Is Columnar Lined Esophagus and Barrett's Esophagus Two Names of A Single Entity?

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Gastro-esophageal reflux disease (GERD) is one of the major pre- requisite for the development of columnar lined esophagus (CLE). The. diagnosis of GERD is best made on barium studies in Trendelenberg position or during a valsalva procedure. Alternatively, upper G.1. endoscopy can show a lax lower esophageal sphincter and gastroesophageal reflux indicating the presence of reflux disease. Twenty-four hours PH manometery using an ambulatory PH meter cannot only help in diagnosing the presence of GERD but also the frequency with which one refluxes during 24 hours. Radioisotopic studies are also used as complimentary tests to check the esophageal transit time and the degree of reflux[1].

The complications of GERD include esophagitis (60-90%), peptic ulceration and dysphagia (40~8O %)[2-9] peptic stricture (20-50%)3, [3-5,7,10] bleeding (3o%)[3,5,11-15] columnar change (5-20%)[1] and development of adenocarcinoma in 20% cases[16-19]

Banet in 1957 was the first one to report the presence of columnar epithelium in the esophagus and since then this entity is called Barrett's esophagus (BE) [20]. The term CLE and BE have since been used interchangeably. BE is commonly defined as the presence of CLE extending 3 cms above the top of lower esophageal sphincter (LES) zone in adults. Occasionally, there is a 2-3 ems dissociation between the Z line and squamo-columnar junction (SC) causing difficulty in the diagnosis of BE[21]. The controversy about the level of SC junction, LES zone and level of CLE is mainl2y because of the reason that BE is a premalignant condition[22]. Studies have shown that some individuals with CLEJBE develop carcinoma while others do not. Adenocarcinoma of the esophagus in about 20% cases arises from this specialised metapiastic epithelium of BE. There has been a debate about the etiology of BE that whether it is congenital or acquired. Borrie and Goldwater[11] proposed a congenital theory by finding a bimodal distribution of BE in their patients, i.e., a group of 0-10 years and a group over 40 years of age. Another point favouring congenital theory is the embryological development of the esophagus. In the early embiyogcnesis i.e., during 3-34mm stage the esophagus is lined by stratified squamous epithelium, at 40mm it is replaced by ciliated columnar epithelium and at 130 mm stage there is caudal and cephalic spread of squamous epithelium from the centre of esophagus[23]. In about 4% cases there is incomplete transformation of the epithelium resulting. in heterotropic epithelium or inlet patch[24-29]. These individuals may later present with stricture of the esophagus at the site of heterotropic epithelium Majority of workers have confirmed an acquired etiology of BE. The association of BE with GERD and its complication[30,31], endoscopically observed extention of Barrett's mucosa in refluxers 33 arid development of Barretts mucosa (BM) after surgery on lower esophagus[34-36] all go in favour of an acquired etiology. The underlying etiology in BE is injury. Most workers believe that squamous epithelium is more resistant to injury than columnar[37,38] epithelium but once a certain amount of injury is inflicted upon the squamous epithelium (may be sudden, high dose as in lye ingestion[39], chemotherapy[40] or chronic as in reflux disease), then squamous epithelium is unable to grow and is taken over by columnar epithelium. Evidence exists that injury causes destruction of squamous epithelium, there is proximal extension of BM showing histologic evidence of ulceration in areas, where columnar epithelium later appears[41-43]. Moreover, BM shows evidence of previous

