

A REVIEW OF HEARING IMPAIRMENT DUE TO BACTERIAL MENINGITIS IN CHILDREN: IMPORTANCE OF EARLY DIAGNOSIS AND TREATMENT

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Abstract

This review underlines the significant role of early diagnosis and treatment of hearing impairment due to bacterial meningitis (BM) in children. BM is a life-threatening neurological condition mostly caused by *Streptococcus pneumoniae*, *Neisseria meningitidis*, or *Haemophilus influenzae*. Hearing loss is the most commonly reported neurological complication of BM; inflammation can damage the inner ear, leading to sensorineural hearing loss or complete deafness. Factors favoring neurological complications, including hearing impairment, are low age, immaturity of the immune system, poor health, anaemia, leukocytosis, and hypoglycemia. To avoid serious complications, quick intervention is necessary – administration of antibiotic in combination with dexamethasone. It is also important to conduct regular audiological tests to monitor hearing, not only immediately after BM, but also in the long term. Otoacoustic emissions (OAEs) and auditory brainstem responses (ABRs) are useful tools, and some researchers also recommend tympanometry. CTs and MRIs are important to visualize the condition of the inner ear after BM, paving the way for qualification for implantation and for pre-operative planning. The best results of implantation come from patients without ossification, whose period of deafness was short, where electrodes were inserted deeply, and who did not have neurological complications after BM. Early implantation is the best option as it promotes proper speech development and allows the child to adapt to their environment.

Key words: bacterial meningitis • hearing loss • deafness • cochlear implants

USZKODZENIE SŁUCHU W PRZEBIEGU BAKTERYJNEGO ZAPALENIA OPON MÓZGOWO-RDZENIOWYCH U DZIECI – ISTOTA WCZESNEJ DIAGNOSTYKI I LECZENIA. PRACA PRZEGLĄDOWA

Streszczenie

W niniejszej pracy przeglądowej podkreślono istotną rolę wczesnego rozpoznania i leczenia uszkodzenia słuchu spowodowanego bakteryjnym zapaleniem opon mózgowo-rdzeniowych (ZOMR, ang. *bacterial meningitis*, BM) u dzieci. BM to zagrażający życiu stan neurologiczny, powodowany głównie przez *Streptococcus pneumoniae*, *Neisseria meningitidis* lub *Haemophilus influenzae*. Utrata słuchu jest najczęściej zgłaszanym neurologicznym powikłaniem BM – stan zapalny może uszkodzić ucho wewnętrzne i doprowadzić do niedosłuchu zmysłowo-nerwowego (odbiorczego) lub całkowitej głuchoty. Czynniki sprzyjającymi powikłaniem neurologicznym w tym uszkodzeniu słuchu są: wiek dziecięcy, niedojrzałość układu immunologicznego, zły stan zdrowia, niedokrwistość, leukocytoza i hipoglikemia. Aby uniknąć poważnych powikłań, konieczna jest szybka interwencja – podanie antybiotyku w skojarzeniu z deksametazonem. Istotne jest również prowadzenie regularnych badań audiologicznych w celu monitorowania słuchu, nie tylko bezpośrednio po BM, lecz także w dalszej perspektywie. Otoemisje akustyczne (OAE) i słuchowe potencjały wywołane pnia mózgu (ABR) są użytecznymi narzędziami do oceny słuchu, a niektórzy badacze rekomendują także tympanometrię. Tomografia komputerowa (CT) i obrazowanie metodą rezonansu magnetycznego (MRI) są istotne do wizualizacji stanu ucha wewnętrznego po bakteryjnym ZOMR i umożliwiają ocenę możliwości implantacji i planowanie przedoperacyjne. Najkorzystniejsze wyniki uzyskują pacjenci, u których nie zakończył się jeszcze proces kostnienia, u których czas trwania głuchoty był krótszy, u których elektrody zostały wszczepione głębiej i którzy nie mieli innych neurologicznych powikłań po bakteryjnym ZOMR. Odpowiednio wczesna implantacja pozwala u dziecka na prawidłowy rozwój mowy i przystosowanie się do środowiska.

Słowa kluczowe: bakteryjne zapalenie opon mózgowych • utrata słuchu • głuchota • implanty ślimakowe

Introduction

Bacterial meningitis (BM) in children can cause damage to the auditory system, leading to hearing impairment involving persistent sensorineural deafness [1]. Immediate treatment can prevent serious complications, and longer term treatments may still be needed [2–5]. In this review, we focus on hearing loss as a complication of BM in children – its epidemiology, risk factors, diagnosis, treatment, and the role for cochlear implants.

