ORIGINAL ARTICLE

Neonatal meningitis: Diagnostic and therapeutic challenges

Les méningites néonatales : Défis diagnostique et thérapeutique

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Abstract

Aim: To describe the clinical, biological, microbiological, and radiological aspects of neonatal meningitis (NNM) as well as its treatment and outcome and to compare between bacterial NNM and viral NNM.

Methods: A Retrospective study of NNM cases, conducted at Pediatric Medicine B department of Bechir Hamza Children's Hospital over a 15-year period (2004-2018).

Results: Forty-seven newborns with meningitis were included. The average age was 15 days (range: 0,62–27 days). The main reasons for consultation were fever (81%), seizures (13%), poor feeding (15%) and digestive symptoms (15%). Physical examination revealed axial hypotonia (26%) and bulging anterior fontanel (4%).

The C-reactive protein level was elevated in 32% of cases with a mean value of 81.5 mg/L. A C-reactive protein value greater than 50 mg/L was statistically associated with bacterial NNM ($p<0.001$).

The analysis of clinical and microbiological data identified 26 cases (55%) of bacterial NNM and 21 cases (45%) of viral NNM. Polymerase chain reaction detected enterovirus genome in two cases.

Bacterial NNM were complicated by hydrocephalus (n=3) and ventriculitis (n=4). Compared to the viral NNM group, the bacterial NNM group had a significantly lower mean age ($p=0.02$), while prematurity, axial hypotonia and grunting were significantly more frequent ($p<0.05$).

Conclusions: Viral NNM are generally benign and rarely require a specific treatment, but they are often underdiagnosed due to the lack of systematic Polymerase Chain Reaction testing in our country. Bacterial NNM are more severe, may lead to neurological sequelae, and are rarely well documented.

Key-words: Newborn, Meningitis, Cerebrospinal fluid, Antibiotherapy.

Résumé

Objectif: Décrire les caractéristiques cliniques, biologiques, microbiologiques, thérapeutiques et évolutives des méningites néonatales (MNN) et de comparer ces caractéristiques entre les MNN bactériennes et virales.

Méthodes: Il s'agissait d'une étude descriptive rétrospective des dossiers des nouveau-nés hospitalisés pour MNN dans le service de Médecine Infantile B, sur une période de 15 ans (2004-2018).

Résultats : Quarante-sept nouveau-nés d'âge médian de 15 jours ont été colligés. Les motifs de consultation étaient une fièvre (81%), des convulsions (13%), un refus de tétée (19%) et des signes digestifs (15%). L'examen a noté une hypotonie axiale (26%) et une fontanelle antérieure bombante (4%). La C-Reactive protein était élevée dans 32% des cas avec une moyenne de 81,5 mg/l. L'analyse des données clinico-microbiologiques a permis d'identifier 26 cas de MNN bactérienne (55%) et 21 cas de MNN virale (45%). La Polymerase Chain Reaction a détecté le génome de l'entérovirus dans deux cas. Les MNN bactériennes se sont compliquées d'une hydrocéphalie (N=3) et d'une ventriculite (N=4). Comparativement au groupe des MNN virales, dans le groupe des MNN bactériennes l'âge moyen était significativement plus jeune ($p=0,02$), alors que la prématurité, l'hypotonie axiale et le geignement étaient significativement plus fréquents ($p<0,05$). Une C-Reactive protein supérieure à 50 mg/l était statistiquement plus fréquentes dans le groupe des MNN bactériennes ($p<0,001$).

Conclusions: Les MNN virales sont bénignes et nécessitent rarement un traitement spécifique, mais elles sont sous estimées en l'absence de Polymerase Chain Reaction systématique. Les MNN bactériennes, plus graves et pourvoyeuses de séquelles neurologiques, sont rarement documentées.

Mots clés: Nouveau-né, Méningite, Liquide céphalo-rachidien, Antibiothérapie

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Introduction

Neonatal meningitis (NNM) is rare. However, among all pediatric age groups, the neonatal period presents the highest incidence of meningitis (1). In 2010, Heath and Okike estimated the global incidence of bacterial NNM to range between 0.22 and 6.1 per 1,000 live births. Moreover, viral meningitis, particularly those caused by enteroviruses, has long been underrecognized worldwide due to the lack of effective diagnostic tools, although it appears to be frequent in newborns (2).

