Small Bowel Obstruction—Who Needs an Operation?

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Occasionally, prophets do come from Mecca. Thus, this study by Zielinski and colleagues from the Mayo Clinic [1] deserves our attention and scrutiny.

The authors reviewed retrospectively 100 consecutive patients with small bowel obstruction (SBO). Not surprisingly, computed tomography (CT) images were available for all patients. (For the non-American readers who may be raising an eyebrow, we should clarify that it is rare in today's United States for a patient with any abdominal complaint to be admitted to the hospital without having passed at least once through the CT scanner.) The patients were divided into two groups based on whether they needed (group 1) or did not need (group 2) surgical treatment.

On univariate analysis multiple variables were significantly associated with the need for an operation: The two clinical variables were: "history of malignancy" (this is not surprising because malignant SBO, unlike the adhesive one, is unlikely to resolve spontaneously) and "vomiting." There were five CT-related predictive variables: free intraperitoneal fluid, mesenteric edema or/and vascular engorgement, small bowel thickening, and the absence of the "small bowel feces sign." However, that progressively obstructed small bowel becomes thick, that mesentery undergoing torsion becomes edematous and engorged, and that compromised bowel tends to "sweat"—producing free fluid—is intuitive and has been described before. (As to the "absence of the small bowel fecal sign," see below.)

Bowel ischemia developed in 11 patients and was associated with the following: clinical peritonitis; closed loop obstruction; increased serum lactate concentration; and radiographic signs that are well known to be associated with strangulating SBO (i.e., pneumatosis intestinalis and portal venous gas, free intraperitoneal fluid, and mesenteric edema, already mentioned above).

Multivariate analysis disclosed four independent predictors of the need for operative management: vomiting; CT evidence of free intraperitoneal fluid; mesenteric edema; and absence of the "small bowel fecal sign." All four features were present in 21 patients, with 19 (90%) eventually requiring surgery. The authors stressed the importance of such combination "because of these 21 patients 13 (62%) were treated initially nonoperatively...." Based on their statistical analysis and prevailing practice dogmas, the authors came up with an algorithm suggesting that its use would decrease mortality, morbidity, and hospital stay of SBO patients.

This elegant study nicely reiterates the truism that there is no *single* clinical, laboratory, or radiographic feature that, if present, can *alone* indicate the need for an operative intervention for SBO. Instead, the clinician has to use his or her wisdom to combine all the information available to reach a correct decision. This study, although none of its results (in isolation) is really novel, provides us with valuable insight.

Whether it is "good" or "bad," the fact of the matter is that in this country abdominal CT has almost replaced the use of plain abdominal radiography for diagnosis of the "acute abdomen." However, do all SBO patients need CT? Of course not! Surely patients with an obviously incarcerated/strangulated groin or abdominal wall hernia, with features of SBO on plain radiography, should be taken directly to the operating room. Also, patients with classic

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features of closed loop obstruction and with a suggestive plain radiographic appearance should go directly for an operation. These patients comprise a small minority, however—what about the rest? Experienced surgeons can be selective when ordering CT, reserving it for patients with an equivocal presentation or if a malignant, inflammatory (e.g., Crohn's), or infective (e.g., acute appendicitis can present like SBO) condition is suspected. Be that as it may, irrespective of whether CT is used, clinicians ought to consider multiple variables—none specific enough in isolation but useful if taken together—in their judgment. For example, signs of inflammation (tachycardia, temperature, leukocytosis) point to bowel ischemia or necrosis; the volume and character of nasogastric (NG) effluent (we have almost never seen a patient with feculent NG tube effluent who avoided an operation—this sign should be as “significant” as the “history of vomiting” stressed by the authors). We add to it the response to therapy: Pain and abdominal tenderness that continue after the abdomen has been decompressed are disquieting signs. Yes, we agree, the presence of classic CT features of intestinal compromise (e.g., pneumatosis) or “fixed” obstruction (e.g., intussusception, torsion of the mesentery) and/or the features described here by the authors make the decision to go to the operating room easier. Furthermore, the presumptive etiology of the SBO should be factored into the decision tree: If the abdomen is “virgin” or there is a history of malignancy, operative intervention is more likely; and preoperative CT could offer a road map or even let us choose to avoid the operation (e.g., carcinomatosis). Regarding “malignancy,” we should point out that in the operated group in the Mayo study there were significantly more patients with an etiology of cancer (35%) than in the nonoperated group (10%), where the main etiology was adhesions (63% vs. 27% in the operated group). Considering the fact that abdominal malignancy is a known cause of intraabdominal fluid formation, such a difference may have biased the results.

