

Case Report – Lessons in Clinical Reasoning

Colby Weil-Lonigan, Celestine He, Robin Levenson and Andrew Sanchez*

Small bowel obstruction from signet-ring cell appendiceal carcinoma

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Abstract

Objectives: To illustrate the diagnostic challenge that arises in patients presenting with symptoms due to small bowel obstruction (SBO) whose initial cross-sectional imaging does not reveal the site or etiology of obstruction, and to review the pathophysiologic mechanisms by which SBO can evade radiographic detection.

Case presentation: A man in his 80s with a remote history of intraabdominal surgeries presented with abdominal pain, bloating, and emesis. Non-contrast enhanced computed tomography (CT) imaging of the abdomen and pelvis showed distended small bowel loops with air-fluid levels, but no transition point or distal decompression. He was initially treated with isotonic fluids and a nasogastric tube to suction with resolution of symptoms, and he was discharged with a diagnosis of ileus of unclear etiology. However, approximately one month later, his symptoms recurred, and repeat CT imaging showed potential SBO at the terminal ileum. Exploratory laparotomy was performed for failure of conservative management, which ultimately revealed appendiceal mucinous adenocarcinoma with peritoneal carcinomatosis as the cause of the patient's initial presentation.

Conclusions: CT findings of obstruction without an identifiable transition point may not reliably distinguish SBO from ileus, and symptom resolution with conservative management may falsely lead to an ileus diagnosis and drive premature closure. Because ileus requires an explanatory trigger, its diagnosis in the absence of one should prompt

consideration of low-grade partial SBO as well as intermittent and functional pathophysiology.

Keywords: small bowel obstruction; malignant small bowel obstruction; appendiceal adenocarcinoma; signet-ring cell carcinoma; peritoneal carcinomatosis

Case presentation and discussion

***Aliquot 1:** A man in his 80s with a history of advanced chronic kidney disease (CKD), laparoscopic radical nephrectomy of the right kidney for renal cell carcinoma (10 years prior to presentation), laparoscopic retroperitoneal lymph node dissection and hepatic wedge resections for local recurrence (eight years prior to presentation), and no subsequent imaging evidence of recurrence presented with acute-onset abdominal pain awakening him from sleep. The pain was followed by severe bloating, recurrent non-bilious emesis preventing oral intake, and watery diarrhea, with three days of progressive worsening prompting presentation to the emergency department.*

The initial work-up for acute abdominal pain prioritizes a search for diagnoses that are life-threatening if left untreated. Laboratory studies may include the complete blood count with differential, complete metabolic panel, serum lactate, and lipase. While certain laboratory derangements may suggest the final diagnosis (e.g. severe lipase elevation → acute pancreatitis), many life-threatening causes of acute abdominal pain do not leave a laboratory fingerprint beyond abnormalities that indicate illness severity (e.g. lactic acidosis and elevated anion gap of organ ischemia). Similarly, symptoms and physical examination findings accompanying acute abdominal pain can vary widely. In light of these limitations, abdominal imaging can be more high yield and more useful for diagnosis.

In the assessment of acute abdominal pain that is diffuse rather than localized, computed tomography (CT) of the abdomen and pelvis enhanced with intravenous (IV) contrast (if no clinical contraindication) is the recommended first-line imaging modality, as CT is often readily available at tertiary care centers and can narrow a large differential

*Corresponding author: Andrew Sanchez, Department of Medicine, Beth Israel Deaconess Medical Center, Harvard Medical School, 330 Brookline Ave, West Span 201, Boston, MA, 02215, USA, E-mail: Asanch21@bidmc.harvard.edu. <https://orcid.org/0000-0001-6910-5554>

Colby Weil-Lonigan and Celestine He, Department of Medicine, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, USA
Robin Levenson, Department of Radiology, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, USA

diagnosis [1]. CT carries a high sensitivity for many intra-abdominal emergencies, including vascular emergencies (i.e. aortic aneurysm, aortic dissection), inflammatory organ pathologies (e.g. appendicitis), bowel obstruction, and viscus perforation, and CT has been described to change clinicians' leading diagnosis in up to half of cases of patients presenting to the emergency department for acute abdominal pain [2, 3]. Importantly, the administration of IV contrast improves diagnostic accuracy, as non-enhanced CT is approximately 30 % less accurate in the identification of the principal cause of abdominal pain [3].

