

BAOJ Surgery

Alalayet Yasen F, et al. BAOJ Surgery 2015, 1:1

1:004

Case report

Early Diagnosis of Acute appendicitis in neonates

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Abstract

Here we are presenting two cases of perforated appendicitis occurred in neonatal Age. Although it is very rare but we should consider it in the differential diagnosis for neonatal peritonitis because it is completely curable if appendectomy done at proper time.

Primary type occurs without associated disease and it is due to obstruction in the lumen by band or kinking of appendix itself.

Any delay in the diagnosis or operation time leads to high mortality rate exceeding 70%.

Laparotomy should be considered even with early symptoms and signs of Neonatal peritonitis.

Key words: Neonatal appendicitis-neonatal peritonitis

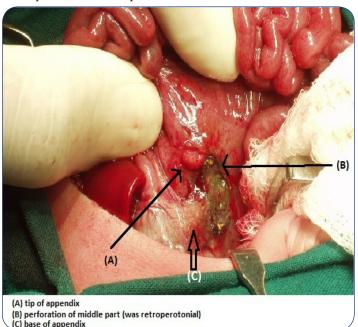
Introduction

Case I

A premature baby boy 35-weeks of gestation, a product of Normal vaginal delivery with birth weight of 2050 gm. routine antenatal ultrasound done at 32- weeks of gestation showed mild fetal ascites, bowel distention and absence of peristalsis without other anomalies. Another ultrasound done at 34-weeks showed that these signs become less prominent and peristalsis reappeared. This baby referred to us at one-day old with greenish vomiting and abdominal distention. Clinical examination revealed mild abdominal distention, mild right flank edema, mild generalized abdominal tenderness and bile stained aspirate coming from nasogastric tube (NGT). Routine blood investigations were within normal limits. Abdominal X-ray showed mild dilated bowel loop especially in right lower quadrant. abdominal ultrasound showed edematous bowel loop in right lower quadrant with posterior abdominal wall edema. The patient diagnosed to have meconium peritonitis, so exploration laparotomy done.

Intra-operatively there was edematous retroperitoneal space with fine calcification deposits and edematous cecum. The middle part of the appendix was retroperitoneal but the tip and the base of the appendix was intra-peritoneal. There was perforation in the middle part (Fig 1), appendectomy done. Histopathology confirmed acute appendicitis as all layers of appendix was inflamed (in addition to presence of ganglion cell in it, so secondary perforation due to Hirschsprung disease is excluded). Retroperitoneal biopsy

shows pigment containing macrophages with micro-calcifications. Feeding started post-operatively on day three and discharged on day fife with final diagnosis of fetal acute appendicitis with inutero perforation. Follow up with ultrasound done at 24 months to check persistent of retroperitoneal calcification, but it wasn't.



Case II

A 19 days old premature baby girl delivered at 29-weeks of gestation with birth weight of 1340 gm. Fetal abdominal ultrasound was

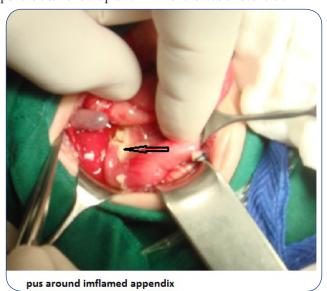
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Rec Date: August 3, 2015, **Acc Date:** August 31, 2015, **Pub Date:** September 1, 2015.

Citation: Yasen FA, Mohamed M, Abdulrahman YA, Nabil S, Abeer SA, Khaled MHA and Fares A (2015) Early Diagnosis of Acute appendicitis in neonates. BAOJ Surgery 1: 004.

