

Introduction

Acute abdomen may be defined generally as an intra-abdominal process causing severe pain and often requiring surgical intervention. It is a condition that requires a fairly immediate judgment.¹

Acute abdomen in pregnancy remains one of the most challenging diagnostic and therapeutic dilemmas today. The incidence of acute abdomen during pregnancy is 1 in 500–635 pregnancies. Despite advancements in medical technology, preoperative diagnosis of acute abdominal conditions is still inaccurate. Laboratory parameters are not specific and often altered as a physiologic consequence of pregnancy. Use of laparoscopic procedures as diagnostic tools makes diagnosis of such conditions earlier, more accurate, and safer.²

The most frequent intra-abdominal disorders encountered during pregnancy are; acute appendicitis, gallbladder diseases, bowel obstruction and perforation, persistent ovarian cysts and twisted adnexal masses.^{3, 4, 5}

Acute abdomen in pregnancy due to nonobstetric causes is accompanied by a high incidence of poor fetal outcome and maternal morbidity. Delay in surgical intervention is the main cause of the poor fetal outcome. Fear of the complication of a negative laparotomy in a

pregnant female makes surgeons wait clear cut symptoms and signs of acute abdomen. In pregnancy, these symptoms and signs are blunted by the anatomical displacement of the pregnant uterus and the masking effect of the physiological symptoms of normal pregnancy.

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The diagnosis of acute abdomen tends to be delayed due to the restrictions imposed on image diagnostic techniques such as x- ray and CT. Surgeons should pay attention in this regard. ⁷

Emergency laparoscopy is the laparoscopic operation which should be performed without any delay in life threatening situations. Gynecologists were the first to start laparoscopy in the diagnosis and treatment; but since 1990 a lot of general surgeons have started to use this technique in abdominal urgency.⁸

Laparoscopic surgery should be performed in the second trimester when possible and appears as safe as laparotomy.⁹

The advantages of laparoscopic procedures over open procedures in the general population are well described. These include reduced post-operative pain, shorter length of hospital stay, a decreased incidence of thromboembolic events, faster recovery, improved cosmesis, and decreased rate of postoperative ileus.¹⁰

The entire peritoneal cavity can be visualized by the laparoscope, and diagnostic laparoscopy is an effective modality for determining pathology within the abdominal cavity. The decision to perform diagnostic laparoscopy is based on clinical judgment, weighing the sensitivities and specificities of other modalities as computed tomography (CT scan) and ultrasound versus the relative morbidity of minimally invasive laparoscopy.¹¹

For these reasons, laparoscopy has become the preferred method for many surgical procedures, including cholecystectomy and appendectomy. Despite this, some surgeons are reluctant to employ this technique when treating a pregnant patient with appendicitis or biliary disease, as there are few data showing the safety of laparoscopy for general surgical procedures during pregnancy.¹²

Aim of the Work

The aim of the study is to evaluate the role of laparoscopy in diagnosis and management of acute abdomen in pregnancy.

Anatomical and Physiological Changes During Pregnancy

During normal pregnancy, virtually every system undergoes anatomical and functional changes that can alter appreciably criteria for diagnosis and treatment of diseases. Thus, understanding of these adaptations to pregnancy remains a major goal of obstetrics, and without such knowledge, it is almost impossible to understand the disease processes that can threaten women during pregnancy.¹³

Certain anatomic and physiologic changes specific to pregnancy may make the cause of the pain difficult to ascertain. As the gravid uterus enlarges, it becomes an abdominal organ at around 12 weeks' gestation and compresses the underlying abdominal viscera. This enlargement may make it difficult to localize the pain and may also mask or delay peritoneal signs.¹⁴

Abdominal assessment during pregnancy is modified by displacement of abdominal viscera by the expanding gravid uterus. For example, the location of maximal abdominal pain and tenderness from acute appendicitis migrates superiorly and laterally as the appendix is displaced by the growing gravid uterus. A rigid abdomen with rebound tenderness remains a valid indicator of peritonitis during pregnancy, but abdominal wall laxity in late pregnancy might mask the classic signs

of peritonitis. An abdominal mass may be missed on physical examination because of the presence of the enlarged gravid uterus.¹⁵