destruction, Le., sparse gland, inflammatory cells, collagen in lamina propriaa and smooth muscle proliferation in muscularis mucos[44,45]. Endoscopic biopsy of ulcers shows a single layer of immature epithelial cells covering the granulation tissue during initial epithelialization, later these immature cells undergo columnar change, if exposed to persistent injury[46]. Some relation has been found between the severity of GERD and epithelial recovery. In mild to moderate reflux disease, the squamous epithelium is replaced by the squamous epithelium while, in severe and prolonged reflux, the squamous epithelium is replaced by columnar epithelium or CLE. On endoscopy, CLE can be diagnosed on the type of Z line which may be smooth, serrate, tongue like, finger like or flame like. Once suspected, it should always be confirmed on biopsy1. Though low lower esophageal sphincter pressure, prolonged period of reflux[47], dysmotility of the esophagus, the quality and quantity of refluxate have been incriminated in the pathogenesis of BE but strongest evidence is for the presence of bile in the refluxat[48] to be very specific. it is the trypsin which causes maximal damage[49] The high risk group prone to develop CLE are male smokers with a prolonged history of GERD. Frequency of CLE varies in different countries and populations. In an autopsy study of 51)() cases, 12% were found to have CLE[50] Of 1 GO0 cases examined radiologically, 4% had CLE[50] and similarly of 6000 subjects endoscoped for various reasons, 5% had GERD and 11% of these 5% had CLE[11]. Overall, 5-20% GERD convert to CLE. Local data available on the frequency of GERD and £LE is scarce. It has been observed that signs and symptoms of GERD are common in our population. Retrospective analysis of the previous two years data at PMIRC showed a 41%frequency ofendospopic esophagitis in patients undergoing endoscopy for various reasons (unpublished data). As a result of frequent signs and symptoms of GERD and endoscopic evidence of esophagitis in our patient population, a collaborative study was conducted between our centre and the Basic Medical Sciences Institute (BMSI) (pathology) to study the frequency of BE in patients having symptoms of GERD. Of 100 biopsies taken from GERD cases, 32% frequency of BE was reported (M. Phil thesis). During the past 2 years at BMSI, 4.6% frequency of adenocarcinoma of the esophagus was reported, which also included adenocarcinoma of stomach coming up into the esophagus (unpublished data). These figures were conflicting because if we have a high frequency of GERD and BE them why is it that we have such a low frequency of adenocarcinoma of the esophagus?

At this time, the new classification of BE was reported[51] according to which diagnosis of BE should be restricted to only those patients who have epithelium which may be premalignant. According to the new classification Barrett's specialised epithelium is CLE with goblet cells and a positive alcean blue stain at PH 2.5. By this definition, BE should be diagnosed only if special epithelium with goblet cells is present for any length in the tubular esophagus. To understand BE, one must clearly understand what is metaplasia? It is abnormal transformation of one fully differentiated mucosal epithelium into another fully differentiated mucosa normally seen in that organ. Normally, distal 3 cm of the esophagus is lined by fundic or cardiac type of mucosa, so this is not metapiasia, but when specialised intestinal epithelium with goblet cells orpenath cells is seen in the esophagus, then this is metaplasia[1].

There are 3 types of gastric epithelia that can be found in the lower esophagus:

1. CLE with cardiac type of glands
2. CLE with fundic type of glands
3. Specialised intestinal epithelium with goblet cells (BE)

Histologically, there is a difference between adenocarcinoma occurring inBE and that of adenocarcinoma of stomach. Carcinoma arising in BE is multifocal and shows dysplasia in the surrounding tissue52.

Using the new classification, another set of 82 biopsies were subjected to histology at BMSI. Of these, only 12 (15%) were found to have CLE but BE was found in none. suggesting that we do have CLE hut the frequency of BE low and that is why frequency of adenocarcinoma is low. CLE has no chance of

malignant transformation but BE has 5-20% chance of malignant conversion[52]. Forthc management, it is suggested thatall patients with symptoms of GERD should be endoscoped and biopsied[1]. Esophageal mucosa should be specifically looked for a stricture, ulcer or erosion and if found, multiple biopsies should be taken in a circular fashion 2-3 cm away[22]. If the esophageal biopsy: shows CLE, reassure the patient and treat reflux but follow-up endoscopy is not suggested; but if BE is confirmed, follow-up biopsies are mandatory as BE is a premalignant condition which requires constant surveillence[22].

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