Nature of BM in children

BM initiates inflammation, including that of the pia mater, arachnoid mater, and the subarachnoid spaces [1]. The most common pathogens causing BM (90% of cases in children) are *Streptococcus pneumoniae*, *Neisseria meningitidis*, and *Haemophilus influenzae* (Hib) – most often in young children and the elderly. Due to its seriousness (a life-threatening neurological emergency) and potentially many complications (hearing impairment and other neurological deficits such as cognitive impairment, motor or sensory nerve deficits, psychiatric disabilities, and even death), it is necessary to quickly administer an antibiotic that crosses the blood-brain barrier (BBB) [2–5].

Despite vaccines against the bacteria causing meningitis (which have led to a significant reduction of frequency of the disease), there are still cases of this disease worldwide, with high risk of paediatric morbidity and mortality [6,7]. In the United States, the incidence of BM is 0.2 to 3.7 cases per 100,000 children [8]. According to Oordt-Speets [9], the frequency of *S. pneumoniae*-induced BM ranges from 22% (Europe) to 41% (Africa), indicating a higher rate of BM in less developed countries. In Africa, bacterial meningitis is most often caused by *E. coli* and *S. pneumoniae*. In Europe, the highest number of cases involve children aged 1–5 years [9]. Estimates made by Baud and Aujard [10] give a mortality rate of 10–15% and complications of 20–50%, with the figure depending on how quickly treatment can be initiated and the type of microorganism involved.

The clinical manifestations of BM are rapid and acute. It classically starts with a fever, headache, or stiff neck, although multiple symptoms may occur. In addition, there may be nausea, vomiting, photophobia, or seizures. Some patients may have positive Kernig and Brudzinski symptoms (although they are not very sensitive symptoms), and increased intracranial pressure may cause damage to the cranial nerves (III, IV, VI, and VII) or Cushing's triad (hypertension, bradycardia, and irregular breathing) [11,12].

Due to the severity of the disease, prompt diagnosis and beginning antimicrobial therapy (immediately after blood collection) is essential. In newborns, the most common combination is ampicillin or cephalosporins (3rd generation, usually cefotaxime) and gentamicin; in infants and those under 50 years, vancomycin with ceftriaxone. After the result of a blood culture are received, adjustment should be made to the most appropriate antibiotic. Additionally, imaging tests should be performed (MRI or CT are preferable) to visualise cerebrovascular lesions and ventriculomegaly [6,10,11,13,14]. Sometimes

cranial ultrasonography (CUS) and electroencephalography (EEG) are helpful [6,10]. A diagnosis of probable BM is made from a positive cerebrospinal fluid (CSF) or blood culture result, while a final diagnosis is made after lumbar puncture and testing of the obtained CSF. Treatment should continue for 3 weeks from the first negative CSF culture [10].

Symptoms of bacterial and viral meningitis can overlap. Posadas believes [15] that determination of procalcitonin (a marker of inflammation) in serum and CSF may be helpful to distinguish bacterial from viral and aseptic etiology; Shorbagy is of a similar opinion [16] after finding significantly higher levels of procalcitonin in patients with BM than in patients with aseptic meningitis. Rapid responses are essential, and delayed treatment may have serious health consequences. BM requires aggressive treatment with antibiotics and supportive therapies (corticosteroids: dexamethasone and also new agents undergoing clinical trials, C5 inhibitors and daptomycin) [15,17,18]. Patients with BM need to be closely monitored in ICUs [11].