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normal.(last one done at 28 weeks). Patient was clinically well on room air, oral feeding and passing stool normally. On day 18 of life, patient becomes irritable, restless and refusing feeding. On day 19, patient developed severe abdominal distention with apnea and intubation & ventilation were required. There was Yellowish secretion came out from NGT. Abdominal examination showed mild distention and Shiny abdominal wall with dilated vessels, erythema in right lower quadrant with generalized abdominal tenderness. On investigations, White blood cell was 14500 ,C reactive protein (CRP 130 mg/L), Abdominal x-ray showed mildly dilated bowel loop without evidence of pneumatosis intestinalis, peritoneal tap revealed turbid yellowish fluid and microscopic examination was positive for White blood cell (polymorph & lymphocyte) with no meconium debris, Red blood cell or bacteria. Abdominal ultrasound detected minor sub hepatic collection and edematous bowel loop in right lower quadrant. The diagnosis was neonatal peritonitis; with suspicion of Necrotizing enterocolitis and sealed perforation, so exploration laparotomy done and revealed yellowish peritoneal fluids with small puss collection around inflamed appendix (Fig2). There were no singes of Necrotizing enterocolitis or Hirschsprung disease or other bowel abnormality, appendectomy done. Patient extubated on 2nd day post op, feeding started on 3rd day. Sweat test also done and it was within normal. Histopathology confirmed acute appendicitis. Pus culture showed no bacterial growth. Patient discharged home at weight of 1800 gram with diagnosis of primary neonatal acute appendicitis. Follow up until 12 months was uneventful.



Discussion

Neonatal Acute appendicitis (NAA) is very rare clinical entity. A survey of literature review showed 150 cases of NAA from 1901 until 2015, out of it only 35 cases primary (true) neonatal appendicitis without associated disease. Preterm and male neonates are commonly affected [1]. The rarity of acute neonatal appendicitis is attributed to the fetal form of the appendix; wide base with conical shape therefore it does not obstructed easily, low-

residue milk diet and decreased incidence of lymphoid hyperplasia. Clinical Features of neonatal acute appendicitis are non-specific, e.g. refuse feeding, vomiting, abdominal distension, Lethargy, irritability, constant crying, abdominal tenderness, respiratory distress and hypoperistalsis [2,3]. Leukocytosis also seen. These non-specific clinical features result in delay in the diagnosis and predisposes to perforation in 70-82% of neonatal appendicitis [4]. Primary (true) appendicitis occurred without associated disease and the secondary one may represent as a complication of underlying disease, i.e., Hirschsprung's disease [5], meconium plug syndrome and cystic fibrosis [6], necrotizing enterocolitis [7] Or with incarcerated inguinal hernia, that called "Amyand's hernia" (the inflamed appendix located within the inguinal hernia sac) [8]. The intra-hernia presentation is about one third of the cases and the abdominal presentation is about two thirds of the cases [9]. Neonatal appendicitis can mimic necrotizing enterocolitis [5] and the diagnosis is often late. Prognosis is uniformly poor with mortality rate 33%-78% [4], the high mortality rate has been attributed to difficulty and delay in establishing an early diagnosis. Also immature immune system, inelastic thin wall appendix [6] and insufficient size of the omentum [7] predisposes to perforation and diffuse peritonitis. The prognosis in scrotal appendicitis in neonates is better than that of intra-abdominal appendicitis. This is due to early presentation and diagnosis of inflammation.

Perforated appendicitis can occur during intrauterine life [10]. Fetal peritonitis can be diagnosed by ultrasound in the presence of ascites, dilated bowel [11], debris, clustering of hyperechogenic intestines, and absence of peristalsis. Also by cytological analysis of peritoneal fluid obtained by fetal paracentesis, showing polynuclear leukocytes, lymphocytes and macrophages containing Intracytoplasmic brownish sediment (meconium). The main types of fetal peritonitis are Chemical (meconium) and infectious (viral; cytomegalovirus, parvovirus or fungal; mucormycosis [12]. Meconioum peritonitis can occur as early as 13 weeks of gestation, calcification may occurs within 24 hours to 8 days. Rarely fetal ascites may regress, intestinal dilatation disappears, and peristalsis reappears. In some cases, fetal ascites could be partially reduced on paracentesis or recently by fetal intra peritoneal injection of urinary trypsin inhibitor, a physiological anti-inflammatory substance, to reduce meconium-induced chemical peritonitis, [13] finally early recognition and intervention improve the survival rate [14].

Conclusion

Moderate Symptoms and signs of neonatal peritonitis with High index of suspicion for neonatal appendicitis can lead to early diagnosis and more timely surgical intervention to reduce the mortality & morbidity. Primary Acute appendicitis can occur not only in neonatal period but also during intrauterine life.

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