CASE REPORT

Children and Chickens: 11-month-old Infant with a Salmonella Deep Neck Infection

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CASE PRESENTATION

An eleven-month-old female presented to the Emergency Department (ED) for decreased oral intake, fussiness, and fever. The infant had been seen twice at her pediatrician's office in the preceding four days with low-grade fevers (Tmax 100.3), nasal congestion, and mild cough, along with progressively decreasing oral intake and urine output thought to be related to a viral upper respiratory infection. Point of care influenza and strep testing were negative. She was ultimately brought to the ED because she was becoming increasingly fussy and refusing to take any fluids for almost 18 hours despite trying small volumes. Per parental report, she had started choking on her post-nasal drip and had a few episodes of posttussive emesis. Her urine output had further decreased with only one damp diaper 8 hours prior to ED arrival. The last dose of antipyretic was 12 hours prior to presentation to the ED. Review of systems was additionally positive for one hard stool the day of admission. Her mother denied respiratory distress, drooling, diarrhea or rash.

Her past medical history was significant for uncomplicated term delivery, normal newborn screen and resolved gastroesophageal reflux. She was undergoing evaluation by occupational therapy for feeding aversion to table foods. She also had a history of a 5-day, self-resolved diarrheal illness at 5 months of age, which was presumed to be *Salmonella* as brother had *Salmonella* PCR positive enteritis at that same time. No other history of recurrent infections and no prior antibiotics. Immunizations were up to date, including seasonal influenza vaccination.

The infant did not attend daycare, rather maternal grandparents cared for her and her sibling during the day. Grandparents live on a farm with numerous animals, including backyard chickens.

Upon arrival to the ED, vital signs included: temperature 99.3, pulse 110 beats per minute, respiratory rate 32 breaths per minute, oxygen saturation 100% on room air, and weight 8 kg (18th percentile). This weight represented a 440 g weight loss in the past 4 days. She appeared listless and was lying in mother's arms. Her conjunctivae and tympanic membranes were normal. Initially she had full range of motion of her neck without swelling and shotty anterior cervical lymphadenopathy. Her oral exam demonstrated chapped lips, mildly erythematous posterior oropharynx and tonsils 2+ without exudates, uvula was midline. Gingivae and dentition normal. Her pulmonary and abdominal exams were unremarkable. She was tachycardic with regular rhythm and no murmur. She was noted to have delayed capillary refill of 4 seconds.

She was given ondansetron followed by an attempt at oral rehydration, which she did not tolerate. After a 20 mL/kg normal saline fluid bolus, she had normalized capillary refill, improved tachycardia and appeared more alert, but she continued to refuse oral intake. Upon re-examination she was noted to have developed a hoarse cry without stridor or respiratory distress. Her oral exam showed prominence of the left pharyngeal wall and she had developed left-sided neck swelling with palpable firmness extending from the angle of the mandible to the mid-neck. There was no warmth on palpation or erythema of the overlying skin.

HOSPITAL COURSE

Given the change in her physical exam with acute-onset left-sided neck swelling, further workup was obtained including complete blood count (CBC), basic metabolic panel (BMP) and C-reactive protein (CRP) along with a computer tomography (CT) of the neck. She was empirically started on clindamycin 40 mg/kg/day divided every 6 hours.

Blood work: CRP 177.8 mg/L (0-5 mg/L), WBC 23.1 (6-17.5), hemoglobin 12.4 (9.5-13.5), platelets 519,000 (150-450,000), neutrophils 69% (35-85), lymphocytes 17% (25-65), monocytes 13% (2-10). BMP was normal.

The contrast CT demonstrated a large left parapharyngeal abscess (PPA) measuring 3.5 cm x 3.0 cm at the greatest dimension and 3.5 cm vertical. There was substantial mass effect with displacement of the hypopharynx and the epiglottis, but the airway remained patent. There were irregular contours and a thin enhancing wall (Image 1). The lesion may have begun as a suppurative lymph node, but had progressed to extracapsular extension and abscess.