A decrease in lower esophageal sphincter pressure leads to heartburn, gastro-esophageal reflux, and even stricture formation. Delayed gastric emptying can lead to increased gastric residual volume, and possibly to nausea and vomiting. The slow colonic transit time may lead to constipation and, subsequently, pain.¹⁴

Other physiologic changes may affect clinical presentation of abdominal pain in pregnancy. Increased progesterone increases respiratory drive; total minute ventilation increases because of a larger tidal volume while respiratory rate is unchanged. Chest films frequently show an increased cardiothoracic ratio largely due to the gravid uterus displacement of the diaphragm. This results in an overall decrease in functional residual capacity. These changes result in an increase in P_{O_2} and a decrease in P_{CO_2} , resulting in a mild respiratory alkalosis.¹⁴

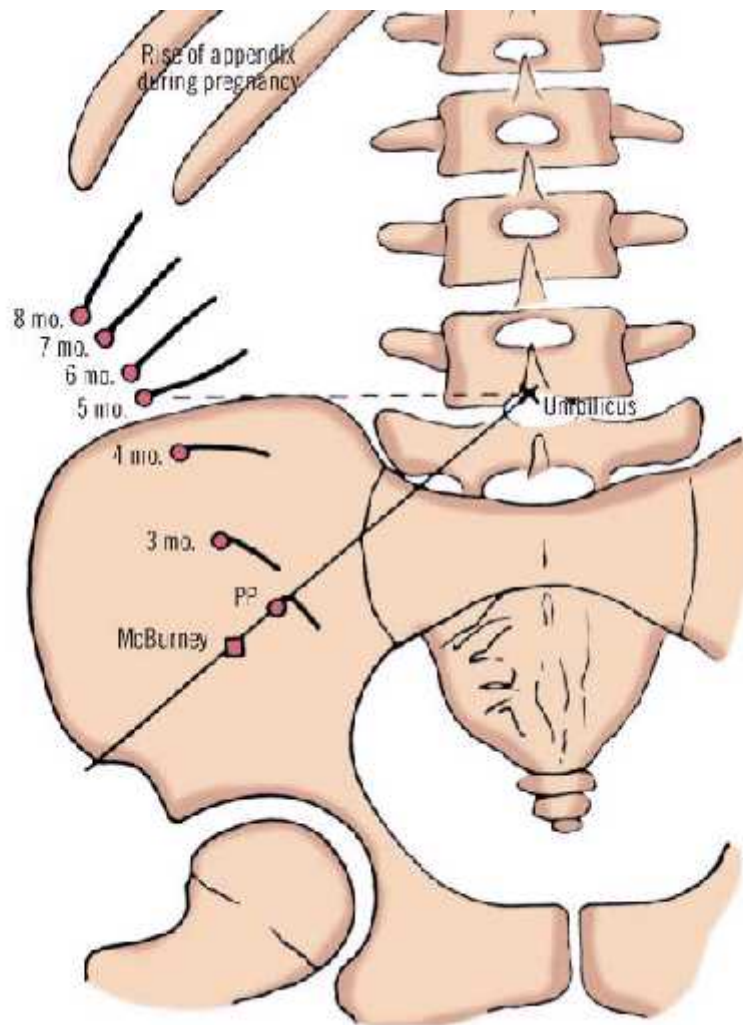


Figure (1): Changes in position and direction of appendix during pregnancy.¹⁶

1. Reproductive Tract

Uterus

In the nonpregnant woman, the uterus is an almost-solid structure weighing about 70 g and with a cavity of 10 mL or less.