Two notable intraabdominal emergencies require particular attention when the contrast-enhanced CT fails to identify the diagnosis yet clinical concern remains. First, acute cholecystitis is not infrequently missed early in its disease course, as the sensitivity of CT has been shown to be approximately 85 % [4]. When CT is non-diagnostic for patients who have abdominal pain accompanied by otherwise unexplained fever or neutrophilic leukocytosis, ultrasonography (US) of the right upper quadrant should be considered, particularly if there are right sided abdominal symptoms or acute cholecystitis is in the differential diagnosis. US is more sensitive than CT in detecting gallstones; CT can fail to detect approximately 20 % of gallstones [4]. US also allows for further assessment of the gallbladder wall as well as for sonographic Murphy sign. Thus, US and CT can be complementary examinations in the evaluation of cases of equivocal acute cholecystitis. However, if the results of US and CT remain equivocal or negative and the pre-test probability for acute cholecystitis remains high, cholescintigraphy should be considered due to its superior sensitivity of approximately 96 % [5].

Second, acute mesenteric ischemia (AMI) should be considered in patients with cardiovascular risk factors who present with pain out-of-proportion to examination, as mortality is as high as 80–100 % when management is delayed beyond 24 h [6]. Suspicion for AMI should be included in the indication for CT because the absence of a stated clinical concern for AMI is associated with a suboptimal imaging protocol (i.e. single-phase rather than triple-phase protocol required for optimal evaluation of mesenteric vasculature) as well as a lower yield of radiology reports documenting the critical findings of AMI, likely reflecting decreased radiologist vigilance for subtle AMI findings [7].

Aliquot 2: *On presentation, the patient was afebrile with a heart rate of 102 beats per minute, blood pressure of 132/78 mm Hg, and peripheral oxygen saturation of 98 % on room air. Abdominal exam was notable for a markedly distended abdomen that was diffusely tender to palpation but without guarding. Laboratory studies were notable for a creatinine of*

4.70 mg/dL (0.40–1.30 mg/dL) from a baseline of 3.0 mg/dL, BUN of 72 mg/dL (6–20 mg/dL), lactic acid of 1.2 mmol/L (0.5–2.0 mmol/L), white blood cell count of 7,840 cells/ μ L (4,000–10,000 cells/ μ L), hemoglobin of 10.6 g/dL (13.7–17.5 g/dL), platelet count of 243,000 cells/ μ L (150,000–400,000 cells/ μ L), and a normal liver function panel. Testing for clostridium difficile was negative, as well as PCR testing for norovirus 1/2 and antigen testing for rotavirus. Stool culture did not identify salmonella, shigella, or campylobacter. A CT scan of the abdomen and pelvis without contrast showed moderately distended small bowel loops with air and fluid levels, no transition point or distal decompression, normal caliber terminal ileum, an unremarkable peritoneal space, and no lymphadenopathy: the radiology team's impression raised the possibility of ileus (Figure 1). A nasogastric tube was placed to suction, and isotonic fluids and anti-emetics were administered with subsequent improvement in the patient's nausea, abdominal pain, and acute kidney injury (AKI).

This presentation of abdominal pain, bloating, and recurrent emesis accompanied by pre-renal AKI and CT demonstrating dilated small bowel loops with air-fluid levels is highly suggestive of small bowel obstruction (SBO). Given the patient's remote history of intraabdominal surgery, adhesive disease was the most likely diagnosis. SBO is a life-threatening surgical emergency that presents with symptoms related to impaired flow of intraluminal intestinal contents: nausea and vomiting due to accumulation of intraluminal contents beyond gastrointestinal (GI) capacitance; paroxysmal abdominal cramping and pain due to increased peristalsis; abdominal distension due to compensatory bowel dilatation; and potentially obstipation in the presence of a complete obstruction [8]. The frequency and severity of SBO symptoms is highly variable and dependent on both the location (i.e. proximal vs. distal) and the degree (i.e. partial vs. complete) of obstruction, with more proximal, complete obstructions generally resulting in a higher acuity and severity of symptoms [8, 9].

