Perforated Appendicitis in the Child: Contemporary Experience

Ofer N. Gofrit MD and Khalil Abu-Dalu MD

Department of Pediatric Surgery, Hadassah University Hospital, Jerusalem, Israel

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Abstract

Background: Despite years of research and clinical experience with acute appendicitis, the rate of complications in the pediatric age group continues to be high.

Objective: To characterize the profile of the child with appendicitis complicated by perforation or intraabdominal abscess.

Methods: Between 1 January 1985 and 31 December 1997 in our department, 581 children under the age of 14 years were clinically diagnosed as suffering from "acute appendicitis." The final diagnoses were: white appendix in 28 cases (4.8%), acute non-complicated appendicitis in 472 (81%), and complicated appendicitis in 81 (13.9%), including 51 cases of free perforation (8.7%) and 30 cases of intraabdominal abscess (5.2%). We retrospectively reviewed the charts of all children with complicated appendicitis and those of 70 randomly selected children with non-complicated appendicitis, and compared patient age, gender, weight percentile, past medical history, and course of the illness.

Results: The children with complicated appendicitis were significantly younger ($P = 4.8*10\text{-}^{7}$), they had higher oral and rectal temperatures ($P = 7.9*10\text{-}^{8}$), higher platelet count ($P = 0.0008$) and lower hemoglobin level ($P = 0.004$). No difference was found in white blood count ($P = 0.41$). Total delay from symptom onset to surgery was 33 hours (SD 23) in the non-complicated group, 60 hours (SD 38) in the perforated appendicitis group, and 176 hours (SD 107) in the intra-abdominal abscess group ($P = 4.6*10\text{-}^{8}$). No difference in intra-hospital delay was found.

Conclusions: Children with complicated appendicitis are characterized by younger age, longer delay from symptom onset to correct diagnosis, and typical laboratory findings. Delays in diagnosis can be avoided by first considering the diagnosis of acute appendicitis in the differential diagnosis when examining any child with abdominal pain.

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"Of great concern, I think, to this audience, is the matter of the significant percentage of children coming into good children hospitals today with ruptured appendices," claimed M. M. Ravitch in 1982 [1]. Can we do anything about it? Is perforation of the inflamed appendix an event that can be prevented by appropriate management of the child with acute non-perforated appendicitis, or is it an inevitable event caused by a specific disease process? The high rate of complicated appendicitis in pediatric population, reaching 72% [2], justifies continued investigation of this subject. In this study we will attempt to characterize the child with perforated appendicitis and perhaps better understand this phenomenon.

Patients and Methods

Between 1 January 1985 and 31 December 1997, a total of 581 children under the age of 14 years were clinically diagnosed in our department as suffering from "acute appendicitis." The final diagnoses were: white appendix in 28 cases (4.8%), acute non-complicated appendicitis in 472 cases (81%), and complicated appendicitis in 81 (13.9%), including 51 cases of free perforation (8.7%) and 30 of intraabdominal abscess (5.2%). The charts of all children with complicated appendicitis and the charts of 70 randomly selected children with non-complicated appendicitis were reviewed retrospectively. Patient age, gender, weight percentile, and past medical history were compared. The history of the present illness was assessed with special attention paid to time schedule.

The first parental delay was defined as the time interval from symptom onset to examination by the first primary physician. The primary physician delay was defined as the time interval from examination by the first primary physician to referral to the emergency department. The second parental delay was defined as the time interval from referral to the emergency department to checking in at the emergency department, and intra-hospital delay was defined as the time interval from checking in at the hospital to the induction of anesthesia. Details of the symptoms, physical examination, laboratory tests, intraoperative findings, postoperative course and complications were recorded.

The treatment policy included a clinical diagnosis of acute appendicitis based on a history of abdominal pain, and on lower abdominal tenderness on physical examination. Neither gastrointestinal manifestations nor high temperature, signs of peritoneal irritation, or elevated white blood count were considered mandatory for diagnosis.

Children with clinical diagnosis of acute appendicitis were given intravenous triple antibiotics including ampicillin, garamycin and metronidazole before surgery, which was undertaken shortly after diagnosis.