NNM is a serious infection. It carries a high risk of mortality, threatening the patient's life, and may also result in severe neurosensory and psychomotor sequelae, compromising long-term neurological outcomes.

While antibiotic therapy has significantly reduced the mortality associated with NNM over the past two decades, its long-term morbidity remains unchanged. Clinically, NNM presents non-specific and often misleading symptoms. Diagnosis relies on the biochemical and microbiological analysis of cerebrospinal fluid (CSF). Brain imaging is essential for detecting complicated forms, which may require prolonged treatment and, in some cases, neurosurgical intervention.

In Tunisia, the therapeutic management of NNM remains hindered by the lack of unified national protocols, particularly for complicated meningitis or cases without bacterial identification. This leads to difficulties in therapeutic decision-making and results in heterogeneous management approaches.

Methods

This was a descriptive, cross-sectional, and retrospective study conducted over a 15-year period, from January 1, 2004, to December 31, 2018. All medical records of newborns hospitalized during the study period in the Department of Pediatric Medicine B at Béchir Hamza Children's Hospital in Tunis, with a confirmed diagnosis of NNM, were analyzed. Patients with central nervous system malformations were excluded.

The diagnosis of NNM was based on the presence of clinical signs suggestive of meningitis combined with the following cerebrospinal fluid (CSF) abnormalities: pleocytosis with more than 20 white cells/mm³, CSF protein level >1.3 g/L, and/or hypoglycorrhachia with a CSF-to-blood glucose ratio < 0.3 (3).

Bacterial NNM was confirmed by the identification of bacteria on direct CSF examination or culture, by the detection of bacterial soluble antigens (AgS) in CSF, or by a positive blood culture. Viral NNM was confirmed by the detection of a virus in the CSF via polymerase chain reaction (PCR). In the absence of microbiological evidence, the bacterial or viral origin of the NNM was determined based on the clinical and biological presentation, as well as CSF cellular and biochemical characteristics.

In addition to the above criteria defining NNM, we established presumptive criteria for a likely bacterial origin. A diagnosis of probable bacterial NNM was made when at least two of the following were present: sepsis or hemodynamic instability, neurological distress, suspected site of bacterial infection, CSF pleocytosis with a predominance of neutrophils (≥70%) or mixed cellularity (30–70% neutrophils), and biological signs of inflammation (elevated C-reactive protein (CRP), leukocytosis, or leukopenia). In the absence of these criteria, a probable viral origin of the meningitis was considered.

Bacterial NNM was classified as early-onset when symptoms appeared between birth and day four of life, and as late-onset when clinical signs appeared between day five and day 28 of life (4).

Data were analyzed using SPSS software version 22.0. We performed a univariate study. Comparisons between two means were performed using the Student's t-test for independent samples or the Mann–Whitney test for small sample sizes. Comparisons of percentages were carried out using Pearson's chi-squared test or Fisher's exact test. A p-value < 0.05 was considered statistically significant. The literature review was conducted using keywords on PubMed, ScienceDirect, and Google Scholar. Anonymity was preserved during data

Results

Forty-seven cases of NNM were recorded, including 30 boys and 17 girls, yielding a sex ratio of 1.76. The average annual incidence of NNM in the study department was 3.13 cases/year. The median age at admission was 15 days (range: 0.62–27 days). Pregnancy follow-up was inadequate in 87% of cases (n=41). Pregnancy was complicated by preeclampsia in 6% of cases (n=3) and gestational diabetes in 13% (n=6). The mean gestational age at birth was 37.9 weeks of amenorrhea (WA), ranging from 31 to 42 WA. Prematurity was noted in eight newborns: spontaneous in five cases and induced in three. The average birth weight was 3248 grams (range: 1600–4670 g), with low birth weight (< 2500 g) recorded in eight newborns (17%).

A positive infectious history was found in 9 cases (19%), including premature and/or prolonged rupture of membranes (≥ 12 hours) in four cases, maternal fever over 38°C during labor in five cases, inadequate maternal antibiotic treatment in four cases, elevated maternal CRP in two cases, and chorioamnionitis in one case. The average duration of symptoms before hospitalization was 34.3 hours (ranging from 2 hours to 6 days), with a median of 24 hours.

