EARLY POSTOPERATIVE SMALL BOWEL OBSTRUCTION: A LITERATURE REVIEW

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Abstract: Early postoperative intestinal blockage is a common and dangerous complication that may slow the recovery of bowel function and necessitate a longer hospital stay, both of which would increase medical costs. The most frequent reasons include adhesions, internal herniation, and inflammation. The trio of colicky stomach pain, vomiting, and complete constipation, together with abdominal distension, are typical symptoms of mechanical SBO. The diagnosis of post-operative intestinal blockage is confirmed by an abdominal x-ray, and a CT scan will show the degree and location of obstruction. The majority of ESBO patients will recover with conservative treatments, however a sizeable percentage of patients will need additional surgery. After taking into account all clinical and radiographic indicators of impending strangling or closed loop obstruction, the amount of time since the last operation, and the specifics of the index operation, the choice to do the procedure again should be decided.

Index Terms—Adhesions , Bowel obstruction, Abdominal pain

INTRODUCTION

Postoperative bowel obstruction could be either mechanical or functional. It was a typical and serious complication that could influence the recuperation of bowel function, thereby leading to prolonged hospitalization which would indeed lead to rising clinical expenses. Literature has mentioned about the increased rate of mortality and morbidity in patients following postoperative abdominal surgery[1,1,2]. Postoperative bowel obstruction is classified for simplification on the basis of the time duration from the initial surgery. Expressly, Early postoperative bowel obstruction was defined as bowel obstruction occurring within the first 30 days post surgery[3] .The incidence of the same was reported by Edna et.al as to be 9 %.[4]

The causative factor causing postoperative bowel obstruction was most importantly either paralytic ileus in abdominal surgery or mechanical obstruction from adhesions. Early postoperative small bowel obstruction (EPSBO), is ordinarily defined as obstruction occurring within the first 30 days after surgery. It is a distinct clinical entity which is often difficult to differentiate from ileus. Conventional surgical teaching states that early postoperative small-bowel obstruction (EPSBO) confronts the clinician with significant diagnostic and therapeutic challenges. Signs and symptoms of small bowel obstruction abdominal distention, pain, nausea, and constipation are the common signs and symptoms observed during the immediate post op period. It becomes difficult differentiating between EPSBO and ileus, and with mortality rates of up to 21 percent; as per the past reports and records, EPSBO can be claimed to be a devastating complication.

The pathophysiology, diagnosis and management of EPSBO differ in individual aspects from those of small bowel obstruction (SBO) in patients who have not undergone intraperitoneal surgery and that of adhesive SBO which develops at a time remote from the operation. Although adhesions account for the majority of SBOs in the early postoperative period, several other aetiologies must be considered.

PATHOPHYSIOLOGY AND CAUSES OF EARLY POST OPERATIVE SMALL BOWEL OBSTRUCTION

Adhesions

Greater number of episodes of EPSBO resolve spontaneously. They are most commonly caused by adhesions. Two case reports stated the cause of such obstruction to be adhesions in 91 per cent [5]and 92 per cent [6]of patients who required surgery. Any operative peritoneal injury or damage caused by peritoneal contamination and infection leads to formation of adhesions. This results in inflammatory response, with in turn activates the complement and coagulation cascades, along with exudation of fibrinogen-rich fluid. Conversion of fibrinogen to fibrin takes place, which thereby attaches to the adjoining damaged surfaces. At this stage, if fibrin is not degraded, fibroblasts cultivate into this matrix and collagen deposition occurs. to the conversion of *fibrinous* adhesions to *fibrous* adhesions. Sometimes, if fibrin degradation is complete, fibrinous adhesions resolve and mesothelial regeneration becomes complete. However, fibrinolytic activity is depressed due to peritoneal injury, caused by surgery or peritonitis. The major determinant of postoperative adhesion formation [7,8,9]is this early balance between fibrin formation and its degradation in the peritoneal cavity.