Epidemiology and risk factors for hearing loss

Hearing loss is the most commonly reported neurological complication of BM [19]. According to Jatto [1], neurological impairment in children after BM occurs in 3–47% of them; however, for hearing loss alone (sensorineural hearing loss, SNHL) the figure rises to 60–90% of children. Zainel finds that neurological complications develop in 71% of infants with BM, 38% of children up to 5 years of age, and 10% in the range 6–16 years [19]. Approximately 10% of children develop unilateral or bilateral hearing loss, and severe or profound bilateral hearing loss occurs in 5% [19]. In the Kenyatta National Hospital, prevalence of hearing loss after BM was reported to be 43% [20]. The highest incidence of neurological complications of meningitis, including hearing loss, is observed in children under 12 months of age [19,21], although one report [20] found that age did not seem to be a factor. Infection with *S. pneumoniae* appears to be the root cause of hearing loss in infants with BM, while Hib is a major risk factor in children over 12 months of age. Another report found a risk of hearing loss from BM caused by *S. pneumoniae* to be 11%, whereas the figure was 5% from Hib or *N. meningitidis* infections [22]; in comparison, later work found the risk to be 14–32% from *S. pneumoniae*, 4–23% from *N. meningitidis*, and 20% from *H. influenzae* [19]. The major risk factors for BM complications are immaturity of the immune system and the poor clinical condition of the patient [22]. Jatto believes that the main risk factors for hearing impairment are anaemia, leukocytosis, and hypoglycemia [1]. There are also direct relationships between the results of CSF analysis and the occurrence of SNHL in infants with acute BM: the predisposing factors are pleocytosis, raised protein, and decreased glucose in the CSF [23]. Immediate provision of antibiotics significantly reduces the risk of neurological complications, while in Hib-induced BM early corticosteroid treatment appears to reduce the risk of hearing loss and neurological complications [19]. There is no correlation between gender and the occurrence and severity of hearing loss [20].

Diagnosis of hearing loss in BM

Audiological tests may include behavioral audiometry, otoacoustic emissions (OAEs), and auditory evoked potentials (AEPs), including auditory brainstem responses (ABRs) and auditory steady-state responses (ASSRs). In children over 2.5 years of age, behavioral audiometry can be used to assess hearing in each ear separately, but it is a subjective test; in younger children, behavioral audiometry with conditioning can be performed [24,25].

If screening includes otoacoustic emissions (OAEs), one needs to consider that in children with simultaneous meningitis and otitis media, there can be a lack of response even when there is no SNHL [24]. One study showed that OAE with tympanometry followed by ABR is the best combination in BM [25]. Abed and colleagues also favour OAE and ABR [28]. Other work has shown that the degree of SNHL cannot be assessed with OAEs [26]. According to Jadia, OAE is an essential screening test, including in BM, allowing for quick intervention in children [27].

Auditory evoked potentials (AEPs), which can test hearing thresholds at high frequencies, is an objective test that is considered the gold standard, but it is time-consuming and needs to be carried out while the child is asleep. New techniques (also objective and requiring the same conditions as AEP) include the auditory steady state response (ASSR), which involves an audiogram to assess hearing thresholds [24,25].

CT and MRI are important preoperative planning tools and necessary for qualifying children for implantation [24,29]. In the opinion of Yan [30], HRCT and MRI are of comparable value in predicting cochlear ossification (HRCT, 53%; MRI, 59%). According to Zhang [31], HRCT allows for better imaging of bone structures and the middle ear, while MRI enables better visualization of intracochlear fluid, semicircular canal, vestibulocochlear nerve, and the cerebellopontine angle. It seems that a combination of MRI (by itself giving 82% sensitivity) and HRCT (62%) allows for greater sensitivity, and in one study the combination gave a figure of 92%.

Persson and colleagues [32] noted that as many as one-third of patients, after recovery, did not undergo an audiological test; they suggest that otoscopy and audiological tests should be undertaken in all patients who have been admitted to hospital with BM. Rodenburg-Vlot [33] also think that audiological tests should be carried out in all children immediately after the end of the acute phase of the disease and that the tests should be repeated if the first result is incorrect. They say that audiological testing is important even for children who do not complain of hearing problems. One Dutch group recommends audiological tests after 1, 2, 6, and 12 months, even if the first test looks clear; they further suggest that all patients with SNHL above 30 dBHL should be referred for testing so that there can be quick detection and timely intervention if profound hearing loss occurs [34]. In 2008, the French-speaking Society of Infectious Pathology issued recommendations that each patient with BM should undergo an audiological examination before leaving hospital, and at the latest within 15 days from the end of

treatment [24]. Worsøe and colleagues [35] suggest that audiometry should be performed in all patients with pneumococcal meningitis. Bilateral impairment appears to increase the risk of becoming deaf [1,35].

Kuschke [36] emphasises the importance of conducting audiological tests in children after BM. A quick referral to an audiologist is needed because hearing loss from BM can lead to ossification of the cochlea, which can make implantation difficult or impossible. However, the same work found that the average time between BM recovery (from a paediatric tertiary hospital in Cape Town, South Africa) to an audiology visit was actually 17 weeks, which is not in keeping with the seriousness of the disease. In fact, testing by tonal audiometry, otoacoustic emissions, tympanometry, and ABR showed that one-third of the children had severe or profound SNHL. Late diagnosis and slow referral to an audiologist may have worsened the children's hearing loss and possibly created delays in qualifying them for cochlear implants. The workers also emphasise the important role of rehabilitation in the form of oral and auditory communication [36].

