A STUDY OF PERITONEAL ADHESIONS

Dissertation submitted in partial fulfillment of regulation for the award of M.S. Degree in General Surgery

(Branch I)

The Tamilnadu
Dr. M.G.R. Medical University
Chennai
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Coimbatore Medical College
Coimbatore - 641 014

CERTIFICATE

Certified that this is the bonafide dissertation done by **Dr. S. SELVA KUMAR** and submitted in partial fulfillment of the requirements for the Degree of M.S., General Surgery, Branch I of The Tamilnadu Dr. M.G.R. Medical University, Chennai.

Date : Unit Chief

Date : Professor & Head
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Date : Dean
Coimbatore Medical College
Coimbatore - 641 014

DECLARATION

I solemnly declare that the dissertation titled **"A Study of Peritnoeal Adhesions"** was done by me from 2006 onwards under the guidance and supervision of **Professor Dr. A. Ramamoorthy M.S.**

This dissertation is submitted to the Tamilnadu Dr. MGR Medical University towards the partial fulfillment of the requirement for the award of MS Degree in General Surgery (Branch I).

Place : **Dr. S. SELVA KUMAR**

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Name of the Candidate : Dr. S. Selvakumar

Course : M.S. (General Surgery)

Period of Study : 2006 - 2009

College : Coimbatore Medical College

Dissertation Topic : A Study Of Peritoneal Adhesions

The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation is accepted / ~~Not accepted~~ and you are permitted / ~~Not Permitted~~ to proceed with the above Study.

Coimbatore - 14.

Date : 8.10.2007

Secretary
Ethics Committee

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INDEX

INTRODUCTION

Peritoneal adhesions are deposits of fibrous tissue that occurs in peritoneal cavity. In majority of the patients, peritoneal adhesions occur as a result of surgery or peritonitis or their combination. In most patients, postopertaive adhesions do not cause any problem. Intraperitoneal adhesions are a major source of morbidity being the commonest cause of small bowel obstruction, secondary female infertility and ectopic gestation. They may also cause chronic abdominal and pelvic pain.

AIM OF THE STUDY

To analyse the various modes of presentation of peritoneal adhesions, investigations and management in patients admitted to the Surgical wards of Coimbatore Medical College Hospital, Coimbatore.

REVIEW OF LITERATURE

ANATOMY OF THE PERITONEUM

The peritoneum is a large serous membrane lining the abdominal cavity. Histologically it is composed of an outer layer of fibrous tissue, which gives strength to the membrane and an inner layer of mesothelial cells which secrete a serous fluid which lubricates the surface, thus allowing free movements of viscera.[19]

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 (Fig.2), 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.[12]

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.[12]

It is convenient to trace this from the back of the abdominal wall at the level of the umbilicus. On following the peritoneum upward from this level it is seen to be reflected around a fibrous cord, the ligamentum teres (obliterated umbilical vein), which reaches from the umbilicus to the under surface of the liver. This reflection forms a somewhat triangular fold, the Falciform ligament of the liver, attaching the upper and anterior surfaces of the liver to the Diaphragm and Abdominal wall. With the exception of the line of attachment of this ligament the peritoneum covers the whole of the under surface of the anterior part of the Diaphragm, and is continued from it on to the upper surface of the right lobe of the liver as the superior layer of the

coronary ligament, and on to the upper surface of the left lobe as the superior layer of the left triangular ligament (fig.1) of the liver. Covering the upper and anterior surfaces of the liver, it continues around its sharp margin on to the under surface, where it presents the following relations:

It covers the under surface of the right lobe and is reflected from the back part of this on to the right suprarenal gland and upper extremity of the right kidney, forming in this situation the inferior layer of the coronary ligament; a special fold, the hepatorenal ligament, is frequently present between the inferior surface of the liver and the front of the kidney. From the kidney it is carried downward to the duodenum and right colic flexure and medialward in front of the inferior vena cava, where it is continuous with the posterior wall of the omental bursa. Between the two layers of the coronary ligament there is a large triangular surface of the liver (fig.1) devoid of peritoneal covering; this is named the bare area of the liver, and is attached to the diaphragm by areolar tissue. Toward the right margin of the liver the two layers of the coronary ligament gradually

approach each other, and ultimately fuse to form a small triangular fold connecting the right lobe of the liver to the diaphragm, and named the right triangular ligament of the liver. The apex of the triangular bare area corresponds with the point of meeting of the two layers of the coronary ligament, its base with the fossa for the inferior vena cava.[12]

It covers the lower surface of the quadrate lobe, the under and lateral surfaces of the gall-bladder, and the under surface and posterior border of the left lobe; it is then reflected from the upper surface of the left lobe to the diaphragm as the inferior layer of the left triangular ligament, and from the porta of the liver and the fossa for the ductus venosus to the lesser curvature of the stomach and the first 2.5 cm. of the duodenum as the anterior layer of the hepatogastric and hepatoduodenal ligaments, which together constitute the lesser omentum. If this layer of the lesser omentum be followed to the right it will be found to turn around the hepatic artery, bile duct, and portal vein, and become continuous with the anterior wall of the omental bursa, forming a free folded edge of peritoneum. Traced downward, it covers the

antero-superior surface of the stomach and the commencement of the duodenum, and is carried down into a large free fold, known as the gastrocolic ligament or greater omentum (fig.2). Reaching the free margin of this fold, it is reflected upward to cover the under and posterior surfaces of the transverse colon, and hence to the posterior abdominal wall as the inferior layer of the transverse mesocolon. It reaches the abdominal wall at the head and anterior border of the pancreas, is then carried down over the lower part of the head and over the inferior surface of the pancreas on the superior mesenteric vessels, and thence to the small intestine as the anterior layer of the mesentery. It encircles the intestine, and subsequently may be traced, as the posterior layer of the mesentery, upward and backward to the abdominal wall. From this it sweeps down over the aorta into the pelvis, where it invests the sigmoid colon, its reduplication forming the sigmoid mesocolon. Leaving first the sides and then the front of the rectum, it is reflected on to the seminal vesicles and fundus of the urinary bladder and, after covering the upper surface of that viscus, is carried along the medial and lateral umbilical

ligaments on to the back of the abdominal wall to the level from which a start was made.

Between the rectum and the bladder it forms, in the male, a pouch, the rectovesical excavation, the bottom of which is slightly below the level of the upper ends of the vesiculae seminales—i.e., about 7.5 cm. from the orifice of the anus. When the bladder is distended, the peritoneum is carried up with the expanded viscus so that a considerable part of the anterior surface of the latter lies directly against the abdominal wall without the intervention of peritoneal membrane (prevesical space of Retzius). In the female the peritoneum is reflected from the rectum over the posterior vaginal fornix to the cervix and body of the uterus, forming the rectouterine excavation (pouch of Douglas). It is continued over the intestinal surface and fundus of the uterus on to its vesical surface, which it covers as far as the junction of the body and cervix uteri, and then to the bladder, forming here a second, but shallower, pouch, the vesicouterine excavation. It is also reflected from the sides of the uterus to the lateral walls of the pelvis as two expanded folds, the broad

ligaments of the uterus, in the free margin of each of which is the uterine tube.[12]

The Epiploic foramen (Foramen Epiploicum; Foramen of Winslow) is the passage of communication between the general cavity and the omental bursa. It is bounded in front by the free border of the lesser omentum, with the common bile duct, hepatic artery, and portal vein between its two layers; behind by the peritoneum covering the inferior vena cava; above by the peritoneum on the caudate process of the liver, and below by the peritoneum covering the commencement of the duodenum and the hepatic artery, the latter passing forward below the foramen before ascending between the two layers of the lesser omentum.

