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BIOLOGICAL PROFILE OF NEONATAL INFECTIONS IN CENTRAL MOROCCO: ABOUT 280 CASES

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Abstract

Neonatal infections are a real public health problem and represent a constant challenge in neonatal units. Their medical and economic consequences justify welldefined surveillance and prevention measures. In order to define the circumstances of their occurrence, as well as the modalities of diagnosis and treatment, we conducted this retrospective study of 280 newborns hospitalized for suspected neonatal infection in the mother-child hospital -Pagnon-of Meknes.Infectious history was positive in 143 newborns (51%), clinical manifestations were dominated by respiratory signs (52%), as well as neurological signs (40%) and thermal disorders (18%) The hemogram showed abnormalities in 22.26% of cases, hyperleukocytosis was present in 12.14%, leukopenia in 3.92%, thrombocytopenia in 13.92% and anemia in 10.71%. The initial C-reactive protein (CRP) was positive in 52.14% of cases, the procalcitonin was positive in 22% of cases. Lumbar puncture was positive in 9% of cases. While blood culture was not realized in our study. The association: Cephalosporin of 3rd generation (C3G) and gentamicin was administered in 1st intention, in 74.28% of the patients, however the association: Cephalosporin of 3rd generation, gentamicin and ampicillin in 22.85% of the patients. The evolution was judged clinically favorable in 63% of cases, on the other hand 20% of patients had sequels, the mortality rate was 15%.The diagnosis of neonatal infection is based on anamnestic, clinical, biological and bacteriological arguments. Since antibiotic therapy is the key to management, prevention involves rigorous surveillance of pregnancies, screening of pregnant women for GBS, and medicalization of deliveries to reduce the rate of neonatal infections.

Keywords

Neonatal infection , Biological markers , Bacteriology, Antibiotics , Prevention

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Main Article

Introduction

Neonatal infections (NNI) are defined as alterations in the organism secondary to the invasion of a harmful pathogen (bacterium, virus or parasite) that can affect the newborn before, during or after birth. They are classified, according to the time of their occurrence, into early neonatal infections and late neonatal infections. Neonatal infection represents a major public health problem worldwide, in both industrialized and developing countries, with an incidence of around 5 to 10 cases per 1000 live births. Maternal infections, although usually benign for the mother, represent a major risk for the unborn child, with neonatal mortality
of around 3%, and morbidity that can be severe (neurological or respiratory sequelae). Bacterial maternal-fetal infection (MFI) is still mainly linked to Group B Streptococcus (GBS), which most often contaminates the newborn during passage through the genital tract at the time of delivery [1].

In the absence of early and appropriate treatment, the mortality rate is high, particularly in the first few weeks of life. Worldwide, an estimated 2.9 million neonatal deaths occur each year, accounting for 47% of deaths in children under 5. In Morocco, neonatal infections are responsible for over 16% of perinatal deaths, so prevention and management are key to meeting the challenge of reducing perinatal mortality [2]. Clinical signs of neonatal infection can range from non-specific symptoms to hemodynamic failure. Early symptoms may include irritability, lethargy or poor feeding. Others may rapidly develop respiratory distress, fever, hypothermia or hypotension with poor perfusion and shock. Sometimes, the diagnosis can only be suspected on the basis of biology results [3]. The immediate diagnosis is far from self-evident, and is based in most cases on simple clinical presumption and biological orientation (in current practice, the markers frequently used are serum levels of the inflammation protein C-reactive protein and leukocyte abnormalities). Bacteriological confirmation is often lacking, due to the difficulty of isolating the causative organisms (between 8% and 10% of positive results). Blood culture results often take a few days, necessitating initial emergency antibiotic treatment of suspected cases, depending on the local bacteriological profile, and careful monitoring for at least the first 48 hours. The longer treatment is delayed in relation to the onset of the disease in newborns, the greater the chance of recovery [4]. The aim of our study is to analyze the epidemiological, clinical, biological and bacteriological profile, as well as the treatment

modalities of neonatal infections in the neonatology department of the Pagnon mother-child hospital in Meknes.

