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Letter to the Editor: Prevalence and Antibiotic Resistance of Neonatal Sepsis Pathogens

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Dear Editor,

We read with great interest the original article by Behmadi et al. (1) in the most recent issue of your journal. We would like to commend the authors for their endeavor but at the same time feel that few necessary clarifications and comments would benefit the general readers of the journal:

1) The abstract mentions that "the aim of this study was to determine the prevalence and evaluate the antimicrobial susceptibility patterns of bacterial infections at a neonatal unit," but the main text states that the objective was "to determine the frequency and antibiotic resistance of pathogens at the neonatal intensive-care unit (NICU) and neonatal ward of our hospital." The study, being "cross-sectional" in design, is able to give prevalence but not frequency or incidence (2). Furthermore, the pathogens associated with neonatal sepsis include viruses and parasites, in addition to bacteria (3).

2) The authors mention the total number of culture (blood, urine and cerebrospinal fluid (CSF)) samples and their positivity rates, but not the total number of newborns being assessed (sample size). Therefore, the percentage of newborns who ultimately have culture (blood, urine or CSF)-positive sepsis indicative of prevalence of bacterial infection, which is one of the study's primary objectives, can never be ascertained.

3) The authors do not provide a definition for terms such as "late-onset sepsis" and "clinical findings suggesting urinary tract infection (UTI)." Different epidemiological studies have used cut-offs ranging from 3 to 7 days to define late-onset sepsis, depending on the gestational age and birth weight of the newborn (4). Also, there are no clinical findings that specifically suggest UTI in a newborn.

4) The methodology does not state how the urine sample for bacterial culture was collected. It should ideally be collected by suprapubic aspiration (5); other methods are associated with high chances of contamination.

5) The authors also do not mention whether hospital-acquired infections were included in the late-onset sepsis group. The causative organisms and the antibiotic sensitivity pattern are expected to vary considerably depending on whether the source of infection is the community or a heath care facility (5). Hospital-acquired pathogens could also explain the high prevalence of antibiotic resistance in the isolated organisms.

6) The finding that coagulase-negative staphylococcus (CONS) was the commonest organism in both early- and late-onset sepsis should be viewed with caution in light of the great controversy about the criteria for defining CONS sepsis in newborns (6). These criteria have often resulted in CONS being overrepresented as a true pathogen in neonatal sepsis (7). In an attempt to decrease false-positivity rates, various investigators have proposed different diagnostic algorithms based upon quantitative culture and colony count, presence of an indwelling central venous catheter (8) or time to positivity of blood culture (9).

Footnote

Authors' Contribution: Both the authors were involved in review of the article and manuscript writing.

References

1. Behmadi H, Borji A, Taghavi-Rad A, Soghandi L, Behmadi R. Prevalence and Antibiotic Resistance of Neonatal Sepsis Pathogens in Neyshabour, Iran. *Arch Pediatr Infect Dis.* 2016;**4**(2):e33818. doi: 10.5812/pedinfect.33818.
2. Levin KA. Study design III: Cross-sectional studies. *Evid Based Dent.* 2006;**7**(1):24–5. doi: 10.1038/sj.ebd.6400375. [PubMed: 16557257].

3. Stoll BJ, Shane AL. In: Nelson Textbook of Pediatrics. Kliegman RM, Stanton BF, St Geme JW, Schor NF, editors. Philadelphia: Elsevier; 2016. pp. 909–25.Infections of the neonatal infant.
4. Dong Y, Speer CP. Late-onset neonatal sepsis: recent developments. *Arch Dis Child Fetal Neonatal Ed.* 2015;**100**(3):F257–63. doi: 10.1136/archdischild-2014-306213. [PubMed: 25425653].
5. Nizet V, Klein JO. In: Infectious Diseases of the fetus and Newborn Infant. Remington JS, Klein JO, editors. Philadelphia: Saunders; 2011. pp. 222–75.Bacterial sepsis and meningitis.
6. Nash C, Chu A, Bhatti M, Alexander K, Schreiber M, Hageman JR. Coagulase Negative Staphylococci in the Neonatal Intensive Care Unit: Are We Any Smarter?. *Neo Rev.* 2013;**14**(6):e284–93.
7. Healy CM, Baker CJ, Palazzi DL, Campbell JR, Edwards MS. Distinguishing true coagulase-negative Staphylococcus infections from contaminants in the neonatal intensive care unit. *J Perinatol.* 2013;**33**(1):52–8. doi: 10.1038/jp.2012.36. [PubMed: 22499081].
8. Craft A, Finer N. Nosocomial coagulase negative staphylococcal (CoNS) catheter-related sepsis in preterm infants: definition, diagnosis, prophylaxis, and prevention. *J Perinatol.* 2001;**21**(3):186–92. doi: 10.1038/sj.jp.7200514. [PubMed: 11503106].
9. Haimi-Cohen Y, Vellozzi EM, Rubin LG. Initial concentration of Staphylococcus epidermidis in simulated pediatric blood cultures correlates with time to positive results with the automated, continuously monitored BACTEC blood culture system. *J Clin Microbiol.* 2002;**40**(3):898–901. [PubMed: 11880412].