PEPTIC ULCER DISEASE: CAN A BEHAVIORAL FACTOR BE IMPLICATED?

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ABSTRACT

This article aims to highlight probable behavioral factors in the etiology of peptic ulcer disease (PUD). Our hypothesis that due to an anatomic predisposition in the foregut blood supply and its gross anatomy, whenever a person with a replete or full stomach changes their position, they may affect/disrupt the blood supply to the lower esophagus, stomach, and proximal part of the duodenum. Our hypothesis states that if a person has a habit of resting after a meal, in a reclined position, on either their right or left side, or is required to do so, the person will develop PUD due to prescribed bed rest, depending on their specific routine. Our hypothesis also proposes that different body positions are linked to other ulcerative peptic processes. The proposed hypothesis has several implications. Firstly, PUD is a somatic disorder and is a disease caused by lifestyle and systematically repeated habitual behaviors. Treatment should focus on the patient discussing their lifestyle habits, making them aware of their after-meal behavior and the link that the behavior has to PUD. Secondly, the primary therapeutic action should focus on postural behavioral therapy, adjusting habit-formed after-eating routines that can reverse PUD once remedied.

Keywords: peptic ulcer disease etiology; ischemia; behavioral factor

INTRODUCTION

Since the first theory regarding peptic ulcer formation, attributed to Dr. Rudolph Virchow in 1853, the commonly accepted peptic ulcer causality view has changed multiple times.[1] To-day, the development of peptic ulcers is assumed to be caused by the bacteria *Helicobacter Pylori* (*H. Pylori*). However, there are continuing concerns about the role that the bacteria play in peptic ulcers' etiology.[2,3,4]

Virchow's theory advanced a vascular origin of peptic ulcer formation. In contrast, consequent theories posed that it was disruptions in regulation, and later, the origin was said to be from bacterial infection. The latter partially explains both etiology and pathogenesis, but there remain questions, and it does not correlate well with Koch's postulates.[2,3,4]

The following hypothesis uses Virchow's thoughts on pathogenesis, Piasecki's findings of an anatomic predisposition in the foregut vasculature, and common behavioral habits identified in small epidemiological studies and in non-published behavioral interventions.[5,6]

Definitions

There are many misconceptions about peptic ulcers. These began in the scientific community and spread to the general public, including the term 'peptic,' which is commonly misunderstood. Therefore, before detailing the hypothesis, it is necessary to define which ulcerative disease is being discussed.

Differentiation of ulcerative processes in the foregut can be made based on their common characteristics. For this article, we define peptic ulcer disease (PUD) as acute. If an etiological factor persists or reoccurs, the disease can transform into chronic with particular morphological features. Peptic ulcers may heal, reactivate, and persist. Gastric peptic ulcers commonly occur on the pyloric region's lesser curvature, while duodenal peptic ulcers occur within the first two and a half centimeters of the duodenum. On average, peptic ulcers are one to three

centimeters in diameter and go deeper than the mucosa layer and over time can extend further, eventually reaching the serosa layer. They are usually round or oval, and most frequently, they occur singly, although they can be found in pairs or other groupings, such as in the proximal part of the duodenum, where they are sometimes called 'kissing' ulcers. Lastly, they can either be in the duodenum, the stomach, or both.[7]

Hypothesis

Our hypothesis is that due to an anatomic predisposition in the foregut blood supply and its gross anatomy, whenever a person with a replete or full stomach changes their position, they may affect the blood supply to the lower esophagus, stomach, and the proximal part of the duodenum, through the linking branches of the celiac trunk [5]. The stomach appears as a soft mobile bag held between two junctions, the immobilized lower esophagus and the duodenum's immobilized proximal part. Consequently, there is a great risk of blood vessels being occluded at the pathway in the proximity of these junctions, and where the occurrence of peptic ulcers is most frequent.

Intensive collateral blood supply is present in anatomic areas that could supply the necessary regions should occlusion occur. However, in many cases, anatomically, it does not function in this way.[6]

In our studies, we identified that duodenal peptic ulcers occurred most frequently when the subject was used to rest, lying on their right side after a meal, with a replete stomach, presumably, in vivo reproduction of "Mayo's anemic spot." While gastric peptic ulcers formed most frequently with a habitual resting body position lying on its left side after a meal. For ulceration to take place, repeated occlusions must take place. A behavioral habit of having a rest after a meal or a prolonged bed rest on either side can lead to such repeated occlusions. Hence, the identification and elimination of such behavior can result in the reversal of PUD.

Hypothesis Evaluation

Anatomic Particularities

An ulcer by itself, its formation in a particular location, especially singly, presumes the presence of a local factor. This has been extensively investigated. Those investigations, and the possible local factors, can be split between vascular and non-vascular origin. Those possible local non-vascular origin factors lack both anatomical and clinical evidence and are reviewed elsewhere.[8]

Piasecki showed in his investigation of local vascular factors, reconfirmed and added to the findings that the lesser curvature in the pyloric region and the first two and a half centimeters of the duodenum had the poorest vasculature, in terms of the number of vessels and their caliber. More importantly, there have been identified end-arteries that arise outside the stomach wall, pierce the main muscle and supply small mucosa areas without the presence of anastomoses with sub-mucosal plexus. In some cases where no anastomoses have been found on the capillary beds.[6]

Therefore, there are areas of the mucosa layer in the foregut where the blood supply depends solely on singular vessels.

