerland in 1955 which is performed through either abdominal or thoracic approach In either case the distal esophagus is fully detached from the margins of hiatus. Several short gastric vessels are ligated and divided for facilitation of a full 360 degree plication of gastric fundus around the abdominal segment of esophagus without any tension and without injury to the spleen. The fundus is brought posteriorly around the esophagus. A pair of sutures are placed through the anterior fundus and the wall of esophagus, where 3-4 cm segment of the intraabdominal esophagus is wrapped by fundus. Fig 2 (11).

Belsy esophagogastroplasty is performed by transthorasic approch that creates a segment of intraabdominal esophagus held in place by a buttress of plicated stomach that surraunds 280 degree of the distal esophagus (12). After the cardia and the lower esophageal segment

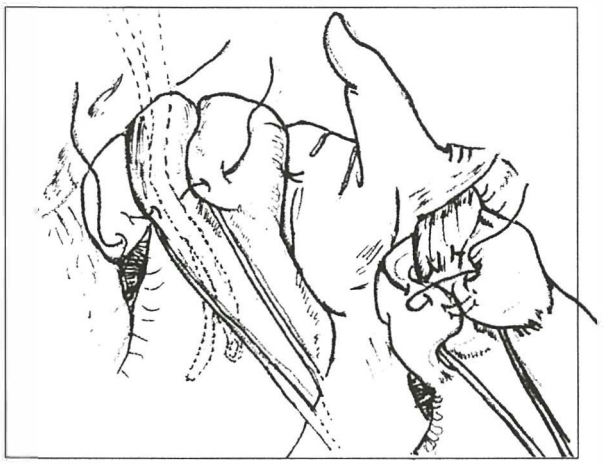

Fig 2: Nissen Fundoplication

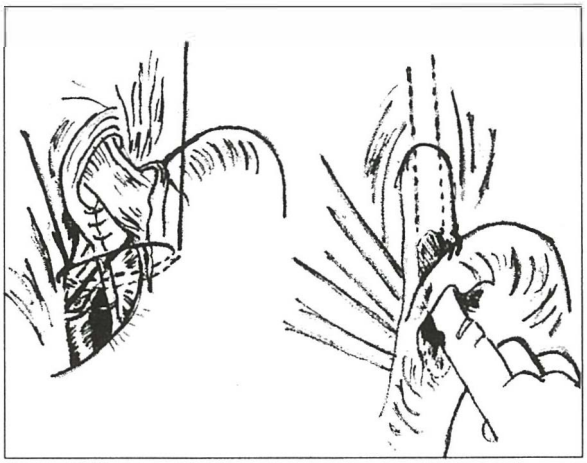

Fig 4: Hill Posterior Gastropexy

is compleatelly cleared of the connective tissue mattress sutures are placed between gastric fundus and muscular leyer of the esophagus 1-2 cm above or below the gastroesophageal junction. After the sutures are tied a second row of mattress sutures is placed to imblicate additional fundus onto the lower esophagus. This second row of sutures passes through hiatus and out through the tendineous portion of diaphragm. Before they are tied curural sutures are placed to narrow the hiatus. Fig. 3

Hill posterior gastropexy and the calibration of the cardia is first performed by Hill 1961. After reducing esophageogastric junction curural sutures narrowing the hiatus are placed behind the esophagus. A single suture is placed incorporating gastrohepatic omentum along the lesser curvator of the stomach and the arcuate ligament of the aorta (11, 12). A pair of sutures are placed on lesser curvatur anchorhing stomach posteriorly. Care is taken to avoid injury to adjacent vagal trunks. The trick of the trade is that when the the sutures are tied, a gastric sling fiber should be sufficiently shortened to permit the distal phalanx of the index finger to invaginate freely into distal esophageal lumen. Fig. 4.