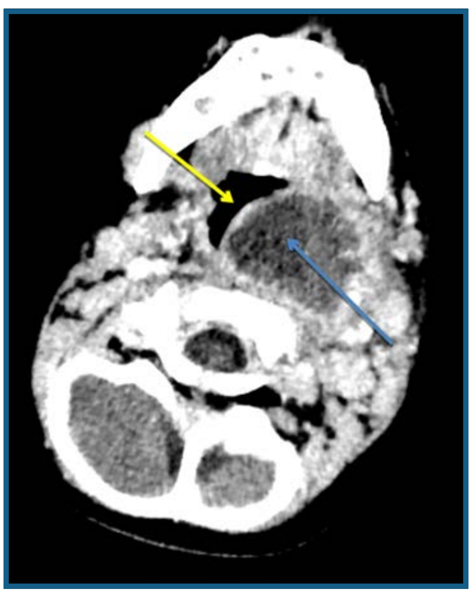

Image 1: Contrast enhanced CT demonstrating a large left parapharyngeal abscess (PPA) measuring 3.5 cm x 3.0 cm at the greatest dimension and 3.5 cm vertical with irregular contours and a thin enhancing wall (blue arrow). Mass effect with displacement of the hypopharynx and the epiglottis (yellow arrow).

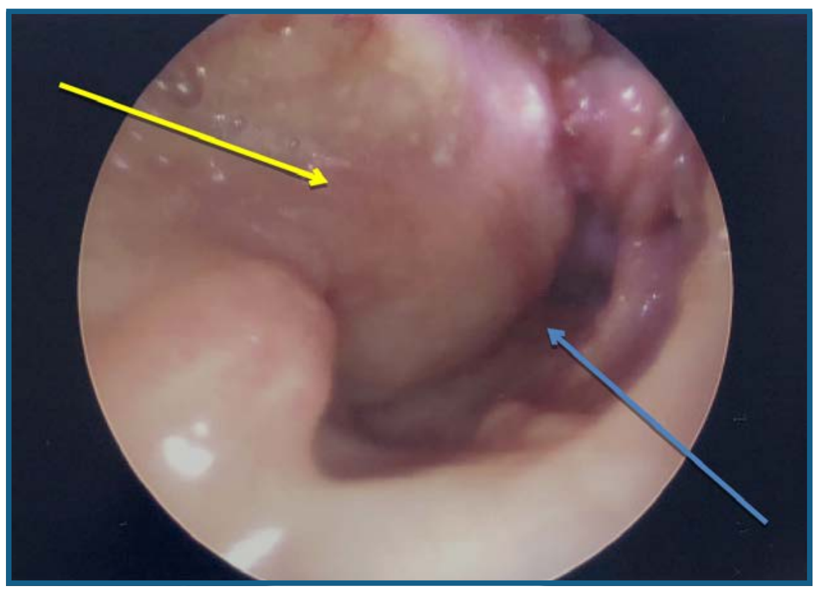

Image 2: Intraoperative image of swelling of the left parapharyngeal space (yellow arrow) with displacement of the epiglottis to the right. Partial collapse of the airway secondary to mass effect (blue arrow).

Pediatric ENT evaluated the infant and surgical incision and drainage was recommended. An intraoral surgical approach was used and 9 mL of purulent material was aspirated and both aerobic and anaerobic cultures were sent (Image 2). She was admitted to the pediatric intensive care unit immediately after surgery due to her high-risk airway, but was transferred to the regular pediatric floor within 24 hours. During her three-day hospitalization, she was continued on IV clindamycin. Within 24 hours following her surgery, she started tolerating oral fluids well. She was discharged home on oral clindamycin to finish a total 10-day course.

FINAL DIAGNOSIS AND PATIENT FOLLOW-UP

The cultures, obtained during surgical incision and drainage, grew 3+ *Salmonella* species, not typhi, and 1+ staph aureus (both sensitive to clindamycin). Fungal culture was negative. No acid-fast bacillus on direct smear.

At her recheck appointment 4 days after hospital discharge, her fevers and fussiness had resolved and oral intake was back to baseline. Physical exam demonstrated resolution of her neck swelling and there was a healing incision in the left parapharyngeal region without drainage.

She had presumed diarrheal illness secondary to salmonella at 5 months of age, but no other history of recurrent bacterial or viral infections. Given the rarity of salmonella as the cause of a PPA, pediatric infectious disease recommended an immunologic work-up including: CBC with differential, CRP, immunoglobulins IgG, IgA and IgM, IgG subclasses (1,2,3 and 4), total complement and vaccination titers (Tetanus Ab IgG, Haemophilus Influenza B IgG, and Pneumococcal Serotypes IgG-14 Ab). These labs were all normal for age.

