

## Introduction

Postoperative adhesions are almost invariable consequences of abdominal and pelvic surgery, no matter whether this performed open or laparoscopic technique. The most important morbidity is small bowel obstruction but other sequelae include female infertility, dyspareunia and increased risk of visceral injury at subsequent laparotomy or laparoscopy (*Ellis et al., 2009*).

Peritoneal adhesions are pathological bonds usually between omentum, loops of bowel and the abdominal wall. These bonds may be a thin film of connective tissue, a thick fibrous bridge containing blood vessels and nerve tissue, or a direct contact between two organ surfaces(*Diamond et al.,2001*).

Peritoneal adhesions are mostly induced by surgical procedures in the peritoneal cavity and their prevalence after major abdominal procedures has been evaluated at 63% - 79%. Small bowel obstruction (SBO) is the most common complication of peritoneal adhesions (*Kössi et al., 2003*).

Colorectal surgery has proved to be the most important type of surgery that may cause intra-abdominal adhesions (*Lower et al., 2000*). This surgery has the highest total number of inpatient episodes, inpatient days, operating time, theater time, and costs due to peritoneal adhesion-related intestinal obstruction (*Lower et al., 2000*).

A diagnosis of bowel obstruction is currently based on a patient's clinical history and physical condition in addition to radiographic analyses and routine blood tests. Common

symptoms include constipation, abdominal distention, abdominal tenderness and persistent nausea and vomiting. Treatment options include medical management or surgical intervention, but determination of the necessity and timing of surgery remains somewhat subjective. Patients whose diagnosis and/or treatment are delayed, risk the development of ischemic bowel with resultant increased morbidity and mortality (*Firoozma et al., 2001*).

The initial evaluation of patients with clinical signs and symptoms of intestinal obstruction should include plain x-ray upright abdominal radiography. Radiography can quickly determine if intestinal perforation has occurred; free air can be seen above the liver in upright films or left lateral decubitus films. Radiography accurately diagnoses intestinal obstruction in approximately 60 percent of cases, and its positive predictive value approaches 80 percent in patients with high-grade intestinal obstruction (*Maglente et al., 2003*), (*Lappas et al., 2001*).

However, plain abdominal films can appear normal in early obstruction and in high jejunal or duodenal obstruction. Therefore, when clinical suspicion for obstruction is high or persists despite negative initial radiography, non-contrast computed tomography (CT) should be ordered (*Stoker et al., 2009*).

It is important to accurately diagnose Small Bowel Obstruction due to adhesions because it may warrant a trial of conservative therapy unless signs of strangulation are present (*Petrovic et al., 2006*).

Treatment options for adhesive small bowel obstruction include early surgery or conservative treatment. There is no consensus with regard to the best procedure to follow (*Williams et al., 2005*).

In the early days of laparoscopy, previous abdominal surgery was a relative contraindication to performing most laparoscopic procedures. Laparoscopic surgery to relieve bowel obstructions was not routinely performed. However, in 1991, Bastug et al., reported the successful use of laparoscopic adhesiolysis for small bowel obstruction in one patient with a single adhesive band. Since then, many case series have documented this technique (*Nagle et al., 2004*).

The laparoscopic approach provides patients with the following benefits: less postoperative pain, decreased incidence of ventral hernia, fewer wound complications, reduced recovery time and return of bowel function, and shorter hospital stay. It also has been shown to decrease the incidence, extent, and severity of intra-abdominal adhesions as compared to open surgery, hence potentially reducing the rate to recurrent adhesive small bowel obstruction(*Tittel et al., 2001*).

Several preventive agents against postoperative peritoneal adhesions have been investigated. Their roles are in activating fibrinolysis, hampering coagulation, diminishing the inflammatory response, inhibiting collagen synthesis, or creating a barrier between adjacent wound surfaces (*Schnüriger et al., 2011*).

