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Perivesical abscesses caused by *Staphylococcus aureus* in two children

Roie Tal [a], Zeev Zonis [b], Yechiel Sweed [c,d], Gabriela Ciceu [d,e], Alon Yulevich [c,d], Daniel Glikman [d,f,*]

[a] Department of Pediatrics, Western Galilee Hospital, Nahariya, Israel
[b] Pediatric Intensive Care Unit, Western Galilee Hospital, Nahariya, Israel
[c] Department of Pediatric Surgery, Western Galilee Hospital, Nahariya, Israel
[d] The Faculty of Medicine in the Galilee, Bar Ilan University, Safed, Israel
[e] Department of Radiology, Western Galilee Hospital, Nahariya, Israel
[f] Infectious Diseases Unit, Western Galilee Hospital, Nahariya, Israel

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ABSTRACT

We present two children with abscesses located adjacent to the urinary bladder without a demonstration of intestinal pathology. The abscesses were caused by *Staphylococcus aureus* and were successfully treated with computerized tomography-guided drainage and antimicrobials. We would like to stress that not every abdominal abscess is secondary to bowel disease or perforation. Therefore, the organisms cultured may differ from the classic mixed gut flora. Hence, especially if there is no evidence of intestinal disease and the location is not typical for intestinal pathology (e.g., perivesical), *S. aureus* must be considered a potential etiologic factor.

Intra-abdominal abscesses are infrequent in the pediatric population. They often occur as a complication of local or generalized peritonitis, commonly secondary to appendicitis, necrotizing enterocolitis, pelvic inflammatory disease, surgery or trauma [1]. The microbiology of intra-abdominal abscesses is usually polymicrobial, and reflects that of the intestinal flora [2,3]. We hereby present two children with intra-abdominal abscesses without intestinal pathology; the abscesses were caused solely by *Staphylococcus aureus* and were located adjacent to the urinary bladder.

1. Child 1

A previously healthy 14-month-old boy was seen at the pediatric emergency department (ED). He had had fever up to 39.3 °C for most of the previous 3 weeks despite being treated with amoxicillin for a positive throat culture for *Streptococcus pyogenes*. He was also restless, but there were no other symptoms. A day before his admission he had watery diarrhea without blood or mucous and he vomited once.

On physical examination he was alert and oriented and did not look ill. His temperature was 37.4 °C, pulse was 100/min and blood pressure was 90/60 mm Hg. The abdomen was soft but tender mostly at the lower abdomen; there was normal peristalsis with no hepatosplenomegaly. The rest of his examination was normal.

Laboratory evaluation revealed a leukocyte count of 26,000 cells/mm^3, of which 86% were neutrophils, the hemoglobin was 8.1 g/dL and there were 860,000 platelets/mm^3. The serum electrolytes, renal function tests, liver enzymes and amylase were normal. Urinalysis was normal. An abdominal ultrasound (US) demonstrated a multilocular collection in the pelvis on the right (Fig. 1A). The collection was located next to the urinary bladder wall on the right, without a visible appendix. Free turbid abdominal fluid and diffuse increased echogenicity of the mesenterial fat were also noted.

The initial working diagnosis was an intra-abdominal abscess, probably periappendicular. Treatment was begun with intravenous ampicillin, gentamicin and metronidazole. An abdominal computerized tomography (CT) with oral and intravenous contrast was done and demonstrated intraperitoneal abscess cranial to the urinary bladder and turbid peritoneal fluid (Fig. 1B). A normal-looking appendix was demonstrated. The abscess was percutaneously drained under CT guidance.

* Corresponding author. Infectious Diseases Unit, Western Galilee Hospital, P.O.B. 21, Nahariya 22100, Israel. Tel.: +972 4 9107579; fax: +972 4 9107280.
E-mail addresses: Danny.glikman@naharia.health.gov.il, dannytamar@gmail.com (D. Glikman).

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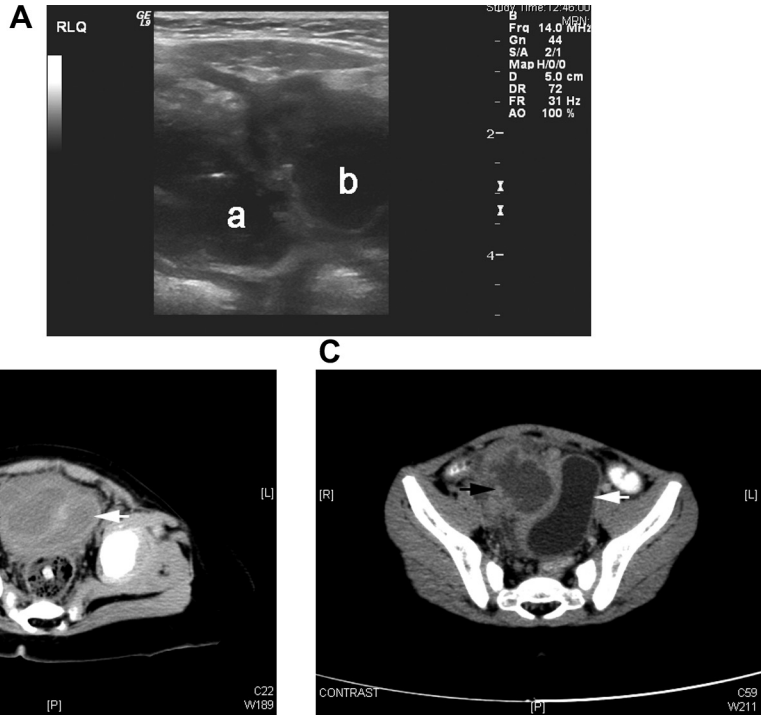