DISCUSSION

The parapharyngeal space is an inverted pyramid, which extends from the base of the skull to the hyoid bone. Deep neck space abscesses, including retropharyngeal (RPA) and parapharyngeal (PPA) abscesses, are uncommon, but do occur more commonly in the pediatric than adult population: national yearly incidence is 4.6 per 100,000 children.[1,2] Studies differ in the mean age at diagnosis from 2.9-4.4 years and male to female ratio from 1.6:1 to 3:1.[3,4] In children, PPA typically occurs by direct continuity of infection or indirectly by lymphatic drainage of the retropharyngeal lymph nodes, which can be more prominent in children.[5] Upper respiratory tract infection (67%) and odontogenic source (25%) are the most common causes of PPA in children.[1] There is a seasonal distribution to PPA with increased incidence in the winter months, which is consistent with the fact that many PPA are often preceded by a viral upper respiratory tract infection.[1]

Initial presenting symptoms of PPA can often be subtle and difficult to discern, especially in young children who are unable to verbalize their symptoms. PPA will frequently begin with a prodromal phase, including fever and upper respiratory symptoms, and then progress to include localized neck and pharyngeal complaints.[4] Common symptoms reported, in patients old enough to articulate, include decreased neck range of motion, neck pain, neck mass, sore throat and pain/difficulty swallowing.[4,6] A Danish retrospective study demonstrated the most consistent physical exam findings of PPA include the following: trismus, posterior pharyngeal erythema and edema, pharyngeal asymmetry, neck swelling and torticollis.[6] Signs of airway compromise are a late finding and if present, need to be taken very seriously.[4]

Although management of PPAs remains controversial, the use of diagnostic imaging, typically by contrast enhanced CT, is recommended almost universally. A CT can help differentiate a drainable abscess from cellulitis and can also assist in surgical planning, if required.[2] Signs on CT that may indicate the presence of PPA include ring enhancement, central lucency and "scalloping" or irregularity of the abscess wall.[4] Ultrasound has been used for diagnosis, more commonly in adults, but sensitivity depends on the skill of the technician and it may not be adequate to visualize deeper abscesses.[2]

Appropriate treatment is important to decrease the risk of developing complications, which occur in 2-3% of cases.[2] Complications can include airway obstruction, sepsis, mediastinitis, carotid artery aneurysm or rupture, Lemierre's syndrome, cranial nerve IX-XII palsy and necrotizing fasciitis.[1,2] Although the incidence of PPA has increased over the past decade, complication rates have decreased secondary to earlier diagnosis, improved surgical methods and superior antibiotics.[4] There is not a consensus regarding treatment protocol for PPA: options include surgical incision and drainage combined with intravenous (IV) antibiotics versus more conservative medical management with IV antibiotics only. This decision often depends on location, size of the abscess and age of the patient. A retrospective case study by Cheng *et al.* concluded a 48-hour trial of IV antibiotics only is a reasonable approach in smaller (<2.2 cm) abscesses in stable children older than 4 years of age.[3] Surgery is recommended as the initial treatment in younger children, larger symptomatic abscesses or if there is no improvement after 48 hours of antibiotics.[5] Surgical approach (external vs intraoral) is determined based on location of the abscess and risk to surrounding structures.[5] More recent studies have demonstrated that the use of the intraoral approach is associated with decreased operating time and need for postoperative IV antibiotics as well as reduced length of hospital stay postoperatively.[4]

The most common organisms that cause parapharyngeal abscesses are *Staphylococcus aureus*, *S. viridans*, group A streptococci and oral anaerobes (*Prophyromonas, Fusobacterium* and *Peptostreptococcus*)[1,2], with recent studies citing a prevalence of up to 61% for methicillin-resistant *S. aureus* (MRSA) as the causative organism.[5] Cheng *et al*. has also reported a higher incidence of PPA caused by MRSA in children less than 15 months of age.[3] Less frequent isolated organisms include *Haempohilus* spp., Gram-negative enteric organisms and polymicrobial infections.[1] Antibiotic therapy is targeted to cover the aforementioned bacteria with empiric regimens including anaerobic coverage (metronidazole or clindamycin), plus penicillin combined with either beta-lactamase inhibitor (amoxicillin with clavulanic acid) or beta lactamase-resistant antibiotic (cefoxitin, cefuroxime, imipenem or meropenem): additionally, clindamycin or vancomycin is added if there is concern for MRSA.[5] If surgical incision and drainage is required, antibiotic selection can then be narrowed based on culture results.