The patient's initial CT report confronts the team with a clinical and radiographic discrepancy. Namely, one of the hallmark CT findings in SBO is a transition point, which describes a discrete caliber change from dilated to decompressed bowel at the site of obstruction. Because a transition point was not identified, the possibility of ileus was raised. The fundamental distinction between SBO and ileus rests in the underlying pathophysiology: SBO involves a physical barrier impeding intestinal flow, whereas ileus reflects impaired or absent peristalsis resulting in functional obstruction.

This distinction drives important differences in management. SBO may require an operative intervention to reduce the obstruction, whereas ileus is managed by

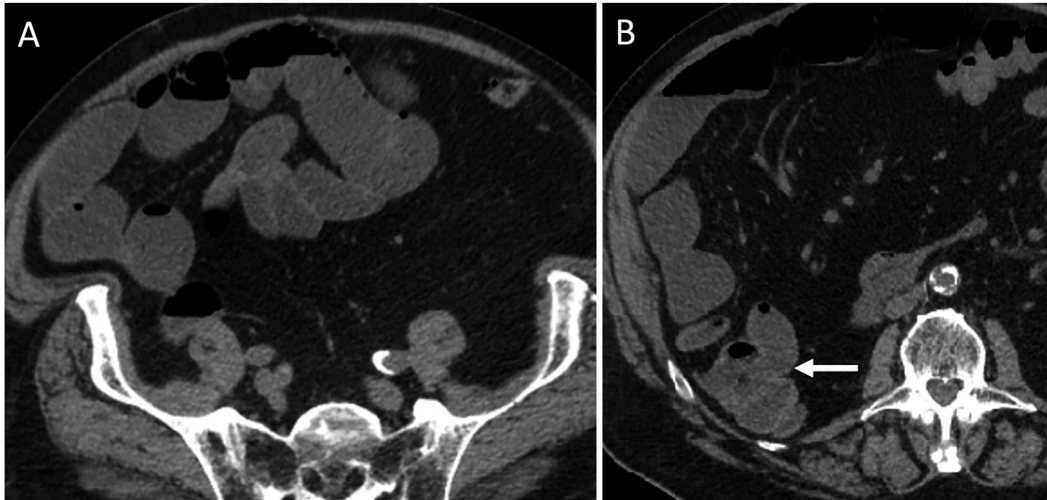


Figure 1: Axial non-contrast enhanced CT images through the abdomen. (A, B) show distended loops of small bowel containing fluid and air-fluid levels. The terminal ileum appears normal in caliber as it enters the cecum (B, white arrow). No transition point was seen.

addressing the underlying cause of paralyzed bowel. Ileus in itself is not an endpoint diagnosis and thus its discovery mandates a search for an explanatory trigger. What is puzzling in this patient whose CT was read as being consistent with ileus is that there is no such accompanying trigger, including recent operation, opioid use, electrolyte disturbance, or systemic illness.

Aliquot 3: On day three of admission, there was new leukocytosis with a white blood cell count of 10,830 cells/ μ L as well as new lactic acidosis measured at 2.7 mmol/L. Due to concern for bowel ischemia, the primary team discussed the need for repeat abdominal imaging. While the primary team preferred repeat CT with the addition of IV contrast, other stakeholders in the patient's care including radiology, nephrology, and the patient and patient's wife preferred to defer IV contrast owing to the risk of contrast nephropathy in this patient with a low GFR (12 mL/min/BSA). Ultimately, a repeat unenhanced CT scan was obtained, which showed similar distension of the small bowel and no evidence of bowel ischemia, as well as collapse of the terminal ileum limiting assessment for a transition point and thus precluding the radiology team's ability to rule-out a partial SBO at the level of the terminal ileum (Figure 2). The leukocytosis and lactic acidosis resolved 48 h later without an identified source beyond severe bowel dilatation. The nasogastric tube was removed, the patient's diet was slowly advanced, and the patient's presenting symptoms resolved on day six of admission. He was discharged home with a presumed diagnosis of ileus of unclear etiology.