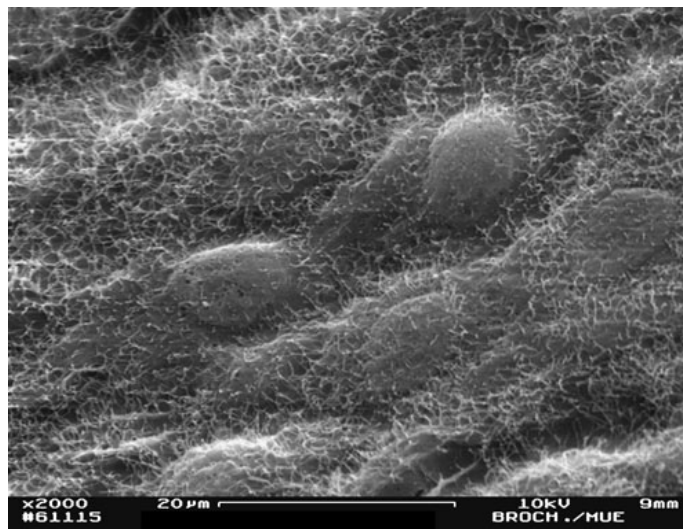
## **Aim of the Work**

The aim of this work is to focus light on the magnitude of the adhesive intestinal obstruction in surgical practice, with emphasis on the pathogenesis of its formation, the diagnostic tools, and treatment options including recent methods and role of laparoscopy in adhesiolysis and the recent methods of its prevention.

## Anatomy of the Peritoneum

### Terminology and Definitions

The peritoneum is a thin, translucent, serous membrane and is the largest and most complexly arranged serous membrane in the body. The peritoneum that lines the abdominal wall is called the parietal peritoneum, whereas the peritoneum that covers a viscous or an organ is called a visceral peritoneum. Both types of peritoneum consist of a single layer of simple low-cuboidal epithelium called a mesothelium. A capillary film of serous fluid (approximately 50–100 ml) separates the parietal and visceral layers of peritoneum from one another and lubricates the peritoneal surfaces (*Kim et al., 2007*).



**Figure (1):**Normal peritoneum consisting of a monolayer of flat mesothelial cells with numerous microvilli (SEM,  $\times 2000$ ) (*Brochhasuen et al., 2012*).

Previously considered as a passive, physical barrier, it is today well known that mesothelial cells play an active role not only in barrier function but also in the exchange of molecules and in the immunologic integrity of the serosa. Physiologically, mesothelial cells are responsible for the frictionless gliding of intraperitoneal organs by secreting substantial amounts of phosphatidylcholine (*Zhong et al., 2000*).

Mesothelial cells also create an antithrombotic surface and possess fibrinolytic activity. They are also involved in the immunological response of the peritoneum by activating lymphocytes and monocytes. The cellular functions of mesothelial cells are regulated not only by various cytokines but also by cell-cell and cell-matrix interactions. Mesothelial cells are able to express a variety of functional pivotal cell surface molecules such as cell adhesion molecules, which control the migration of leucocytes after peritoneal damage and during peritoneal inflammation. In addition, activated mesothelial cells are able to synthesize biologically active mediators such as nitrogen monoxide, plasminogen activator inhibitor and tissue plasminogen activator (*Yao et al., 2003*).

A unique property of the peritoneum is its delicacy. Because mesothelial cells are poorly interconnected through very loose intercellular bridges, the peritoneal surface is highly susceptible to trauma (*Mutsaers et al., 2007*).

Minimal mobilization or damage to the peritoneum can result in denudation of peritoneal surfaces, which can trigger the formation of adhesions. Another unique property of the peritoneum is its uniform, relatively rapid rate of surface mesothelialization after trauma. Irrespective of the size of injury, peritoneal mesothelialization is complete within 5–7 days (*DiZerega et al., 2001*).

The peritoneum is normally less than 1 mm thick and is not commonly visualized unless thickened by disease and/or surrounded by fluid. The peritoneal cavity is a potential space, only becoming apparent on cross sectional imaging when filled with abnormal fluid or gas (*Meyers et al., 2011*).