Fig. 1. A: An abdominal ultrasound demonstrating a multilocular collection ($a + b$) in the right pelvis, measuring 3 × 4.5 cm with echogenicity of the surrounding fat. B: A contrast media CT of the pelvis showing a right multilocular abscess compressing the urinary bladder (*black arrow – abscess*; *white arrow – urinary bladder*). C: A contrast media CT of pelvis showing an abscess on the right, compressing the urinary bladder (*black arrow – abscess*; *white arrow – urinary bladder*).

Direct microscopy of the aspirated fluid revealed gram positive cocci. The antibiotic regimen was changed to vancomycin, gentamicin and clindamycin. He also received packed red blood cells due to symptomatic anemia.

A blood culture which was drawn on admission and the abscess aspirate culture grew *S. aureus* susceptible to methicillin and clindamycin. The antibiotic treatment was changed to cefazolin and clindamycin. An echocardiography was performed; it showed a normal heart anatomy and function without vegetations. Repeat blood cultures were sterile as was the urine culture.

On the 4th day of admission the drain was removed. On the 5th day the C-reactive protein (CRP) level was 100 mg/L (normal range up to 8 mg/L). The child's respiratory and hemodynamic condition returned to normal. The fever, however, persisted. His abdomen was tender and pain was noticed especially prior to and during defecation. Thus, a repeat US was performed on day 8 of hospitalization. It showed 2 attached collections in the mid-lower abdomen, to the right of the midline, each measures about 1.5 × 2 cm. A third, slightly larger collection was seated deeper in the pelvis, anterior to the urinary bladder. A repeat CT was performed in the following day (9th hospitalization day). It demonstrated multiple small abscesses in the upper and lower abdomen between the bowel loops. At this time the child defervesced, was less irritable, and the CRP level decreased to 84 mg/L. A decision was made to continue the same antibiotics without surgical drainage of the abscesses, assuming the abscesses were either spillage from the drainage of the first abscess or organization of the peritoneal turbid fluid seen before.

The child's condition continued to improve. Repeat US examinations demonstrated decreasing volume of the abscesses. The child was discharged on day 21 of hospitalization without fever or pain and with a normal CRP and blood count. He received additional 3 weeks of oral antibiotics (initially cephalexin and clindamycin followed by cephalexin). Repeat US 6 weeks after his admission showed the disappearance of the abscesses. Because of the severe unusual infection an immunological workup was done: Human immunodeficiency virus serology was negative, immunoglobulin levels (IgG, IgA, IgM) were normal (including adequate post-vaccination levels against measles, mumps and rubella), and neutrophil function (including chemotaxis, oxidation and bactericidal activity) was normal. The blood staphylococcal isolate was submitted to the Israeli *S. aureus* Central Laboratory and was found to be Panton–Valentine leukocidin (PVL) negative by polymerase chain reaction. Currently, over 2 years after his hospitalization, the child's medical and developmental status is normal for his age.

2. Child 2

A previously healthy 3.5-year-old boy presented to the ED with 7 days of dysuria and 2 days of fever. His medical history was remarkable only for air enema reduction of intussusception at 6 months of age. His temperature was 37.2 °C and the abdomen was noted to be soft but tender in the lower region without signs of peritonitis.

Blood count demonstrated mild leukocytosis (16,000 cells/mm^3) with 66% neutrophils and microcytic anemia (hemoglobin 10.5 g/dL) with normal platelet count (413,000/mm^3). Urinalysis revealed 2–3 red blood cells/mm^3. CRP was 103 mg/L (normal range up to 5 mg/L).

A localized inflammatory process in the pelvis was seen by US. Abdominal CT revealed an abscess 4.5 cm in diameter, adjacent to the urinary bladder on the right (Fig. 1C). It also showed a normal-looking appendix.

The abscess was drained percutaneously, guided by CT. Culture of the drained pus grew *S. aureus* susceptible to methicillin and clindamycin. The boy was treated initially with ampicillin, gentamicin and clindamycin which were changed to cefazolin according to the culture result. Blood and urine cultures were negative. The abscess decreased in size, and the symptoms resolved. CRP on

discharge decreased to 10 mg/L. He was discharged 12 days after his admission with no further antibiotic therapy.

Twelve days later he was admitted to another hospital because of abdominal pain. Abdominal US demonstrated the abscess with dimensions of 3.4 × 2.7 cm. He was treated with piperacillin–tazobactam with decreasing volume of the abscess and was discharged a week later. Currently, 3 years after his hospitalization he is well.