Materials and methods

This study was carried out in the neonatology department, which is a neonatal medicine and intensive care unit at the Pagnon Mother and Child Hospital in Meknes. This was a retrospective, analytical study of the medical records of 280 neonates hospitalized for suspected neonatal infection over a 5-month period, from August 1, 2020 to December 31, 2020. For each patient included in the study, an information sheet on the following data was completed from the medical records:

- Epidemiological characteristics (age, sex, weight, etc.).
- Maternal data: medical follow-up of pregnancy, term of delivery, gestiture, parity, mode of delivery, history of infection (leucorrhoea, micturition burns, maternal temperature at delivery ≥ 38.0°C, premature rupture of membranes (RPM), prolonged labour, stained or meconium-stained amniotic fluid, other).
- Reason for hospitalization.
- Clinical data.
- Biological data: mainly blood count and C-reactive protein (CRP) determination.
- Bacteriological data: includes cerebrospinal fluid and urine cytobacteriological examination.
- Radiological data.
- Antibiotic treatment and duration of administration.
- Length of stay on the ward.
- Evolution.

Data entry and processing were carried out using EXCEL statistical and graphical spreadsheet software, version 2016. Continuous quantitative variables were described as appropriate by mean and standard deviation.

Results

The number of deliveries in 2020 was 9095, of which 8114 were vaginal and 981 were vaginal. The number of deliveries during the 05 months of the study was 4205, of which 725 newborns (17.24%) were hospitalized for suspected neonatal infection. The study population comprised 280 newborns, of whom 266 were admitted between 0-7 days of life, i.e. 94%, 7 newborns were

admitted between 7-15 days of life, i.e. 3%, and 7 newborns were admitted between 15-28 days of life, i.e. 3%. Mean age at admission was 4.15 days ± 3.08 days. The extremes were 0 days and 28 days (Table 1). Our study revealed a predominance of male newborns, who represented 58% of our series, i.e. a total number of 161 boys for 119 girls (42%). The sex ratio was 1.38 (Figure 1). In our study, 43% of the mothers (121 cases) were multiparous, while 33% of them (93 cases) were primiparous; 33% were not mentioned on the parthogram (66 cases). 10 cases had diabetes, 09 cases gestational diabetes, 06 cases preeclampsia, 04 cases arterial hypertension and threatened premature delivery (CPB), 02 cases asthma, one case thrombocytopenia and one case goiter. (Figure 2). In our study, 32% (89 cases) were premature (<37 SA), 67% (190 cases) were at term: between 37 and 40 SA and 1 case was over term (>42 SA). The notion of pregnancy follow-up was not mentioned on the parthogram in 76% (233 cases), 7% (21 cases) of pregnancies were well monitored, 2% (7 cases) of pregnancies were poorly monitored and 6% (19 cases) were not monitored. The mode of delivery was predominantly instrumentless vaginal delivery with a percentage of 68% (189 cases), followed by Caesarean section with a percentage of 31% (87 cases) and finally instrumental vaginal delivery with a percentage of 1% (4 cases).

A blood count was performed on 274 newborns in the study population:

- Hyperleukocytosis was found in 34 newborns, i.e. 12.14% of the series;
- Leukopenia was found in 11 newborns, i.e. 3.92% of the study population;
- 30 newborns presented with anemia, representing 10.71% of cases studied;
- Thrombocytopenia was found in 39 newborns, or 13.92% of cases;
- And 6 newborns showed thrombocytosis, representing 2.14%.

The most frequent association was hyperleukocytosis + thrombocytopenia. It was present in 26 newborns (9.28%). All patients were tested for CRP, which was elevated in 146 cases (52.14%). The positive rate in our study was over 20 mg/l. Extreme values were 2.1mg/l and 269mg/l. Procalcitonin was measured in 35 patients, and was positive in 8 cases (22%). The positivity rate in our study was greater than 2 ug/l. The extreme values were 0.1ug/l and 100 ug/l.