An incision in the right lower quadrant was used in all patients, the extent of the disease was assessed, and samples of

peritoneal fluid were sent for gram stain, culture and sensitivity. If the appendix appeared normal, it was removed and the abdomen was carefully examined to exclude other pathologic findings. Appendectomy was performed in patients with acute, suppurative or gangrenous appendicitis. If the appendix was perforated, it was removed and every effort was made to obtain a secure closure of the stump. A sump suction was inserted into the Douglas pouch and peritoneal fluid aspirated with the child placed in the anti-Trendelenburg position. Routine peritoneal irrigation was not performed. Postoperative management was according to the pathology found: In the child with acute non-complicated appendicitis, antibiotics were stopped and the child was hospitalized until temperature normalized and a bowel movement occurred. In the child with perforated appendicitis, intravenous antibiotics were continued for at least 3 days, after which the child was discharged from hospital with instruction to continue oral antibiotics to complete a 7 day course. For children with palpable abdominal mass on physical examination, however, the management was different. After confirming the clinical diagnosis of intraabdominal abscess with either abdominal ultrasonography or computerized tomography, it was then the surgeon's decision whether to:

- Perform immediate surgery, drain the abscess and remove the appendix
- Perform immediate surgery, drain the abscess, and perform appendectomy 6 weeks later
- Drain the abscess percutaneously and perform appendectomy 6 weeks later
- Give intravenous antibiotics and perform appendectomy 6 weeks later.

Ambulatory follow-up was continued for at least 3 months after discharge.

Statistical analysis was done using the chi-square test and Fisher's exact test for comparison of categorical data, and Student's *t*-test for two samples assuming unequal variance for comparison of continued variables.

Results

All children with clinical diagnosis of acute appendicitis without a palpable abdominal mass were sent immediately for surgery. The management of 30 children with a palpable abdominal mass and radiologically confirmed intraabdominal abscess was as follows: immediate surgery with drainage of the abscess and removal of the appendix in 18 cases; open drainage of the abscess and interval appendectomy in 5 cases; percutaneous drainage of the abscess in one case (unfortunately this child did not return for follow-up); and intravenous antibiotic treatment and interval appendectomy in 6 cases. A total of 11 interval appendectomies were performed.

Demographic data are presented in Table 1. Children with complicated appendicitis were significantly younger ($P = 4.8*10^{-7}$). There were no cases of non-complicated appendicitis in children under the age of 3. The proportion of girls was higher among children with complicated appendicitis, but this difference did not reach statistical significance. The weight percentiles were similar in all groups and were around the 40th percentile. A significant medical history was present in three children with non-complicated appendicitis (bronchial asthma, chronic sinusitis, and history of prematurity, each in a single child), and in one child with intraabdominal abscess (celiac disease).

Table 1. Demographic parameters of the children in the study

Variable (SD)	Acute appendicitis (n-70)**	Perforated appendicitis (n = 51)	Appendicitis with abscess (n = 30)	*P* *
Mean age (yr)	9.8 (2.8)	7.6 (3.4)	6.2 (3.8)	$4.8*10^{-7}$
Female gender (%)	35.2	37.3	46.7	0.5
Weight percentile (%)	41 (39)	38 (32)	42 (37)	0.89

* Comparing the cases of acute non-complicated appendicitis with the combined group of patients with perforated appendicitis and patients with intraabdominal abscess.

** A random sample out of a population of 471 cases.

Table 2. Physical and laboratory characteristics of children with appendicitis upon presentation to the emergency department

Variable (SD)	Acute appendicitis	Perforated appendicitis	Appendicitis with abscess	*P* *
Oral temperature (°C)	37.4 (0.71)	37.9 (0.85)	38.2 (0.56)	0.017
Rectal temperature (°C)	37.9 (0.7)	38.7 (0.85)	38.6 (0.76)	$7.9*10^{-8}$
Abdominal rebound	59 (84.3%)	45 (88.2%)	21 (70%)	0.1
White blood count ($10^3/mm^3$)	16.9 (5.5)	17.8 (6.2)	17.6 (5.8)	0.41
Hemoglobin (g/dl)	13.4 (1.1)	13.1 (1.4)	12.7 (1.7)	0.004
Platelet count ($10^3/mm^3$)	263 (87)	308 (106)	401 (185)	0.0008

* Comparing the cases of acute non-complicated appendicitis with the combined group of patients with perforated appendicitis and patients with intraabdominal abscess.

The physical and laboratory findings are shown in Table 2. It is notable that children with complicated appendicitis had significantly higher oral and rectal temperatures ($P = 7.9*10^{-8}$), but no difference was found in the proportion of children demonstrating abdominal rebound on physical examination. Platelet count was significantly higher ($P = 0.0008$) and hemoglobin level lower ($P = 0.004$) among patients with complicated appendicitis, but no difference was found in white blood count ($P = 0.41$).

The causes of delay in diagnosis and treatment are given in Table 3. Primary physicians were responsible for the longest delay, reaching a mean of 100 hours from examination of children with intraabdominal abscess to referral to the emergency department. This delay was most often due to false initial diagnosis (urinary tract infection, upper respiratory infection, or pneumonia). Total delay from symptom onset to

Table 3. Causes of delays in diagnosis and treatment

Variable* (SD)	Acute appendicitis	Perforated appendicitis	Appendicitis with abscess	P**
First parental delay	22 (19)	32 (16)	43 (32)	0.002
Delay of primary MD	2.3 (13.3)	8 (16)	100 (108)	0.002
Second parental delay	4.2 (11.9)	0.9 (3.4)	11.3 (36)	0.85
Intra-hospital delay	8.4 (10.5)	13.4 (23)	7.9 (79)	0.21
Total parental delay	25.1 (20.6)	32.2 (14.9)	59.4 (45)	0.004
Total delay	33 (23)	60 (38)	176 (107)	$4.6x10^{-8}$

* As defined in the Patients and Methods section.