Internal herniation

EPSBO can be caused by internal herniation accounting to any defect within the peritoneal cavity. A small bowel may herniate through, when a mesenteric or omental defect is created, or an opening is left in the 'gutters' behind a colostomy or enterostomy, thus; leading to EPSBO. In one of the studies, herniation through a pelvic floor defect following abdominoperineal resection was reported to be responsible for five of 13 cases of EPSBO[10]. However, procedures such as rectal resection, which commonly produce potential spaces and peritoneal 'windows', rarely give rise to internal herniation. In a case series of 1061 patients undergoing rectal resection for cancer, only 18 patients reported to develop SBO within the first 6 weeks of resection. Each obstruction was most commonly related to postoperative complications, majorly intra-abdominal sepsis. In a study, the paracolostomy lateral space was

not closed in patients treated by abdominoperineal excision of the rectum, nevertheless there was no paracolostomy obstruction[11]. A case report by Fasth *et al.* stated that they were unable to close the pelvic peritoneal defect after abdominoperineal rectal excision combined with extensive lymph node clearance in 31 patients. Only one patient of all developed EPSBO. Franchi *et al.*[12] randomized patients undergoing radical abdominal hysterectomy and pelvic node dissection to closure and non-closure of the pelvic peritoneum and concluded that non-closure was not associated with an increased risk of adhesion-related complications.

It appears, therefore, that large peritoneal defects and spaces are less likely to cause EPSBO. On the contrary, narrow peritoneal defects have proven to be of a greater risk.

Inflammation

A local inflammatory lesion can also be a contributing factor towards EPSBO. A localized inflammatory lesion in the form of intra-abdominal abscess or a phlegmon, may adhere to the small bowel leading to EPSBO. Such 'septic' or 'inflammatory' adhesions were responsible for EPSBO in three of 41 [13]and 4 per cent[14] of patients. In a study reported, intraperitoneal abscess was present in 12 of 26 patients with EPSBO[15]. In such cases, it is impossible to judge retrospectively whether the obstructing process was adhesive or paralytic (ileus).

Other causes

Apart from the usual causes, there are some uncommon factors that also need some focus of light. Some of these include intussusception and intramural intestinal haematoma[1314]Postoperative intestinal intussusception develops in the absence of an identifiable 'leading point', and may be associated with prolonged operation and postoperative ileus[16]. It appears to be more usual after operations in paediatric patients, with or without a leading point; the latter may even be represented by an inverted appendicular stump after appendicectomy[17].EPSBO caused by intussusception is also one of the familiar complications of retro colic gastrojejunostomy. This occurs usually without gastric resection, when the efferent jejunal limb intussuscepts into the stomach[18]. Postoperative haematoma formation at the bowel wall or mesentery may present as EPSBO in some of the cases. [13].

Recurrent or persistent postoperative SBO may result due to failure to resolve the primary cause of obstruction at the first operation. Estimate the frequency of adhesive EPSBO following operations for adhesive SBO cannot be estimated. Reobstructions are caused by adhesions that were missed or left undivided. Intestinal phytobezoars causing SBO are commonly multiple; missed bezoars may therefore cause EPSBO that requires reoperation. This was reported in six of 77 patients treated for intestinal obstruction caused by persimmon bezoars[1923]. Similar situation may arise following surgery for gallstone ileus when an overlooked intraenteric stone or subsequent passage of another gallstone via the cholecystoenteric fistula may lead to EPSBO[20].

The diffusely inflamed and friable bowel in patients undergoing surgery for complications of radiation enteritis is prone to early postoperative complications, including obstruction[25]. Likewise, patients operated for peritoneal carcinomatosis are at high risk of persistent and recurrent EPSBO. This is majorly observed when multiple obstructive sites are identified at the index operation[21].

Eventually, a whole array of intestinal anastomotic complications, including anastomotic narrowing due to oedema or leakage, may present as EPSBO. Also, additionally, a so called self-limiting 'mini' anastomotic leak, associated with local phlegmon, is often responsible but moreover always underdiagnosed.