(table 2) Lumbar puncture with cerebrospinal fluid study was performed in 113 cases (41%) in the presence of neurological signs, fever, very high CRP or signs of sepsis. ECBU was performed in 14 patients (5% of all cases). Bacteriological results were sterile in 10 cases (71%), while 4 cases (29%) were non-sterile (03 cases of E. coli and 01 case of acinobacter spp).

Discussion

Neonatal infection is one of the major causes of neonatal morbidity and mortality worldwide. Recently, the global occurrence of neonatal deaths due to infection has been estimated at 0.6 - 0.7 million, with the highest mortality observed in areas of sub-Saharan Africa [5]. Its incidence in developed countries is estimated at between 5 and 10 per 1000 live births. In developing countries, on the other hand, the incidence is higher, around 30 to 50 per 1000 live births. In contrast to developed countries, infected newborns in developing countries are often cared for late, due to a lack of facilities and specialists, especially in rural areas requiring travel to distant hospitals. Epidemiological and etiological data vary greatly from one region to another. There is a lack of up-to-date, accurate data, particularly in developing countries [6, 7]. Neonatal infection is a public health problem, not only because of its mortality and morbidity rates, but also because of the additional costs associated with prolonged hospitalization and antibiotic administration, both in industrialized and developing countries, where incidence and mortality are highest. The rate of hospitalization and probabilistic antibiotic therapy due to suspected neonatal infection varies between 11% and 80% [8, 9]. It is well established that neonatal mortality reflects the performance of a country's public health system. These findings call for the reinforcement of this program in terms of preventive measures, early diagnosis and rapid, effective management of infected newborns in Morocco.

Transplacental hematogenous contamination may be secondary to maternal bacteremia or septicemia, with transmission of germs to fetal blood either directly through the umbilical vein, or from a placental focus or infected amniotic fluid. The most frequently incriminated germs are T.pallidum and L.monocytogenes. The ascending route is the most frequent, and is secondary to colonization of the amniotic fluid (AF) by a pathogenic or non-pathogenic germ from the vaginal flora (S. agalatiae, E.coli...), with or without rupture of the membranes. While postnatal contamination via breast milk is very rare, the germ most frequently found is S. agalatiae. Vaginal flora is extremely diverse in its physiological state, dominated by Gram-positive bacteria (Lactobacillus de Doderlein). The presence in a vaginal swab from a pregnant woman of bacteria in pure culture, with or without conservation of the Doderlein lactobacillus flora, corresponds to carriage or a local infectious process (*Table 3*). [10]

The potential severity of certain neonatal infections, and their rapid evolution, explain the need for a sensitive, specific and early biological marker of infection, so as not to delay the

management and initiation of antibiotic treatment in a contaminated newborn, but also to avoid treating a large number of healthy children on the basis of more or less significant presumptive elements, and thus avoid the abusive use of antibiotics [3].

Biological evaluation of symptomatic newborns is most often based on two types of marker: increased serum levels of inflammatory proteins (CRP) and leukocyte abnormalities. Other markers such as procalcitonin and cytokines are increasingly used in the management of infected newborns [11]. There are significant physiological variations in blood count data, especially leukocytes, between premature and full-term infants, children at different stages of developmental stages and the adolescent. All three bone marrow lineages may be altered during neonatal infection, but the most interesting abnormalities for the diagnosis of infection concern the granular lineage in particular. In addition to variations linked to gestational age, there are important physiological changes in this line during the first days of life [12]. The classic chronology of the leukocyte response to infection is as follows: neutropenia, myelhemia, neutrophil polynucleosis. Neutropenia is a fairly early usually of short duration, and is linked to the trapping of neutrophils of neutrophils at the site of infection. The appearance of young forms of white blood cells in the bloodstream reflects strong bone marrow stimulation, It precedes and accompanies hyperleukocytosis. Thus, the discovery of neutropenia or hyperleukocytosis and/or myelimia is a sign of infection. However, the reliability of these markers is very limited, as several factors can modify the circulating leukocyte count, such as the presence of hemolysis fetal hypoxia, maternal fever, maternal toxemia, in addition to physiological variations related to gestational age and postnatal age (*Table 4*). [13]

