Gastroesophageal Reflux in the Intensive Care Unit Patient

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KEYWORDS

• Gastroesophageal reflux • Critically ill patients • Microaspiration
• Nursing interventions • Positioning • Acid suppression • Enteral nutrition

KEY POINTS

- The incidence and prevalence of gastroesophageal reflux disease (GERD) is increasing worldwide.
- The number of patients admitted to an intensive care unit with GERD is unknown.
- Current treatment of GERD includes medications, behavioral and life style modifications, and surgical interventions.
- Interventions used in critically ill patients may increase the risk of reflux and complications.

INTRODUCTION

Globally, the prevalence of gastroesophageal reflux disease (GERD) continues to increase at more than 25% in North America and Europe,[1] and notably since 1995, an increased incidence has been noted in Asia as well.[2] The prevalence rate of GERD in developed countries is also linked to age with adults ages 50 to 70 being the most commonly affected. Longer life expectancy combined with an aging population will likely contribute to an increased prevalence of GERD into the future. However, morbidity and the associated economic burden of GERD reaches across the age spectrum, with risk concerns extending even to premature infants, infants and neonates, and those with compromised respiratory health.[3–5] In the United States, the prevalence range for GERD is estimated to be between 10% and 20% of the population, with the most common symptom of GERD, heartburn, estimated to affect 10 million adults in the United States on a daily basis.[6]

Disclosure Statement: The authors have no financial interests, affiliations, or conflicts of interest that relate to the publication of this material.
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Crit Care Nurs Clin N Am ■ (2017) ■–■
https://doi.org/10.1016/j.cnc.2017.10.011
ccnursing.theclinics.com

The specific incidence of GERD in the patient in the intensive care unit (ICU) is unknown; however, it is paramount that critical care nurses possess an understanding of the causes of GERD and the associated risks and added morbidity it poses to the critically ill patient. The purpose of this article is to briefly review the pathophysiology, risk factors, and current treatment modalities used in the management of GERD, and to discuss the assessment of, risks for, and potential complications of undiagnosed, unrecognized, or known GERD in the adult ICU patient.

PATHOPHYSIOLOGY

Specific anatomic structures known as the esophagogastric junction are designed to be protective. The esophagogastric junction is a circular structure composed of strong, smooth muscle fibers of the lower esophageal sphincter (LES) that are surrounded by oblique gastric fibers, which collectively are attached to the striated muscles of the crural diaphragm, and supported by the diaphragmatic hiatus through which the esophagus passes.[1] The esophagogastric junction essentially functions as a 1-way valve to prevent the flow of gastric acid back into the esophagus during varying degrees of pressure changes occurring in the stomach during digestion.[7]

Gastroesophageal reflux (GER), in which gastric contents reenter the esophagus, is a normal physiologic response occurring several times over the course of a day in all individuals. It is important to note that this physiologic reflux occurs transiently and spontaneously in the esophagus for a variety of reasons, most frequently after meals, when swallowing, in gamma-aminobutyric acid–mediated stretch responses, or from gastric distension as a means of expelling gas that has accumulated in the stomach,[8] with gastric distention as the primary reason. GER is also more likely to occur after a large and/or high-fat meal. Intact protective mechanisms involving the esophageal sphincters return any refluxed content to the stomach, and there are typically no symptoms reported or associated with GER.

Reflux that is significant in terms of the amount of gastric contents, that occurs frequently, and that produces symptoms patients describe as problematic or that impact their quality of life, is characterized as GERD.[9] In GERD, regurgitation of the gastric contents into the esophagus is due to a sensorimotor malfunction that results in an ineffective protective antireflux barrier, or additionally, but rarely, from an overproduction of acid by gastric tumors that form in the stomach in Zollinger-Ellison syndrome.[10] A number of factors affect protection of the esophageal mucosal from erosive gastric or duodenal contents. Factors include impaired motility of the esophagus to clear refluxed contents, acid neutralization by saliva, esophageal resistance at the cellular level, impaired barrier function (relaxation) of the LES, and the presence of a sliding hiatal hernia,[1] as well as gastric factors such as delayed gastric emptying (in up to 40% of patients with GERD), pepsin activity, and the presence of *Helicobacter pylori* infection.[9,11] Of interest is that *H pylori* may have a protective role in esophagitis and GERD symptoms, although the association is not well-understood and continues to be investigated.[12–14] Abnormal amounts of reflux can also lead to mucosal changes in the esophagus, including Barrett's esophagus, which is associated with the development of esophageal cancer.[15]

Signs and Symptoms

Efforts in understanding the complex relationship between severity of symptoms and the extent of GERD remains the focus of research. Symptoms of GERD (**Table 1**) may be similar to symptoms of a number of health conditions and diseases. A diagnosis of GERD and its underlying cause may be delayed or even unrecognized because