



Case Report

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Spontaneous Bacterial Peritonitis during Pregnancy: A Rare Occurrence

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Abstract

Acute abdomen during pregnancy is not an uncommon emergency in obstetrical practice. But dilemma occurs when no cause is found despite thorough workup and investigations, even on laparotomy. Peritonitis though may occur during pregnancy following tubal rupture in ectopic pregnancy or other intra abdominal conditions but spontaneous bacterial peritonitis is rarest of rare occurrence during pregnancy and that too with staphylococci. Only 2-4% cases of spontaneous bacterial peritonitis with staphylococcus aureus have been mentioned in literature. In a rare case we came across, a primigravidae who presented with pain abdomen, fever and ascites which later on diagnosed to be a case of spontaneous bacterial peritonitis and the microorganism isolated was found to be staphylococcus aureus. We are reporting this case because of rare occurrence of this entity in pregnancy and the paucity of literature.

Keywords: Peritonitis; Ascites; Primary bacterial peritonitis; Staphylococci

Introduction

Microbial contamination of the peritoneal cavity is termed as peritonitis or intra-abdominal infection, and is classified according to etiology [1]. Peritonitis has been categorized as primary, secondary, or (more recently) tertiary. Peritonitis complicating peritoneal dialysis can be considered as an additional category [2]. Acute peritonitis may be classified as primary (spontaneous), where an infection has arisen *de novo* within the peritoneum, or secondary, where the inflammatory process involving the peritoneum is the result of an identifiable primary process [3]. Bacterial peritonitis is usually polymicrobial, both aerobic and anaerobic organisms being present. The exception is primary peritonitis ('spontaneous' peritonitis), in which a pure infection with streptococcal, pneumococcal or *Haemophilus* bacteria occurs. Infecting organisms may reach the peritoneal cavity via a number of routes viz gastrointestinal perforation e.g. perforated ulcer, diverticular perforation; exogenous contamination e.g. drains, open surgery, trauma; transmural bacterial translocation (no perforation) e.g. inflammatory bowel disease, appendicitis, ischaemic bowel; female genital tract infection e.g. pelvic inflammatory disease; haematogenous spread (rare) e.g. septicaemia [4]. Although the route of infection in primary peritonitis is usually not apparent it is thought to be hematogenous, lymphogenous, transmural migration through an intact gut wall from the intestinal lumen or, in women, from the vagina or via the fallopian tubes [5] and

rarely require surgical intervention while the effective therapy for secondary peritonitis requires source control to resect or repair the diseased organ; debridement of necrotic, infected tissue and debris; and administration of antimicrobial agents directed against aerobes and anaerobes [1]. We are presenting here a rarest of rare case of spontaneous bacterial peritonitis with staphylococcal infection in a primigravida at 26 weeks with successful pregnancy outcome.

Case Report

A 21 years old primigravida reported to labour room at Kamla Nehru Hospital, Shimla at 26+6 weeks with complaints of pain abdomen and fever for 3-4 days. She was admitted for similar complaints at local practitioner where USG abdomen and pelvis revealed as cistae with thick septations for which she was referred to our hospital. There was no significant past and family history. On examination, vitals were stable. Height of uterus corresponded to 26 weeks with tense and distended abdomen. Fetal heart sound could not be localised. Laboratory investigations revealed hemoglobin 9.5g/dl, total lymphocyte count 6800/uL with differential lymphocyte count $N_{79}L_{20}M_0E_0$, ESR-65mm, renal and liver function tests were within normal limits; total serum proteins were 6.9 g/dl with serum albumin 3.2g/dl and blood sugar was normal. USG abdomen and pelvis showed normal liver and gall bladder, moderate maternal ascites

with thick septations, single live fetus with gestational age 26+4 weeks with severe oligohydramnios. She was started on antibiotics, received two doses betamethasone and nifedipine tocolysis. USG guided diagnostic ascitic tap was done at 27+2 weeks. Microscopic examination revealed field full of pus cells and gram staining showed predominant polymorphonuclear cells with gram positive cocci (*Staphylococci*) in groups and clusters. Culture was sterile. Glucose-1mg/dl, proteins-4.9g/dl (exudate), LDH->20000U/l, ADA-323u/l, mycobacterium tuberculosis PCR negative. Repeat USG at 27+6 weeks suggested massive multiloculated septated collection in abdomen occupying whole of bilateral lumbar regions and epigastric region. Decision for exploratory laparotomy was taken. Intraoperatively, 1.5-2 litres multiseptated collection of thick non foul smelling pus was drained. The uterus was 28 weeks size. Bilateral ovaries could not be visualised. Pouch of douglas was completely obliterated. Peritoneal toileting was done after breaking all loculi. Abdomen was inspected carefully but primary source of peritonitis could not be identified. The post operative period was uneventful. She received antibiotics and tocolysis. Pus culture was sterile. She went into preterm labour at 30 weeks and had preterm vaginal delivery of a female child 1.25kg. Both mother and baby were discharged under satisfactory conditions.

Discussion

Idiopathic peritonitis is un common, constituting about 1 per cent of all cases of peritonitis and occurs when no obvious source for the peritoneal infection can be demonstrated. It is a diagnosis by exclusion and is confirmed in retrospect when the results of blood cultures or peritoneal swabs become available. Formerly, pneumococci were implicated, but in recent years haemolytic streptococci, *Escherichia coli*, and *Klebsiella spp.* are more frequently cultured. It was classically described in young girls where the port of entry was presumed to be through the fallopian tubes. Adult primary peritonitis arises via haematogenous spread or translocation of bacteria through the bowel wall, especially in the presence of exogenous (e.g. steroid therapy) or endogenous (intercurrent disease) immunosuppression [3]. Spontaneous bacterial peritonitis (SBP) is a known complication of ascites due to cirrhosis; it has also been reported in some non-cirrhotic conditions with ascites [6]. Spontaneous bacterial peritonitis is thought to occur as a result of prolonged bacteraemia due to impaired host-defense mechanisms and decreased bactericidal activity in the ascitic fluid. Bactericidal activity parallels the total protein concentration in the fluid [7]. Spontaneous bacterial

peritonitis can vary in its presentation from being clinically dramatic to totally asymptomatic [3]. Fever of about 38°C (100 °F) is the most common presenting feature and occurs in 50 to 80 per cent of patients [2,3,5]. Abdominal pain, usually diffuse, occurs in 25 to 72 per cent of patients; rebound tenderness is elicited in over 50 per cent of patients. However it can occur in the absence of abdominal pain or fever [3]. A diagnostic abdominal paracentesis essential [3,8]. Ascitic fluid absolute white blood cell count lower than 250 polymorphonuclear cells/mm³ usually denotes uninfected ascites, while counts above this suggest infection, but a count above 500/mm³ establishes with more than 80 per cent certainty the diagnosis of spontaneous bacterial peritonitis [3]. Pelvic infection via the fallopian tubes is responsible for a high proportion of 'non-gastrointestinal' causes of peritonitis. Idiopathic streptococcal and staphylococcal peritonitis in adults is fortunately rare. In streptococcal peritonitis, the peritoneal exudate is odourless and thin, contains some flecks of fibrin and may be blood-stained [4]. *Staphylococcus aureus* is an unusual cause of primary peritonitis, accounting for only 2-4% of cases [5]. The use of intravaginal tampons has led to an increased incidence of *Staphylococcus aureus* infections: these can be associated with 'toxic shock syndrome' and disseminated intravascular coagulopathy [4].

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