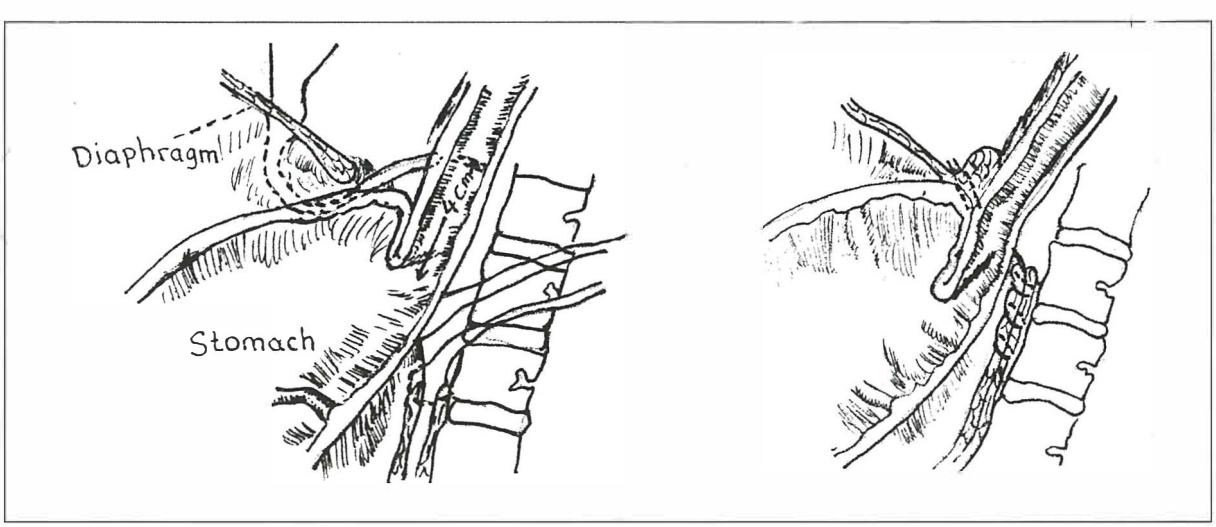

Fig 3:Belsy Esophagogastroplasty

Vagatomy may be needed for patients whose stricture requires continual dilation (4). If high-grade dysplasia is detected in a biopsy specimen from mucosa that showed no gross endoscopic abnormality it is recomended to repeat the endoscopy with multiple biopsy specimens taken of the area of the dysplasia to determine its extend and search for coexisting adenocarcinoma. In some patients the condition with high-grade dysplasia may regress, in others it may remain stabile for many years (13) and in others it may progress to adenocarcinoma. Therefor every patient must be evaluated individually. Hospital mortality rates for esophagectomy are pyhsician-spesific, ranging from 1.4 per cent to 37.5 per cent (2).

Overall, the prognosis for patients with esophageal adenocarcinoma is poor with a 5 year survival only 7 per cent. However endoscopic surveialance of the patients with Barrett's Esophagus permits early detection of Adenocarcinoma. Once the intramucosal adenocarcinoma is documented in Barrett's Esophagus surgical resection is recommended. Because the Barret's adenocarcinomas are frequently multicentric or associated with high-grade dysplasia and because specialized metaplasia can become dysplasia and finaly adenocarcinoma the entire columnar-lined segment must be resected.

Acknowledgment: We are greatful to Dr. Musa Akoğlu for critical review of the manuscript.

REFERENCES

1. Phillips RW, Wong RKH: Barrett's esophagus. Gastroentorology Clinics of North America 1991; 20: 791-816.
2. Reid BR: Barrett's esophagus and esophageal adenocarcinoma. Gastroenterology Clinics of North America 1991; 20: 817-834.
3. Katzka DA: Barrett's Esophagus: Detection and Management. Gastroenterology Clinics of North America 1989; 19(2): 339-57.
4. Pollepalle SC, McCallum RW: Barrett's Esophagus, Currennt assesment and Future Perspective. Gastroenterology Clinics of America 1990; 19(3): 733-44.
5. Schwartz SI, Ellis H: Gastroesophageal Reflux Disease, Hiatus Hernia, Achalasia of Esophagus and Spontaneous Rupture. Maingot's Abdominal Operation 1990; 520.
6. Lackey C, Rankin RA, Welsh JD: Stricture Localisation in Barrett's Esophagus. Gastrointestinal Endoscopy 1884; 30(6): 331-33.
7. Clark GWB, Jenkins HJ: Endoscopy in Diagnosis of Foregut Disease. Problems in General Surgery 1992; 9(1): 1-13.
8. Dalay R, Görgül A, Caner E, Boyacıoğlu, S, Ateş B et al: Barrett Ösofagus Tanısında Endoskopik Boyamanın Değeri. Gazi Tıp Dergisi 1990; 3: 137-40.
9. Kayaalp O; Antiemetik ilaçlar. Tıbbı Farmakoloji 1986; 3 baskı; 2771-73.
10. Lee FI, Isaacs PET: Barrett's Ulcer: Responde to Standard Dose Ranitidine, High Dose Ranitidine, and Omeprazole. The Am J. of Gastroenterology 1988; 83(9): 914-16.
11. Starnes VA, Adkins B, Hallinger JF, Sawyers JL: Barrett's Esophagus. Arch Surg., 984, 119: 563-67.
12. Schwarts S, Shires GT, Spencer FC: Esophagus and Diaphragmatic Hernies. Principles of Surgery 1988; Fifth Edition; 1117-1125.
13. Sabiston DC: The Esophagus. Textbook of Surgery 1991; Fourteenth Edition: 711-712.