Numerous peritoneal folds extend between the various organs or connect them to the parietes; they serve to hold the viscera in position, and, at the same time, enclose the vessels and nerves proceeding to them. They are grouped under the three headings of ligaments, omenta, and mesenteries.

There are two omenta, the Lesser and the Greater.

The Lesser omentum (fig.2) (omentum minus; small omentum; gastrohepatic omentum) is the duplicature which extends to the liver from the lesser curvature of the stomach and the commencement of the duodenum. It is extremely thin, and is continuous with the two layers of peritoneum which cover respectively the antero-superior and postero-inferior surfaces of the stomach and first part of the duodenum. When these two layers reach the lesser curvature of the stomach and the upper border of the duodenum, they join together and ascend as a double fold to the porta of the liver; to the left of the porta the fold is attached to the bottom of the fossa for the ductus venosus, along which it is carried to the diaphragm, where the two layers separate to embrace the end of the esophagus. At the right border of the omentum the two layers are continuous, and form a free margin which constitutes the anterior boundary of the epiploic foramen. The portion of the lesser omentum extending between the liver and stomach is termed the hepatogastric ligament, while that between the liver and duodenum is the hepatoduodenal ligament. Between the two layers of the lesser omentum, close to

the right free margin, are the hepatic artery, the common bile duct, the portal vein, lymphatics, and the hepatic plexus of nerves—all these structures being enclosed in a fibrous capsule (Glisson's capsule). Between the layers of the lesser omentum, where they are attached to the stomach, run the right and left gastric vessels.

The Greater omentum (fig.2) (omentum majus; great omentum; gastrocolic omentum) is the largest peritoneal fold. It consists of a double sheet of peritoneum, folded on itself so that it is made up of four layers. The two layers which descend from the stomach and commencement of the duodenum pass in front of the small intestines, sometimes as low down as the pelvis; they then turn upon themselves, and ascend again as far as the transverse colon, where they separate and enclose that part of the intestine. These individual layers may be easily demonstrated in the young subject, but in the adult they are more or less inseparably blended. The left border of the greater omentum is continuous with the gastrolienal ligament; its right border extends as far as the commencement of the duodenum. The

greater omentum is usually thin, presents a cribriform appearance, and always contains some adipose tissue, which in fat people accumulates in considerable quantity. Between its two anterior layers, a short distance from the greater curvature of the stomach, is the anastomosis between the right and left gastroepiploic vessels.

The mesenteries are: the mesentery proper, the transverse mesocolon, and the sigmoid mesocolon. In addition to these there are sometimes present an ascending and a descending mesocolon.

The mesentery proper (mesenterium) is the broad, fan-shaped fold of peritoneum which connects the convolutions of the jejunum and ileum with the posterior wall of the abdomen. Its root—the part connected with the structures in front of the vertebral column—is narrow, about 15 cm. long, and is directed obliquely from the duodenojejunal flexure at the left side of the second lumbar vertebra to the right sacroiliac articulation. Its intestinal border is about 6 metres long; and here the two layers separate to enclose the intestine, and form its peritoneal coat. It is narrow above, but widens rapidly to about 20 cm., and is

thrown into numerous plaits or folds. It suspends the small intestine, and contains between its layers the intestinal branches of the superior mesenteric artery, with their accompanying veins and plexuses of nerves, the lacteal vessels, and mesenteric lymph glands.

The transverse mesocolon (fig.2) (mesocolon transversum) is a broad fold, which connects the transverse colon to the posterior wall of the abdomen. It is continuous with the two posterior layers of the greater omentum, which, after separating to surround the transverse colon, join behind it, and are continued backward to the vertebral column, where they diverge in front of the anterior border of the pancreas. This fold contains between its layers the vessels which supply the transverse colon.

The sigmoid mesocolon (fig.1) (mesocolon sigmoideum) is the fold of peritoneum which retains the sigmoid colon in connection with the pelvic wall. Its line of attachment forms a V-shaped curve, the apex of the curve being placed about the point of division of the left common iliac artery. The curve beings on

the medial side of the left Psoas major, and runs upward and backward to the apex, from which it bends sharply downward, and ends in the median plane at the level of the third sacral vertebra. The sigmoid and superior hemorrhoidal vessels run between the two layers of this fold.

In most cases the peritoneum covers only the front and sides of the ascending and descending parts of the colon. Sometimes, however, these are surrounded by the serous membrane and attached to the posterior abdominal wall by an ascending and a descending mesocolon respectively. A fold of peritoneum, the phrenicocolic ligament, is continued from the left colic flexure to the diaphragm opposite the tenth and eleventh ribs; it passes below and serves to support the spleen, and therefore has received the name of sustentaculum lienis.

The appendices epiploicae are small pouches of the peritoneum filled with fat and situated along the colon and upper part of the rectum. They are chiefly appended to the transverse and sigmoid parts of the colon.

The blood supply to the abdominal parietal peritoneum is from the branches of the arteries of the abdominal wall. The blood supply of the pelvic parietal peritoneum is from the blood vessels of the pelvic wall. Blood to the visceral peritoneum is from branches of the celiac trunk and from branches of the superior and inferior mesenteric arteries, or the pelvic visceral blood vessels.[18]

The lymphatics of the parietal peritoneum join the lymphatics of the body wall, and all drain to parietal lymph nodes. However, the lymphatics of the visceral peritoneum join the lymphatics of the related organs and are drained accordingly.[18]

Allen and Weatherford described the removal of particles of 10-20 microns from the peritoneal cavity through openings of the basement membrane with the help of the peritoneal lymphatics. These peculiar lymphatics were found only in the peritoneum covering the abdominal surface of the diaphragm.

In 1863, Von Recklinghausen was the first to describe the modified lymphatics which are able to remove particles from the peritoneal fluid during the process of respiration. The relaxed diaphragm permits opening of the stomata of these lymphatic vessels, and the fluid enters the lymphatic circulation. Higgins et al.reported that contractions of the diaphragm pump the lymph and its contents (particulate matter and molecular substances) upward, aided by one-way valves which are located within the lymphatics of the retrosternal area.

The parietal peritoneum contains somatic afferent nerves. The peritoneum contains many sensory fibers for the sensation of pain; the anterior portion of the parietal peritoneum is especially sensitive. The parietal peritoneum, therefore, is similar in sensitivity to the parietal pleura of the thorax.

