

INTRODUCTION

The term acute abdomen designates symptoms and signs of intra-abdominal disease usually treated best by surgical operation, so the evaluation of patients with abdominal pain must be methodical and careful. The proper management of patients with acute abdominal pain requires a timely decision about the need for surgical operation. This decision requires evaluation of the patient's history and physical findings, laboratory data, and imaging tests. so that timely treatment can minimize morbidity and mortality (*Jones et al., 2004*).

Diagnostic laparoscopy (DL) is minimally invasive surgery for the diagnosis of intra-abdominal diseases. The procedure enables direct inspection of large surface areas of intra-abdominal organs, facilitates the acquisition of biopsy specimens, cultures, and aspirates, allows the use of laparoscopic ultrasound and makes therapeutic intervention possible. Diagnostic laparoscopy has been applied to many clinical situations with variable success (*Richardson et al., 2009*).

Laparoscopy is not analternative to physical examination/good clinical judgment or to conventional noninvasive diagnostic methods in treating the patient with symptoms of an acute abdomen. However it must be considered an effective option in treating patients in whom these methods

fail and as a challenging alternative to open surgery in the management algorithm for abdominal emergencies (*Agresta et al., 2008*).

Laparoscopic surgery has now been described in treatment of many abdominal emergencies, such as acute appendicitis, blunt and penetrating trauma, perforated peptic ulcer disease, acute cholecystitis, Non-Specific abdominal Pain and Gynaecological Emergencies as Ectopic Pregnancy and Ovarian Cyst Torsion, and this variety of conditions seems to expand further (*Warren et al., 2006*).

Laparoscopy has been advocated for patients in intensive care units based on the unreliable nature of diagnosis in the patient with pharmacological and metabolic obtundation, prior intubation, compounding comorbidities, unexplained sepsis, acidosis, or multiorgan failure, and whose transportation to the radiology suite or subjection to other investigative procedures is problematic. Acalculous cholecystitis, gastrointestinal tract perforation, intestinal ischemia, pancreatitis, bowel obstruction, and intra-abdominal hemorrhage are frequent diagnoses, easy to make laparoscopically (*Shukla et al., 2010*).

AIM OF THE WORK

To discuss different applications of laparoscopy in diagnosis and treatment of acute abdomen.

PATHOPHYSIOLOGY OF ACUTE ABDOMEN

Definition of acute abdomen

The term acute abdominal pain generally refers to previously undiagnosed pain that arises suddenly and is of less than 7 days' (usually less than 48 hours') duration. It may be caused by a great variety of intraperitoneal disorders, many of which call for surgical treatment, as well as by a range of extraperitoneal disorders which typically do not call for surgical treatment. Abdominal pain that persists for 6 hours or longer is usually caused by disorders of surgical significance (*David et al., 2007*).

The complaint of abdominal pain is probably one of the more common surgical complaints of patients attending both an out-patient clinic and also as an emergency at hospital. All the organs contained in the abdomen, pelvis and retroperitoneum can be the cause of abdominal pain. However, one must not forget that acute abdominal pain can also be caused by some acute medical problems (porphyria, diabetic ketoacidosis, etc.) in addition to both acute cardiac and pulmonary disorders (*Williams et al., 2008*).

Pathway of abdominal pain

The peritoneal membrane is conveniently divided into two parts the visceral peritoneum surrounding the viscera and

the parietal peritoneum lining the other surfaces of the cavity. The peritoneum has a number of functions:

- Pain perception (parietal peritoneum)
- Visceral lubrication
- Fluid and particulate absorption
- Inflammatory and immune responses
- Fibrinolytic activity

The parietal portion is richly supplied with nerves and, when irritated cause severe pain accurately localised to the affected area. The visceral peritoneum, in contrast, is poorly supplied with nerves and its irritation causes vague pain that is usually located to the midline (*Williams et al., 2008*).

Abdominal pain can be divided into two components that correlate with the innervation of the visceral and parietal peritoneum. The visceral peritoneum is innervated by C fibers that course with the autonomic ganglia. Visceral pain is a response to injury of the organ and its adherent visceral peritoneum. Distension, stretch, traction, compression, torsion, ischemia, and inflammation of the visceral peritoneum trigger visceral pain fibers. Contrarily, abdominal organs are insensate to heat, cutting, or electrical stimulation. C fibers are unmyelinated, polymodal nociceptors that conduct slowly (0.5–5 m/s) producing a dull, crampy pain. C fibers travel bilaterally

with the sympathetic and parasympathetic chains, and will often be interpreted as vague, central abdominal pain (*Nadine et al., 2008*).