The reasons for consultation were fever (81%), hypothermia (6%), seizures (13%), feeding refusal (19%), grunting (6%) and digestive symptoms (15%). Neurological signs were absent at history-taking in 64% of cases. Physical examination revealed axial hypotonia (26%), a bulging anterior fontanel (4%), respiratory distress (6%), and poor hemodynamic status in one case (2%).

Biological findings showed leukocytosis in one case, leukopenia in two cases, and thrombocytopenia in three. CRP was elevated in 32% of cases, with a mean value of 81.5 mg/L (range: 23.7–156.6 mg/L). Procalcitonin was measured in 9% of patients (n=4) and was negative in all

cases, with a mean value of 0.13 µg/L (range: 0.05 to 0.19 µg/L).

As for the microbiological results, lumbar puncture (LP) was performed while the newborn was already on antibiotic treatment in 32% of cases (n=14). In 6% of cases (n=3), the CSF was hemorrhagic, and cell counts could not be assessed. The characteristics of CSF analysis are shown in table 1.

Table 1. Cytological and biochemical characteristics of cerebrospinal fluid in newborns treated for neonatal meningitis

Characteristic		Results (N=47)
Liquid appearance	Clear	15
	Cloudy	14
	Hematic	15
	Hemorrhagic	3
Mean cell count (cells/mm³)		564 (Range : 3 to 6 000)
Cell formula	Neutrophil predominance	15
	Lymphocyte predominance	17
	Mixed formula	12
Elevated CSF* protein (g/L)		12 (Mean= 4.08; Range : 1.6 to 6.57)
CSF*/serum glucose ratio < 0.3		10

***CSF** : Cerebrospinal fluid

Direct CSF examination was positive in 6% of cases, CSF culture isolated a pathogen in 15%, and blood cultures were positive in 9%. The identified pathogens were E. coli (3 cases), Klebsiella pneumoniae (2 cases), Group B Streptococcus (1 case), Pseudomonas aeruginosa (1 case), and Proteus mirabilis (1 case). Review of the medical records revealed no evidence of nosocomial infection, and transmission during delivery could not be assessed due to insufficient peripartum bacteriological data. PCR was performed in 11% of cases (n=5), detecting enterovirus genome in two cases (2/5).

A total of 26 cases of bacterial NNM (8 documented) and 21 cases of viral NNM (2 documented) were identified. Among bacterial cases, 9/26 were early-onset and 17/26 were late-onset.

The first-line antibiotic therapy most commonly prescribed was the combination of ampicillin (200 mg/kg/day), cefotaxime (200 mg/kg/day), and gentamicin (3–5 mg/kg/day). This regimen was administered in 19 out of 26 bacterial NNM cases and in 15 out of 21 viral NNM cases.

For bacterial NNM, antibiotic treatment was adjusted based on clinical evolution and bacteriological results. Fluoroquinolones were added in 6 out of 26 newborns, with an average duration of 14.63 days (range: 5 to 35 days). Ciprofloxacin was prescribed prophylactically for five days in three neonates, based on antibiogram results: ESBL-producing E. coli in two, and multidrug-resistant Pseudomonas aeruginosa in one, sensitive to ceftazidime and ciprofloxacin. The average total duration of antibiotic treatment was 22 days (range: 10 to 60 days). Complications included ventriculitis (4 cases), hydrocephalus (3 cases), intraventricular hemorrhage (3 cases), and cerebral venous thrombosis (1 case).

For viral NNM, antibiotics were administered during hospitalization in 18 newborns, with an average duration of 6.7 days (range: 1 to 15 days). Three newborns did not receive any antibiotics. Herpetic meningitis was suspected in two newborns who received intravenous acyclovir at a dose of 20 mg/kg three times daily. The treatment was discontinued after 10 and 12 days, respectively, once CSF PCR for herpes was found negative.

No deaths were reported. Medium- and long-term follow-up was conducted in the research department for 23 patients, for 11/26 patients with bacterial NNM (average follow-up of 10.5 months) and for 12/21 patients with viral NNM (average follow-up of 5.5 months). This follow-up revealed neurological sequelae in newborns treated for bacterial NNM (n=6/26), including psychomotor delay in four cases and epilepsy in three. No sequelae were reported in the viral NNM group.