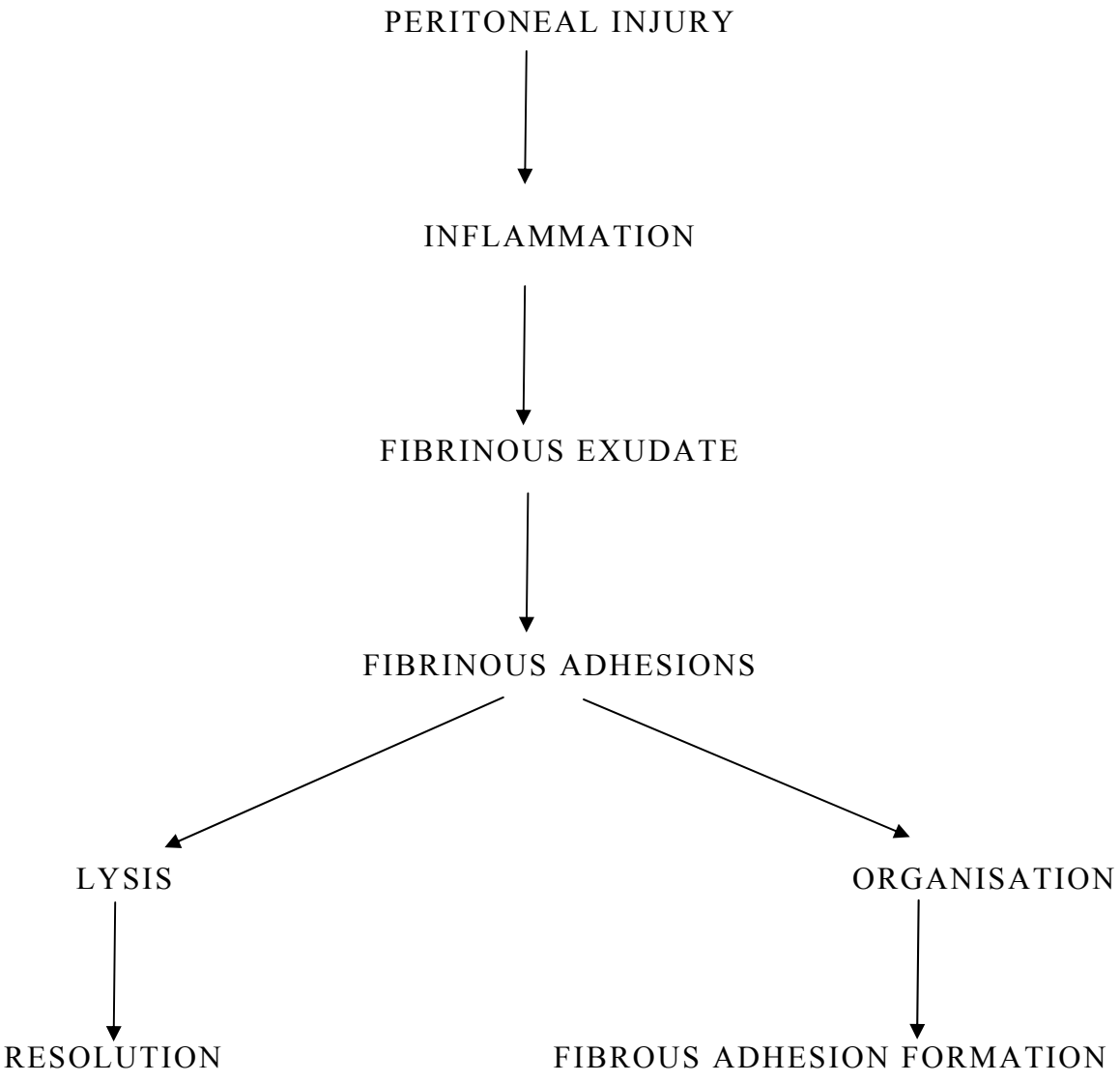
In contrast, the visceral peritoneum has no somatic afferent nerves and is relatively insensitive to pain. Sensations which do occur are poorly perceived and not clearly localized by the brain, as is characteristic of visceral afferent fibers carried by autonomic nerves to viscera in general. The principal stimulus which can evoke pain from visceral peritoneum is tension upon or stretching of the tissue, or ischemia. A perforated viscus may, perhaps, produce anterior abdominal wall rigidity, and an intraperitoneal fluid collection may produce pain like sensations of traction or tension on the mesentery in the retroperitoneal space, but not localized pain. A similarity can be seen here also between visceral pleura and visceral peritoneum, in that the visceral pleura which invests the lungs is relatively insensitive to pain.[18]

AETIO-PATHOGENESIS

The causes of peritoneal injury and adhesion formation are congenital, operative trauma, bacterial peritonitis, radiotherapy, ischaemic injury, foreign body reactions, (e.g. starch, talc), and chemical injury.[17]

Intra-abdominal adhesions are usually the result of peritoneal injury. A wide range of recognised inflammatory stimuli can cause peritoneal injury. Iatrogenic operative injury to the peritoneum and / or bacterial peritonitis are the leading causes of intraperitoneal adhesions. Histopathological studies demonstrate a clear sequence of events from injury to the formation of adhesions. Peritoneal inflammation leads to the formation of an inflammatory exudates which contains strands of fibrin. This fibrinous exudates is organized and fibroblast invasion is followed by deposition of collagen and the formation of permanent fibrous tissue. This process is not the inevitable result of peritoneal inflammation because mesothelial surfaces such as peritoneum possess fibrinolytic activity which, if not

impaired, will lyse fibrin within the inflammatory exudates before organization takes place.[4]



PERITONEAL FIBRINOLYSIS

Experimental and clinical studies have identified the presence of plasminogen-activation activity (PAA) in the mesothelium. Biopsies from both visceral and parietal peritoneum taken from different abdominal sites have shown similar levels of PAA. Tissue plasminogen activator (tPA) has found in human peritoneal tissue and is now considered to be the main physiological mediator of PAA.[17]

EFFECT OF PERITONEAL INJURY OR INFLAMMATION ON FIBRINOLYTIC ACTIVITY

Animal and human studies demonstrated that both mechanical and chemical injury reduced peritoneal PAA. This reduction in peritoneal PAA is currently regarded as central to the pathogenesis of adhesion formation. Studies of post operative peritoneal drain fluid have shown a progressive reduction in PAA in the first few hours following operation, followed by completed loss of fibrinolytic activity upto 72hrs after operation.

This reduction in peritoneal PAA caused by injury to peritoneum has been found to be due to the production and release of plasminoen activator inhibitors. Both plasminogen activator inhibitors, 1&2 (PAI-1 & PAI-2), have been isolated in high concentration in inflamed peritoneum and also in postoperative peritoneal fluid. These inhibitor reduce and subsequently abolish all peritoneal fibrinolytic activity. It appears that there is a biphasic response to surgery by the peritoneum; an early reduction in peritoneal PAA as a result of loss of tPA, followed by a later complete loss of fibrinolytic activity as a result of the marked increase in levels of PAI-1 &PAI-2.