During pregnancy, the uterus is transformed into a relatively thin-walled muscular organ of sufficient capacity to accommodate the fetus, placenta, and amniotic fluid. The total volume of the contents at term averages about 5L but may be 20 L or more. By the end of pregnancy, the uterus has achieved a capacity that is 500 to 100 times greater than in the nonpregnant state. The corresponding increase in uterine weight is such that, by term, the organ weighs approximately 1100 g.¹³

Uterine hypertrophy early in pregnancy probably is stimulated by the action of estrogen and perhaps that of progesterone. It is apparent that hypertrophy of early pregnancy does not occur entirely in response to mechanical distention by the products of conception, because similar uterine changes are observed with ectopic pregnancy.¹³

Access to the peritoneal cavity must be based on the size of the uterus. Some investigators suggest the use of Hasson's trochar, although others feel comfortable with Veres needle insufflation. A surgeon experienced in

laparoscopic surgery is required. In general, when planning the procedure, an open laparoscopic procedure using Hasson's trochar and a more upward placement of the laparoscopic camera to a supraumbilical location appears logical, as there has been a report of Veres needle placement into the amniotic cavity with insufflation at 21 weeks with subsequent fetal loss. Otherwise, insufflation and camera placement in the midclavicular line, 1 to 2 cm inferior to the costal margin may be considered. The goal is to avoid the gravid uterus and to limit pneumoperitoneal pressure to no more than 12 to 15 mmHg in an attempt to decrease the likelihood of fetal acidosis.¹⁴

Keeping the intra-peritoneal pressure at 12 to 15 mmHg may preclude adequate visualization, especially in the obese patient or those with adhesive disease from prior surgery, and must be kept in mind when planning surgery. After insufflation is performed, the placement of other trocars depends on the procedure and the size of the gravid uterus.¹⁴

Uterine Size, Shape, and Position

For the first few weeks, the uterus maintains its original pear shape, but as pregnancy advances, the corpus and fundus assume a more globular form, becoming almost spherical by 12 weeks. Subsequently, the organ increases more rapidly in length than in width

and assumes an ovoid shape. By the end of 12 weeks, the uterus has become too large to remain entirely within the pelvis. As the uterus continues to enlarge, it contacts the anterior abdominal wall, displaces the intestines laterally and superiorly, and continues to rise, ultimately reaching almost to the liver. With ascent of the uterus from the pelvis, it usually undergoes rotation to the right. This dextrorotation likely is caused by the rectosigmoid on the left side of the pelvis.

As the uterus rises, tension is exerted on the broad and round ligaments with the pregnant woman standing; the longitudinal axis of the uterus corresponds to an extension of the axis of the pelvic inlet. The abdominal wall supports the uterus and unless it is quite relaxed, maintains this relation between the long axis of the uterus and the axis of the pelvic inlet. When the pregnant woman is supine, the uterus falls back to rest on the vertebral column and the adjacent great vessels, especially the inferior vena cava and aorta.¹³

Cervix

The cervical glands undergo such marked proliferation that by the end of pregnancy they occupy approximately half of the entire cervical mass, rather than a small fraction as in the nonpregnant state. These normal pregnancy-induced changes represent an extension, or eversion, of the proliferating columnar

endocervical glands. This tissue tends to be red and velvety and bleeds even with minor trauma, such as with Pap smear sampling.¹³