Comparison between bacterial and viral NNM groups revealed several statistically significant differences (Table 2).

Table 2. Comparison of clinical, biological, microbiological, and outcome characteristics in bacterial and viral neonatal meningitis

Characteristics		Bacterial Meningitis (N = 26)	Viral Meningitis (N = 21)	P value
Age (days)		11.1	15.8	0.02
Mean gestational age (weeks)		37.4	38.6	0.04
Prematurity		7	1	0.03
Mean birth weight (grams)		3 077	3 461	0.04
Perinatal infectious risk factors	Maternal fever > 38°C	5	0	0.03
	Others (rupture of membranes delay, maternal antibiotic)	4	1	0.6
Clinical symptoms	Fever	19	20	0.1
	Digestive symptoms	1	6	0.03
	Viral exposure	0	9	<0.001
	Signs of viral infection	0	6	0.01
Physical examination	Axial hypotonia	10	2	0.02
	Bulging fontanel	2	1	0.4
	Grunting	7	1	0.05
C reactive protein	Mean level (mg/L)	51	4.3	<0.001
	< 20 mg/L	11	21	<0.001
	> 50 mg/L	11	0	<0.001
Cerospinal fluid analysis	Mean pleocytosis (cells/mm³)	776	314	0.1
	Neutrophil predominance	14	0	<0.001
	Lymphocyte predominance	0	17	<0.001
	Mixed cell formula	9	4	<0.001
	Mean protein (g/L)	1.65	0.94	0.03
Mean antibiotic therapy duration (days)		22	6.7	<0.001
Mean hospital stay length (days)		23.35	9.36	<0.001
Outcomes	Favorable outcome	17	22	0.01
	Complicated meningitis	5	0	0.01
	Neurological sequelae	6	0	0.01

Discussion

NNM presents a significant management challenge. First, from an etiological standpoint, distinguishing between bacterial and viral NNM is difficult, especially in the absence of comprehensive microbiological testing and/or cerebrospinal fluid (CSF) sampling before initiating antibiotic therapy. Second, therapeutic management is also problematic due to the lack of a standardized treatment protocol, particularly for cases of NNM without identified pathogens and for complicated bacterial NNM. Our study is among the few national and international studies to have established the clinical, microbiological and outcome profile of NNM based on a relatively large case series. It also identified clinical and biological risk factors for bacterial NNM, thereby providing valuable guidance to clinicians in determining first-line antibiotic therapy. However, our study faced a major methodological limitation in determining whether NNM was viral or bacterial. Indeed, limited access to viral PCR testing (only 11% of patients underwent viral PCR) forced us to classify NNM as viral based on clinical and biological assumptions, CSF analysis, and the absence of bacteriological confirmation. These limitations probably led to bias in the judgment and analysis of the results, and confirm the difficulty of making a positive diagnosis of bacterial or viral NNM. Our results cannot be generalized and should be considered with caution. Our study was also limited by its retrospective design, which led to incomplete data and variations in clinical management over the years.

In our study, 26 cases of bacterial NNM were identified. The incidence is estimated at 0.3‰ live births in developed countries and between 2.4 and 6.1‰ in developing countries (1). A positive infectious history was found in 27% of cases, compared to 88.6% reported by Ben Hamouda et al. (5) and 43.6% by Kamoun et al. (6). The mean age at admission was 11.1 days, consistent with findings from the literature: 9 days in the Tunisian study by Kamoun et al. (6), and 9.45 days in the study by Bouskraoui et al. in Marrakech, which included 20 cases of bacterial NNM (7). This can be explaned by the fact that our department is not close to the maternity ward of the newborn, making difficult to collect prenatal data and leading to a bias in patient recruitment, hospitalized especially during the postnatal period in our department. Two clinical forms are recognized: early-onset bacterial NNM (day 0 to day 4) and late-onset (day 5 to day 28). In our study, late-onset forms were the most frequent (65%). Clinical manifestations are diverse and nonspecific, making diagnosis particularly difficult (2). Neurological signs are inconstant and nonspecific; in our cohort, they were absent in 64% of cases at history-taking and in 74% of cases on physical examination.