In related work, Saha and colleagues [37] tested OAEs in children after BM; ABRs and tympanometry were also performed. Testing showed a hearing loss of 40 dB or more. There was a hearing deficit in 33% of children 30–40 days after discharge and in 18% some 12–24 months from discharge. The investigators point to the importance of hearing screening in children, especially after BM. Zeeshan and co-workers [38] performed a study in 2018 on 175 children diagnosed with BM (aged from 1 month to 13 years). Two weeks after admission, children with type A tympanograms had their hearing assessed using otoacoustic emissions; sensorineural hearing loss was diagnosed if there was an absence of OAEs. The scientists found that hearing impairment was strongly correlated with severe cases of the disease. The OAE screening reflects the functional state of the inner ear, although it does not allow the degree of hearing loss to be determined [38]. A similar study by Jatto showed that one-third of children with BM who passed an OAE test (10% of 183 ears) had normal ABR hearing thresholds [1]. The difference in hearing thresholds in children after meningitis (the research group) and in those without the disease (a control group) was significant.

According to Roine [39], hearing impairment in children develops soon after BM and rarely returns to its previous level of function. In their study, the scientists observed considerable hearing impairment in post-meningitis children: ABR showed that 41% of those were affected by hearing impairment. Some 10% of the ears had a temporary hearing loss from less than 60 dB to over 80 dB, and 7% had a permanent loss. Improvements were found in 22% of ears from above 80 dB to below 60 dB, and in half of the ears with damage above 80 dB. In 68% of children, the evolution of hearing thresholds was the same in both ears, while in the others the evolution differed [39]. Karppinen and colleagues [40] tested the ABRs of children following BM. They found the typical ABR wave at 80 dBHL was absent in 48% of patients. No correlation was found between an absent ABR and severe neurological complications in the studied group of children. There was a correlation between

long latencies (waves III or V) and mortality and neurological complications. Thus, according to Karppinen, the ABR is the most important test in the prognosis of BM, including hearing loss [40].

In the opinion of De Barros and colleagues [41], bilateral deafness may appear immediately or several months after BM, so hearing screening for 2 years after the disease is crucial. While an ABR can confirm changes seen in audiometric tests, an MRI should also be performed in cases of severe or profound hearing loss, in order to quickly detect inflammation or visualise cochlear ossification and to perform early cochlear implantation.

According to Kapelovich and co-workers [42], gadolinium-enhanced MRI is highly effective and allows one to detect early-stage *labyrinthitis* and assess whether there is likely to be future hearing loss after BM. This opinion is shared by Orman, who evaluated the accuracy of MRI in predicting the development of SNHL in infants suffering from BM [43]. T1-weighted MRI with contrast and FLAIR hyperintensity were used to visualise inner ear abnormalities. In the prognosis of SNHL, good accuracy was found between the T1-weighted assessment and hyperintense FLAIR (both methods have high specificity, 94–96%, and moderate sensitivity, 51–60%). Disturbing factors may include low glucose levels and high protein levels in CSF, but nevertheless these imaging modalities can be extremely helpful in predicting the development of SNHL [43].

Treatment of hearing loss in BM

Inflammation releases nitric oxide, superoxides, and peroxynitrites, which have cytotoxic effects on the cochlea, leading to damage (by breaking the blood-labyrinth barrier). Obstruction of the blood vessels reaching the inner ear (embolism or thrombus) may lead to further destruction and nerve damage as a result of ischemia and hypoxia [1,9].

Guidelines from 2016 by the European Society of Clinical Microbiology and Infectious Diseases (ESCMID) and the Study Group on Infections of the Brain (ESGIB) recommend that, in any patient with suspected BM, antibiotic treatment be started within 1 hour of admission to hospital (the choice of antibiotic depending on age and risk factors) [44]. According to ESCMID, it is advisable to administer dexamethasone at the same time as antibiotics. ESCMID also advise early assessment of the ear, nose, and throat to detect hearing loss [44]. According to a Cochrane systematic review in 2015, corticosteroids significantly reduce hearing loss and other neurological complications, although they do not reduce the overall mortality from BM [45]. In the subgroup of people with severe hearing loss, the first dose of antibiotic appears to have a slightly better effect than a first dose of corticosteroid (dexamethasone, hydrocortisone, or prednisone), although the difference was not significant. Adjunctive corticosteroids gave a remarkable reduction in the frequency of hearing loss, although they do not affect the incidence of long-term neurological complications and the use of dexamethasone is associated with a higher risk of recurrent fever [45]. This opinion is shared by Wang [46] whose work showed that dexamethasone reduced the risk of hearing loss and