CRP is a cyclic homo-pentameric protein of the acute phase of inflammation. It belongs to the pentraxin family, which plays an important role in innate and adaptive immunity. It is normally undetectable in the serum of healthy subjects. Its synthesis and hepatic release are triggered by IL-6, IL- 1β and TNFα following an inflammatory process of infectious or non-infectious origin. It activates the classical complement pathway to promote phagocytosis. Its kinetics are delayed, with serum levels rising between 6 and 12 hours after the onset of inflammation. It peaks after 24-48 hours, then declines rapidly to normalize within 4-7 days. Its half-life is 19 hours [14]. CRP is positive at 20 mg/L and negative at less than 6 mg/L [15]. This protein does not cross the placental barrier, and a high level in neonatal blood reflects the presence of neonatal inflammation [16]. What's more, its measurement is simple, rapid, routinely available and inexpensive, using immunonephelometry or immunoturbidimetry. This makes it a reference

marker for the investigation and monitoring of inflammatory processes [17]. Consequently, it is frequently used in many neonatal intensive care units for the diagnosis of neonatal infection, despite the fact that it has a number of limitations. This marker is not very effective, as it is aspecific and delayed. Before 12 hours, it is usually of little interest (sensitivity 50%) and its contribution can be optimized by repeated assay, which can even guide antibiotic therapy [18]. False positives are rare and can be linked to a number of perinatal events: aspiration of meconium fluid, asphyxia, perinatal trauma, cerebral haemorrhage, surgery and administration of exogenous surfactant. False negatives can occur, especially in the early stages of infection. Different performances at variable thresholds of serum CRP positivity have been reported in the literature [19]. These differences can be explained by the heterogeneity of the studies, including gestational age, post-natal age, birth weight, inter-individual variability, type of infection (early, late) and assay technique [20]. The main conclusions drawn from studies investigating serum CRP are as follows:

- ✓ The 12-72-hour CRP is the best test for differentiating infected from uninfected newborns, at a threshold of 20 mg/L with a sensitivity of almost 78% and a specificity of almost 94%.
- ✓ CRP on the sixth day of treatment is reliable for stopping antibiotics, and can therefore be used to reduce the duration of antibiotic therapy in infected newborns.

A negative CRP with a negative culture excludes infection [9].

Recently, evidence of the usefulness of CRP determination in umbilical cord blood for the diagnosis of early neonatal infection has been suggested. for the diagnosis of early neonatal infection. A recent case-control study in Egypt, involving 70 full-term full-term neonates showed that CRP determination in saliva is also useful for differentiating between infected and uninfected newborns at a threshold of of 3.48ng/L with a sensitivity of 94.3% and a specificity of 80% [20]. In our series, CRP was performed in all patients. It was elevated in 52.14%, which is higher than that reported in the study by El Mehdi Mourtada (Marrakech) [21], where it was positive in 168 cases, or 38% of patients this test. In the study by Romuald Edgard Mongo (Rabat), CRP was positive in in 86.93% of cases [22].