Using in situ messenger RNA hybridization techniques , whawell and coworkers localized PAI-1 production to the mesothelium and the endothelial cells lining the subendothelial blood vessels, and PAI-2 production to the mesothelium and to the monocytes within the submesothelial tissue in inflammed peritoneum.

Surgeon have often observed a wide variation among patients in their tendency to form adhesions. Following an equivalent operative procedures, some patients develop extensive , dense and thick adhesions, while other have flimsy adhesions . It has been found that patients who developed severe and dense abdominal adhesions have lower levels of t-PA activity and 10 fold higher PAI-1 levels in their peritoneal fluid compared with those who developed milder and softer adhesions. The same phenomenon was also observed in adhesion tissues taken from patients with varying propensity of adhesion reformation. These observation might explain the individual variation in the susceptibility to adhesion formation. Components of plasmin system with in the peritoneum and / or adhesion tissue may be useful predictor for the degree of intraperitoneal adhesion formation or reformation.[17]

ROLE OF CYTOKINES IN ADHESION FORMATION

During peritoneal inflammation, cytokines and other inflammatory mediators are produced by resident cells and infiltrating leucocytes. Inflammatory cytokines have mainly paracrine effects, and their concentrations within the inflamed peritoneal fluid are several 100 folds higher than those in the measured plasma.

Mesothelial cells derived from human omentum or from a human mesothelial cell lines have demonstrated that the proinflammatory cytokines tumour necrosis factor –α , interleukin-1, interleukin-6, and transforming growth factor –β, together with lipopolysaccharide, all resulted in increased PAI-1 release. Studies of postoperative peritoneal fluid following peritoneal injury have shown that the time course of peritoneal cytokine production is in keeping with their de novo stimulation of plasminogen-activator inhibitor production and release. Thus it appears that operative injury results in an increase in peritoneal cytokine production with subsequent stimulation of synthesis and release of plasminogen activator inhibitors. These

act to reduce peritoneal fibrinolytic activity and enhance adhesion formation. This concept is supported by experimental studies , which have shown that postoperative and postirradiaton administration of IL-1 increased adhesion formation.[17]

PREVENTION OF PERITONEAL ADHESIONS

It is important from the prevention point of view, to recognize and define two types of adhesion formation.

1) de novo adhesions that occurs where no adhesion existed prior to the operation – either the operative site or at other intraperitoneal locations

2) adhesion reformation that recur following adhesiolysis – either at the main operative site or at other sites where adhesiolysis has been undertaken.[4]

SURGICAL TECHNIQUE

Good surgical technique remains an important part of adhesion prevention. The principle is to minimize peritoneal injury by careful handling of tissues, the use of atraumatic instruments, starch-free gloves, non-linting swabs, less reactive sutures and, in some procedures, operative magnification. Tissue ischaemia should be avoided and bacterial contamination minimized to avoid postoperative peritoneal infection. Careful haemostasis is an integral part of good surgical technique. Blood clots may adhere to the injured peritoneum and provide fibrin matrix necessary for adhesion formation, the type of surgical incision is an important factor in adhesion formation. Adhesions are more frequent following midline incision than Pfannenstiel incision and following gynaecological operation than obstetric surgery. The role of peritoneal fluid irrigation using crystalloid solutions during surgery to reduce adhesion formation is not proven and there is some evidence that these solutions are adhesiogenic. Ultra sonic scalpel showed no benefit in reducing adhesions in patients undergoing tubal surgery for infertility.[4]

PHARMACOLOGICAL AGENTS TO PREVENT ADHESIONS

A wide variety of agents have been used to reduce the peritoneal inflammatory response. These agents includes non-steroidal anti-inflammatory drugs, corticosteroids, histamine antagonists, anti oxidants and calcium channel blocking agents. A number of fibrinolytic enzymes have been used including streptokinase, urokinase, plasmin and t-PA. These enzymes when incorporated into slow release gel there is experimental evidence of localized reduction in adhesion formation.[4]

COMPLICATIONS OF PERITONEAL ADHESIONS

Inadvertent enterotomy during reopening of the abdomen or subsequent adhesion dissection is a feared complication of surgery after previous laparotomy. The incidence can be as high as 20% in open surgery and between 1% and 100% in

laparoscopy depending on the underlying disease. Delayed postoperative detection of enterotomy is a particular feature of laparoscopy associated with significant morbidity and mortality especially in the hands of untrained and inadequately trained laparoscopic surgeons.

Adhesions to the ventral abdominal wall are responsible for the majority of trocar injuries. Both trocar injuries and inadvertent enterotomies result in conversion from laparoscopy to laparotomy in almost 100% of cases.

Dissecting adhesions before executing the planned operation takes on average 20 min, being one-fifth of the total operating time in patients having had previous open colorectal surgery. There is some evidence that postoperative morbidity and mortality of patients who need adhesiolysis is higher than that of patients with a virgin abdomen. The necessity to dissect adhesions is associated with increased hospital stay. Postsurgical adhesions are considered a main reason for conversion from laparoscopy to laparotomy in many types of procedures including laparoscopic colonic resection.[9]

MATERIALS AND METHODS

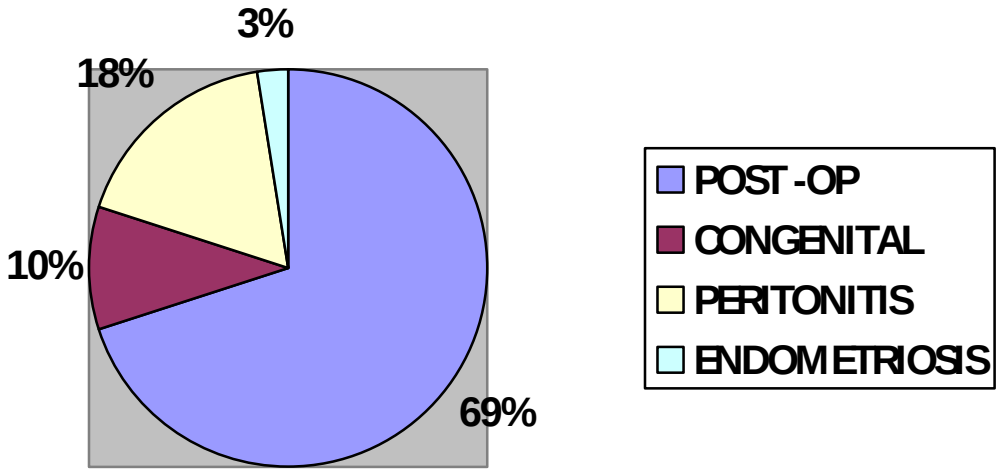
Patients who presented with symptoms of peritoneal adhesions in all surgical units in the Department of General Surgery, Coimbatore Medical College Hospital between 2006 – 2008 were evaluated.