The repeat CT on hospital day three adds new information but does not resolve the original discrepancy between the clinical picture and CT results. The collapse of the

terminal ileum now limits the radiologist's ability to exclude a partial SBO at that level, making the study indeterminate rather than negative for SBO: this is an important distinction. An indeterminate imaging result in a patient with no clear trigger for ileus should heighten diagnostic concern rather than resolve it.

Forgoing IV contrast was clinically reasonable given the patient's advanced CKD and recent AKI, and it reflects a tradeoff reached through multidisciplinary discussion. It spared the patient the risk of contrast nephropathy, but it also reduced the scan's sensitivity for contrast-enhancing pathologies.

While the discharge diagnosis of ileus is suspect due to the lack of an accompanying trigger, it should be noted the patient's presenting symptoms and laboratory derangements completely resolved with non-operative management. Indications for operative management of SBO include signs of bowel compromise (e.g. peritonitis, evidence of bowel ischemia) as well as specific SBO subtypes carrying a high risk for strangulation (e.g. closed-loop obstruction, internal hernia) or failure of non-operative management (e.g. primary bowel malignancy) [10]. In patients presenting without these features, a trial of non-operative therapies defined by nasogastric decompression, bowel rest, and IV fluid resuscitation can be safely initiated, and serial evaluation of the patients' clinical evolution guides remaining management [10].

Management of ileus is essentially identical but with added emphasis on identifying and treating underlying triggers. In both conditions, the resolution of obstructive symptoms with conservative management often signals that no further diagnostic work-up is needed [10]. Adhesive

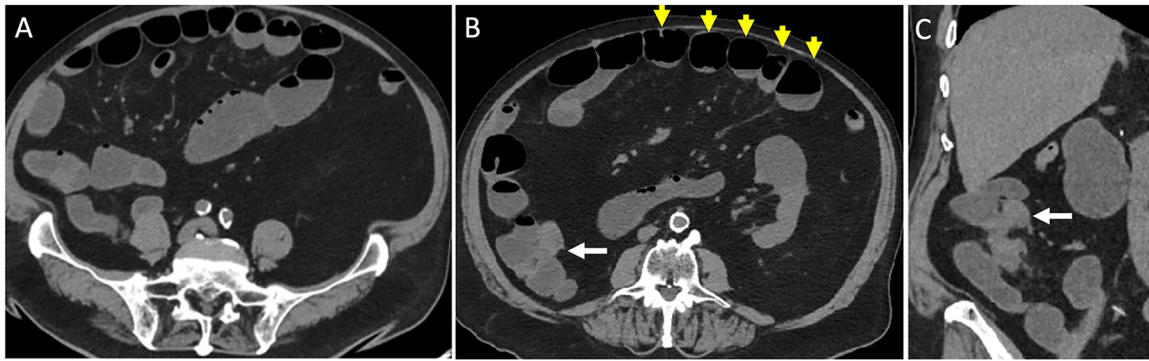


Figure 2: Axial non-contrast enhanced CT images of the mid abdomen. (A, B) show distended loops of small bowel with air-fluid levels (yellow arrows in B). Axial and coronal non-contrast enhanced CT images and (B, C) demonstrate the terminal ileum is collapsed (white arrow).

disease illustrates this well, as it is the most common cause of SBO in both surgical and non-surgical patients, and non-operative management is successful in up to 85 % of patients [10].

In patients with SBO undergoing non-operative management, the gastrografin challenge can also be used to study whether non-operative management is likely to succeed. This procedure involves the administration of a water-soluble, radiopaque solution by mouth or nasogastric tube followed by the acquisition of serial radiographs over 24 h, thus allowing the assessment of contrast passage over time. The presence of contrast within the colon by 24 h predicts non-operative success with a sensitivity of 92 %, and the absence of contrast within the colon by 24 h indicates non-operative failure [10].