Conclusion

Neonatal infections are a real public health problem, and remain a constant preoccupation in neonatal departments. Their medical and economic consequences justify well-defined surveillance and prevention measures. Effective management of neonatal infection requires close coordination between pediatricians and obstetricians, with the aim of obtaining reliable anamnestic data to help identify at-risk newborns and ensure better care. The diagnosis of NIN is based on a combination of clinical, biological and bacteriological evidence. The diagnosis of NIN is based on a combination of clinical, biological and bacteriological evidence. The medical biology laboratory plays an important role in this diagnosis, through the use of relevant and reliable biological markers and the isolation of pathogens responsible for infection. The main bacterial agents responsible for these infections are GBS and E. coli. For this reason, GBS screening of pregnant women is recommended to reduce the rate of neonatal infections. In most cases, newborns are put on probabilistic antibiotics as soon as a bacterial infection is suspected. This overuse of antibiotics increases the risk of resistance and immediate and long-term deleterious effects on the newborn. Improving prognosis requires comprehensive management, with appropriate use of antibiotics, and the development of therapeutic strategies adapted to local epidemiological conditions. We cannot conclude this work without stressing the importance of multicenter prospective studies in our country, in order to draw up a global profile of neonatal infection, which will enable us to better adapt our therapeutic strategy, to pursue a well-targeted prevention policy that could lead to a reduction in the risk of this scourge, and to standardize national protocols.

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Figures :

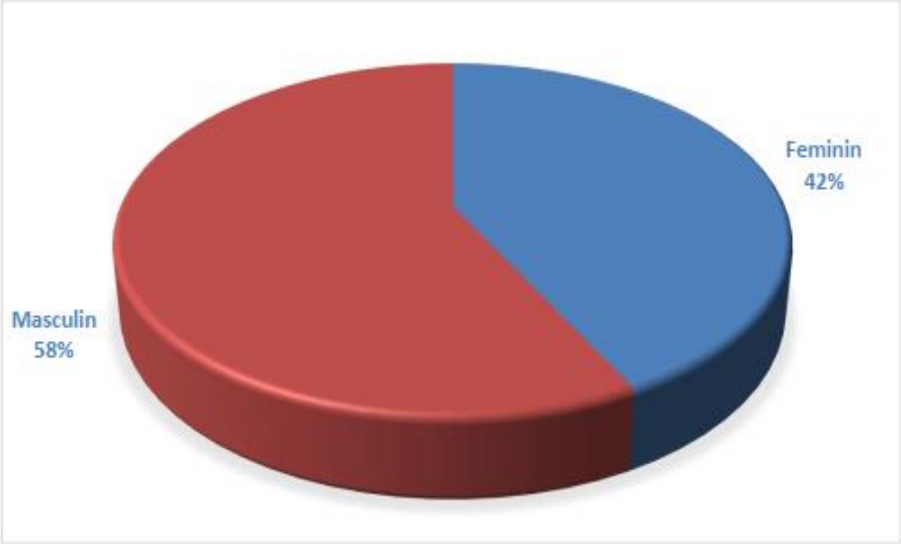

Figure 1: Distribution of patients by gender.