The present study is based on the study of 40 cases of symptomatic peritoneal adhesions admitted into the surgical units of Coimbatore Medical College Hospital during the period from 2006 to 2008. The study includes all patients above 13yrs and both the sexes. The etiology, clinical features, investigations and management were studied. All patients were evaluated using a carefully prepared proforma, a through history, clinical examination and necessary investigations were carried out and recorded in proforma.

CLINICAL FEATURES

ETIOLOGY

Out of 40 cases of symptomatic peritoneal adhesions 28were due to previous sugery, 7 cases were due to peritonitis, 4 cases were due to congenital bands, one patient HIV positive with peritoneal adhesions and mass in the parietal wall presented with symptoms of adhesions, biopsy report of the mass came as endometriosis. None of the patient in this study group adhesions can be attributed to previous history of blunt injury abdomen



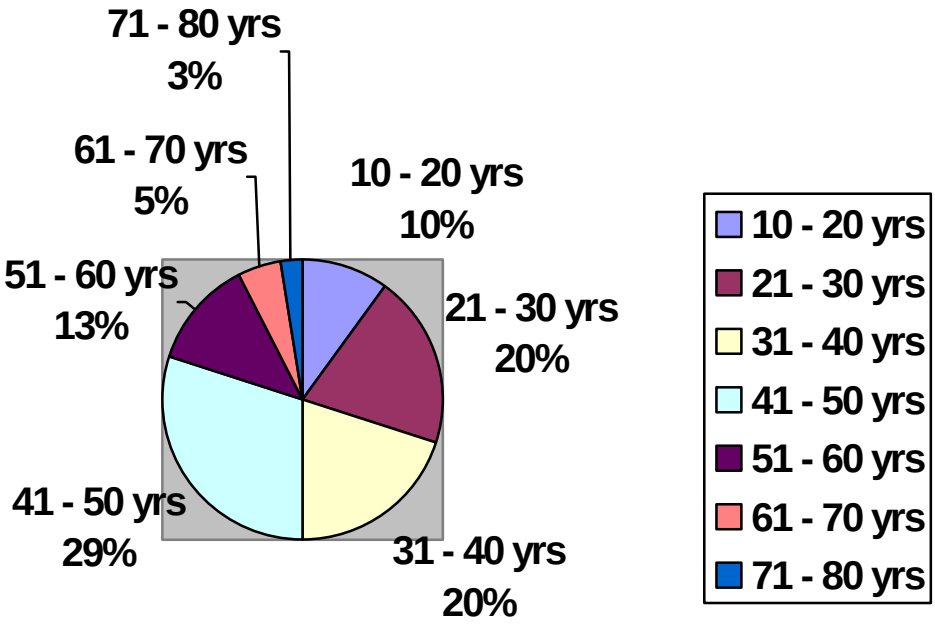
AGE

Out of 40 cases studied, about 12 patients belongs to the age group between 41-50 yrs of age, next is the 21-30 & 31-40yrs age group with 8 patients belonging to each of the age group. Four cases due to congenital bands were between 19 & 30 yrs of age.

AGE GROUP	NO OF CASES
10 -20 Yrs	4

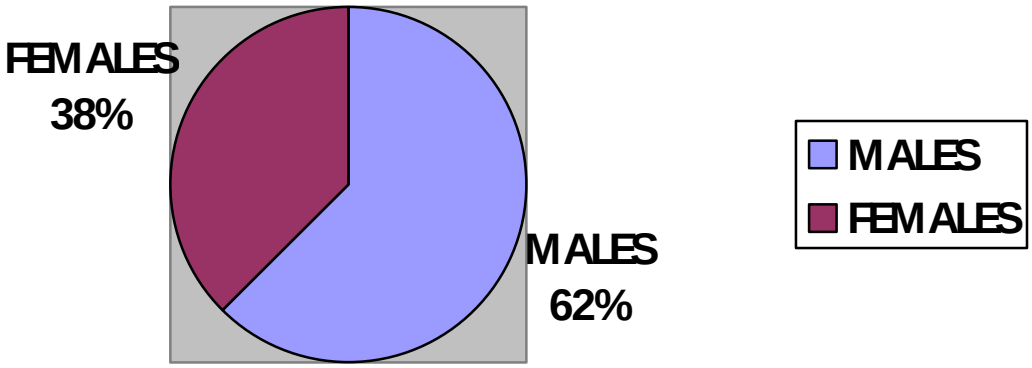
21- 30 Yrs	8
31 -40 Yrs	8
41-50 Yrs	12
51-60 Yrs	5
61-70 Yrs	2
71-80 Yrs	1

AGE



SEX

Out of 40 cases studied 25 patient were males and 15 patients were females. The male : female ratio is 1.7

