INTRODUCTION

Barrett's esophagus is refer to an abnormal changes (metaplasia) in cells of the lower end of the esophagus, (non keratinized epithelium to columnar epithelium) BE is found in about 10% of patients who seek medical care for heartburn. ***(Koppert L and Wijnhoven B., 2005).***

Barrett's Esophagus is sometimes called Barrett's syndrome (CELLO) Columnar epithelium lined lower esophagus or Colloquially as Barrett's. ***(Stein H and Siewert J., 1993).***

It is estimated that 5% to 15% of patients with GERD will have BE, patients with long standing GERD are at greater risk for developing BE, which is considered the precursor lesion to adenocarcinoma. The presence of BE increases the individual's relative risk of cancer 30 to 120 times compared with persons without BE. ***(Miros M and Kerlin p., 1991).***

The esophagus is a muscular tube that service as food passage between pharynx and stomach. The first element of the Greek term oisopagos is derived from oisum " to carry ", the second may have its origin from phagos " to eat " or phagema "food ". (***Liebermann et al., 1995).***

It's length is about (18-26 cm) .There are three distinct regions: cervical, thoracic and abdominal. The abdominal part extends from the diaphragmatic hiatus (T_{10} vertebral level) to the orifice of the cardia of the stomach, it forms a truncated cone, about 1 cm long, lies in the esophageal groove on the posterior surface of the left lobe of the liver. The lower esophageal sphincter is not a clear anatomical demarcation but it is a high pressure zone that prevents the back flow of food. (***Long JD and Orlando RC., 2002***).

The rich arterial supply of the esophagus is segmented, branches of the inferior thyroid artery provide arterial supply to upper esophageal sphincter and cervical part, paired aorto-esophageal arteries or terminal branches of bronchial arteries supply the thoracic esophagus, the left gastric artery and branch of the left phrenic artery supply LES and the most distal segment of the esophagus. Arteries that supply the esophagus form dense network in sub mucosa of esophagus. (***Skandalakis JE and Ellis H., 2000***).

Innervations of the esophagus like the viscera, receives dual sensory innervations, traditionally referred to as parasympathetic and sympathetic but more properly based on the actual nerves, vagal and spinal. (***Goyal R and Sivarao D., 1999***).

The esophageal Wall consists of four layers: Mucosa, sub mucosa, musclaris propria and adventitia. Unlike other areas of the gastrointestinal tract, the esophagus does not have a distinct serosal covering, this allows esophageal tumors to spread more easily and makes them harder to treat surgically. (***Boyce H and Boyce G., 2003***).

The mucosa is thick and reddish cranially and more pale caudally. It is formed of non keratinized squamous epithelium which covers the entire inner surface of the esophagus except LES, where both squamous and columnar epithelium may co exist. Lamina propria is thin a layer of CT. Muscularis mucosa is a thin layer composed of irregularly arranged smooth muscle fibers. ***(Christensen J and Wingated DL., 1983).***

The sub mucosa contains connective tissue as well as lymphocytes, plasma cells, nerve cells (Meissner's plexus), vascular network (Heller plexus) and mucous glands. The muscularis propria is responsible for motor function, the upper 5% - 33% is composed exclusively of striated

(skeletal) muscle and the distal 33% is composed of smooth muscle. In between there is mixture of both, called the transition zone. The adventitia is an external fibrous layer that covers the esophagus and connecting it with neighboring structures. ***(Ghosh SK., 2006).***

For unknown reasons, Barrett's esophagus is found three times often in males than in females. Between the seventh and eighth decade of life (around age of 40years), in some instances, Barrett's esophagus appears to be not congenital (present above the age of five). ***(Cameron AJ and Lomboy CT., 1992).***

There is a relationship between chronicity of heartburn and the development of BE, but there is no relationship between the severity of heartburn and development of BE. The exact cause of BE is not known, it may occur as a complication of chronic GERD. Symptoms are similar to those produced by chronic GERD, such as heartburn, reflux of stomach acid into the mouth, dysphagia, vomiting of blood and unintentional weight loss. ***(Cameron AJ and Lomboy CT., 1992).***

The diagnosis of BE is not easy, at the present time it can not be diagnosed on the basis of symptoms, physical exam or blood tests. The only useful test is upper gastrointestinal endoscopy and biopsy. Further diagnostic evaluation is indicated when there is no response to empiric therapy or in the presence of warning symptoms of complicated GERD, i.e., dysphagia, bleeding, weight loss and/or chest pain. (***Devanult KR and Castell DO., 1999***).

There are two requirements for the diagnosis of BE, endoscopy show an abnormal pink lining is seen as replacing the normal whitish lining of the esophagus, for short distance (usually less than 2.5 inches) up the esophagus from GEJ and biopsy (tissue sampling) under the microscope

the presence of intestinal cell types that are called goblet cells, because of their shape, if intestinal goblet cells are not present the diagnosis of BE shouldn't be made. The most important predictor of cancer risk in patients with BE is the presence of a degree of dysplasia. Biopsy specimens are graded as negative, indefinite for dysplasia, low-grade dysplasia (LGD), and high-grade dysplasia(HGD). ***(Reid B J, et al., 1988).***

The patient with LGD progresses to HGD in a median period of 11monthes ranges (2-43monthes). ***(Skacel M, et al., 2000).***

In study, 32% of patient with HGD developed adenocarcinoma in an average of 3years, diffuse HGD associated with a 3.7fold increase in the risk of adenocarcinoma compared with focal HGD. ***(Buttar N S, et al., 2001).***

Identification of tumor markers and other specific risk factors may be helpful in predicting who is at risk for dysplasia, the current recommendations include routine endoscopy and biopsy (looking for dysplastic changes) every 12monthes or so while the underlying reflux is controlled with proton pump inhibitor drugs in combination with measures to prevent reflux. (***Sampliner R E., 2002***).

Laser treatment is used in severe dysplasia, while overt malignancy may require surgery, radiation therapy or systemic chemotherapy. Presently there is no reliable way to determine which patient with Barrett's esophagus will go on to develop esophageal cancer. Endoscopic mucosal resection (EMR) has also been evaluated as a management technique. ***(Reshamwala P and Darwin P., 2006).***

Additionally an operation known as a Nissen fundoplication can reduce the reflux of acid from the stomach into the esophagus. ***(Abbas A, et al., 2004).***

Other experimental strategies for eliminating Barrett's esophagus, such as Thermal ablation and Photodynamic therapy, are based on the principle that long-term regression may require reinjury of the metaplastic epithelium, followed by regeneration of normal squamous epithelium. ***(Overholt BF, et al., 2003).***

Thermal ablation using multipolar electro coagulation, Argon plasma coagulation, and Laser therapy ablation are all feasible in the sense that they are minimally invasive and cause less morbidity and mortality than surgery. ***(Attwood SA, et al., 2003).***

The aim of endoscopic screening and surveillance is to both identify BE and detect early neoplastic changes, defined as dysplasia, The goal of such monitoring is to improve early recognition of invasive esophageal cancer, presumably at a curable stage. ***(Falk GW., 1999).***

Published guidelines for the surveillance of BE suggest different intervals for surveillance endoscopy, depending on histological finding of previous endoscopy, once inflammation related to GERD is controlled and endoscopic diagnosis of BE is established, current guidelines recommend obtaining four-quadrant jumbo biopsies at 2-cm intervals. ***(Sampliner RE., 2002).***

The true incidence of BE is 0.5 percent per year, at less frequent endoscopy interval of three to five years is also reasonable. As already mentioned, once in a life time endoscopy may be sufficient in patients without dysplasia. ***(Inadomi JM, et al., 2003).***