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An Editorial Note on Fatal Pediatric Streptococcal Infection: A Clinico-Pathological Study

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Abstract

In this study we analysed the clinical and the autopsy findings of 38 pediatric cases, above the age of 28 days, when the death was due to Streptococcal infection. Cases with underlying risk factors for infections, were excluded from the study. The two main infective organisms were *Streptococcus pneumoniae* (SPn; 45%) and *Streptococcus pyogenes* (SPy; 37%). Almost all (92%) decedents had some prodromal symptoms prior to death and the majority had a very rapid terminal course: 89% were found unresponsive, suddenly collapsed, or died within 24 hours of hospital admission. Sepsis was the most common immediate cause of death (64%), which was more common in the SPy group than in the SPn group (71% *vs.* 48%). Pneumonia was found in 18% of the cases with similar number of cases in both SPn and SPy groups (18% *vs.* 21%). Meningitis, found in 16% of cases, was exclusively caused by SPn. Additional molecular studies of autopsy samples to identify more virulent Streptococcal strains would be a great value to vaccine developments and to prevent sudden and unexpected pediatric deaths related to Streptococcal infection.

Keywords: *Streptococcus*; Postmortem; Pediatric; Microbiology; Infection

Editorial Note

There are clinical studies on Streptococcal Infection (SI) among hospitalized children, but only a few provide any postmortem correlation. Similarly, there is limited published autopsy data available on fatal pediatric SI. In our autopsy-based study we analyzed the clinical and the pathological features of pediatric deaths due to SI [1]. Our cohort included 38 Coroner-warranted and next-kin-consented autopsied cases between 1997 and 2019. Neonates, under the age of 28 days, and children with pre-existing factors, which would have predisposed for SI, were excluded from the study [1].

The largest number of SI deaths was due to *Streptococcus pneumoniae* (SPn) (45%), followed by *Streptococcus pyogenes* (SPy) (37%) [1]. Indeed, SPn and SPy are the two most commonly reported Streptococcus species associated with invasive infection in children. *Streptococcus agalactiae* (SAg) usually causes severe disease in neonates and this age group was excluded from the study. However, there were three deaths in our cohort attributed to SAg infection: two infants and one 23 month-old child [1].

SPn causes more infections under the age of 5 years, especially in those younger than SPn typically causes bacteraemia and meningitis in children under 2 years of age, whereas pneumonia is more prevalent in the older age group [2]. The majority of SPy infections occurs in school-aged children, between 5-17 years of age, and SPy most frequently causes acute pharyngotonsillitis and cutaneous infections [3]. Our data align with this age-related prevalence. Most deaths occurred in children over 2 years of age with similar number of deaths in both SPy and SPn groups. Under the age of 24 months, more deaths were due to SPn infection [1].

Children are at increased risk for SIs for many reasons. They have high incidences of nasopharyngeal colonization, their immune system is relatively immature and they are more frequently exposure to bacteria in daycare centres and schools [4]. Moreover, early signs and symptoms of invasive SI are often non-specific, such as fever, irritability, rhinitis and decreased appetite, and making early diagnosis challenging.

Our data showed that almost all children (92%) had prodromal symptoms or signs of an illness prior to death. Most of them (79%) had a history of being unwell for at least a day, and 34% were seen by a doctor in the days prior to their terminal presentation. Fever (80%) was the most common clinical sign, followed by gastrointestinal and respiratory symptoms (60% and 51%, respectively). These included vomiting, diarrhoea, nausea, abdominal pain, difficulty drinking, feeding, cough, shortness of breath, tachypnoea, wheezing, grunting, chest pain, sore throat and runny nose. Skin rash was more documented in the setting of SPy infection [1].

Another interesting finding our study demonstrated was that the terminal events were generally very brief. 89% of decedents had a rapid terminal course, including 13% were found unresponsive at home, 21% had witnessed collapse at home or en route to the hospital in an ambulance, and 55% died within 24 hours of hospital admission. 11% survived beyond 1-day post-admission and died later [1]. In a study from England, analysing SPn infection related pediatric deaths, it was found that 13.3% died at home, 5.5% en route to hospital, and 16.7% in the emergency department. 64% died later in the hospital, though, there was no specification on the length of hospital admission [5,6].

Our study showed that majority (64%) of death was attributed to sepsis, which was more frequent in the SPy group (71%) than in the SPn group (48%). All SAg infection related deaths were due to sepsis. In half of the septic deaths, source of infection was demonstrated at postmortem examination. These included pneumonia, meningitis, lung abscess, ruptured appendicitis, neck abscess and purulent peritonitis. Non-septic deaths (37%) were due to localized organ inflammation and resultant organ failure, for example necrotizing pneumonia, purulent meningitis, and diffuse purulent peritonitis. Pneumonia was present in a similar number of cases in both SPn and SPy groups. All meningitis-associated deaths were caused by SPn infection [1].

Diagnosis of sepsis at autopsy examination has many challenges. Pre-mortem clinical information might be limited or unavailable, especially in cases of sudden unexpected deaths. Macroscopic and histological findings in septic deaths are non-specific, and the result of postmortem microbiology cultures can be affected by multiple factors. Bacteria detected in a postmortem culture can be a true pathogen contributing to death, or be 'artefactual' due to agonal spread, postmortem translocation or contamination [7]. Moreover, negative culture results might be due to premortem antibiotic administration, low circulating microbial loads and insufficient amounts of blood sampled for microbiology [8]. Therefore, in our study, autopsy diagnosis of sepsis was based on the combination of available clinical history and premortem culture results, postmortem stigmata of septic death and postmortem culture results.

Recommendations on post mortem microbiology sampling in cases which are clinically suspicious of sepsis include heart blood and spleen, with additional sampling of lung, liver and kidney to be able to confirm presence of the same pathogen at multiple sites [7,9]. Middle ears should always be examined in children with swab culturing as this may be a primary site of infection in cases of meningitis. To optimize interpretation of any positive culture results, histological examination of the area sampled for culture is advised i.e., vital inflammatory response seen on histology would corroborate that an isolated organism in the culture is pathologically significant. This relates not only to internal organs but to any abnormal skin lesions, as skin is the most common portal of entry in invasive SPy infection [3,10,11].

Infection screen should also include virology testing as viral infection, such as Varicella and Influenza viruses have been identified as risk factors for SI [12-14]. In our study two children had a history of viral infection (chickenpox and Influenza A virus) shortly before death.

Autopsy examination continues to be a source of valuable information, which enhances our understanding of fatal pediatric infectious deaths. It helps to identify specific infective organisms, and their portals of entry, through internal organ/tissue sampling, not readily accessible in the living.

Though, multivalent pneumococcal conjugate vaccines (PCV-7 and PCV-13) and improved antimicrobial treatment have decreased the number of invasive SPn infections, non-vaccine preventable serotypes are demonstrated to be responsible for a more recent increase in invasive pneumococcal diseases [15,16]. Currently, there is no licensed vaccine against SPy. Therefore, additional molecular serotyping and/or genotyping of the bacteria in autopsy samples to identify more virulent Streptococcal strains associated with a high risk for sudden death would be of great value in future vaccine development.

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