Clinical Observations

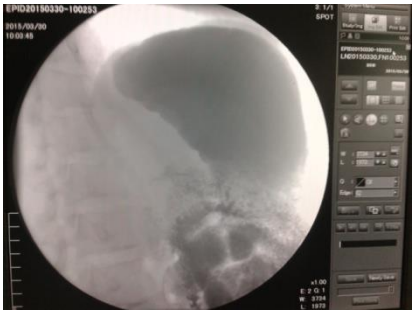
In 1908 Mayo reported his clinical observation on what was later called "Mayo's anemic spot." In his observation, he pulled the stomach down and to the left during an operation, which resulted in the formation of a white well-demarcated area on the proximal anterior duodenal wall, an area of local ischemia that resolved after traction ended. For illustrations, please refer to Piasecki and Kirk.[6, 8, 9, 10]

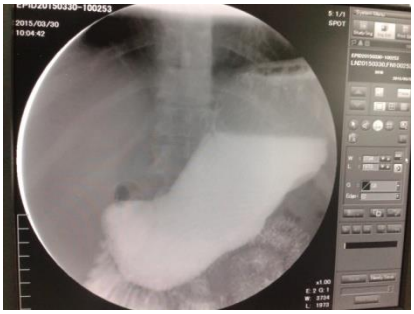
Kirk later reproduced Mayo's experiment in his investigation of duodenal ulcer etiology. He proposed that patients with duodenal peptic ulcers should "eat frequent small meals and so prevent gastric distention and displacement" and that the local factor leading to ulceration was the ischemia.

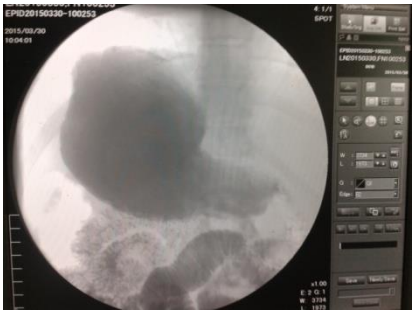
X-Ray imaging of the stomach

The X-Ray images with contrast demonstrate well the extent of deviation of the stomach, filled with contrast-rich fluid (1,250ml), concerning the spine. While in vertical position vertebral column almost equally divides the stomach into halves (Picture B), the filled stomach location with horizontal body positioning becomes almost completely beyond and down re the vertebral column at the respective side. (Picture A and C). Areas of significant distention are also evident in Picture C at the pylorus.

Figure 1 Mobility of the stomach filled with contrast-containing fluid


Picture A. Left side horizontal


Picture B. Vertical


Picture C. Right side horizontal

Pathogenesis of PUD

PUD's pathogenesis begins with the presence of acute or chronic hypoxia, the result of obstruction of blood flow caused by the bending of a blood vessel. This happens whenever a person has a habit of resting on their side after a meal or is required to do so as part of their prescribed bed rest. Due to these obstructions, and as a mechanism to eliminate blood stasis, arterial-venous anastomoses form before the obstruction's site to shunt the blood away. This reduces edema, but at the same time also causes greater ischemia of the mucosa.[6]

Eventually, the mucosa and the layers up to the level of the obstruction become necrotized. The stomach's aggressive acidic environment could participate in ulcer formation, but this is not obligatory, as ulceration can equally occur in an achloridic environment.

The ulcerative process involves a local inflammation that progresses to erosion, that erosion becomes an ulcer. Then, a continuation of the persistent and reoccurring etiological factors will lead to a chronic ulcer formation.

The affected part of the stomach or duodenum is limited to the area supplied by the occluded vessel. This factor explains the right-shaped, well-demarcated forms of peptic ulcers.

Restoration of blood supply to the area of ulceration reverses the process, and the ulcer begins healing. The time taken to heal depends on the size and severity of the ulcer. On average, chronic ulcers heal in forty days, but this figure could differ if the proposed etiology-based treatment is applied.[5]

Chronic conditions such as functional dyspepsia, gastritis, and duodenitis, or a combination of both, cause pre-ulcer conditions, as they appear as a local response to hypoxia.

Empirical Testing of the Hypothesis

Our hypothesis can be tested empirically by conducting controlled studies on patients who suffer from PUD. Using a control group, on placebo medication, and a test group who have theirs after meal habits adjusted to prevent reclining after eating. This study result would reveal the efficacy of both the treatment and the veracity of the hypothesis.

CONCLUSION

This article aimed to highlight probable behavioral factors in the etiology of PUD. Our hypothesis stated that if a person has a habit of resting after a meal, in a reclined position,

on either their right or left side, or is required to do so due to prescribed bed rest, the person will develop PUD or pre-PUD conditions, depending on their specific routine.

Our hypothesis also proposed that different body positions are linked to different ulcerative peptic processes' other locations. The location where the peptic ulcers form depends on whether the patient lies more on their left or right side.

The proposed hypothesis, if correct and accepted, has several implications. Firstly, PUD is a somatic disorder and is a disease caused by lifestyle and systematically repeated behavior. Treatment should focus on the patient discussing their lifestyle habits, making them aware of their after-meal behavior and the link that the behavior has to PUD.

Secondly, the primary therapeutic action should focus on postural behavioral therapy, adjusting habit-formed after-eating routines that can reverse PUD once remedied.

Disclosure of interest

No conflict of interest

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