We know that the CT is not a panacea: Most patients do not have the combination of the four features described in this study; in fact, most do not have even three of them. The commonly used radiological term “high-grade SBO,” denoting a visible small bowel “transition” point, is not helpful because in at least half of such patients the SBO resolves spontaneously. Commonly, we encounter the patient a few hours after their CT scan, at which point things may have changed. Therefore, when the emergency room physician had ordered CT no free intraperitoneal fluid was visualized, and there was no mesenteric edema. But what about *now*? SBO can progress, right? Should we be repeating the CT to search for the development of the “predictive features,” or should we continue with conservative “drip and suck” treatment, provided of course that there is no clinical

deterioration? As serial rescanning of SBO patients is not in vogue, most surgeons would attempt conservative treatment—but for how long? Traditionally, surgeons tend to procrastinate. In the discussed article, the average time from admission to laparotomy when strangulation was suspected was 1.5 days (which seems reasonable considering the usual emergency room, CT, and the “chain of command” delays). On the other hand, the time to operation in the absence of strangulation was 5.3 days, obviously contributing to the long hospital stay. This period of waiting for the resolution of SBO, which never would materialize, seems to us excessive and is typical of the current state of art. We believe that the way to shorten the triage to surgery for those who need it, while sparing those who do not need it, is the routine use of oral, water-soluble contrast material.

In their discussion, Zielinski et al. mention the diagnostic and therapeutic use of oral water-soluble contrast material only to dismiss its value based on selective and biased review of the literature and in the absence of personal experience. We have to differ: Based on our understanding of the literature (we lack space to review it here) and our own experience, this is what we do when we encounter a patient with SBO: We use the “Gastrografin challenge.” After the initial gastric decompression we instill 100 ml Gastrografin via the NG tube, which is then clamped. After 4 to 6 hours, a simple plain abdominal radiograph is obtained. The presence of contrast in the large bowel proves that the obstruction is not complete. In most instances, the hyperosmolar Gastrografin is soon rushing into the rectum as well: the patient smiles and his room stinks. On the other hand, failure of Gastrografin to reach the colon within 4 to 6 hours indicates to us a complete obstruction (some would wait longer, up to 24 hours; but in our experience, at that late stage the Gastrografin has been diluted or absorbed). The probability of spontaneous resolution after a failed Gastrografin challenge is low—if the Gastrografin cannot pass through the obstruction, nothing can pass. Thus, these patients require surgery anyway, so why not operate on them now? Another sign of a failed Gastrografin challenge is the failure of Gastrografin to leave the stomach and enter the small bowel. This signifies significant back-pressure in the obstructed bowel and the need for an urgent operation. Naturally, the Gastrografin challenge can be combined with CT. So if we are the ones ordering the CT we would combine it with oral Gastrografin and follow the CT with plain abdominal radiography a few hours later to see whether the contrast has reached the colon. (You may need to convince your radiologist that nondiluted oral contrast is appropriate.) If Gastrografin has been omitted during the CT we would order the “challenge” the next day.

Surprisingly, only one-third of Zielinski et al.'s patients received oral contrast during their CT as if their

radiologists had no interest in seeing the fate of the contrast in the obstructed bowel. Finally, one aspect in their article mystified us—the “small bowel feces sign.” We assume that this is what our radiologists call pseudofeces, fecalization, or colonization of the small bowel (the small bowel looks like the colon). We cannot understand, however, why the “absence” of this sign should be one of the CT signs predicting an operation because to us feculent vomiting or/and accumulation of debris in the small bowel indicates advanced, persistent obstruction. However, as some would say: “If you torture the statistical data long enough, you can make it say anything.”

In conclusion, specific CT features of SBO, as emphasized by the Mayo group, should be incorporated within the whole clinical picture. However, the use of water-soluble

contrast is a necessity. The old famous adage “never let the sun rise and set on a complete small bowel obstruction” is of course passé. The motto we suggest instead is, “never let a patient with a complete intestinal obstruction (i.e., no Gastrografin in his colon) avoid the knife for longer than is necessary.”

Reference

1. Zielinski MD, Eiken PW, Bannon MP et al (2010) Multivariate model to predict need for exploration and presence of ischemia in small bowel obstruction. *World J Surg*. doi:[10.1007/s00268-010-0479-3](https://doi.org/10.1007/s00268-010-0479-3)