Pathogenesis, Management and Prevention of Adhesive Intestinal Obstruction

An Essay

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الاتجاهات الحديثة في علاج ومنع حدوث الإنسداد المعوي الالتصاقي

رسالة

توطئة للحصول علي درجة الماجستير في الجراحة العامة

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List of abbreviations

CT	Computed tomography
ECM	Extracellular matrix
FDP	Fibrin degradation products
ICAM	Intracellular adhesion molecule
IL	Interleukin
IPAA	Ileal pouch anal anastomosis
MMP	Matrix metalloproteinases
MR	Magnetic resonance
NK	Natural killer
NSAIDs	Non steroidal anti-inflammatory drugs
PAI- 1	Plasminogen activator inhibitor-1
PLGA	poly-lactideco-glycolide
PMNs	polymorpho-nuclear granulocytes
PPV	Positive prédictive value
PTFE	Poly-tetra-fluoroethylene
rtPA	Recombinant tissue plasminogen activator
SBO	Small bowel obstruction
SCAR	Surgical and Clinical Adhesions Research
SP	Substance P
TF	Tissue factor
TGF-β	Transforming growth factor-β
TIMP	tissue inhibitors of matrix metalloportinase
TNF	Tumor necrosis factor
tPA	Tissue type plasminogen activator
uPA	Urokinase-like plasminogen activator
US	Ultrasound
VCAM	Vascular cell adhesion molecule

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Introduction

Adhesions are abnormal fibrinous structures predominantly in the abdominal cavity. These structures are connections between serosal and/or non-serosal surfaces of the internal organs and the abdominal wall, at where there should be no connection **(Hammoud et al., 2004 and Vrijland et al., 2003).**

Intestinal obstruction is a common problem in surgical practice. It is responsible for more than 9000 deaths annually **(Russell et al., 2000).**

Adhesive intestinal obstruction makes up to 60 to 80 percent of admissions for intestinal obstruction **(Russell et al., 2000).**

Adhesive intestinal obstruction caused by a wide range of recognized inflammatory stimuli which cause peritoneal injury including; operative injury, bacterial peritonitis, radiotherapy, ischemic injury and foreign body reactions, e.g. starch talc and chemical injury **(Hellebrekers et al., 2000).**

Since adhesion formation occurs after trauma to visceral or parietal peritoneum, surgery is major etiologic factor associated with adhesion formation **(Peters et al., 2001 and Hammoud et al., 2004).**

Damaged peritoneum evokes a local inflammatory reaction, which leads to the formation of fibrinous exudates. This deposition of fibrin is necessary for normal tissue repair. During the reparation process, the fibrin deposit needs to be resolved to restore normal tissue composition. This resolution is mediated by the fibrinolytic system. An insufficient fibrinolytic system causes persistence of the fibrin deposition. An imbalance between fibrin deposition and dissolution will eventually leads to formation of permanent fibrous adhesions, with deposition of collagen **(Hammoud et al., 2004 and Hellebrekers, 2005).**

Adhesive intestinal obstruction can be diagnosed by a history of previous operation and four cardinal features, pain, vomiting, distention and constipation.

These features vary according to

a- The location of the obstruction

b- The duration of the obstruction

c- The underlying pathology

d- The presence or absence of intestinal ischemia

Late manifestations which may be encountered include dehydration, oliguria, hypovolemic shock, pyrexia, septicemia and respiratory embrassement **(Russell et al., 2000).**

The sensitivity of plain X ray for detection of complete intestinal obstruction is about 50% or may be less **(Al-Musawi and Thomposon, 2001)** or small bowel enema or oral gastrografin which can differentiate between complete and incomplete intestinal obstruction or US **(Ogata et al., 1994)** or CT or MRI **(Al-Musawi and Thompson, 2001).**

The initial management of adhesive intestinal obstruction is nasogastric suction and correction of fluids and electrolytes when clinically indicated. The surgical treatment of intestinal adhesion is adhesiolysis. This may be performed either laparoscopically or by laparotomy.

Adhesive intestinal obstruction can be prevented through:

Experimentally product as physical barriers **(Tudan et al., 2003)** or membrane barriers **(Dania and Togas, 2004)** and good handling with peritoneum to reduce peritoneal injury resulted from uses of surgical needles and retractors **(Boys, 1942)** and prevent presence of foreign bodies as starch and talc.

Aim of the work:

This essay is a trial to focus light on the magnitude of the adhesive intestinal obstruction in surgical practice, with emphasis on the pathogenesis of its formation, their treatment and the current methods of their prevention.