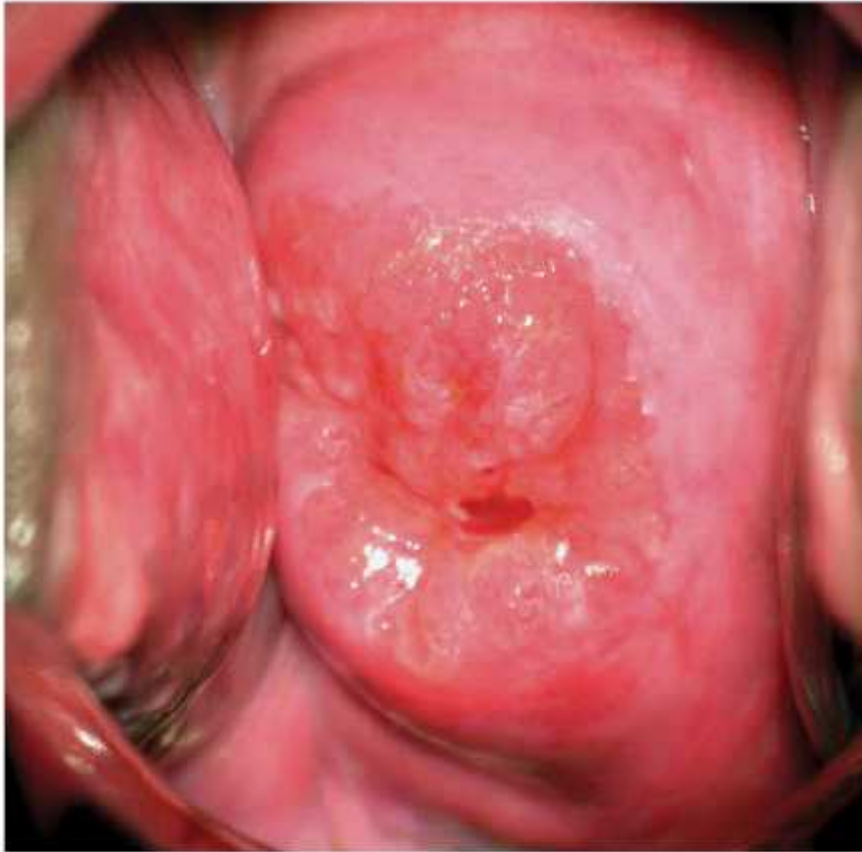


Figure (2): Cervical changes during pregnancy. ¹³

2. Abdominal Wall

During pregnancy reddish, slightly depressed streaks commonly develop in the abdominal skin and sometimes in the skin over the breasts and thighs. These

are called striae gravidarum or stretch marks. In multiparous women, in addition to the reddish striae of the present pregnancy, glistening, silvery lines that represent the cicatrices of previous striae frequently are seen.¹³

Occasionally, the muscles of the abdominal walls do not withstand the tension to which they are subjected. As a result, rectus muscles separate in the midline, creating a diastasis recti of varying extent. If severe, a considerable portion of the anterior uterine wall is covered by only a layer of skin, attenuated fascia, and peritoneum. True fascial defects lead to ventral hernia, which uncommonly require antepartum surgical correction.¹³

3. Respiratory System

Under the influence of progesterone, there is an early (first trimester) 25% increase in alveolar minute ventilation (MV) caused by increases in both respiratory rate (15%) and tidal volume (40%). At full term, MV increases 45% to 70% higher than nonpregnant values. This produces a slight chronic respiratory alkalosis ($P_{aCO_2} = 28-32$ mmHg; $pH = 7.44$), which shifts the maternal oxyhemoglobin dissociation curve to the right, promoting oxygen delivery to fetus. The increase in the arterial pH level is limited by an increase in renal bicarbonate excretion. Despite the expected pregnancy

induced increase in metabolic demand, P_{aO_2} remains normal or increases slightly during pregnancy.¹⁷

Gravid uteri exert a restrictive effect on respiratory mechanics, with a reduction in functional residual capacity of 20% at term.¹⁷

Airway management in pregnancy is further complicated by the anatomical changes related to both weight gain and edema of the upper airway and vocal cords, which occurs secondary to a generalized increase in capillary permeability, so there is a significant risk of failed intubation.¹⁷