The peritoneum 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 (*Elsayes et al., 2006*).

Three terms are used to describe the parts of the peritoneum that connect organs with each other or to the abdominal wall:

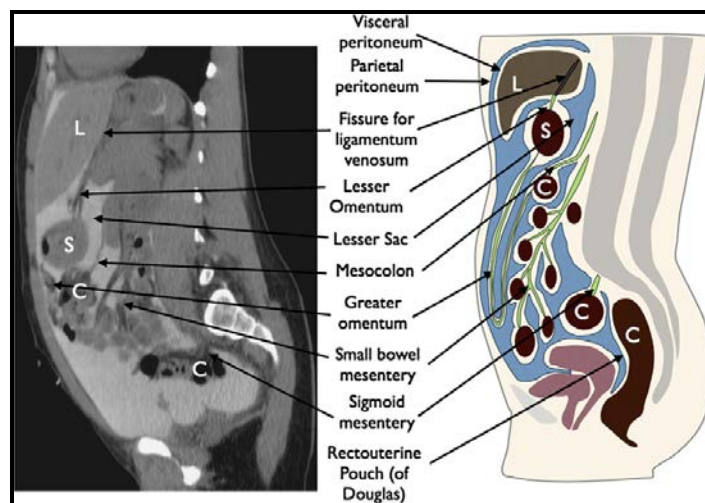
- A) A mesentery suspends the small and large bowel from the posterior abdominal wall by a double layer of peritoneum. It acts as a conduit for neurovascular and lymphatic structures between the organ and retroperitoneal structures. True mesenteries are connected to the posterior abdominal wall. Specialized mesenteries (the omenta and meso-appendix) are not attached to the posterior abdominal wall.

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B) A ligament is formed by two layers of peritoneum and supports a structure within the peritoneal cavity. It is named according to the structures it connects. For example, the spleno-renal ligament connects the kidney to the spleen; the gastro-splenic ligament connects the stomach to the spleen.

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C) An omentum refers to a double-layered continuation of peritoneal ligaments joining the stomach and proximal duodenum to adjacent structures. The greater omentum extends from the greater curvature of the stomach. It is formed from the fusion of the dorsal mesogastrium with the anterior border of the transverse colon and is, therefore, made up of four layers of peritoneum. The lesser omentum extends from the lesser curvature of the stomach to the liver(*Elsayes et al., 2006*).



**Figure (2):**Sagittal CT peritoneogram (CT post-intraperitoneal instillation of contrast medium via a peritoneal dialysis catheter) correlated with a schematic diagram demonstrating the peritoneal cavity. C, colon; S, stomach; L, liver(*Patel et al.,2012*).



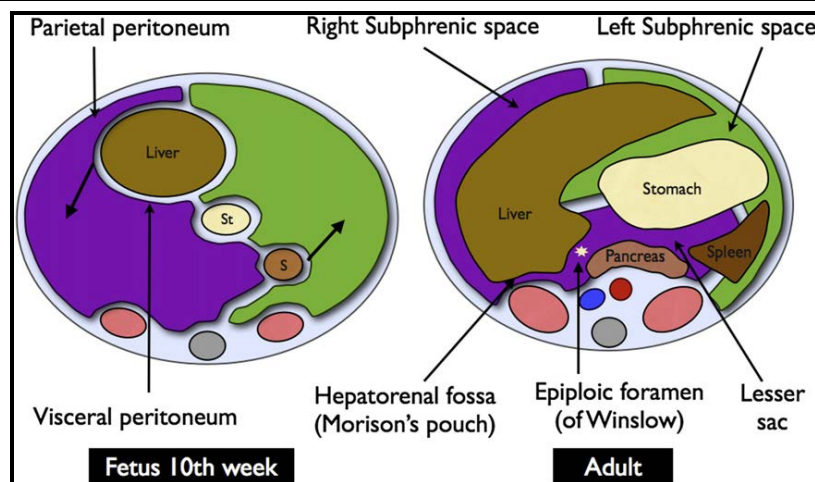
## Embryological development

An understanding of peritoneal embryological development is helpful to explain the configuration of the peritoneal spaces and how these spaces communicate. The primordial parietal peritoneum is of mesodermal origin and lines the embryonic body cavity. It is a closed sac and the lumen is the peritoneal cavity. Initially, the developing peritoneal cavity is divided into right and left by the ventral and dorsal mesenteries of the primitive gut, the remnants of which form many of the ligaments and mesenteries (*Moore et al., 2008*).