serious neurological complications, although it did not affect the mortality rate of children with BM. The same work showed that dexamethasone decreased the risk of hearing loss more effectively than did antibiotics [46]. It seems that dexamethasone (even before antibiotic therapy) suppresses ossifying labyrinthitis and progressive ossification of the lumen of the cochlea, both of which limit the treatment options and subsequent disability in children [42,47]. Work by Esposito [48] demonstrated good results of dexamethasone (given before or with the first dose of antibiotic) in preventing Hib-induced BM hearing loss in children. The use of dexamethasone in pneumococcal BM should be considered whenever there is late admission to hospital, as antibiotics may be ineffective, especially if there is cephalosporin resistance to pneumococci. Dexamethasone does not appear to be effective in meningococcal BM, but more studies are needed in larger groups of patients [48].

In a study on a group of children treated with ceftriaxone after BM, it has been shown that, in adjunctive therapy, dexamethasone and glycerol did not prevent hearing loss [49]. Major predictors of hearing loss are the child's age (each additional month of life reduced the risk of hearing loss by 2%) while the time of antibiotic administration did not affect the hearing outcome [49]. Another study used acetaminophen as adjunctive therapy, and claimed that it had little or no effect on hearing loss in BM, although it may increase the risk of other neurological complications [50]. Clearly, more work is required.

Turning to non-corticosteroid adjuvants – immunoglobulins, heparin, pentoxifylline, and mixtures of succinic acid, inosine, nicotinamide, and riboflavin mononucleotide – which have been tried by various research groups, the results are inconclusive [50]. Apart from immunoglobulins, these drugs also tend to cause allergic reactions in some patients. It has been noted [51] that, for pneumococcal meningitis in mice, combining daptomycin with an anti-C5 antibody is more effective than ceftriaxone with dexamethasone: the treatment reduced the acute stage of hearing loss in BM as well as improved cognitive function and disease symptoms. At the same time, daptomycin combined with anti-IL-1 β antibody or rosovitin gave no significant effects on hearing, but did reduce CSF inflammation [51].

Early cochlear implantation in children after BM

Currently, the only treatment for post-meningitis deafness is cochlear implantation. Association of BM with ossification of the cochlea was found in one study which highlighted the role of pneumococci and meningococci [52]. Cochlear ossification can be divided into 3 stages according to CT results: grade A indicates the cochlea shows no signs of ossification; grade B indicates a degree of ossification but covering no more than two-thirds of the lumen; and grade C indicates complete ossification [52,53]. Similar stages have been described by [30], while another recent work distinguished four stages [54]. Some work has found a higher impedance in implanted children with deafness after BM compared to a control group (deaf implanted children who did not have BM), regardless of the degree of ossification of the cochlea [52].

The goal of cochlear implantation is better speech and hearing performance, and the faster the implantation after BM, the better the audiological result [55]. For optimal results, account needs to be taken of the child's age and how long they have been deaf. Children with cochlear ossification have worse speech perception and a poorer prognosis than children without ossification [55]. One systematic review [56] found significantly better audiological outcomes in all studies compared to pre-intervention. However, it found that the patients who benefited the most were those without cochlear ossification, who had been deaf only a short time, who had deep electrode insertion, and who had no neurological complications after BM. These children had good speech intelligibility and a hearing threshold much better than those who had cochlear ossification. Early bilateral implantation appears to be the most effective treatment [30,56], especially in children without mastoidectomy [57].

Zhang and colleagues [31] maintain that, since deafness may promote ossification in the inner ear, implantation should be considered in all cases of profound SNHL. Ossification is a problem as it can make deep electrode insertion impossible, thus preventing the implant from working at its best. Effective performance of an implant depends on the appropriate number of electrodes being inserted: for Med-El at least 8 pairs of electrodes and up to 12, for Cochlear 10 to 22, and for Advanced Bionics 9 to 16. Partial implantation is likely to give poor results. This points to the need for regular audiological examinations and imaging of the inner ear (MRI, HRCT, or both), allowing for early intervention, especially since ossification can occur fairly quickly [31].