Given the patient's young age and lack of risk factors, it was surprising when the identified pathogen causing her PPA was *Salmonella.* A PubMed literature review confirmed the rarity of this diagnosis with only 2 previous case reports of children under 18 years of age with *Salmonella* as the cause of a deep neck abscess.[7,8] A majority of the 21 documented cases of *Salmonella* identified in neck infections occurred in older (ages 24-70) and/or immunocompromised patients.

A case report by Su et al reported that localized salmonellosis infections rarely occur in patients without underlying illness and are present in less than 8% of patients hospitalized for a *Salmonella* infection.[7] Thus, a localized *Salmonella* infection in a healthy child, as seen in our patient, is exceedingly rare. Her only known risk of exposure to *Salmonella* was backyard chickens at her grandparent's home. Although the infant did not personally touch the chickens, the grandparents did care for the chickens while she was at their home and used their eggs for cooking.

The backyard chicken (BYC) movement in the USA has increased human contact with poultry and subsequently, human contact with the *Salmonella* pathogen.[9] With this change, there has been a subsequent increase in live poultry-associated salmonellosis (LPAS) outbreaks.[10] In the United States from 1990 to 2014, 53 outbreaks of human salmonellosis linked to live poultry were verified which resulted in 2,611 documented illnesses, 387 recorded hospitalizations and five known deaths.[10] Median patient age at the time of infection was 9 years.[10] Of those infected by LPAS, chick and duckling exposure were reported by 85% and 38% of patients, respectively.[10] The incidence of *Salmonella* infection due to live poultry exposure is increasing. In a 6-month period alone in 2018, 334 people were infected with *Salmonella* from confirmed contact with live poultry, and of those infected, 31% were younger than 5 years of age, as was our patient.[11]

It is well known that ingestion of inadequately cooked eggs or chicken meat can lead to *Salmonella* infection, but people often do not understand or acknowledge the risk of infection through direct handling of the birds or indirect contact with contaminated bedding and living structures.[12] Poultry will often have no clinical signs of infection, but still intermittently shed *Salmonella* bacteria, which can lead to infection.[12] Review of the LPAS outbreak data from 1990-2014 determined that high-risk practices were common, including keeping poultry inside households (46% of patients) and kissing poultry (13% of patients).[10] Despite the increase in LPAS, there is limited research surrounding the BYC movement and private poultry owners' practices. One such study was performed in Seattle to better understand the knowledge base and hygiene practices of urban backyard poultry owners regarding *Salmonella.* It showed that while almost all of the participants knew that exposure to *Salmonella* is an inherent risk associated with raising poultry and harvesting eggs, their reported and observed practices promoted risk of transmission of *Salmonella*.[12] Approximately 25% of participants reported snuggling and kissing birds or eating/drinking near them.[12]

While infection from salmonella secondary to live poultry exposure is clearly on the rise, there are few studies of BYC and their prevalence of *Salmonella*. A study conducted in 2019 of 50 residents in Boston investigated the habits of backyard chicken-human interactions and assessed the prevalence of *Salmonella* in those flocks.[9] Interestingly, they discovered that many chickens were treated as pets (75%) with frequent handling and inadequate hygiene practices but that the overall incidence of *Salmonella* was low (<2% of flocks).[9]

CONCLUSION

Despite lower than expected carrier status of *Salmonella* by chickens, in a limited number of studies, there has been a growing burden of BYC-associated diseases. Since children are at higher risk for improper hand hygiene, parents must remain vigilant and teach proper handling techniques. Pediatricians should ask about backyard poultry exposure and discuss the importance of proper hygiene to help decrease the risk of LPAS infection. Simple rules for parents include: washing hands after touching chickens or touching their roaming areas, never letting chickens inside the house, wearing separate shoes while taking care of chickens, keeping children under 5 years of age from handling chickens and not eating food near chickens.[11]

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