** Comparing the cases of acute non-complicated appendicitis with the combined group of patients with perforated appendicitis and patients with intraabdominal abscess.

surgery was 33 hours (SD 23) in the non-complicated group, 60 hours (SD 38) in the perforated appendicitis group, and 176 hours (SD 107) in the intraabdominal abscess group ($P = 4.6*10\text{-}^{8}$). No difference in intra-hospital delay was found between the groups, but patients with appendiceal perforation had a slightly longer intra-hospital delay due to the need for preoperative resuscitation with intravenous fluids and antibiotics. Despite the obvious difference in mean delay time between the groups, there were 7 cases (10%) of acute non-complicated appendicitis with a total delay in excess of 72 hours, and 5 cases (9.8%) of perforated appendicitis with a total delay time of less than 24 hours. All cases of intraabdominal abscess had delays longer than 60 hours from symptom onset to diagnosis.

Peritoneal cultures were positive in 6% of the patients with acute non-complicated appendicitis, in 80% of the patients with perforated appendicitis, and in 94% of the patients with intraabdominal abscess. The commonest bacteria grown in cultures from the peritoneum were *Escherichia coli* (41 cases), *Streptococcus* species (20 cases), *Bacteroides fragilis* (17 cases), and *Pseudomonas aeruginosa* (10 cases). In 26 cases (42%) more than one bacterial species was found.

There was no mortality due to appendicitis. The only postoperative complications recorded in the simple appendicitis group were a single case each of intraabdominal abscess and prolonged ileus. Complications in the perforated appendicitis group included: intraabdominal abscess in seven cases, wound infection in two cases, and late abdominal pain leading to rehospitalization after discharge in five children. Two of the five children with late abdominal pain had intestinal obstruction; one underwent surgery and the other was conservatively treated. The other three children with late abdominal pain improved with observation only, without a definitive diagnosis. Complications in the intraabdominal abscess group included wound infection in three patients, and late intestinal obstruction that was treated conservatively in another child.

Discussion

Appendicitis is the most common surgical condition of the abdomen in children. Unfortunately, perforation rates in the pediatric population, especially among younger children, are high reaching 18–72% [2–4]. This is often associated with significant complications such as intraabdominal abscess formation and wound infection, and occasionally with mortality [5].

We present a series of 581 children clinically diagnosed in our institution over 14 years as suffering from acute appendicitis. These cases were divided into three groups: acute non-complicated appendicitis (81%), appendicitis with perforation (8.7%), and appendicitis with intraabdominal abscess (5.2%). Normal appendix was found in 4.8% of the cases. Analysis of the database shows that children with complicated appendicitis were significantly younger (there were no cases of non-complicated appendicitis in children under the age of 3), and had higher oral and rectal temperatures, higher platelet count and lower hemoglobin level. Of special interest was the remarkably elevated platelet count, reaching a mean of $401{,}000/mm^3$ in children with intraabdominal abscess. This point can be of clinical significance: the finding of an elevated platelet count in a child with abdominal pain may direct the clinician to the diagnosis of intraabdominal abscess.

Striking differences in delay from symptom onset to surgery were found [Table 3]. Mean total delay time was 60 hours (SD 38) in the perforated appendicitis group and 176 hours (SD 107) in the intraabdominal abscess group compared to 33 hours (SD 23) in the non-complicated-group ($P = 4.6*10\text{-}^{8}$). All the delays were due to pre-hospital factors (parents and primary care physicians) and not to intra-hospital factors. Prolonged delay from symptom onset to correct diagnosis in cases of complicated appendicitis is well documented in the literature [6–9].

Discussion of the pathogenesis of acute appendicitis can be limited to two basic entities, namely acute non-perforated appendicitis and perforated appendicitis. The latter entity contains two pathological conditions: free perforation associated with peritonitis, and intraabdominal abscess where the inflammatory process is localized by the body's defense mechanisms.