Because this patient's symptoms resolved with conservative management alone, the primary team's decision to discharge with a documented diagnosis of ileus was technically consistent with surgical management algorithms for both SBO and ileus. While the possibility of SBO with a falsely negative CT was never raised, even if SBO was more explicitly considered in the patient's discharge documentation, adhesive disease would have remained the most likely explanation [11].

Aliquot 4: Twenty-six days after discharge, the patient re-presented to the emergency department after experiencing acute-onset recurrence of his prior symptoms. A CT scan without contrast showed increased dilation of the small bowel compared to the initial CT scans, a gradual taper of bowel with collapse of the distal terminal ileum, and trace ascites with an otherwise normal-appearing peritoneal space (Figure 3). General surgery was consulted, and operative management for SBO was pursued given failure of recent conservative management, with highest concern for culprit adhesions. However, exploratory laparotomy instead revealed a rubbery mass at the ileocecal valve as well as extensive serosal and mesenteric tumor deposits. Pathology

identified the neoplasm as high-grade appendiceal mucinous adenocarcinoma with signet-ring cell features. The patient passed away one month later due to an intracranial hemorrhage from a traumatic fall. Figure 4 provides a summary of aspects of the case that contributed to the diagnostic delay.

Clinical teaching points

The role of CT in the diagnosis of SBO

In any patient presenting with symptoms concerning for SBO, imaging is an essential component of the diagnostic work-up, potentially leading to identification and further characterization of the underlying pathology. While abdominal X-ray can be both a more rapid and less expensive option, CT enhanced with IV contrast remains the gold standard, which carries a sensitivity of up to 91 % and specificity up to 89 % for SBO of all types [12, 13]. The increased sensitivity and specificity of CT over radiography is predominantly garnered from its ability to produce cross-sectional images that allows for study of the bowel wall and intraabdominal space in much greater detail. This can not only reveal the secondary imaging findings of bowel obstruction (e.g. proximal dilated bowel, air-fluid levels, fecalized small bowel), but often the site of obstruction (e.g. transition point with distal decompression) and cause (e.g. incarcerated hernia) [12, 13]. IV contrast administration is generally recommended during the initial CT assessment of SBO as it both increases the sensitivity for SBO-related complications (e.g. bowel ischemia) and sensitivity for the detection of the underlying etiology (e.g. enhancing tumor) [13]. While the sensitivity of CT for SBO is quite high for patients with symptoms of SBO of all types, this case of a primary bowel neoplasm presenting with recurrent SBO symptoms and rather unremarkable CT results exemplifies

