Brief communication - Esophagus

Barrett’s esophagus: participation of the esophageal wall

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Abstract

The modern inhibitors of gastric acid secretion reduce very efficiently the aggressiveness of gastroesophageal reflux but they appear to modify the pathophysiological situation in a way that favours the development of Barrett’s esophagus (BE). During the last two decades, the prevalence of BE in the population submitted to endoscopy has clearly increased while the peptic stricture of the esophagus is fast disappearing. With a reduced tendency to stricture and fibrosis, the esophagus seems more prone to the columnar metaplasia. This suggests that the factor that promotes the columnar metaplasia reaches the areas of esophagitis through the esophageal wall. In the conditions of an advanced reflux disease, the veins crossing the gastroesophageal junction would be well suited to the transport of this factor, provided they have not been obliterated by thrombosis or fibrosis. The veins do not normally participate in the metabolic activity but their permeability may be altered in a pathological environment. In the esophageal wall submitted to an important reflux, the environment cannot be normal, as evidenced by the alterations of the esophageal motricity. Furthermore, these veins are submitted to the peristaltic activity of the esophagus.

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In Barrett’s esophagus (BE), a varying amount of the squamous epithelium of the lower esophagus is replaced by a columnar epithelium. This happens in a minority of patients suffering from gastroesophageal reflux disease (GERD), at a mean age estimated around 40 years [1]. At the time when BE is recognized, the reflux is pronounced and often contaminated with bile. These characteristics are not specific to BE, however, and it is still unclear what factor drives the healing of the epithelial lesions of esophagitis towards the columnar epithelium [2]. According to clinical observations, this factor could reach the epithelium not from the lumen but from the esophageal wall as it does obviously in other instances of epithelial injury, during chemotherapy or graft versus host disease [3].

The first observation goes back a few decades, when esophageal stricture was a common complication of GERD: it has been noted that the stricture is situated at the transition zone from squamous to columnar epithelium, either at the esogastric junction or at the upper end of a segment of Barrett’s mucosa [4]. In other words, Barrett’s mucosa does not form above an established stricture, even when the esophagitis remains active above the stricture and causes a progressive shortening of the esophagus. It may be indicated incidentally that strictures within Barrett’s mucosa are not unknown but most of them are related to Barrett’s ulcers [5]. The capacity of a stricture to prevent columnar epithelium from appearing higher up in the esophagus is confirmed by a second observation made in recent years: since the potent inhibitors of gastric acid secretion are available, the incidence of strictures has been sharply reduced but the incidence of BE is on the rise [2]. Admittedly, the true incidence of BE is difficult to assess but the incidence of the adenocarcinoma of the esophagus that is closely related to BE increased up to 10-fold [6,7].

These observations suggest that the factor that promotes the Barrett’s mucosa reaches the areas of esophagitis from the esophageal wall after crossing the parietal junction between the stomach and the esophagus and that this progression is prevented by a stricture. The venous blood, that is apt to flow in either direction along the lower esophagus, is the first potential candidate for the carriage of this factor [8]. When chronic esophagitis reaches the stage of BE, the lower esophageal sphincter is very defective and there is nearly always a hiatal hernia [9]. Under such
conditions, the pressure gradient between the abdomen and the thorax tends to direct through the esophagus the venous blood of the part of the gastric wall that forms the hiatal hernia, towards the azygos vein rather than towards the portal vein (Fig. 1). This collateral circulation is apparently prevented in most cases, however, because the tiny veins of the ‘palisade zone’ that are running just underneath the epithelium at the level of the lower esophageal sphincter are directly exposed to the peptic inflammation and to the fibrosis that goes with it [8]. The potent inhibitors of the gastric acid secretion reduce the aggressiveness of reflux and likely prevent in some cases the venous disconnection between stomach and esophagus as they prevent the strictures. This could account for the increased incidence of BE.

In GERD, the conditions for the apparition of BE are not satisfied from the outset. If the venous circulation at the esogastric junction is really involved in the apparition of BE, the time gap between the onset of the symptoms and the apparition of Barrett’s mucosa would give the opportunity to achieve the esogastric venous disconnection and prevent the BE [10].

References