In our study, lumbar puncture (LP) yielded hemorrhagic CSF in 38% of cases, similar to what is reported in the literature: 50% and 47% in the studies by Baziomo et al. (8) and Bouskraoui et al. (7), respectively. LP remains technically challenging in neonates, especially in preterm or low birth weight infants, complicating accurate diagnosis.

Hypoglycorrhachia in bacterial NNM has prognostic value. Its persistence 48 hours after initiation of appropriate antibiotic therapy is considered a poor prognostic indicator, often reflecting complications such as ventriculitis (4cas). In our study, hypoglycorrhachia was present in 7 of 26 cases. Direct CSF examination was positive in only 12% of cases (all gram-negative bacilli), and CSF culture was positive in 39%, making definitive diagnosis of bacterial NNM difficult. These results are comparable to those from Kamoun et al. (6) and Ben Hamouda et al. (5), where CSF cultures were positive in 50.9% and 36.4% of cases, respectively. The pathogens isolated in our study included E. coli (3/26), extended-spectrum beta-lactamase (ESBL)-producing E. coli (1/26), and Pseudomonas aeruginosa (1/26).

In developing countries, the bacteriological profile is dominated by enterobacteria, whereas in developed countries, 50–80% of cases are due to Group B Streptococcus (GBS) and E. coli, and 5–10% to Listeria monocytogenes (5,10). Early-onset forms are primarily caused by GBS, while E. coli is predominant in late-onset cases (10).

Blood cultures are negative in 15–55% of bacterial NNM cases (1), potentially due to low bacterial load or prior antibiotic exposure. In our study, blood cultures were positive in 15%, isolating E. coli (n=1), Klebsiella pneumoniae (n=2) and GBS (n=1).

No universally defined CRP threshold exists in the literature; however, levels above 60–80 mg/L are considered warning signs by some authors (2). In our study, CRP was elevated in 62% of patients (n=16), with a mean value of 51 mg/L (range: 1–146 mg/L).

Therapeutic management of bacterial NNM involves two essential components. In our study, empirical triple antibiotic therapy was prescribed in 21/26 cases, and dual therapy guided by clinical presentation in 5/26. The average duration of antibiotic treatment was 18.4 days in cases with favorable outcomes (range: 10–24 days) and 36.8 days in complicated cases (range: 15–60 days), with an overall mean of 22 days.

Ciprofloxacin was prescribed prophylactically for five days in three neonates, with favorable outcomes and no sequelae. It was added based on antibiogram results in three other cases (ESBL-producing E. coli in two, and multidrug-resistant Pseudomonas aeruginosa in one, sensitive to ceftazidime and ciprofloxacin). Several studies have proved the efficacy of fluoroquinolones, particularly ciprofloxacin, in treating neonatal meningitis caused by E. coli, as well as nosocomial or multidrug-resistant meningitis. Ciprofloxacin provides faster CSF sterilization, better antibiotic diffusion in cerebral abscesses and a reduced risk of neurosensory sequelae (10, 11). Therefore ciprofloxacin is administered during the first 5 days of treatment at a dose of 20 mg/kg/day in the first week of life and 30 mg/kg/day thereafter. In cases of established brain abscesses, treatment is extended based on lesion evolution (12).

Corticosteroids have no role in bacterial NNM, as studies have not shown efficacy in reducing mortality or neurological sequelae (13). No corticosteroid therapy was prescribed in our study.

Cranial ultrasound (CU) is accessible, non-irradiating, but operator-dependent and limited in precision. It plays a key role in the early detection of complications: ventriculitis, subdural empyema, brain abscess, and hydrocephalus (14). In our study, CU was performed in all patients, and abnormalities were found in four cases (triventricular hemorrhage, increased echogenicity of basal ganglia, ventriculitis, and sagittal sinus thrombosis).

Cranial CT has limited value in NNM evaluation and was used in only three cases due to the unavailability of MRI. It revealed hydrocephalus in one case, a frontal hygroma with widening of the interhemispheric fissure in another, and sinus thrombosis in the third. MRI with contrast helps detect subtle or asymptomatic CNS lesions. In our study, MRI was performed in three cases and revealed hydrocephalus in all three.