Organs proximal to the ligament of Treitz, embryologically derived from the foregut (stomach, duodenum, pancreas), will refer to the celiac chain. This is perceived as epigastric pain. Likewise, the midgut (small bowel and right colon) presents as periumbilical pain and the hindgut (transverse, left, sigmoid colon, and rectum) as hypogastric pain. Somatic pain travels via A δ somatic fibers coursing with the spinal nerves T7 through L2. A δ fibers are thinly myelinated, fast conductors that, when fired, are perceived as sharp, pricking pain. When an abdominal process inflames the parietal peritoneum, or peritonitis, the pain becomes severe and localizes to the source of inflammation. Movement or aggravation of the parietal peritoneum will exacerbate the pain (*Nadine et al., 2008*).

Of course, most causes of abdominal pain will incorporate both visceral and parietal pain, producing a picture that changes as the inflammation increases and spreads. This evolving picture may be picked up within the history-taking process or subsequently by regular and repeated review of the patient. Pain from small bowel obstruction will usually be central and colicky in nature but, as the obstructed loop becomes

ischaemic and starts to inflame the overlying peritoneum, the pain will become continuous, more widespread and be associated with signs of 'peritonitis' (*Williams et al., 2008*).

Specific characteristics of abdominal pain

The site, onset, character and duration of the abdominal pain provide important pointers to the diagnosis. Radiation of the pain, progression or alteration of its site or character, factors that aggravate the pain or relieve it and any associated symptoms are also helpful in refining the diagnosis.

1-The site of the abdominal pain

The site of abdominal pain is usually related to one of nine areas. These regions are demarcated by the mid-clavicular lines in the vertical axis and by the transpyloric and transtubercular lines in the horizontal axis (*Williams et al., 2008*).

A-The location of pain

The location of pain serves only as a rough guide to the diagnosis-"typical" descriptions are reported in only two thirds of cases. This great variability is due to atypical pain patterns, a shift of maximum intensity away from the primary site, or advanced or severe disease. In cases presenting late with diffuse peritonitis, generalized pain may completely obscure the

precipitating event. Pain confined to either upper quadrant may be evaluated by anatomic consideration of acute conditions that affect the underlying organs. Abdominal pain may be referred or may shift to sites far removed from the primarily affected organs (*Doherty, 2010*).

B-Referred pain

The perception of pain removed from the location of its source, or referred pain, can be predicted by the nerves with which the pain fibers travel. For example, appendiceal obstruction leading to its distension will present as vague, dull, referred visceral pain to the umbilicus. An inflamed gallbladder causing parietal irritation of the diaphragm will refer via the phrenic nerve as shoulder pain. The retroperitoneal genitourinary organs and pancreas share innervation with the abdominal viscera and can also present as abdominal pain. The pain may be vague from referral via the autonomic ganglia of the abdomen and pelvis, or more severe from direct irritation of the abutting parietal peritoneum (*Nadine et al., 2008*).

C- Shifting pain

Spreading or shifting pain parallels the course of the underlying condition. The site of pain at onset should be distinguished from the site at presentation. Beginning classically in the epigastric or periumbilical region, the incipient visceral

pain of acute appendicitis (due to distension of the appendix) later shifts to become sharper parietal pain localized in the right lower quadrant when the overlying peritoneum becomes directly inflamed. In perforated peptic ulcer, pain almost always begins in the epigastrium, but as the leaked gastric contents track down the right may paracolic gutter, pain descend to the right lower quadrant with even diminution of the epigastric pain (*Doherty, 2010*).

2-Mode of Onset and Progression of Pain

The mode of onset of abdominal pain may help the examiner to determine the severity of the underlying disease. Pain that has a sudden onset suggests an intra-abdominal catastrophe, such as a ruptured abdominal aortic aneurysm (AAA), a perforated viscus, or a ruptured ectopic pregnancy. Rapidly progressive pain that becomes intensely focused in a well-defined area within a period of a few minutes to an hour or two suggests a condition such as acute cholecystitis or pancreatitis. Pain that has a gradual onset over several hours, usually beginning as slight or vague discomfort and slowly progressing to steady and more localized pain, suggests a subacute process and is characteristic of processes that lead to peritoneal inflammation. Numerous disorders may be associated with this mode of onset, including acute appendicitis, diverticulitis, pelvic inflammatory disease (PID), and intestinal obstruction (*David et al., 2007*).

3-Character of pain:

The nature, severity, and periodicity of pain provide useful clues to the underlying cause. Steady pain is most common. Sharp superficial constant pain due to severe peritoneal irritation is typical of perforated ulcer or a ruptured appendix, ovarian cyst, or ectopic pregnancy. The gripping, mounting pain of small bowel obstruction (and occasionally early pancreatitis) is usually intermittent, vague, deep-seated, and crescendo at first but soon becomes sharper, unremitting, and better localized. Unlike the disquieting but bearable pain associated with bowel obstruction, pain caused by lesions occluding smaller conduits (bile ducts, uterine tubes, and ureters) rapidly becomes unbearably intense (*Doherty, 2010*).