As the viscera develop, they acquire a visceral peritoneal covering as they protrude into the peritoneal sac. As the organs protrude into the peritoneal sac they remain connected to their extraperitoneal origins via neurovascular and lymphatic structures(*Moore et al., 2010*).

Cranial to the transverse mesocolon, the ventral mesentery contains the liver bud and the dorsal mesentery and the splenic bud. As development continues, these organs migrate anticlockwise taking their attached mesenteries with them. This migration divides the right peritoneal cavity into the perihepatic space and the lesser sac. The left peritoneal space forms the left subphrenic space(*Moore et al., 2010*).

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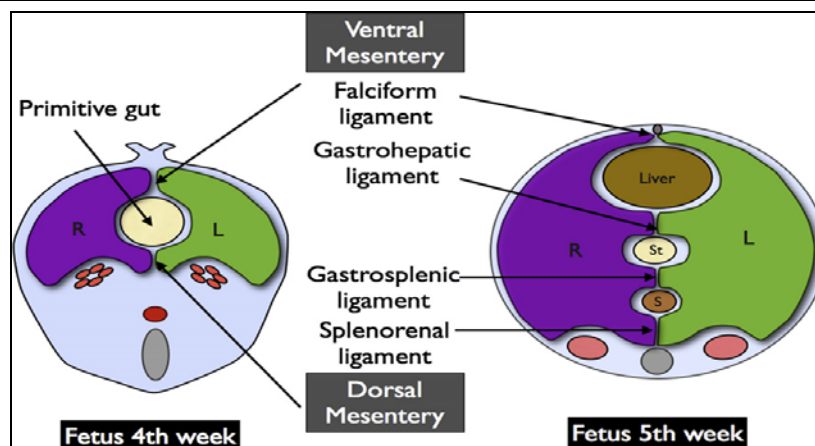
**Figure (3):** Schematic diagram demonstrating the formation of the peritoneal spaces from fetal life to adulthood. St, stomach; S, spleen(Patel et al.,2012).

## The Intraperitoneal Compartments

The peritoneal cavity is divided into interconnecting spaces, the supramesocolic and inframesocolic spaces and the pelvic cavity.

### Supramesocolic compartment

The supramesocolic compartment refers to the intraperitoneal spaces above the transverse colon. It contains the stomach, liver, and spleen. The ventral mesentery forms the falciform ligament, which divides the supramesolic compartment into left and right. It also gives rise to the lesser omentum, made up of the gastrohepatic and hepatoduodenal ligaments. The dorsal mesentery forms the greater omentum, gastroduodenal, gastrosplenic, gastrophrenic, gastropancreatic, splenorenal, and phrenicocolic ligaments (Eunhye et al., 2007);(Elsayes et al., 2006).



**Figure (4):** Schematic diagram demonstrating the formation of the peritoneal spaces from foetal life to adulthood. R, Right peritoneal space; L, Left peritoneal space, St, stomach; S, spleen(*Patel et al.,2012*).