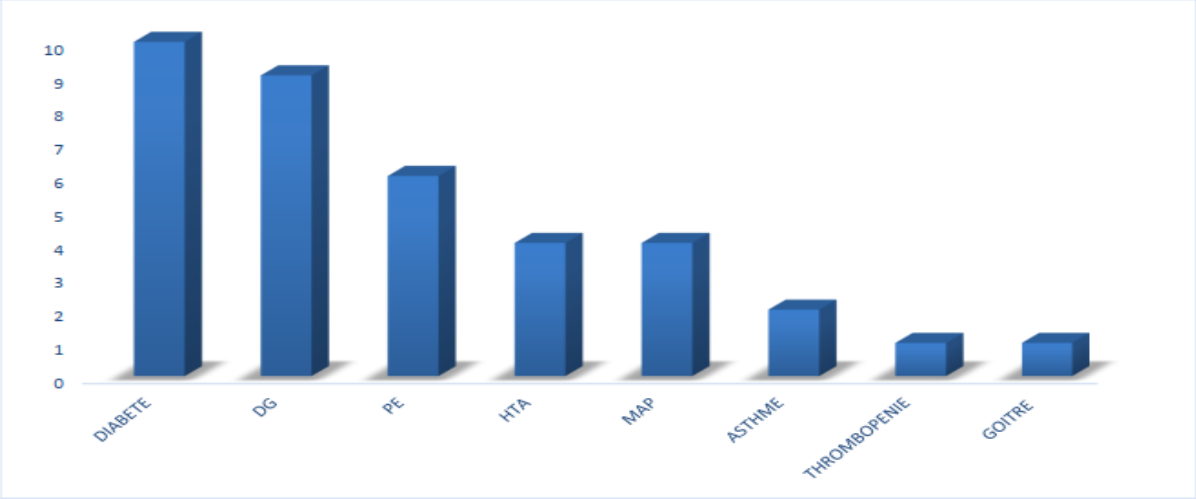

Figure 2: Distribution according to maternal medical and obstetric history.

(DG : diabète gestationnel ; PE : prééclampsie ; MAP : menace d'accouchement prématuré)

Tables:

Age moyen	Écart type	Extrêmes
4.1 jours	3.08 jours	0-28 jours

Table 1: Distribution by average age and standard deviation

Marqueurs biologiques et biochimiques		Nombres	Pourcentage
NFS	Hyperleucocytose	43	12,14%
	Leucopénie	11	3,92%
	Anémie	30	10,71%
	Thrombopénie	39	13,92%
	Thrombocytose	6	2,14%
CRP> 20mg/l		146	52%
Procalcitonine> 2ug/l		8	3%

Table 2 : Récapitulatif des données biologiques et biochimiques

Bactéries des infections hématogènes	– *L. monocytogenes* – *E. coli, Proteus*	
Bactéries vaginales	Groupe 1	Flore de portage habituel **sans risque** majeur d'infection : –*Lactobacillus* –*Streptocoque alpha hémolytique*
	Groupe 2	Flore issue de la flore digestive **à risque infectieux néonatale :** –*SGB* –*Entérobactéries (E. coli K1)* –*Staphylococcus aureus* –*Gardnella vaginalis* –*Mycoplasme hominis* –*Bactéries anaérobie*
	Groupe 3	Flore oropharyngée colonisant le vagin à **haut risque** d'infection néonatale : –*Streptocoque pyogène, Pneumocoque* –*Haemophilus influenzae, Méningoques.*

Table 3: Maternal bacteria at neonatal risk [3].

	Naissance	J1	J2	J3	J4 - J7	J8 - J14	J15 -1 mois	2 Mois
Hématies (10^6/ml)	3,9 - 6,4	-	3,8 - 6,1	3,8 - 6,2		3,7 - 5,7	3,1 - 5,2	3,1 - 4,3
Hémoglobine (g/dl)	14,1 - 21,6	-	13,8 - 22,1	13,5-21,9		12,3 - 19,9	10,8 - 17,2	9,7 - 13,5
Hématocrite (%)	43,4 - 67,5	-	40,6 - 64,1	39,2 - 62,7		35,7 - 58,5	30,2 - 49,4	27,3 - 41,2
Plaquettes (10^6/ml)	150 - 400						150 - 600	
Leucocytes (10^6/ml)	9,1 - 30	11,2 - 27	7,9 - 22,5	6,2 - 17,1	6,3 - 17,4	7,3 - 16,6	7,1 - 15,9	6,4 - 12,5
Lymphocytes (10^6/ml)	2 - 6,7	2 - 6,6	2 - 6,8	2 - 6,4	2 - 8	3 - 9,4	3,5 - 10,2	3,7 - 10,5
PNN (10^6/ml)	5 - 26	4 - 19	3 - 16	3 - 11	1,5 - 9,5	1 - 9,3	7,1 - 15,9	1 - 4,9
PNE (10^6/ml)	< 0,8							
PNB (10^6/ml)	< 0,2							
Monocytes (10^6/ml)	0,4 - 3						0,3 - 1,5	

Table 4: Reference intervals for blood counts from birth to 2 months of age of life [81]

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