Pain is appropriately referred to as colic if there are pain-free intervals that reflect intermittent smooth muscle contractions, as in ureteral colic. In the strict sense, the term "biliary colic" is a misnomer because biliary pain does not remit. The reason is that the gallbladder and bile duct, in contrast to the ureters and intestine, do not have peristaltic movements. The "aching discomfort" of ulcer pain, the "stabbing, breathtaking" pain of acute pancreatitis and mesenteric infarction, and the "searing" pain of ruptured aortic aneurysm remain apt descriptions. Despite the use of such descriptive terms, the quality of visceral pain is not a reliable clue to its cause (*Doherty, 2010*).

4- Aggravating features of pain

Obtaining information on aggravating or relieving factors can be particularly helpful to the assessing surgeon. Pain made worse by moving and coughing suggests peritoneal inflammation 'peritonism', whereas pain which makes the patient roll around or double up is typical of 'colic'. Factors that aggravate or relieve pain are also important in making a diagnosis, and information on the influence of movement, position, food, antacids, vomiting, bowel action and micturition on the pain must always be sought. A history of previous trauma, however minor, may also be important. At the same time, it is important to ask about associated symptoms such as vomiting, diarrhoea, dysuria or a missed period that preceded or followed the onset of the pain as these may again provide important diagnostic clues (*Williams et al., 2008*).

Differential diagnosis of acute abdomen

- Surgical causes of acute Abdomen
- Haemorrhage
 - Solid organ trauma
 - Leaking or ruptured arterial aneurysm
 - Ruptured ectopic pregnancy
 - Intestinal ulceration

- Hemorrhagic pancreatitis
- Bleeding gastrointestinal diverticulum
- Spontaneous rupture of spleen
- Mallory- Weiss syndrome
- Arteriovenous malformation of gastrointestinal tract
- Aortoduodenal fistula after aortic vascular graft

- **Infection**

- Appendicitis
- Cholecystitis
- Meckel's diverticulitis
- Hepatic abscess
- Diverticular abscess
- Psoas abscess

- **Perforation**

- Perforated gastrointestinal ulcer
- Perforated gastrointestinal cancer
- Boerhaave's syndrome
- Perforated diverticulum

- **Obstruction**

- Adhesion related small or large bowel obstruction
- Sigmoid volvulus

- Cecal volvulus
- Incarcerated hernias
- Inflammatory bowel disease
- Gastrointestinal malignancy
- Intussusception
- **Ischemia**
 - Strangulated hernias
 - Mesenteric thrombosis or embolism
 - Ovarian torsion
 - Testicular torsion
 - Ischemic colitis

2- Nonsurgical Causes of Acute Abdomen

- **Endocrine and Metabolic Causes**
 - Uremia
 - Diabetic crisis
 - Addisonian crisis
 - Acute intermittent porphyria
 - Hereditary Mediterranean fever
- **Hematologic Causes**
 - Sickle cell crisis

- Acute leukemia
- Other blood dyscrasias
- **Toxins and Drugs**
 - Lead poisoning
 - Other heavy metal poisoning
 - Narcotic withdrawal
 - Black widow spider poisoning

(Russell et al., 2008)

Pathophysiology of peritonitis as a common cause of surgical acute abdomen

Introduction of bacteria or irritating chemicals into the peritoneal cavity can cause an outpouring of fluid from the peritoneal membrane. The peritoneum responds to inflammation by increased blood flow, increased permeability, and the formation of a fibrinous exudate on its surface. The bowel also develops local or generalized paralysis. The fibrinous surface and decreased intestinal movement cause adherence between the bowel and omentum or abdominal wall and help to localize inflammation. As a result, an abscess may produce sharply localized pain with normal bowel sounds and gastrointestinal function, whereas a diffuse process, such as a perforated duodenal ulcer,

produces generalized abdominal pain with a quiet abdomen. Peritonitis may affect the entire abdominal cavity or a portion of the visceral or parietal peritoneum (*Russell et al., 2008*).

Peritonitis is peritoneal inflammation from any cause. It is usually recognized on physical examination by severe tenderness to palpation, with or without rebound tenderness, and guarding. Peritonitis is usually secondary to an inflammatory insult, most often gram-negative infections with enteric organisms or anaerobes. It can result from noninfectious inflammation, a common example being pancreatitis (*Russell et al., 2008*).

Types of peritonitis

- **Primary peritonitis**

Occurs more commonly in children and is most often due to Pneumococcus or hemolytic Streptococcus species infection, Adults with end-stage renal disease on peritoneal dialysis can develop infections of their peritoneal fluid, with the most common organisms being gram-positive cocci. Adults with ascites and cirrhosis can develop primary peritonitis, and in these cases, the organisms are usually Escherichia coli and Klebsiella species (*Russell et al., 2008*).

- **Secondary peritonitis**