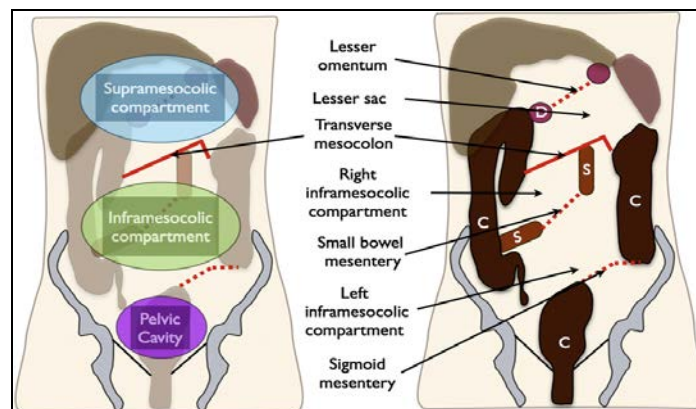
The right subphrenic space is limited postero-inferiorly by the coronary ligament of the liver but otherwise communicates freely with the perihepatic and subhepatic spaces, including the hepatorenal recess (Morison's pouch). It also communicates with the lesser sac via the epiploic foramen (foramen of Winslow). There is no compartmentalization of the left subphrenic space allowing communication between left subphrenic and perisplenic space(*Meyers et al., 2011*).

### Inframesocolic compartment

The inframesocolic compartment lies posterior to the greater omentum, below the transverse mesocolon and medial to the ascending and descending colon. It is divided into right and left by the oblique small bowel mesentery. It contains the small bowel and ascending and descending colon. The dorsal

mesentery gives rise to the transverse mesocolon, small bowel mesentery, sigmoid mesentery, and the mesoappendix. The ventral mesentery regresses below the transverse mesocolon. The mesorectum attaches to the posterior pelvis creating the perirectal space (Elsayes *et al.*, 2006).

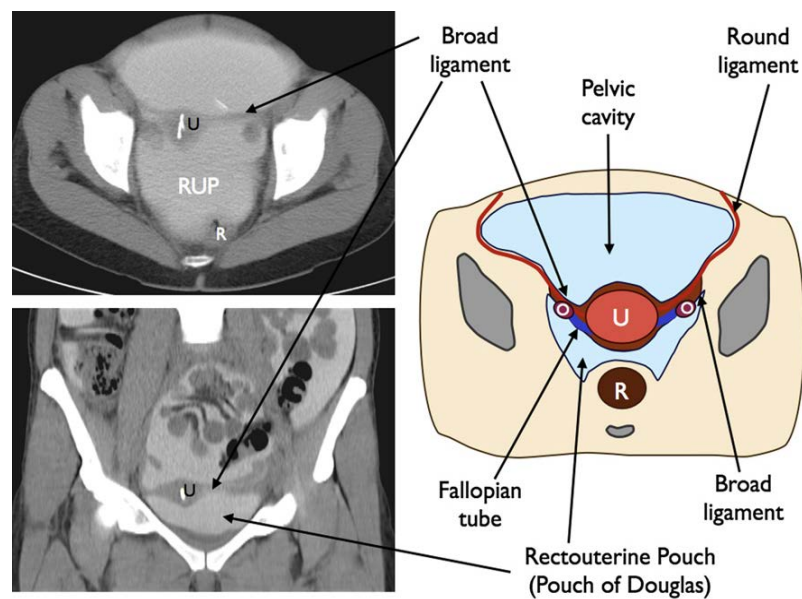
Lateral to the ascending and descending colon are the right and left paracolic gutters. The right paracolic gutter is continuous with the right perihepatic space. On the left, the phrenicocolic ligament prevents direct communication between the left paracolic gutter and the left subphrenic space. This helps contain disease entities such as a left subphrenic abscess. The supramesocolic compartment communicates with the inframesocolic compartments by way of the right paracolic gutter (Meyers *et al.*, 2011).



**Figure (5):** Coronal schematic diagram with the intraperitoneal bowel removed to illustrate the supramesocolic, inframesocolic, and pelvic compartments, and the origin of the omenta, mesenteries, and spaces. S, small bowel; C, colon D duodenum (Patel *et al.*, 2012).