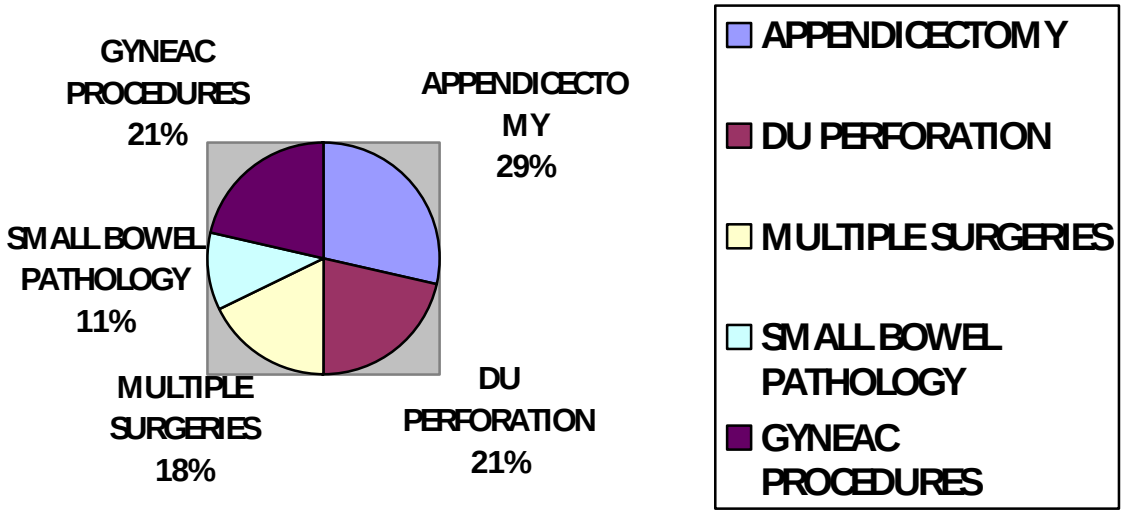


TYPE OF PREVIOUS SURGERY

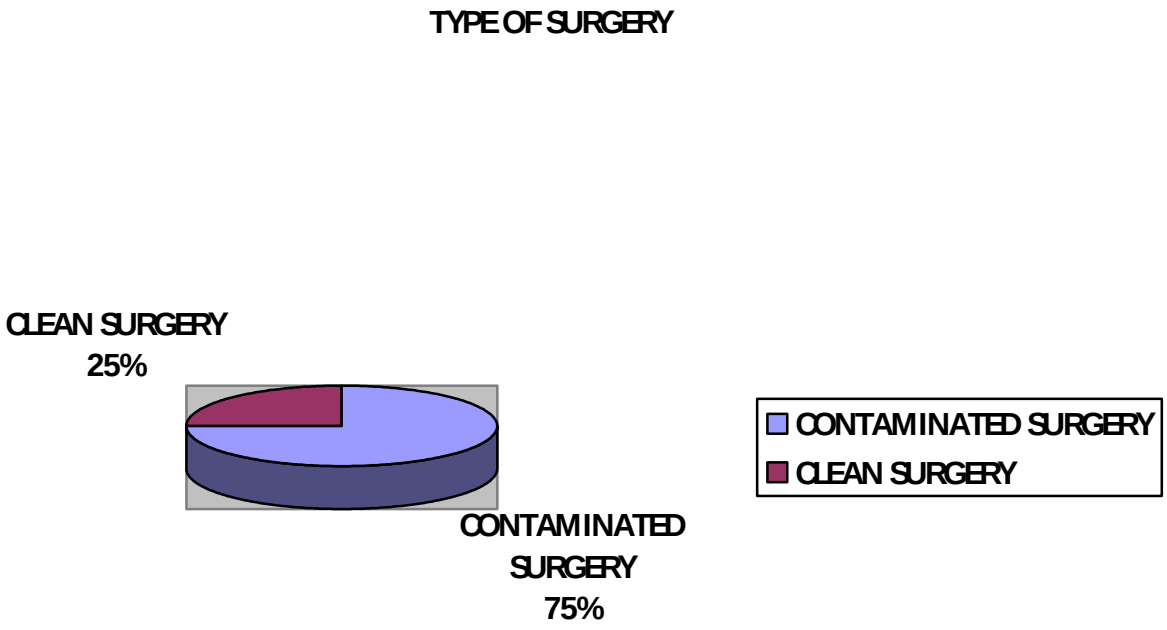
Of the 28 patients with adhesions due to previous surgery, 8 were due to previous appendicectomy, 6 were due to perforated duodenal ulcer, 5 were due to multiple surgery, 3 patients had undergone surgery for small bowel pathology and 6 patient had

undergone gynaecological procedures. The study shows a high incidence of adhesion formation after appendicectomy, duodenal perforation closure and patients who had undergone multiple surgeries as suggested in the literature. All the patients who presented with adhesions were due to previous surgery.

TYPE OF PREVIOUS SURGERY



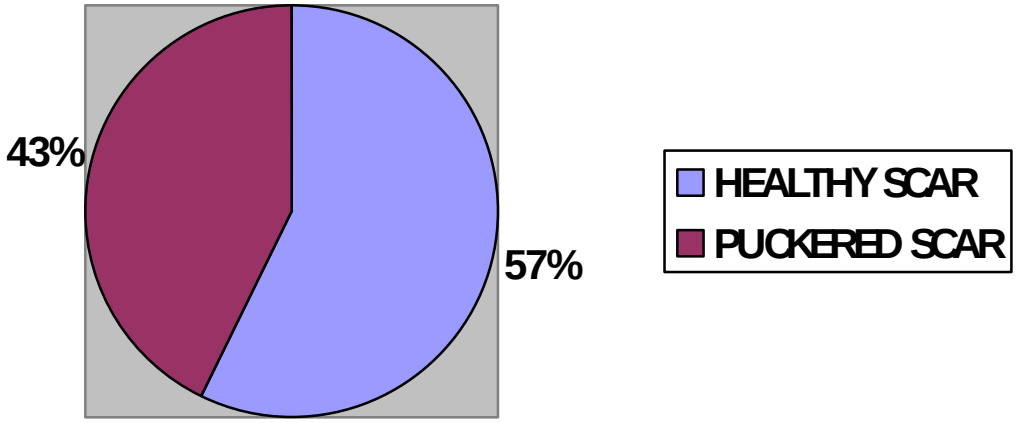
Out of 28 patients of postoperative adhesions, 21 patients had undergone surgery for infected pathology while 7 patients for non infected pathology. Adhesions more frequently occurred in patient with peritonitis and peritoneal contamination than patient undergone surgery without peritonitis.



TYPE OF SCAR

Out of 28 patients with postoperative adhesions, 16 patients had healthy linear scar which has healed by primary intension and 12 patients had puckered scar. Patients with

puckered and dimpled scar is more likely to have postoperative adhesions.

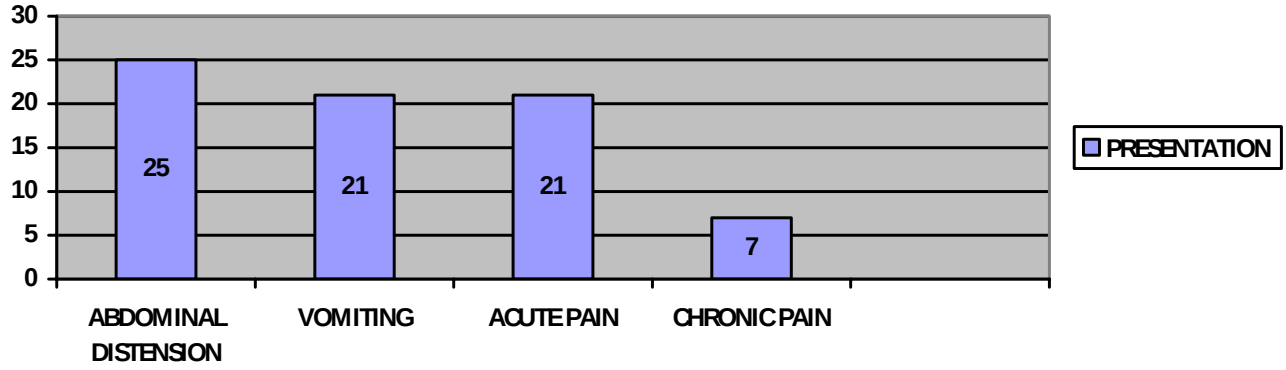


MODE OF PRESENTATION

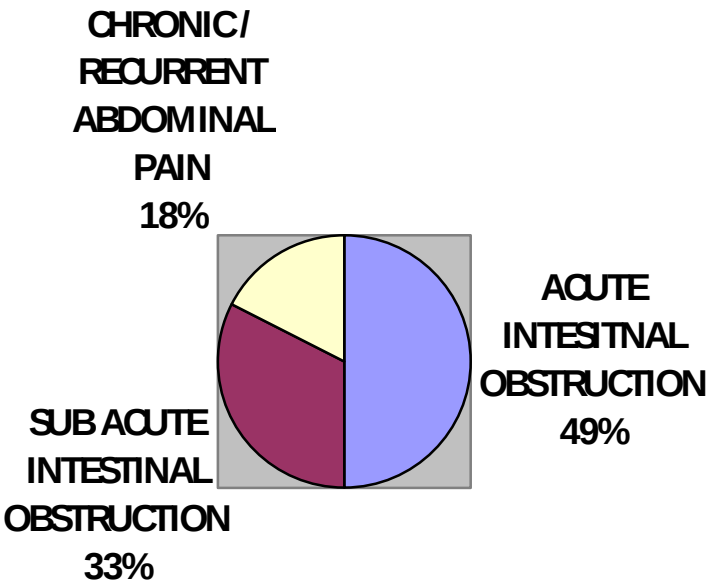
Most of the patient presented with peritoneal adhesions presented with abdominal distention, acute abdominal pain and

vomiting. A few of the patient with partial instestinal obstruction presented with recurrent chronic abdominal pain.