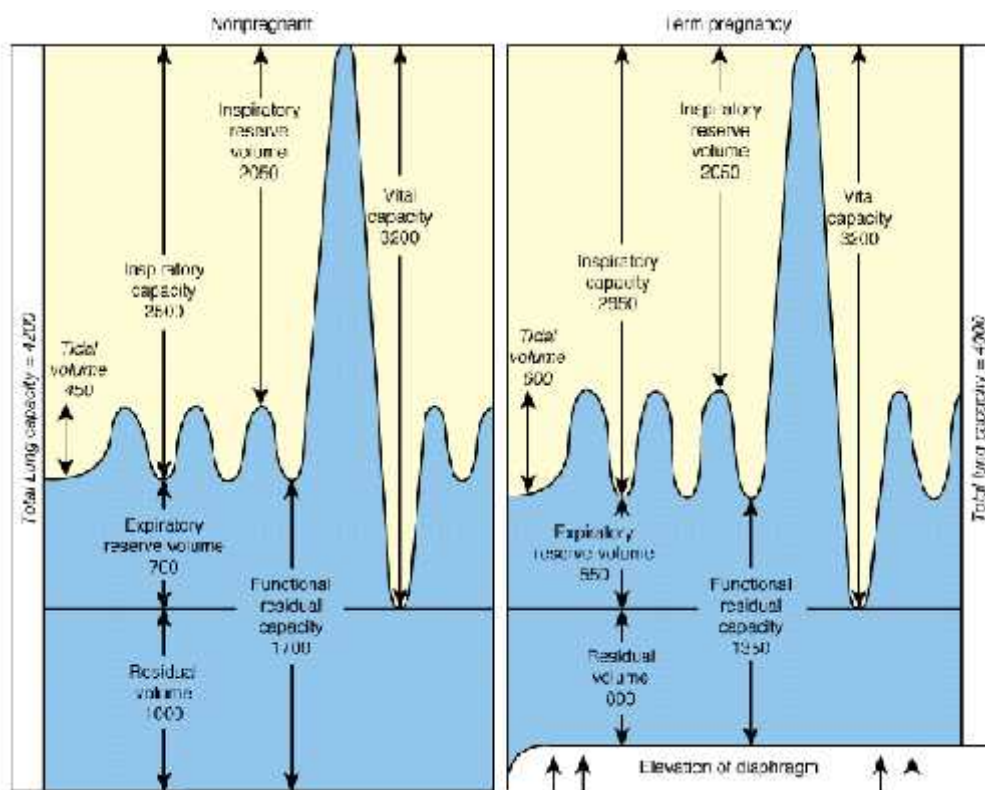


Figure (3): Respiratory changes during pregnancy.¹³

4. Cardiovascular System

Cardiac output begins to increase early in the first trimester and peaks in the second trimester (up to 50% higher than the baseline value), at which time heart rate has increased by 25% and stroke volume has by 30%.¹⁷

The increased metabolic demands of fetus and the presence of placenta as a low-pressure system in parallel with systemic circulation necessitate this increase in cardiac output. Systemic and pulmonary vascular resistances decrease in response to increased synthesis of vasodilators such as prostacyclin.¹⁷

Blood pressure undergoes minor changes during pregnancy, falling slightly in the first trimester, rising in the second, and approximating its prepregnancy level in the third. At 12 weeks' gestation, the uterus rises out of the pelvis to encroach upon the abdominal viscera. Aortocaval compression in the supine position becomes clinically relevant.¹⁷

Supine hypotensive syndrome is associated with cardiac output reductions of up to 20%. Inferior vena caval and aortic compression are demonstrable in the supine position and require left lateral tilts of 15° and 30°, respectively, to reliably restore adequate circulation.¹⁷

5. Hematological System

Blood volume expansion occurs in the first trimester and increases by 35% to 50% at term. The greater increase in plasma volume relative to red cell mass leads to dilutional anemia. This serves a protective physiologic function at birth where lost blood is relatively hemoglobin poor but leads to early compromise of oxygen-carrying capacity in acutely hemorrhaging patients. The reduction in blood viscosity improves flow through the uteroplacental circulation.¹⁷

A benign leukocytosis up to 15000 mm³ during pregnancy and that up to 20000 mm³ during labor are commonly seen and can confound diagnosis of systemic infection.¹⁷

6. Coagulation System

Pregnancy is a procoagulable state with alterations in both coagulation and fibrinolysis. The changes in the coagulation system during pregnancy appear to be aimed at minimizing blood loss at delivery. Unfortunately, these changes also predispose to thromboembolism, particularly in those with additional risk factors.¹⁸

In terms of absolute risk, pregnancy is associated with a four-to-six-fold increase in venous thromboembolism compared with non-pregnant age-matched controls.¹⁸
