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Acute Cholecystitis in Children, Report of Three Cases.

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Abstract:

Acute cholecystitis is rare in pediatric age group and usually is not diagnosed. Recent reports of pediatric acute cholecystitis are increasing which can be because of more clinical suspiciousness or increased long term TPN usage. This article reports a three year old boy, a six year old girl and a seven year old girl with acute cholecystitis which referred to primary care because of chills, fever, vomiting and generalized abdominal tenderness. With impression of acute peritonitis, these patients were operated and acute acalculous cholecystitis was the diagnosis. Culture of billiary secretions of them was positive for Salmonella Typhi.

Key Words: Acute cholecystitis, children.

Introduction:

Cholecystitis is a inflammatory disease of gall bladder which can be seen in acute or chronic forms, and each can be either with or without gall stone. Incidence of cholecystitis in children is reported to be from less than 1% to 4% in different studies. It is more prevalent in girls and is seen in whites twice as black people. Although cholecystitis is reported in all age groups, it is very unusual in pediatric age group.

In acute form, abdominal pain is the most common symptom which is usually located in right upper quadrant and epigastric area. Other symptoms include nausea, vomiting, chills, fever and jaundice. In physical examination, the most common finding is right upper quadrant tenderness. Other signs include abdominal distension, guarding, rebound tenderness and mildly enlarged and tender liver. In some children, palpable mass is present.

Laboratory data are not usually diagnostic, however, radiologic studies such as plain abdominal films (visualization of radioopaque stones), ultrasound and radioscan are very useful in diagnosis. Treatment of acute cholecystitis in children is the same as adults.

Case Presentation:

1- A three year old boy was referred to clinic due to abdominal pain for four days. The pain was initially around umbilicus and then was generalized. The pain was colicky and aggravated with food and not radiated to any specific site. Anorexia and vomiting has occurred after the pain appearance. Similar pains were present sometimes during the past two months.

In physical examination, the boy was lethargic with a distended abdomen. Decreased bowel sounds and generalized tenderness of abdomen was present. Rest of the physical exam including digital rectal examination was normal. Vital signs and laboratory data is abstracted in table1. Abdominal X ray was normal and abdominal ultrasound was not available.

With impression of acute peritonitis, midline laparatomy was performed. There was bile in peritoneal cavity, liver and choleductal duct were normal in size and shape, gall bladder was inflamed and distended without perforation. Stomach, appendix and small and large bowels were normal. Finally, peritoneal fluid was send for smear and culture, peritoneal irrigation, appendectomy and cholecystectomy were performed. Histologic diagnosis was acute cholecystitis.