3. Discussion

Intra-abdominal abscesses are infrequent in the pediatric population; they often occur as a complication of local or generalized peritonitis, commonly secondary to appendicitis [1]. The microbiology of intra-abdominal abscesses is usually polymicrobial, and reflects that of the intestinal flora. In a study of 30 patients with complicated appendicitis, an average of 10 different bacterial species were isolated from abdominal cultures [2]. Although *S. aureus* can be a part of the intestinal tract flora (intestinal carriage rate of 8–30% in healthy adults is reported), it is an infrequent cause of abdominal abscesses; the bacterium was isolated in only 4% of 1237 microbiologically confirmed complicated intra-abdominal infections [3,4].

Psoas, hepatic and renal abscesses are described as caused by *S. aureus* [5–7]. *S. aureus* is also implicated in pancreatic and splenic abscesses and can complicate omphalitis in neonates [8]. Bladder wall abscesses were described in association with neurogenic bladder, and their differential diagnosis includes infected urachal cysts and enterovesical fistulas; in these cases *Pseudomonas aeruginosa* and *Escherichia coli* grew in culture in the first example and *S. aureus* in the latter [9,10]. In both of our children, the abscess was adjacent to the urinary bladder from the right without involvement of the bladder wall. One child was bacteremic with probable seeding of *S. aureus* in this location via the blood. We do not know why the organization of the abscesses took place in that specific anatomic location; urachal abnormalities are usually located in the midline and no intestinal pathology was evident on CT. Furthermore, *S. aureus* was solely identified without typical gut flora organisms such as anaerobes and enteric Gram negatives. PVL, a staphylococcal toxin believed in the past to be the primary virulence factor, is commonly found in community-associated methicillin-resistant *S. aureus* isolates but can also be found occasionally in methicillin-susceptible strains. Recently, the importance of PVL in the pathogenesis of staphylococcal infections has come into question but it is still considered by many a marker of enhanced virulence [11]. Our first child had a severe staphylococcal infection but the isolate tested negative for PVL, thus strengthening the virulence debate.

The diagnosis and treatment of abdominal *S. aureus* abscess poses several dilemmas to the physician. The first is whether the growth of *S. aureus*, isolated from the abscess, is not a contamination from the skin. Naturally, a meticulous preparation of the skin prior to invasive procedures is mandatory. Growth of the bacterium from another sterile site is helpful as was the case in our first child who also had *S. aureus* bacteremia. The second dilemma is the origin of the abscess. The absence of other bacteria (such as aerobic Gram negatives and anaerobic pathogens) growing in the cultures derived from the abscesses and absence of a gut perforation or hints for a bowel disease on CT are evidence against gut-origin of the abscesses in our cases. We did not aspirate the secondary abscesses that were found after the drainage in the case of the first child because we assumed that either a local dissemination occurred during the drainage procedure or organization of the peritoneal turbid fluid, seen before, has taken place. The clinical response to narrow-spectrum anti-staphylococcal antimicrobials supports our assumption.

After a diagnosis is made, consideration should be given to the optimal treatment. The percutaneous drainage, guided by CT, is considered currently to be the first line of treatment [1]. A surgical intervention is indicated if the former modality is not available or if drainage fails. Adjunctive antibiotic treatment should be given, and may be the sole modality if the abscess is small. The management of a patient with multiple abscesses is probably dependent on the number and size of foci, their location and proximity to each other, the assumed microbial etiology and response to prior treatment, and the host's immune status and co-morbidities. In such cases it is reasonable to percutaneously drain the large collections, to hasten their resolution as well as to obtain pus for culture, and treat other foci with antibiotics while maintaining a close clinical and radiologic follow up.

Once the decision is made to treat conservatively with antibiotics, the next dilemma is for how long. The optimal duration of therapy is unknown. Our decision on the length of antibiotic therapy was made according to the clinical status, the laboratory data (mainly CRP and erythrocyte sedimentation rate when the CRP already normalized) and the imaging. Reports of 3–6 week duration of antimicrobial therapy were published for hepatic and renal abscesses [6,7]. Guidelines published by the Infectious Diseases Society of America for complicated intra-abdominal infection in adults and children recommend a 4–7 day treatment if control of the source of infection is achieved, but do not specify recommendations for the case of our first child, who had multiple abdominal abscesses, and do not address the issue of *S. aureus* being the major or sole pathogen [3].

We present here 2 rare cases of children with perivesical abscesses caused by *S. aureus* and secondary multiple abdominal abscesses in one child, who were successfully treated with CT-guided drainage and antimicrobials. We would like to stress that not every abdominal abscess is secondary to bowel disease or perforation. Therefore, the organisms cultured may differ from the classic mixed gut flora. Hence, especially if there is no evidence of intestinal disease and the location is not typical for intestinal pathology (e.g., perivesical), *S. aureus* must be considered a potential etiologic factor.

Conflict of interest statement

The authors declare that they have no conflict of interest.

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Informed consent

Written informed consent was obtained from the patients for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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