Anatomy and physiology of peritoneum

Anatomy:

Peritoneum is a serous sac consisting of a thin mesothelial membrane that lines the abdominal and pelvic cavities and covers most of the abdominal organs contained therein **(Healy and Reznek, 1998).**

The peritoneum is the largest serous membrane in the body, and consists, in the male, of a closed sac, a part of which is applied against the abdominal parietes, while the remainder is reflected over the contained viscera. In the female the peritoneum is not a closed sac, since the free ends of the uterine tubes open directly into the peritoneal cavity. The part which lines the parietes is named the parietal portion of the peritoneum; that which is reflected over the contained viscera constitutes the visceral portion of the peritoneum **(Elsayes et al., 2006).**

Although the peritoneum is a single continuous sheet, it is divided arbitrarily into two types, the visceral peritoneum and the parietal peritoneum. The parietal peritoneum lines the abdominal and pelvic cavities. The visceral peritoneum covers the external surface of most abdominal organs, or viscera. The small and large intestines are suspended from the posterior

aspect of the peritoneal cavity by the mesentery, a double layer of parietal peritoneum that has fused during embryologic development **(Healy and Reznek, 1998).**

The space between the parietal and visceral layers of the peritoneum is named the peritoneal cavity; but under normal conditions this cavity is merely a potential one, since the parietal and visceral layers are in contact. The peritoneal cavity gives off a large diverticulum, the omental bursa, which is situated behind the stomach and adjoining structures; the neck of communication between the cavity and the bursa is termed the epiploic foramen (foramen of Winslow). Formerly the main portion of the cavity was described as the greater, and the omental bursa as the lesser sac **(Elsayes et al., 2006).**

The peritoneum differs from the other serous membranes of the body in presenting a much more complex arrangement, and one that can be clearly understood only by following the changes which take place in the digestive tube during its development **(Healy and Reznek, 1998).**

The mesentery serves as a conduit for the blood vessels, nerves, and lymphatic vessels going to and from the abdominal organs. The omentum is a double-layer extension of visceral peritoneum that extends from the stomach. The lesser omentum, also known as the gastrohepatic ligament, arises from the lesser

curvature of the stomach and extends to the liver. The greater omentum arises from the greater curvature of the stomach and extends inferiorly in the peritoneal cavity **(Elsayes et al., 2006).**

Other peritoneal ligaments, such as the gastrosplenic ligament and splenorenal ligament, are also formed by fused double layers of peritoneum. The peritoneal cavity consists of several communicating spaces **(Meyers et al., 1987).**

Fused layers of peritoneum form the transverse mesocolon, which is the mesentery suspending the transverse colon. The transverse mesocolon divides the peritoneal cavity into supramesocolic and inframesocolic components, the transverse mesocolon acts as the floor of the lesser sac. The transverse mesocolon provides a pathway of spread for pancreatic disease to the transverse colon **(Elsayes et al., 2006).**

Supramesolic Compartment:

The supramesocolic compartment is divided into right and left peritoneal spaces by the falciform ligament. The left supramesocolic peritoneal space is bound on the right by the hepatic falciform ligament and consists of anterior and posterior perihepatic components. The anterior and posterior perihepatic spaces communicate freely below the lower border of the liver **(Healy and Reznek, 1998).**

This space can be affected by abnormalities involving the left hepatic lobe, lesser gastric curvature, anterior gastric and duodenal walls, and anterior wall of the gallbladder. The right supramesocolic peritoneal space comprises an anterior perihepatic region, bound medially by the falciform ligament, and a posterior component, known as the lesser sac. The two right supramesocolic spaces communicate via the foramen of Winslow. Morison's pouch (also known as the hepatorenal fossa) is a recess between the liver and right kidney. Fluid collections in the right perihepatic space are usually explained by abnormalities involving the right hepatic lobe, gallbladder, and duodenum **(Elsayes et al., 2006).**

Inframesocolic Compartment

The inframesocolic compartment is divided into two spaces by the obliquely oriented small-bowel mesentery. The right inframesocolic space is to the right of the small-bowel mesentery but medial to the ascending colon. The left inframesocolic space is to the left of the small-bowel mesentery. The right and left paracolic gutters run laterally to the ascending and descending colonic reflections, respectively. The right paracolic gutter is continuous superiorly with the right perihepatic space. On the left, the phrenicocolic ligament represents a barrier between the left paracolic gutter and the left

supramesocolic peritoneal space. Finally, the midline pouch of Douglas and the lateral paravesicular spaces form the most dependent portions of the peritoneal cavity, where infected fluid and malignant ascites usually pool by means of gravity **(Elsayes et al., 2006).**