No deaths occurred among the 26 bacterial NNM cases. Previous Tunisian studies by Ben Hamouda et al. (5) and Kamoun et al. (6) reported mortality rates of 15.9% and 40%, respectively. In developed countries, mortality rates have declined significantly, reaching 13.7% in France (15), whereas in developing countries, they remain high (27–58%) (16).

Neurological sequelae (hearing loss, motor delay, cognitive impairment, epilepsy, speech delay, hydrocephalus) occur in 20–50% of bacterial NNM cases (17, 18). In our study, the prevalence of neurosensory sequelae was 23%. This relatively low rate can be explained by the lack of follow-up for all patients in the study department (n=11/26) and the limited average follow-up period at the end of the study (average follow-up period = 10.5 months). Other neurosensory sequelae may not have been reported or detected.

As for viral NNM, we estimated 21 cases of neonatal viral meningitis, with a predominance in summer. Diagnosing viral NNM remains difficult due to challenges in identifying the causative virus and the risk of overlooking bacterial NNM, which requires urgent treatment and carries a high risk of severe complications. As a result, many viral NNM are treated as probable bacterial cases.

The availability of multiplex PCR testing in recent years has significantly improved the detection of viral NNM. In our study, PCR for herpes simplex virus types 1 and 2 and enterovirus genome was performed in only five cases, yielding two positive enterovirus results.

Viral meningitis treatment is primarily symptomatic. Empirical antibiotic therapy is often initiated until bacterial infection is ruled out. In our study, 17 of 21 viral NNM cases received empirical antibiotics, with a mean duration of 6.7 days (range: 1 to 15 days). The lack of microbiological confirmation of the viral nature of NNM in most cases, combined with lingering doubts about the bacterial nature of NNM and the possibility of neurosensory sequelae, explains the widespread prescription of antibiotics in these patients and the difficulty of taking the decision to stop first-line antibiotic therapy, a decision that depends on the expertise level of the treating physician.

Acyclovir was prescribed in suspected herpes cases (2 neonates) at 60 mg/kg/day IV for 21 days but discontinued after 10 and 12 days respectively, following negative CSF PCR.

CU remains important in viral NNM management for detecting white matter lesions (19). It was performed in 14 of 21 viral NNM cases and revealed no abnormalities. Unlike bacterial NNM, viral cases are rarely associated with long-term neurological sequelae (20). In our study, all patients who were followed up (n=12/21) had favorable outcomes, with a mean follow-up of 5.5 months. This rate may be underestimated due to the lack of follow-up for all patients and the relatively limited duration of follow-up.

Comparison between bacterial and viral NNM revealed that bacterial cases involved younger age, more frequent prematurity, and lower birth weights. Clinically, maternal fever, axial hypotonia, and grunting were more common in bacterial NNM, while digestive symptoms, viral exposure, and signs of viral infection were significantly more frequent in the viral group. Biologically, bacterial NNM showed significantly higher neutrophil counts and CRP levels. On LP, neutrophil predominance and elevated CSF protein were also statistically associated with bacterial NNM.

These clinical, anamnestic, and biological differences may assist clinicians in distinguishing between bacterial and viral NNM when microbiological investigations are inconclusive, thus guiding therapeutic decisions.

In terms of management, the duration of antibiotic therapy and hospitalization was significantly longer in bacterial NNM. Acute complications and neurological sequelae were also significantly more frequent in bacterial cases, leading to higher treatment burden, cost, and long-term neurological impact.

Conclusions

NNM is a rare but serious infection, particularly bacterial NNM, which compromises both the vital and functional prognosis due to the risk of severe neurosensory sequelae. However, it is often difficult to clearly differentiate bacterial from viral NNM.

Despite the difficulty of discriminating between bacterial and viral NNM, due to the lack of microbiological confirmation in most cases, our study tried to identify clinical and biological characteristics that could help clinicians distinguish between bacterial and viral NNM during the first hours of patient management in order to better target first-line treatment. However, our results cannot be generalized, and further larger studies involving precise microbiological testing are needed in order to establish clearly defined guidelines.

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