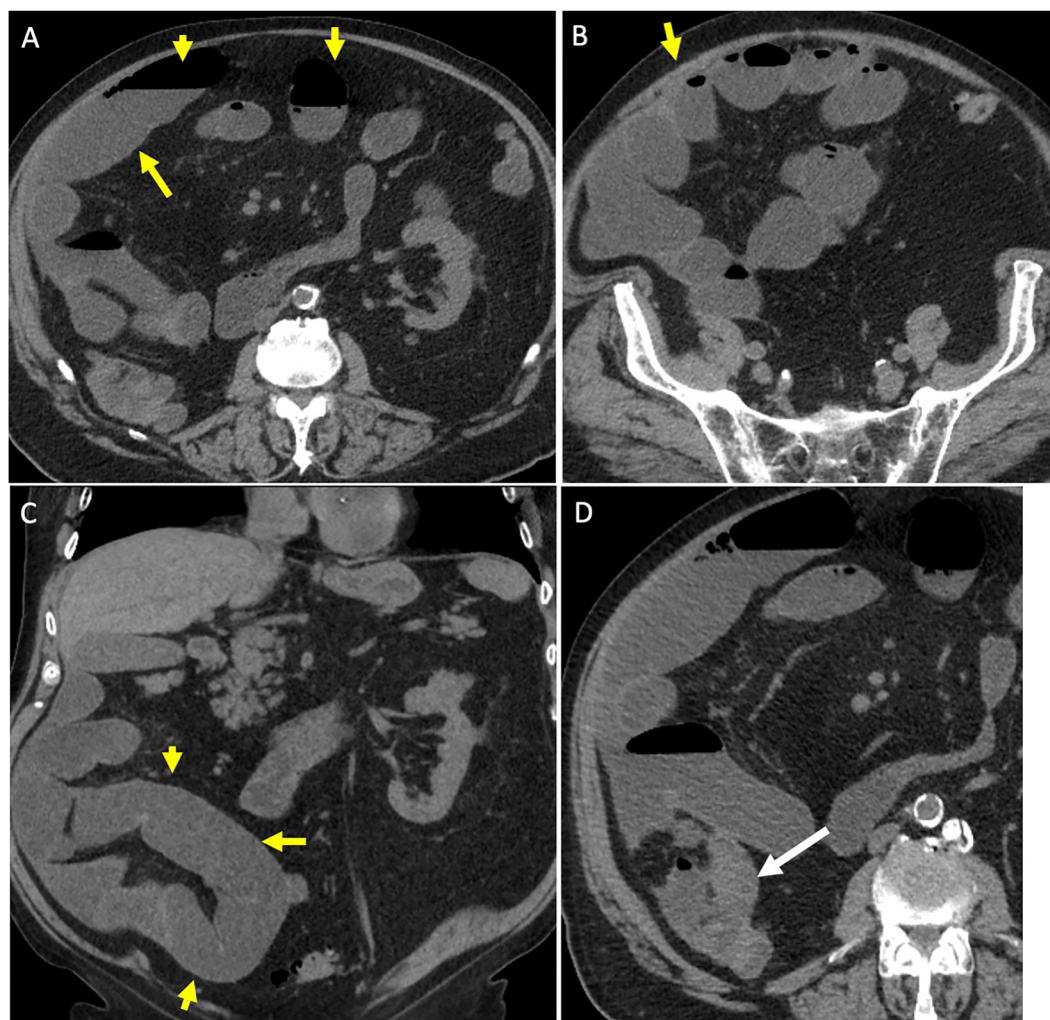


Figure 3: Axial (A, B) and coronal (C) non-contrast enhanced CT images of the mid abdomen show increased dilation of loops of small bowel which contain fluid and air-fluid levels (yellow arrows). Axial non-contrast enhanced CT image (D) shows the collapsed terminal ileum. There is a gradual decrease in caliber of the ileum, with the terminal ileum (white arrow) collapsed. No discrete mass was identified.

the diagnostic crux that emerges when CT results have identified imaging findings of SBO without identification of the precise site and/or etiology of the obstruction.

Malignant SBO: diagnostic challenges related to divergent mechanisms

While epidemiologic studies have elucidated that adhesive disease is the culprit of SBO in approximately two-thirds of cases, they do not stratify prevalence by clinical presentation [9, 11]. Failure to account for the precise mechanism of SBO can lead to diagnostic error, as the etiologies behind chronic partial as well as recurrent intermittent SBO are more elusive to identify [9]. This diagnostic challenge is perhaps best demonstrated by the difficulty of the diagnosis of SBO related

to occult malignancy. Despite widespread awareness that SBO related to malignancy constitutes up to 20 % of cases of SBO, the mechanisms of SBO related to malignancy are divergent and include functional obstruction (e.g. peritoneal carcinomatosis resulting in decreased bowel compliance, intramural intestinal metastasis resulting in decreased peristalsis) and intussusception related to primary bowel neoplasms [14].