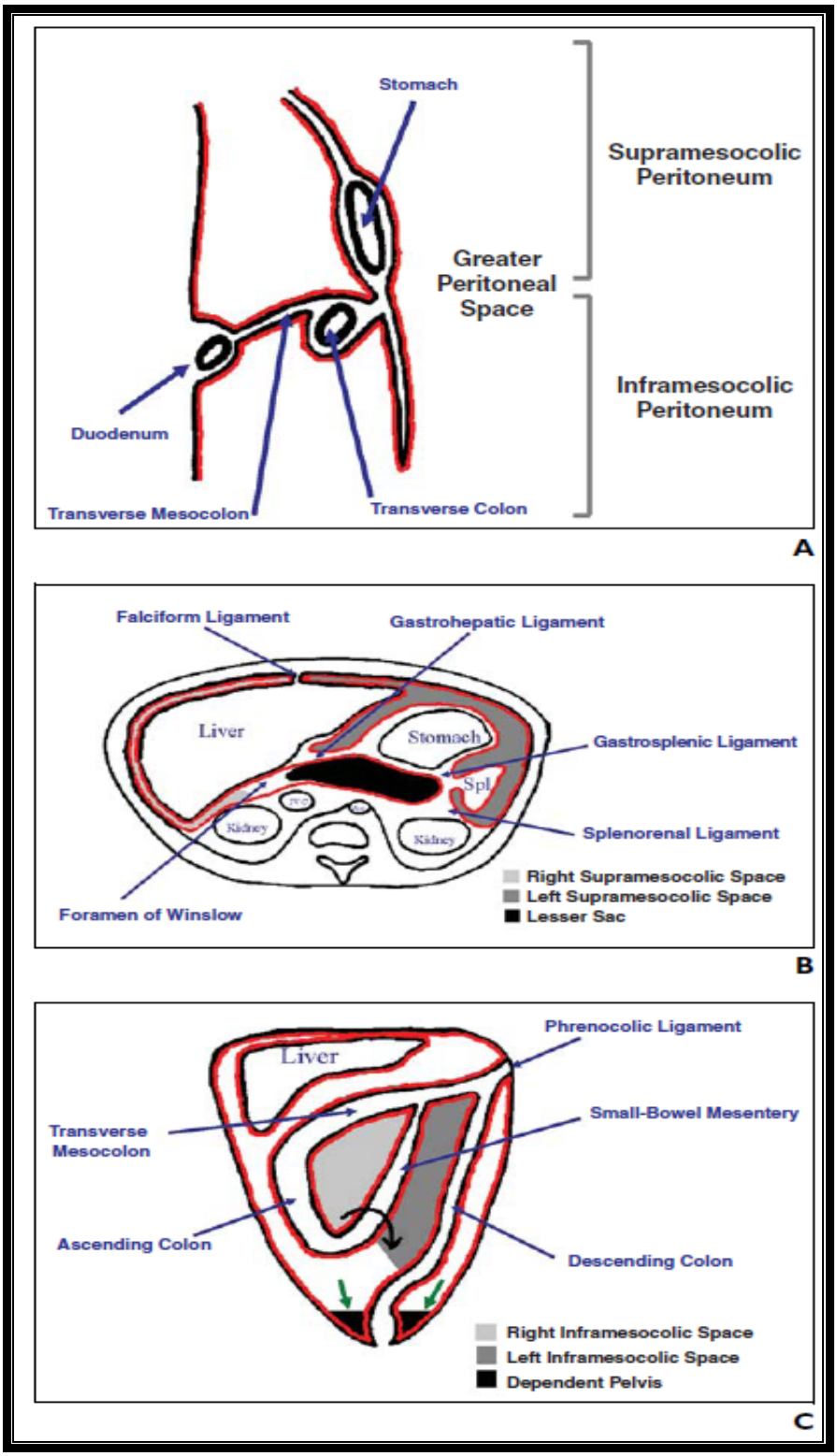

Figure (1): Schematics of peritoneal anatomy. A–C, In these sagittal (A), axial (B), and coronal (C) views, pouch of Douglas and lateral paravesicular spaces are seen to communicate (green arrows) with peritoneal cavity. Peritoneum is shown in red. Ao = aorta, IVC = inferior vena cava, Spl = spleen **(Elsayes et al., 2006).**