

Sensorineural hearing loss after neonatal meningitis: a single-centre retrospective study

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To cite: Sharma A, Leaf JM, Thomas S, *et al*. Sensorineural hearing loss after neonatal meningitis: a single-centre retrospective study. *BMJ Paediatrics Open* 2022;**6**:e001601. doi:10.1136/bmjpo-2022-001601

Received 5 July 2022
Accepted 29 August 2022

ABSTRACT

Babies in intensive care are at higher risk for meningitis and sensorineural hearing loss (SNHL). We reviewed the rate of SNHL among definite cases of bacterial/fungal meningitis in our neonatal intensive care unit over a 16-year period (2006–2021). We identified 16 confirmed meningitis cases among 16 070 admissions: 8 of 10 surviving infants with available diagnostic audiology had normal/satisfactory hearing while 2 of 10 had SNHL. Both infants with permanent hearing loss had been born extremely preterm and received potentially ototoxic antimicrobials. Larger studies are needed to clarify whether SNHL occurs mainly due to meningitis itself or to its antimicrobial drug treatment.

Neonatal meningitis is a well-recognised risk factor for sensorineural hearing loss (SNHL),¹ and indicates automatic referral for early auditory brainstem response testing and other formal audiological testing.² There are no recent UK data reporting detailed hearing outcomes of neonates who suffered neonatal meningitis. Our aim was to study the incidence of SNHL in neonates admitted to our tertiary-level neonatal intensive care unit (NICU) with proven meningitis.

We reviewed electronic neonatal records (BadgerNet, Clevermed, UK) and our local microbiology database for the 16-year period 1 January 2006 to 31 December 2021 to identify all neonates diagnosed with unequivocal bacterial or fungal meningitis. We defined proven meningitis cases as those with positive bacterial or fungal growth on culture of cerebrospinal fluid (CSF) obtained from infants with clinical signs of suspected meningitis/sepsis and who had received at least 2 weeks of antibiotic therapy for the episode. We excluded any neonates with meningitis who were never admitted to our NICU, those treated for suspected meningitis despite a negative CSF culture, and cases of viral meningitis. Babies with CSF isolates of a coagulase-negative *Staphylococcus* plus a concomitant CSF leucocyte count $<20 \times 10^6/L$ were considered false positive cases and also

excluded.³ For all confirmed cases, we reviewed microbiological isolates, concomitant blood cultures, CSF cell counts, newborn hearing screening results and later diagnostic audiological testing at 0.5, 1, 2 and 4kHz pure tone audiometry thresholds. SNHL was diagnosed for infants with hearing loss thresholds >20 decibels.⁴

We had 16070 neonatal admissions to our NICU during the 16-year study period. Twenty-eight babies had a culture-positive CSF result and of these 16 were confirmed as definite meningitis cases (table 1). Twelve infants were born preterm and four were born at term. Overall, the definite meningitis rate was 16/16 070 (0.1%), or 1 case per 1000 admissions. Fifteen were bacterial meningitis cases and one fungal. Three babies had meningitis caused by a Coagulase-negative *Staphylococcus*. All except four babies had concomitant blood cultures positive with the same organism. CSF specimens showed very wide variation in white to red blood cell ratios (table 1). The incidence of meningitis was three-fold higher among preterm compared with term admissions, 12/7185 vs 4/8885, $p=0.02$, χ^2 test). Two infants did not undergo newborn hearing screening (1 deceased, 1 contraindicated). Of 14 who underwent the screening, 9 had clear responses and 5 required referral (1 bilateral, 4 unilateral screen fails).

Definitive follow-up audiology outcomes were unavailable for six babies (two died in infancy before completion of diagnostic testing; two had no formal testing despite meningitis history; two failed to attend appointments).

Diagnostic audiology outcome data were available for 10 of the 14 surviving infants: 8 had hearing within normal limits (normal/satisfactory), and 2 have SNHL (1 severe bilateral; 1 moderate mixed bilateral), a rate of SNHL among surviving neonatal meningitis cases of at least 14% (2/14) (95% confidence limits: 2%, 43%). Both SNHL cases were infants born extremely preterm who



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Table 1 Summary of baseline characteristics, CSF isolates and microscopy, and hearing outcomes of the 16 neonates with definite meningitis

Birth gestational age, completed weeks	28 (24–41; 25–38)
Birth weight, grammes	935 (589–3630; 740–2280)
Postnatal age at diagnosis, days	14 (2–89; 8–40)
Isolate, n cases:	
<i>Escherichia coli</i>	5
Group B Streptococcus	5
Coagulase-negative Staphylococcus	3*
<i>Pseudomonas aeruginosa</i>	1
<i>Enterobacter cloacae</i> complex	1
<i>Candida albicans</i>	1
CSF cell counts	
WCC, $\times 10^6/L$	745 (6–7420; 78–3015)
% polymorphs of total WCC	75% (0%–95%; 40%–90%)
RCC, $\times 10^6/L$	2525 (2–21 600; 540–6520)
CSF ratio WCC:RCC	1:4 (1:550 –1:6 $\times 10^{-4}$; 1:135–1:0.32)
Hearing outcomes†, n	
Sensorineural hearing loss	2
Normal	3
Satisfactory‡	5
N/A	6

Data are median (range; IQR).

*1 *Staphylococcus haemolyticus* 1 *Staphylococcus epidermidis*, 1 not further speciated.

†Normal hearing ≤ 20 dB; mild hearing loss 21–40 dB minimum detectable threshold; moderate 41–70 dB; severe loss 71–95 dB; profound loss > 95 dB.

‡Satisfactory hearing, based on otoacoustic emissions and visual reinforcement audiometry with sound field testing down to 25 dB (no audiogram), but unable to rule out mild loss (n=3) or based on tone pip auditory brainstem responses/otoacoustic emissions (no audiogram) (n=2).

CSF, cerebrospinal fluid; N/A, not available; RCC, red cell count; WCC, white cell count.

had suffered meningitis in their second postnatal week: one had fungal meningitis caused by *Candida albicans*; one had bacterial meningitis caused by *Staphylococcus epidermidis*. Their antimicrobial treatments included the potentially ototoxic agents flucytosine, vancomycin and rifampicin.

Our single-centre series covering a 16-year period showed that the incidence of definite meningitis among neonatal admissions was very low overall. SNHL caused by neonatal meningitis (or its treatment) was thus relatively rare, with only two confirmed cases in 16 years in our NICU. However, among cases of proven meningitis, the associated rate of SNHL was not insignificant. A large epidemiological study—for example, one linking meningitis cases contained in large infection surveillance databases with hearing outcomes as logged in national audiological databases—would provide a more accurate indication of SNHL risk after neonatal meningitis. Such linking, along with related biochemical antimicrobial therapeutic drug monitoring data, may also help to clarify whether SNHL occurs mainly due to meningitis itself or to its antimicrobial drug treatment in extremely preterm neonates.

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Contributors PC devised this project. AS, ST, and PC collected clinical data, JML, CC and JF collected audiological data, and CS interrogated the microbiological database. PC and CT reviewed and adjudicated the meningitis cases, and JF provided audiological expertise. PC analysed the data. PC and AS wrote the first manuscript draft and PC wrote the final draft. All authors contributed to manuscript revisions. PC is guarantor.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent for publication Not applicable.

Provenance and peer review Not commissioned; internally peer reviewed.

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