Surgical Critical Care
Gastrointestinal Complications

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KEYWORDS

• Intra-abdominal hypertension • Intra-abdominal pressure • Stress ulceration
• Abdominal compartment syndrome • Intestinal ileus • Olgilvie's Syndrome
• Enterocutaneous fistula • Pseudo-obstruction

KEY POINTS

- Intra-abdominal hypertension (IAH) represents an often under-recognized threat to the critically ill patient population.
- A wide range of associated causes, as well as difficulty attributing early organ dysfunction to intra-abdominal pressure (IAP), may lead to insidious progression.
- Recognition of risk factors, vigilant monitoring of abdominal pressures, early implementation of noninvasive measures to reduce IAP, and prompt decompressive laparotomy for refractory or progressive IAH are the keys to successful prevention and management of this condition.

Critical illness and injury affect the gastrointestinal tract almost uniformly to some extent. Complications of critical illness range from the sequelae of direct intestinal injury and repair to impaired motility, intra-abdominal hypertension (IAH), and ulceration, among others. Contemporary clinical practice has incorporated many advances in the prevention and treatment of gastrointestinal complications during critical illness. In this article, the epidemiology, risk factors, means of diagnosis, treatment, and prevention of some of these compilations are discussed.

STRESS ULCERATION

Extensive trauma, critical illness, and shock states may result in persistent relative hypotension and catecholamine release leading to prolonged splanchnic hypoperfusion. This can then be exacerbated by vasopressor requirements and overall low cardiac output. As a result, gastrointestinal vessels may fail to autoregulate or adequately compensate, resulting in mucosal ischemia. The cells then become dysfunctional, causing a decrease in bicarbonate release and lack of sufficient acid neutralization. As a consequence of this progressive intramucosal acidosis and hypoxemia, cell death outpaces cellular regeneration and ulceration occurs.

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Epidemiology

Early studies surrounding the risk of stress-induced ulceration in the critically ill have demonstrated that some level of gastric ulceration will occur in nearly all patients requiring intensive care unit (ICU) care.[1] The critical question, however, is how often does this insult result in clinical significance. This becomes more difficult to assess due to variations in definitions. Maier and colleagues[2] studied 98 consecutive subjects who were critically ill, requiring intubation and ICU care for at least 72 hours without gastric feeding. Gastric aspirates yielded heme-positive results in 99%, with 12% being deemed grossly positive for blood. However, only 2% required a blood transfusion and 1% required an operation. Additional studies report the incidence of stress-induced bleeding to range from 0.05% to 2.3% of trauma patients, with the lower incidence being defined as requiring at least 2 units of blood transfusion and the higher incidence failing to define bleeding in its methods.[3,4] Despite the difficulty in interpreting the incidence, the increased morbidity and mortality of those who do develop bleeding is significant. A Canadian prospective multicenter cohort study encompassing 2252 subjects demonstrated a 48.5% mortality rate in the group with bleeding as opposed to 9.1% in the group that did not.[5] Although the higher associated mortality rate likely reflects the overall systemic burden of illness and injury in those with stress ulceration, the risk seems relevant in all critically ill surgical ICU patients.

Risk Factors

Stress ulceration is a result of hypoperfusion and vasoconstriction. Therefore, the 2 most prominent risk factors include persistent hypotension and vasopressor use. Mechanical ventilation also seems to play an important role in the biochemical pathway of this disease. The mechanism for this is proposed to be made up of 3 separate causes. First, the use of positive end-expiratory pressure (PEEP) is known to reduce cardiac preload, thereby exacerbating preexisting hypotension. The use of positive pressure ventilation has also been shown to result in the elevation of sympathetic nervous tone, resulting in exacerbation of vascular shunting by way of splanchnic vasoconstriction. Finally, newer data suggest that mechanical ventilation causes the release of interleukin (IL)-1b, IL 6, and macrophage inhibitory protein 2.[6] These proinflammatory cytokines augment the cascade of ongoing cellular damage and add to the already present mucosal damage. As a sum total, multiple studies have identified prolonged mechanical ventilation as an independent predictor of clinical bleeding and, thus, a target for prophylaxis. In fact, a study of 2252 consecutive subjects admitted to 1 of 4 affiliated medical-surgical ICUs suggested that the odds of developing a bleed after 48 hours of mechanical ventilation was as high as 15 times that of someone breathing spontaneously.[5]

Two populations that merit special attention are patients with traumatic brain injuries and burn injury. In patients with significant brain injury, the stress reactions previously noted are compounded by additional insult to the hypothalamic-pituitary-adrenal axis. Specific injury in this area has been noted to result in an upregulation of terminal parasympathetic activity. Initially proposed by Rokitansky in 1841 and clarified by Cushing in 1932, the cause is thought to be a disruption of the parasympathetic centers of the hypothalamus and its connections to the vagal nuclei in the medulla. Normally exhibiting a static inhibition of the vagal nuclei, the loss of this connection, therefore, causes an upregulation of the nerve and its actions. As a result, acetylcholine release on antral G-cells is disinhibited, yielding abnormally high acid secretion. Although typically thought to be part of a multifactorial cause of ulcer