Out of 40 patients 25patient gave history of abdominal distension or fullness / bloating of the abdomen. 21 out of 40 had atleast one episode of vomiting. 28 patients presented with abdominal pain of which 21 are acute in nature lasting for less than 2 weeks and 7 are chronic in nature lasting for more than 2 weeks.



Out of 40 patients, 20 patients presented with acute intestinal obstruction, 13 patients presented with subacute intestinal obstruction and 7 patients presented with chronic / recurrent abdominal pain.



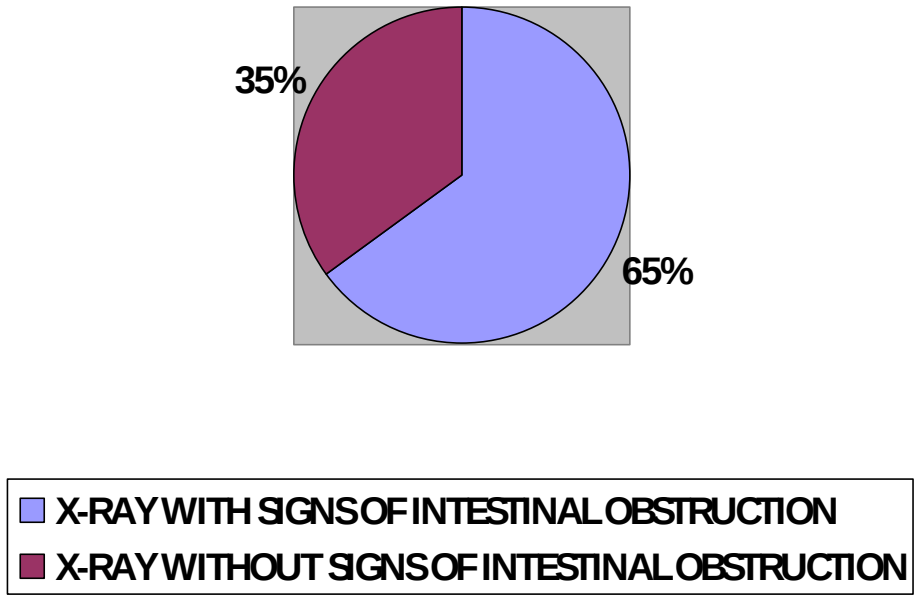
MANAGEMENT

INVESTIGATIONS

X-RAY ABDOMEN

X-ray abdomen erect was taken for all the patient studied. X ray abdomen showed sign of intestinal obstruction (dilated small bowel loop & multiple air fluid level) in 26 out of 40 patients. They are more predictive for cases with acute obstruction than for patients with subacute obstruction.

X-RAY ABDOMEN



USG ABDOMEN

USG abdomen was done for patients with subacute obstruction and in patients with chronic abdominal pain. USG showed dilated bowel loops and hyperactive peristalsis in patients with subacute obstruction. USG abdomen for one patient who presented with chronic abdominal pain revealed a mass measuring 2*2 cm in the parietal wall in the subumbilical area, biopsy proved it as endometriosis. In other patients with chronic

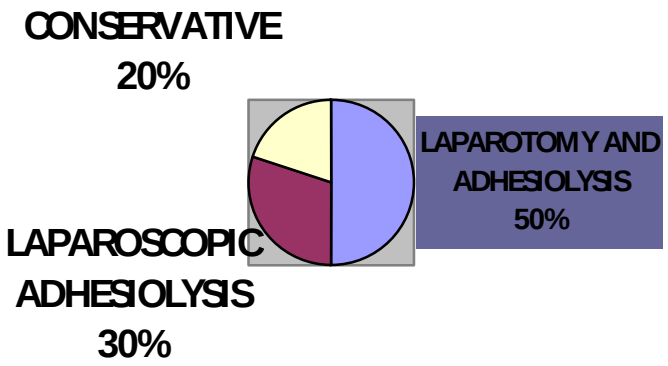
abdominal pain without features of intestinal obstruction USG didn't reveal any abnormality.

DIAGNOSTIC LAPAROSCOPY AND ADHESIOLYSIS

6 out of 7 Patients, who presented with chronic abdominal pain without features of intestinal obstruction had normal x-ray and ultrasound abdomen, and were subjected to diagnostic laparoscopy. All the patients had peritoneal adhesions and adhesiolysis done .

TREATMENT

Out of 40 patients who presented with symptomatic peritoneal adhesions 20 who presented with acute intestinal obstruction underwent emergency laparotomy and adhesiolysis, 12 patients who presented with subacute intestinal obstruction and chronic abdominal pain underwent laparoscopic adhesiolysis, 8 patient had their symptoms resolved with conservative management (ryle's tube aspiration and IV fluids).



One of the patient who underwent laparoscopic adhesiolysis had omental adhesions to the site of Pueperal sterilisation scar and a swelling measuring 2*2 cm in size in close proximity to the adhesions, biopsy of the swelling came as endometriosis. Endometriosis is one of the cause of peritoneal adhesions in females.

Accidental enterotomy happened with two patients who underwent laparotomy for acute intestinal obstruction while opening the abdomen . For one of the patient it was closed in two layers, While the other patient had cooconed small bowel with dense adhesions to the anterior abdominal wall so resection and anastomis of cooconed small bowel and ileotransverse anastomosis done.

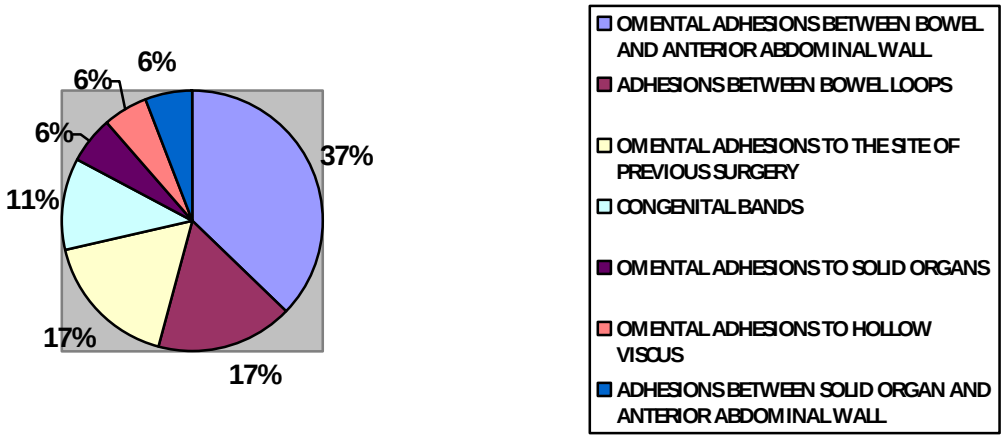
Two patients in this study out of 20 patients who underwent emergency laparotomy had gangrenous change in the small bowel due to internal herniation of small bowel, resection and anastomosis of small bowel done along with adhesiolysis in both the patients.