Can delaying diagnosis and treatment in a child with acute non-perforated appendicitis lead to perforation, and does the temporal association between delayed diagnosis and perforation, as found in our study and in previous studies, prove a causal relationship? Two hypotheses were suggested to explain the pathogenesis of acute appendicitis: the bacterial infection theory and the luminal obstruction theory. The most accepted theory explaining the pathogenesis of acute appendicitis is that of luminal obstruction by a fecalith, by a foreign material, or by an expanded lymphoid tissue in the appendiceal wall. According to the obstructive theory, fluid accumulation behind the obstruction compromises perfusion of the appendiceal wall. Bacterial invasion of the ischemic wall leads to acute inflammation and eventually to perforation. This theory is supported by the pathological findings in experimentally obstructed rabbit appendix – findings similar to those in human acute appendicitis [10]. However, when measuring intraoperatively the intraluminal pressure in patients with acute appendicitis it was found that in most cases of acute appendicitis the

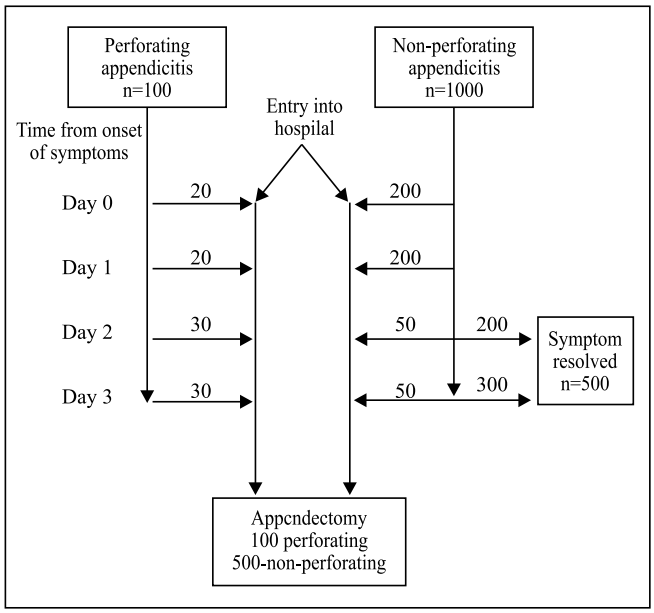

Figure 1. Time course from onset of symptoms to hospital admission for two hypothetical types of appendicitis. Numbers over arrows indicate cases admitted to the hospital on the day after symptom onset as shown on the same line. (Modified with permission from Luckmann R. *Am J Epidemiol* 1989;129:916)

intraluminal pressure is zero. Elevated intraluminal pressure (above 20 cm of water) was found only in cases of gangrenous appendicitis [11,12]. These findings suggest that obstruction is not an important causative factor in the pathogenesis of acute appendicitis but rather a late event in the natural history of this disease. Alternatively, acute appendicitis could represent two different pathological processes: non-perforating appendicitis where the main pathogenic process is bacterial infection (these patients rarely if ever perforate), and perforating appendicitis where the main process is obstruction.

What can account for the difference in delay from symptoms onset to surgery between the perforating and non-perforating appendicitis? A model explaining this phenomenon was proposed by Dr. Luckmann [Figure 1]. The model assumes two different disease processes with no conversions from one disease process to the other. The model also assumes that many patients with non-perforating appendicitis recover spontaneously over a few days as the bacterial infection subsides. These patients do not achieve medical attention, while patients with appendiceal obstruction inevitably get worse and continue to present to the medical system for a few days after disease onset [13]. This model is supported by the attendance of 10% of cases of non-perforated appendicitis with delay in excess of 72 hours, and 10% of cases with perforated appendicitis with delay of less than 24 hours.

Why are the children with complicated appendicitis younger, as found in our study and in previous reports [2–4]? It was proposed that young children have a thin-walled appendix that is predisposed to perforation [2]. Young children are also unable to give a reliable history and to cooperate during physical diagnosis. They commonly suffer from other intercurrent diseases like upper respiratory infection, otitis media or gastroenteritis, making their clinical picture even more complicated. All these factors lead to confusion and to delay in diagnosis.

The perforation rate in our study (free perforation plus intraabdominal abscess), 13.9%, is remarkably low [7]. This low rate most likely reflects a trend in the pathogenesis of the disease and not necessarily better care of the children. We hope that this trend will continue.

Despite the proposed mechanism of two different disease processes, mistakes in diagnosis do occur, and shortening the interval from symptom onset to surgery is definitely desirable. In order to avoid mistakes, acute appendicitis should be considered first in the differential diagnosis in any child with abdominal pain. The diagnosis of acute appendicitis should be entertained in areas endemic for familial Mediterranean fever, and if there is a positive family history of this disease. Perforated appendicitis with intraabdominal abscess sometimes presents indolently with weight loss, malaise, and pelvic mass on physical examination. A malignant disease may be suggested. Ultrasonography or computerized tomography should point toward the correct diagnosis.

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Correspondence: Dr. K. Abu-Dalu, Dept. of Pediatric Surgery, Hadassah University Hospital, Ein Kerem, P.O.Box 12000, Jerusalem 91120, Israel. Phone: (972-2) 677-6477, Fax: (972-2) 643-4434, email: abu-dalu@md2.huji.ac.il