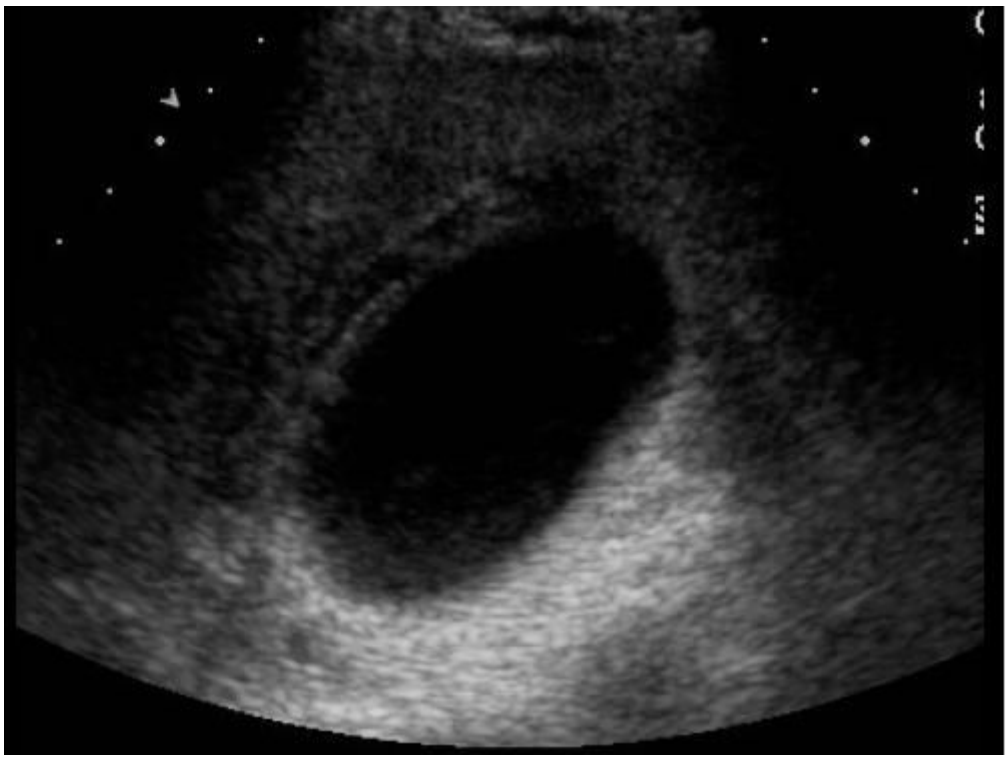
2- A six year old girl was brought to the clinic because of abdominal pain, chills and fever. The symptoms occurred since 10 days ago, starting with abdominal pain and then fever. The pain was initially in upper abdomen, colicky, on and off, without radiating and gradually become constant and aggravated with eating. Finally she developed chills and fever, anorexia, nausea and vomiting. In the course of present illness, she had received medication for acid peptic disease and parasitic infestations with no improvement. She has no similar history in the past. Physical examination revealed letharginess, abdominal distension, decreased bowel sounds, tenderness and rebound. Rest of the physical examination was within normal limits. Vital signs and laboratory findings are summarized in table 1. Plain abdomen radiography was normal and ultrasound not available. Patient was admitted to the hospital with impression of acute appendicitis. After NG Tube and Foley catheter insertion, antibiotic administration and circulatory resuscitation, the abdomen was incised in midline. There was no fluid in the peritoneal cavity. Liver, choleductal duct, stomach, small intestine, appendix, large bowel and contents of pelvis were completely normal. Gall bladder was distended and inflamed with thickened wall and with no stone in. The diagnosis was acute acalculous cholecystitis. Sample from peritoneal fluid taken, cholecystectomy and appendectomy performed. Pathology confirmed the diagnosis.

3- A seven year old girl was referred to the primary care clinic due to severe colicky abdominal pain, chills, fever and vomiting since three days ago. The pain was constant (not colicky), not radiating and started from periubmlical area and then localized to the right upper quadrant and aggravated with eating. It was associated with anorexia, chills and fever. Physical examination revealed an acutely ill patient with letharginess, abdominal distension, decreased bowel sounds, tenderness and rebound . Rest of the physical exam was within normal limits. Vital signs and laboratory data is shown in table 1. Abdominal X ray was normal and ultrasound was compatible with acute cholecystitis (fig. 1). With impression of acute peritonitis, midline laparatomy was performed. There was no fluid in peritoneal cavity, liver and choleductal duct were normal in size and shape, gall bladder was inflamed and distended without perforation. Stomach, appendix and small intestine and large bowel were normal. Finally, peritoneal fluid was send for smear and culture, appendectomy and cholecystectomy were performed. Histologic diagnosis was acute cholecystitis.

Table 1: Vital signs and laboratory data of the patients.

	Patient 1	Patient 2	Patient 3
Pulse Rate	140	120	120
Respiratory Rate	28	30	26
Temprature	38.5	38	38.5
Blood Pressure	80/50	90/50	95/50
Hb	11	12.3	12
WBC	18200	23000	21500
PMN (%)	87	91	88
Lymphocyte (%)	10	5	8
Band Neutrophill (%)	2	3	3
Blood Sugar	102	91	100
Sodium	135	138	133
Potassium	3.5	4	3.8
Peritoneal Culture	S. Typhi	S. Typhi	S. Typhi
Widal test	Negative	Negative	Negative

fig.1. Ultrasound capture of the third patient.



Discussion:

In acute cholecystitis, inflammation and wall edema appears in gall bladder. In 95% of adults, it is accompanied by gallstone but it is less prevalent in children. In the gall stone cholecystitis, obstruction the cystic duct seems to be the starting event, although the cause of evoked inflammatory response is not clearly

