Pathogenesis and Clinical and Economic Consequences of Postoperative Ileus

Michael G. Doorly, MD, MS, Anthony J. Senagore, MD, MS, MBA*

KEYWORDS

- Postoperative ileus • Abdominal surgery
- Early postoperative small bowel obstruction • Ogilvie syndrome
- Alvimopan

Postoperative ileus (POI) is perceived as an unavoidable outcome of major abdominal surgery, primarily because of poorly understood multifactorial pathophysiology.[1] Although ileus is thought to be a disease of the small intestine, the duration of POI may be primarily dependent on the return of colonic motility.[2] Physicians should understand the risk factors contributing to the development of POI to help prevent, recognize, and treat this morbid and financial problem. POI is multifactorial in origin and the causative factors include neuromuscular, inflammatory, and pharmacologic influences.[1] Understanding the various causes of POI helps to guide its prevention, diagnosis, treatment, and reduction of cost. The enhanced recovery protocol (fast-track) provides a consistent checklist that benefits all postoperative patients. Not all postoperative abdominal distention is POI. Recognizing the differences between POI, early postoperative small bowel obstruction (EPSBO), and acute colonic pseudo-obstruction can expedite care and improve survival.

FISCAL BURDEN OF POI

Although not life-threatening, POI can prolong postoperative recovery, increase the length of stay in hospital, and the use and costs of health care resources.[3–6] For example, up to 25% of colectomy patients suffer POI, which doubles the cost of their care.[7] POI may lead to other costly morbidities. The resultant abdominal distention of POI increases the risk of hernia formation and wound dehiscence, while nausea and vomiting affect the resumption of enteral nutrition and increase the risk of malnutrition

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Keck School of Medicine of USC, 1441 Eastlake Avenue, Suite 7418, Los Angeles, CA 90033-4612, USA
* Corresponding author.
E-mail address: Anthony.Senagore@med.usc.edu

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and impaired wound healing. The need for nasogastric decompression and prolonged venous access inhibit ambulation, which may increase rates of pulmonary complications and thromboembolism. Prolonged fasting requires parenteral nutrition, which costs 3.5 times more per day than enteral nutrition,[8] without including the cost of central venous catheter placement or its complications. The morbidities of POI and the added care place a greater economic burden on the health care system.

The prolonged hospitalization associated with POI is an important issue at hospitals with a limited number of beds and high demand for inpatient services. Efforts to minimize POI and lengths of stay in hospital without increasing the risk of postdischarge readmission have the potential to improve the financial bottom line for hospitals, private and governmental health insurers, and society at large. Approximately 161,000 Medicare recipients undergo major intestinal or rectal resection each year, which consumes an estimated 1.8 million days in hospital at a cost of $1.75 billion.[9,10] Safely shortening this length of stay would produce significant dividends because the economic impact of POI is estimated at $750 million per year.[11–13]

Recurrence of primary (delayed) POI is the cessation of flatus or stool, with bloating and nausea or vomiting after a period of apparent resolution.[14] This process usually occurs 1 or more days after the patient has been discharged home, subsequently requiring readmission to the hospital. Readmission occurs in approximately 10% of patients who undergo major abdominal surgery, and about half of these patients are readmitted for gastrointestinal (GI) failure or some measure of recurrence of their POI.[15] This is a particularly important problem because the economic costs of readmission are exaggerated in part from tests such as computed tomography (CT) scans. The readmission cost of delayed POI is comparable to the readmission cost of more serious adverse surgical complications.[7] These formidable costs obligate the pathophysiologic understanding of this frequently preventable disease.

PATHOGENESIS OF POI

The multifactorial approach to prevention and treatment of POI is due in part to its complex pathogenesis. Studies have identified 3 major precipitating mechanisms for POI: neurogenic, inflammatory, and pharmacologic (**Fig. 1**). These mechanisms overlap to cause the disease, but the neuronal mechanism seems to disproportionately affect the early postoperative period.[14] Endogenous neuromuscular inhibitors of the bowel include norepinephrine, corticotropin-releasing hormone (CRH), nitric oxide, somatostatin, glucagon, gastric inhibitory peptide, and opioids.[16] Neurogenic bowel inhibition relates to pain-induced neural reflexes, which in turn result in sympathetic hyperactivity and inhibition of GI motility.[17,18] Another response to tissue trauma is the release of CRH from the central nervous system. CRH is recognized to contribute to the induction and duration of ileus.[19] Pain-induced reflexes also generate endogenous opioids that contribute to GI inhibition. Strategies to reduce postoperative pain and POI include the use of laparoscopic surgery and epidural local anesthetics.[17,20] POI may occur after minimally invasive surgical procedures, despite obvious reductions in surgical trauma and manipulation of the bowel, possibly because of the effects of opioid analgesics mediated by the stimulation of GI opioid receptors by exogenous opioids.[21,22]

Localized postoperative inflammation inhibits GI smooth muscle, and the duration of POI seems to correspond to the magnitude of intestinal inflammatory response.[22,23] Surgical manipulation of intestine activates quiescent macrophages and mast cells to induce several inflammatory cascades, primarily via the arachidonic acid pathway. As with pain response, these inflammatory mediators cause the release of endogenous