RISK OF EARLY POSTOPERATIVE SMALL BOWEL OBSTRUCTION TO THE INDEX PROCEDURE

Stewart *et al.* conducted a prospective study[6] with a conclusion that operations for SBO associated with groin hernia, surgery on the left side of the colon and rectum, and small bowel operations were mainly the procedures most frequently followed by EPSBO. Pickleman and Lee[5], conducted a study on 101 patients, thereby noted that EPSBO was more common after colectomy, small bowel resection and exploratory laparotomy. A lower risk of EPSBO was reported post surgeries in the supracolic compartment as compared to those performed in the infracolic compartment[613] [14]An increased risk of EPSBO was observed for perforated but not non-perforated appendicitis, following appendicectomy[15]. Andersson[22] in a cohort study of 245 400 patients who underwent open appendicectomy, stated that SBO developed within 4 weeks of surgery in 0·4 per cent. The associated risk factors were perforated appendicitis, negative appendicectomy and advanced age. EPSBO is a frequent complication of total colectomy. It developed in 5·5 per cent after total or subtotal colectomy[23] and 12·3 per cent of patients undergoing ileal pouch–anal anastomosis procedures[24]. Removal of omentum in such operations is thought to increase the risk of adhesive SBO, as it promotes adhesions of the intestine to the abdominal wall.

EARLY POSTOPERATIVE SMALL BOWEL OBSTRUCTION AFTER LAPAROSCOPY

A multicentre study from France stated that the prevalence of SBO after abdominal laparoscopic procedures was 0·2 per cent, with episodes of EPSBO representing 88 per cent of all obstructions[25]. Most commonly associated procedures with EPSBO are cholecystectomy, transperitoneal hernia repair and appendicectomy after laparoscopy. The mechanism of obstruction majorly included adhesions or 'bands' in half of the patients and small bowel incarceration at the port site in the other half. All port-site herniations were associated with the use of 10- or 12-mm trocars and the umbilical port was the commonest site observed. In the majority (66 per cent) of port-site herniations adequate fascial closure was not performed[25]. However, adequate amount closure of the fascial defect does not preclude the possibility of trocar-site incarceration of bowel; a strangulated Richter's hernia was reported in which the bowel was caught in the preperitoneal space behind a well repaired fascial defect of a subcostal trocar site[26] .Additionally , 5-mm port sites can also provide a nidus for bowel incarceration leading to EPSBO[27]. A case report described herniation of bowel into the port site immediately after removal of a peritoneal drain, leading to EPSBO[28]. One of the other causes of EPSBO following laparoscopic surgery is spilled gallstones during cholecystectomy, which may lead to the development of an inflammatory mass to which the bowel adheres[29] .One of the rare causes reported was an episode following laparoscopic appendicectomy[30]due to a loose linear staple from a load fired across the appendiceal stump.

CLINICAL FEATURES

The diagnosis of EPSBO is frequently delayed, or confused with that of postoperative paralytic ileus. Typical features of mechanical SBO project through the triad of colicky abdominal pain, vomiting and absolute constipation, along with abdominal distension. Presenting clinical features of early obstruction are generally less transparent as ileus. The reason documented for this is that

incisional pain and analgesia tend to mask the clinical picture. In one of the case reports, abdominal pain was present in 15 of 26 patients[317], another case reported vomiting in 82 per cen[5] of the patients , whereas in two case series 20 of 26 vomiting was observed. Absolute constipation is rare possibility; patients may continue to pass small amounts of flatus and even stool, that does not exclude EPSBO[31]. Abdominal distension is the only clinical feature that has been most consistently reported with EPSBO. It occurs in 76–81 per cent of patients[31]. Although 'obstructive' bowel sounds that accompany colicky abdominal pain are highly suggestive of mechanical SBO, differentiation of these from the high-pitched tinkling and uncoordinated bowel sounds attributed to postoperative ileus is difficult. According to some of the authors[31], when two or more of the above clinical features are present, the likelihood of reoperation is greater; 20 of 26 patients with three of four clinical signs ultimately required reoperation for persisting EPSBO[31].

DIAGNOSIS

EPSBO or persistent ileus is characterized mainly by failure of the patient to eat, pass flatus or evacuate the bowel within 5 days after a laparotomy. Obstruction is a more certain diagnosis if the patient has already passed flatus or stool and then ceases to do so. Nevertheless, the patient improves spontaneously sometimes without the cause being determined. On the contrary, abdominal imaging should be performed to help identify the cause of the problem and plan management accordingly.