## Pelvis

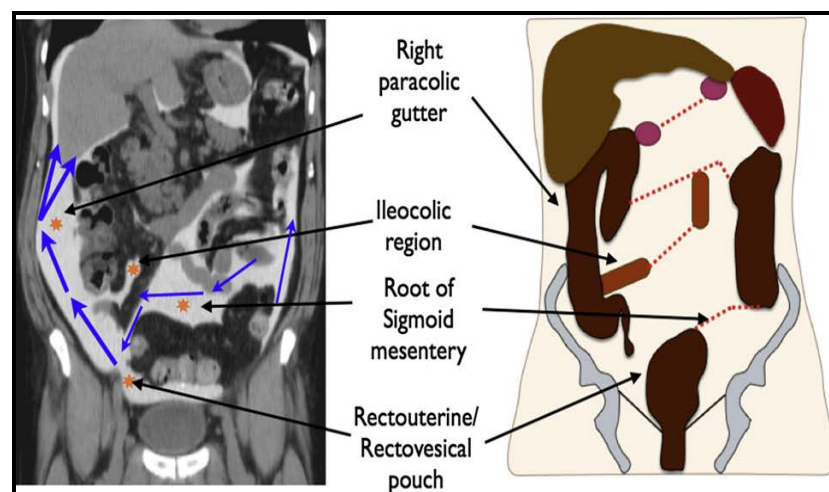
The urogenital peritoneum reflects over the pelvic organs to form most of the pelvic ligaments and mesenteries. These include the broad and round ligaments of the uterus in females. The median, medial, and lateral umbilical folds create the midline recto-vesical pouch in males and the recto-uterine pouch (pouch of Douglas) in females and the paravesical fossae. In females, the broad ligament is a peritoneal reflection forming the mesentery for the ovaries, fallopian tubes, and posterior myometrium. It also drapes over the ureters and round ligament. These ligaments and mesenteries act as a pathway for local spread of disease between structures (*Meyers et al., 2011*).



**Figure (6):** Axial and coronal CT peritoneograms demonstrating pelvic peritoneal folds and spaces in a female patient. U, uterus; R, rectum; RUP, rectouterine pouch; O, ovary (*Patel et al., 2012*).

## Peritoneal fluid circulation

The peritoneal cavity normally contains only a thin film of fluid, approximately 100 ml. This fluid is continually produced, circulated, and reabsorbed. Direction of flow is determined by diaphragmatic movement and bowel peristalsis. During inspiration, pressure decreases in the upperabdomen creating an intra-abdominal pressure gradient that encourages fluid to flow up the paracolic gutters even in a standing position. Limitations to flow are imposed by peritoneal attachments and ligaments. The peritoneal fluid takes the path of least resistance resulting in flow up the right paracolic gutter as this is wider than the left. The majority of the fluid is resorbed via lymphatics in the subphrenic space (*Meyers et al., 2011*).



**Figure (7):** Coronal CT peritoneogram demonstrating the direction of flow of peritoneal fluid, blue arrows, and regions of preferential fluid stasis (\*) correlated with a schematic diagram with the intraperitoneal bowel removed (*Patel et al., 2012*).

## Pathogenesis of Adhesive Intestinal Obstruction

Intraperitoneal adhesions are defined as cicatricial adherences between two contiguous peritoneal surfaces that are normally unattached. Following surgical interventions that result in peritoneal trauma, abnormal scar tissue may form between peritoneal surfaces that are normally free, resulting in definitive adhesion formation (*Duron, 2007*).

Development of fibrous adhesions is a nonspecific response to tissue injury, cutting, surgical denudation, ischemia, desiccation, or abrasion that occur as a consequence of peritoneal trauma during surgery. The subsequent healing process in the peritoneal cavity occurs by combination of mesothelial regeneration and fibrosis, resulting in either restoration of the tissue serosa or adhesion development between damaged serosal surfaces. Although adhesions per se are usually considered deleterious, there are times (such as when omentum adheres to occlude a site of leaking bowel anastomosis or other perforated viscous) where adhesions may potentially be advantageous (*Alpay et al., 2008*).

Adhesions can be considered a teleological response to tissue injury, which results from interference with the vascular supply to tissue (from cutting vessels, ligation of