Teissier and co-workers [24] audiotically tested 283 children who had received cochlear implants, 16 of whom had suffered BM before implantation. After the disease, SNHL occurred about 8.3 months later on average (median 1.5 months), while implantation was performed some 2 years and 3 months after infection (median 7 months). Prior to implantation, all 16 children had severe to profound deafness (threshold above 80 dB). Before implantation, cochlear or vestibular ossification was seen in 12 of the 16 children examined by CT. After implantation, most of the children (11) had near to normal speech intelligibility, while 5 children had poorer results. The investigators emphasise the importance of regular audiological testing for detection of SNHL, since patients are likely to experience SNHL some time after the disease (range 1 month to 13 years). It is important to perform implantation before ossification occurs. If hearing worsens, an MRI is required [24].

Kazemi and colleagues [58] assessed patients unilaterally implanted after BM using the Categories of Auditory Performance (CAP) and Speech Intelligibility Ratings (SIR). Post-implantation, the mean CAP and SIR scores improved significantly over time. Cochlear implants bring many benefits, but the authors point out that ossification is the main cause of complications and presents a challenge for surgeons [58]. In a related study by Nichani [59], cochlear implantation was performed in children under 16 years of age who became deaf after BM, looking for differences between those with ossification compared to

those without. Outcomes in the group with ossification (27 children) and without (25) were assessed on the basis of the CAP scale and the Manchester Speech and Language Development scale. For the children without ossification, 22 had a hearing performance rating of 5 or more and 19 children were able to use simple phrases (3 or more words) according to the Manchester scale. The children with partial ossification all received a standard electrode. But in those with gross ossification, 3 children needed a scala vestibuli insertion, 7 needed split electrode insertion, and in 3 children only partial electrode insertion was possible. The authors conclude that implantation of children with SNHL after BM brings enormous benefits, even in children with gross cochlear ossification. They also mention that bilateral implantation is necessary due to the risk of obstructive osteoneogenesis [59].

Philippon and colleagues [60] recommend early bilateral cochlear implantation if children become deaf after BM. They find that fibrosis in the inner ear can lead to ossification in the future. It is assumed that early cochlear implantation and thus early electrical stimulation allows greater survival of neurons in the spiral ganglion. Implantation can partially reverse neuronal degeneration. They note that bilateral implantation is preferred because it allows better sound localisation and understanding of speech in noise [60]. A related study by Yan [30] showed that bilateral cochlear implantation after 6 months allowed children to achieve an average hearing threshold of 30 dBHL (in stages I and II of cochlear ossification) while for those with stage III there was an average level of 40 dBHL (for comparison, before implantation, the mean threshold was 96 dBHL). In stages I and II, CAP scores were similar, but in stage III they were significantly lower. The SIR results in children with prelingual deafness were comparable in stages I and II, but higher in the case of postlingual deafness – hence the age at which the child suffers BM is significant [30]. While cochlear implants play an important role in restoring sound perception and speech detection, work by de Brito [61] showed that those children whose hearing loss was due to BM did not achieve, a year after receiving an implant, such good results in speech recognition tests (open and closed sets) as children who had received implants for other reasons. According to Liu [62], labyrinthitis ossificans can adversely affect speech perception after implantation, but other preimplantation (and pre-BM) factors can also play a part, including neurological deficits, age at deafness, and length of deafness – further research is needed.

Farinetti [63] claims that cochlear implantation should be considered in all people with severe or total hearing loss because of its safety and low complication rate. However, there are obstacles in children with infections like BM, but familiarising doctors with them can often avoid complications – Farinetti estimates that 15% will have minor complications, 5% serious ones, and that 43% are associated with implant dysfunction. In case of device failure, sometimes reimplantation is necessary. In general, bilateral implantation in children after BM is recommended [63]. Rubin and Lorry [64] emphasise the importance of vaccinations in children who have been qualified for cochlear implantation or who have already received a cochlear implant; vaccines against pneumococci, Hib, and influenza

are particularly recommended due to such children having greater susceptibility to infections.

As a result of BM, there may be impairment of vestibular function as well as hearing. If it affects gait, it may impair or delay post-motor development. Therefore, if a child becomes unsteady after BM, vestibular assessment is essential [65].

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Conclusions

Bacterial meningitis often damages children's hearing, and a child suffering BM should be regularly tested audiotically in order to check whether there is a possible deficit. The testing should be conducted not only immediately after the disease but also long term, as research has shown that hearing impairments can develop months or years after the disease. Regular testing will allow quick intervention, possibly with a cochlear implant, should cochlear ossification occur. Implantation is expected to provide good sound perception and speech development, allowing the child to function normally and adapt to their environment.

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