SBO pathophysiology: ‘constitutive’ obstruction

There are multiple pathophysiologic mechanisms by which SBO can evade detection by CT and result in misclassification as ileus. First, one must consider if the underlying pathophysiology of SBO is constitutive (i.e. continuous) or

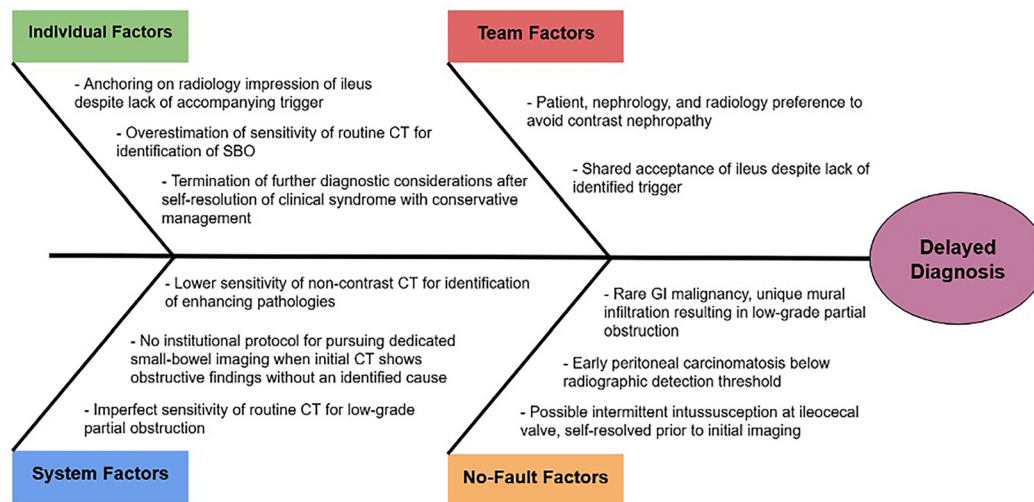


Figure 4: A fishbone diagram demonstrating several factors that contributed to the diagnostic delay.

intermittent. While constitutive pathophysiology, such as one due to a fixed luminal narrowing (e.g. stricture), implies that the pathology is always visualizable during a radiographic study, consideration of the severity of the underlying obstruction is essential. For example, the sensitivity of CT for low-grade partial SBOs is substantially lower than for high-grade partial SBOs (63 % vs. greater than 95 %) [15, 16]. This is particularly important to consider in cases of malignancy, which may only result in partial obstruction. For this reason, patients presenting with subacute to chronic SBO symptoms without an identifiable site of obstruction on initial CT should receive further work-up. While a procedure known as enteroclysis carries a sensitivity for low-grade, partial SBO of 100 %, enteroclysis is intensive and requires intubation of the duodenum using a nasoenteric tube and installation of a large volume of air and contrast into the small bowel [16]. Alternative studies that do not require nasoenteric tube placement but still require ingestion of large volumes of contrast include CT and MRI enterography. As acutely ill patients with SBO generally cannot tolerate the oral contrast volume required for these studies, they are generally reserved for the outpatient setting in patients with recurrent, low-grade presentations (e.g. suspected small bowel stricture).

SBO pathophysiology: ‘intermittent’ obstruction

‘Intermittent’ obstruction implies a dynamic process and that the obstruction may not be captured if the obstructive episode has spontaneously resolved prior to image acquisition. Hernias are a common example of this phenomenon, as

abdominal wall and groin hernias can present with intermittent SBO symptoms should the hernia remain reducible. In the case of intermittent SBO due to hernias, however, the culprit is still generally identifiable by CT. More diagnostically challenging is the phenomenon of intussusception, which involves intermittent telescoping of a proximal bowel segment into a distal loop [17]. Symptomatic intussusception in the adult is rare, with an incidence of less than 5 % of all intussusception cases, but its diagnosis is essential given its strong association with underlying malignancy [17]. Intussusception confined to the colon is the most common subtype of symptomatic intussusception in the adult, with enteric intussusception accounting for approximately 13.3 % of cases and ileocecal and ileocolic intussusception accounting for approximately 5.7 % of cases [17]. At any site, the most frequent malignant lesions associated with intussusception are adenocarcinoma, GI stromal tumor, and lymphoma, while lipomas and polyps are the most common benign lesions [17]. Identifying cases of intermittent SBO requires a high index of suspicion derived from recurrent obstructive symptoms, patient demographics, and review of the past medical history.