understood. In animal models, ligature of the cystic ducts will not lead to inflammatory response. Perhaps mechanical distension, ischemia, released mediators or a bacterial cause play a role. Phopholipase A enzyme, present in gall bladder mucosa, hydrolyses the biliary lecithin to lysolecithin which in turn increases the synthesis of prostoglandine E2 in mucosa of gall bladder. PGE2 transforms the absorptive mucosa of the gall bladder into secretive mucosa and increases the mucus secretion. Accumulated mucus in obstructed sac causes increased pressure and a vicious cycle. The last step in this scenario is bacterial infection which does not seem to play in important pathophysiological role, although intestinal bacteria are frequently grown in the culture media. Therefore, cholecystitis is primarily a chemical inflammation. In acalculous acute cholecystitis, inflammation occurs in absence of gallstone. In more than half of pediatric cases of cholecystectomy is performed due to acalculus cholecystitis. This situation in children is usually associated with infections (e.g., viral gastroenteritis, bacterial intestinal infections (such as Salmonella, Shigella, E. Coli), Streptococcal infections, Pneumonia, Giardiasis, and Leptospirosis), metabolic, vascular, traumatic, malignant or congenital diseases. Bile slow down which for example happens in total parenteral nutrition (TPN) or following surgery is also a known cause. Oddi sphincter spasm following surgery which can occur as a result of opium analgesics can aggravate the bile stasis, causing the entry of bacteria and pancreatic secretions into the biliary tract, leading to chemical cholecystitis and infection. In the other hand, vascular bed of the gall bladder may be affected in systemic diseases which in turn, results in weakened mucosal defense mechanisms and contents of bile such as lysolecithin can cause damage. Congenital or inflammatory stenosis of cystic duct or external pressure effect (such as lymph node enlargement) can cause obstruction.

Clinical symptoms of gallstone and acalcucus cholecystitis are similar. In the acute attack, abdominal pain is the most common which is usually severe and can be constant or colicky in nature. Pain is localized in right upper quadrant or epigastric area, and can radiate to right shoulder or back. Sometimes pain is in periumbelical area or back and iin fact, only a few of pediatric patients have merely right upper quadrant pain. Anorexia, nausea and vomiting are common complaints in acute attack, sometimes accompanied with jaundice or dark urine. Fever and chills are uncommon. In chronic cases, history of fatty meal

intolerance and several attacks may be present.

In physical examination, right upper quadrant tenderness is the most common finding. Other signs may include guarding, rebound tenderness, minimally enlarged and tender liver, palpable gall bladder (specially in the first attack, because it gets fibrotic in subsequent attacks). Jaundice is present in 20% of patients which is commonly due to choledocal stone or other causes are billiary duct obstruction such as mesenteric lymph node enlargement. Murphy sign (inspiratory pause with right upper quadrant pressue) and Boa sign (right scapula hyperesthesia) may be present.

Routine laboratory findings are not usually helpful in diagnosis of acute cholecystitis and are usually within normal limits except for patients with jaundice or underlying disease leading to acute cholecystitis (e.g., haemolytic anemia). Leukocytosis is a common finding, but WBC count rarely exceeds 12000 to 15000/ml in uncomplicated cases. One third of patients have normal WBC count, although shift to the left may be seen in this group.

Increased billirubin, if present, is of mixed type and rarely is more than 2-5 mg/dl. If higher quantities detected, one should think of hemolysis or choledocal obstruction. 15-20% of patients have increased alkaline phosphatise, AST and ALT (Tranaminases). Urine billirubin is increased and urobilinogen is decreased in jaundiced patients.

In gallstone cholecystitits with pigment stone, plain abdominal radiography can show the stone (negative in cholesterol cholelithiasis). Ultrasound findings can include stone visualization, obstruction the neck of gall bladder with stone, wall thickness, local tenderness over gall bladder (radiologic Murphy sign), mural sonoluscent layer, echogenic material in the gall bladder, gall bladder distension and surrounding fluid accumulation. It should be emphasized that presence of gallstone does not necessarily mean cholecystitis. Radiologic (Ultrasonic) Murphy sign, although highly specific, is not so sensitive and is seen just in two thirds of the patients. Wall thickness and mural sonoluscent layer is highly associated with acute cholecystitis. Other differential diagnoses include hypoalbuminemia and hepatitis. In scintigraphy, absence of gall bladder after one hour almost always means acute cholecystitis (normal isotope scan rules out the diagnosis). False positive causes include long fasting, total parentral nutrition (TPN) and severe hepatocellular disease.

Treatment of gallstone cholecystitis is early cholecystectomy (within the

first 24 hours of diagnosis) after patient stabilization and antibiotic administration. In case of acalculus cholecystitis, the treatment of choice is urgent cholecystectomy (because of higher incidence of perforation and mortality in comparison with gallstone cholecystitis).

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