One of the patient who presented with acute intestinal obstruction had miliary tubercles studded throughout the abdomen with a band of peritoneal tissue encircling the distal ileum on laparotomy. Biopsy from the tubercle showed tuberculous aetiology. Patients was started on ATT in post operative period period and patient recovered well.

Two patient out of 40 patients succumbed to peritoneal adhesions. The cause of death in both the cases is septiceamia with cardiorespiratory arrest. Both the patient had gangrenous change in the small bowel.

TYPES OF ADHESIONS

Patients presented with different types of adhesions, 13 patients had adhesion between bowel loop and anterior abdominal wall, 6 had adhesions between bowel loops, 6 patients had omental adhesions to the site of previous surgery, 4 patients had congenital band, 2 patients had omental adhesions to solid organs, 2 patients had omental adhesions to hollow viscus, 2 patients had adhesions between anterior abdominal wall and solid organ.



DISCUSSION

There is some evidence from animal and clinical studies that laparoscopic procedures cause fewer abdominal adhesions compared to open operations. The possible factors for decreasing adhesions post – laparoscopy are; meticulous technique with the co-axial illumination and magnification, good haemostasis, liberal irrigation, and use of fine electrodes. Gauze swabs, retractors and foreign bodies such as lint and talcum powder are not used in laparoscopic surgery. Prolonged peritoneal exposure to air during laparotomy and subsequent mesothelial dessication may contribute to de novo adhesion formation at sites remote from the operative procedure. As the peritoneal cavity is normally sterile, warm and wet, peritoneal injury during laparoscopy may be further minimized by using filtered, heated and hydrated insufflating gas instead of the currently used unconditioned dry gas.

A prospective randomized clinical trial reported by Lundorff and colleagues used second-look laparoscopy to

evaluate adhesion formation in a 105 women with ectopic pregnancy randomised to either laparoscopy or laparotomy treatment. The authors concluded that laparoscopic surgery induced fewer de no adhesions and less adhesion formation at the operation site. Recently, Levrant and co- workers have demonstrated that the incidence of incisional adhesions is less with laparoscopic surgery. Some author have claimed that laparoscopic procedures decrease adhesion reformation, but this conclusion was not supported in a recent meta- analysis. Common problems with such studies are the variable timing of second-look laparoscopy and the classification of adhesions severity. Most of the available clinical data comes from gynaecological studies of fallopian tube surgery and there are few data from general surgical laparoscopic procedures. In summary, laparoscopic procedures appears to reduce incisional and de novo adhesion formation away from the operative site.

In this study 40 patients who presented with symptomatic peritoneal adhesions were studied, 69% of the adhesions were due to previous surgery, 18% were due to peritonitis, 10% of the

patient had congenital band and 3% due to endometriosis and PID. Perry Jr JF, Smith GA, Yonehiro EG. Intestinal obstruction caused by adhesions. A review of 388 cases. Ann surg 1955; 142: 810 – 816 in a review of 388 patients with abdominal adhesions, 79% had a history of surgery, 18% had a history of peritoneal infection, and 11% had congenital adhesions

75% with symptomatic adhesions had undergone contaminated surgery only 25% of the patient had undergone clean surgery.

29% Of the patients belong to the 41 -50 yrs age group, while 20% of the patients belong to 21 – 30 & 31 – 40 yrs group. 62% of the patients who presented with symptomatic adhesions are males, while 38% are females.

Appendicectomy is the main cause of adhesions with 29% of the cases due to previous appendicectomy, laparotomy and duodenal ulcer perforation comes next with 21% of the cases. 18% of the patients with adhesions had undergone multiple

surgeries. Fuzun M, Kaymak E, Harmancioglu O, Astarciolu K, Principal cause of mechanical bowel obstruction in surgically treated adults in Western Turkey. Br J Surg 1991; 78: 202 -203 stated that acute appendicitis and appendicectomy are potent cause of adhesions, 36% of patients presenting with postoperative adhesions had undergone appendicectomy

Most of the patient presented with complaints of abdominal distension. Vomiting and acute abdominal pain was present in more than half of the patients. Patients with subacute obstruction presented with chronic abdominal pain.

43% of patients with adhesions due to previous surgery had puckered scar. Patients with puckered scar is more likely to have problem due to adhesions. Salim R, Kadan Y, Nachum Z, Edelstein S, Shalev E. Department of Obstetrics and Gynecology, Ha `Emek Medical Center, Afula, Israel; Rappaport Faculty of Medicine, Technion–Israel Institute of Technology, Haifa, Israel. Abdominal scar characteristics as a predictor of intra-abdominal adhesions at repeat cesarean delivery, Fertility and sterility

2008, Pub Med PMID: 18178195, concluded that A depressed abdominal scar of a previous cesarean delivery is correlated with the incidence and severity of intra-abdominal adhesions.

50% undergone laparotomy and adhesiolysis, all the patients in this group presented with acute obstruction. 30 % undergone laparoscopic adhesiolysis, all the patient in this group presented with subacute obstruction and chronic abdominal pain. 20% of the patients recovered by conservative management.

5% of the patient in this study died of the disease, the cause of death is due to septic complication of the disease.

CONCLUSION

This study concludes that

- The commonest cause of symptomatic peritoneal adhesions is previous surgery especially contaminated surgery.

- Patients with acute obstruction need emergency laparotomy, these patients can be managed conservatively and can be taken up for laparoscopy and adhesiolysis once the acute crisis resolves.

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PROFORMA

NAME: AGE/SEX: IPNO:

ADDRESS:

DATE OF ADMISSION :

DATE OF SURGERY :

DATE OF DISCHARGE :

CHIEF COMPLAINTS:

1) Abdominal distension :
2) Abdominal pain :
3) Vomiting :
4) Constipation
5) Fever :
6) Others :

PAST HISTORY :

PREVIOUS SURGERY DETAILS :

PERSONAL HISTORY :

MENSTRUAL HISTORY :

CLINICAL FINDINGS :

TYPE OF PREVIOUS INCISION :

TYPE OF SCAR : 1) HEALTHY 2)PUCKERED 3)IRREGULAR

INVESTIGATIONS:

BLOOD INVESTIGATIONS :

X-RAY CHEST & ABDOMEN :

USG ABDOMEN :

OTHER INVESTIGATION :

PLAN OF MANAGEMENT :

CONSERVATIVE LAPAROSCOPY LAPAROTOMY

OPERATIVE FINDINGS :

TYPE OF ADHESIONS :

POST OPERATIVE PERIOD :

FOLLOW UP :