RADIOLOGICAL INVESTIGATIONS

The first radiological study is ordered when features of EPSBO develop, is plain abdominal radiography with the patient erect and supine. The presence of distended small bowel loops with fluid levels, with inadequacy of colonic gas, indicates mechanical EPSBO. However, the interpretation of plain radiographs is difficult as EPSBO may manifest with distended small bowel loops and some air in the large bowel. There is only 19 to 73 per cent [31]level of accuracy of plain radiography in this setting [32].

Upper gastrointestinal contrast studies are regularly used. Failure of the contrast to reach the large bowel denotes mechanical EPSBO. The opposite is not necessarily true; as patients in whom orally administered contrast passes into the colon may also present with an obstruction and need a further operation[33]. In one of the cases [5], contrast follow-through studies identified a definite point of obstruction in 72 per cent of patients.

Clinicians favour use of water-soluble contrast for several reasons, contrarily radiologists prefer barium accounting to its superior imaging qualities. Firstly, barium tends to stay in the obstructed or paralysed bowel for many days. This may confound interpretation of subsequent computed tomography (CT) images. On the other hand, water-soluble contrast is harmless and, unlike barium[34], will not produce peritonitis even if it extravasates into the peritoneal cavity.

WHEN TO OPERATE?

There are several queries that can cross a surgeon's mind. Taking into consideration that persistent ileus and its causes have been ruled out, and there is no clinical and radiological evidence of strangulating obstruction, for how long should non-operative management be continued? Though the risk of intestinal strangulation in the setting of EPSBO is small, non-operative management should be attempted initially in every patient. There are however various schools of thoughts, regarding when to operate. Ellozy *et al.*[14]recommended re-exploration for patients whose symptoms fail to resolve within 1 week[14]. Resolution of the condition without operation usually occurs within 2 weeks, as was the case in 96 per cent of patients in Pickleman and Lee's series[5,13] and 30 (73 per cent) of 41 patients in the series by Quatromoni *et al*[13]. However, spontaneous resolution beyond 10 days after operation is unlikely[5]. The number of patients who eventually require operation varies between series, probably depending on the case mix. The comprehensive variation in reoperation rates also reflects the attitude of surgeons.

OBSTRUCTION AFTER LAPAROSCOPIC SURGERY

When SBO develops after laparoscopic surgery, it is mandatory to consider whether the bowel is caught partially or fully in one of the trocar sites. As per given in the literature, all patients who developed EPSBO after a laparoscopic procedure eventually required reoperation. The reason behind this can be because the small bowel was incarcerated in a peritoneal defect created either by trocar placement or peritoneal incision for herniorrhaphy [35]. Physical findings suggestive of this condition, such as exceptional tenderness at the trocar site or a mass, were not noted in any patient. To obtain an early diagnosis, CT and ultrasonographic examination of the abdomen are recommended. CT can help detect the trocar site responsible, allowing immediate operation to relieve the obstruction. This can be carried through the (extended) actual trocar site thereby, avoiding a formal laparotomy[36]. EPSBO after laparoscopy is a specific entity that demands immediate action.

PREVENTION

EPSBO can be prevented by sound operative technique and attention to detail. Gentle dissection and handling of tissues, leaving as little foreign material as possible (for example non-absorbable suture material), careful haemostasis to avoid haematoma formation, thorough peritoneal cleansing, avoiding potential orifices for internal hernias, not denuding the peritoneum unnecessarily, and not catching loops of bowel during abdominal closure, are clearly essential. Measures to prevent the development of EPSBO comprise of the routine closure of 10-mm trocar sites. This should be carried out under direct vision, incorporating all layers of the abdominal wall in order to eliminate peritoneal defects. Closure of 5-mm port sites should be taken into consideration if the fascial defect has been enlarged by operative manipulation.

CONCLUSIONS

ESBO is one of the commonest challenging problems faced by every surgeon. Although, most patients with ESBO will resolve with conservative measures, a significant proportion of patients do require re-operation. The decision to re-operate should be made after accounting all clinical and radiographic signs suggesting impending strangulation or closed loop obstruction, elapsed length of time of non-operative management, and the nature of the index operation. Early re-operation should be considered after certain laparoscopic procedures.

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