SBO pathophysiology: ‘functional’ obstruction

‘Functional’ obstruction implies a pathophysiology that ultimately results in impaired GI motility, such as neuromuscular diseases resulting in myenteric plexus dysfunction or infiltrative diseases resulting in decreased bowel compliance. Malignancy is an important consideration, as dissemination of tumor deposits throughout the peritoneal cavity

(i.e. peritoneal carcinomatosis) can result in diffusely decreased bowel compliance, particularly in cases of metastatic GI and ovarian malignancies [14, 18]. The sensitivity of CT for peritoneal carcinomatosis is variable and dependent on the size and distribution of tumor deposits, and the presence of ascites may increase the detection rate of parietal implantation [18]. While MRI and PET/CT carry higher sensitivity for peritoneal carcinomatosis, even PET/CT can miss micronodular disease and malignancies with inherently low ^{18}F -FDG uptake (e.g. mucinous neoplasm) [18, 19].

Signet-ring cell carcinoma

This case of appendiceal mucinous adenocarcinoma with signet-ring cell features presenting with SBO despite unrevealing initial CT imaging highlights signet-ring cell carcinoma as a prototypical example of how primary GI neoplasms can evade radiographic detection. Signet-ring cell carcinoma, a rare subtype of adenocarcinoma, is characterized by poor neoplastic cell cohesion that results in a propensity to diffusely infiltrate the bowel wall and preclude formation of a visible mass [20]. This histopathology suggests that the initial CT imaging in this case may have been negative because circumferential mural infiltration produced only a low-grade, partial obstruction, a setting in which CT sensitivity is diminished [15, 16]. Furthermore, the lack of IV contrast may have further limited abnormal soft tissue detection. It is also possible that intussusception at the ileocecal valve may have resulted in intermittent obstruction not captured at the time of image acquisition. While this patient likely did not have a sufficient burden of peritoneal carcinomatosis to cause overt functional obstruction, the unexpected intraoperative finding of carcinomatosis further underscores the imperfect sensitivity of CT for carcinoma with small nodule size. This case highlights the need for increased awareness of the variable clinical presentations of SBO and how an understanding of the divergent mechanisms of obstruction is crucial for accurate interpretation of intraabdominal imaging.

Pitfalls – myths – pearls

Pitfalls

“An obstructive syndrome that resolves with bowel rest and carries no transition point on CT is an ileus.” SBO can be misdiagnosed as ileus when CT fails to identify the site and cause of obstruction. Because conservative, non-operative therapies define the cornerstone of management for both

conditions when neither are complicated, self-resolution of the obstructive syndrome can inappropriately confirm the diagnosis of ileus and lead to premature closure. Ileus, however, describes a state of paralyzed bowel and requires an endpoint diagnosis.

Myths

“A CT of the abdomen and pelvis showing no transition point, discrete mass, or peritoneal carcinomatosis effectively rules out malignant SBO.” The sensitivity of CT for low-grade partial SBO is substantially lower than for high-grade partial SBO (63 % vs. >95 %), and primary GI malignancies are well described to cause low-grade partial SBO. Therefore, the absence of a visible mass or transition point on CT does not exclude primary GI malignancy in patients presenting with recurrent obstructive symptoms. Additionally, CT sensitivity for peritoneal carcinomatosis is variable and dependent on nodule size and distribution, meaning even extensive peritoneal deposits can be missed if nodules are small.

Pearls

When ‘ileus’ is diagnosed in a patient with secondary CT findings of obstruction (e.g. air-fluid levels) but no trigger is identified, the unrevealing CT should prompt consideration of three mechanisms by which SBO can evade radiographic detection. First, some obstructions are constitutively present but result in only *low-grade partial* obstruction, which are often missed on routine CT and require more sensitive small-bowel imaging (e.g. CT vs. MR enterography). Second, obstructions can be *intermittent* and spontaneously resolve before image acquisition (e.g. intussusception). Finally, *functional* obstruction can result from decreased bowel compliance without a fixed mechanical lesion (e.g., peritoneal carcinomatosis). Rapid recurrence of an obstructive GI syndrome after conservative management necessitates surgical consultation for consideration of diagnostic laparoscopy or exploratory laparotomy, especially in patients without a surgical history and risk of adhesive disease.

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