

Neonatal Bilirubin Encephalopathy: Study of 30 Cases at Albert Royer National Children Hospital of Dakar

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Abstract

Introduction: Unconjugated bilirubin jaundice is a common symptom in neonatal period. In some babies, excessive serum bilirubin concentrations can place them at risk of acute bilirubin encephalopathy (BE) when the unconjugated pigment crosses the blood-brain barrier. Our study aimed to describe epidemiology, diagnosis and prognosis of BE at the Neonatology Department of Albert Royer Children's Hospital of Dakar. **Materials and Methods:** It was a retrospective, descriptive study of cases of BE from January 1, 2015 to June 30, 2019. Obstetric and perinatal data as well as postnatal jaundice data (onset time, associated signs, signs of encephalopathy, treatment and evolution) were collected and analyzed by SPSS software version 2.0. **Results:** We collected 30 cases of BE (1.14% des admissions) with average age of 6.7 days and sex-ratio of 1.3. Majority were term babies (29 cases; 96.7%) and 7 (23.3%) had intrauterine growth retardation. Almost all newborns (27 cases; 90%) were exclusively breastfed. At admission, all children exhibited blunt jaundice and signs of encephalopathy dominated by the abolition of archaic reflexes (76.7%), low suction (22 cases; 73.3%), central apnea (12 cases, 40%). The mean serum bilirubinemia was 322 mg/litre. Neonatal infection (10 cases; 33.3%) and fetal-maternal incompatibility (16 cases; 53.3%) were the main causes. All children received intensive phototherapy and exsanguino transfusion was performed for 7 newborns (23.3%). Nine children died (30% mortality rate). **Conclusion:** Only better organisation of perinatal care with enhanced postnatal follow-up can reduce the incidence of EB.

Keywords

Neonatal Hyperbilirubinemia, Encephalopathy, Perinatal Care

1. Introduction

Unconjugated bilirubin jaundice is the most common symptom in neonatal period, affecting up to 50% - 70% of newborns [1]. In presence of some risk factors, such as hemolysis, sepsis, prematurity, low birth weight, it can be complicated by bilirubinic encephalopathy (BE), when the unconjugated pigment crosses the blood-brain barrier and attaches to basal ganglia (pallidum, subthalamic, cochlear and oculosic). Clinical manifestations of BE can be observed in the acute phase (drowsiness, lethargy, low suction, hypotonia, hypertonia with opisthotonos, apnea, convulsions ...), or as chronic sequelae (dystonia, choreo-athetosis, deafness, oculomotor paralysis ...) [2] [3] [4].

The prevalence of BE is particularly high in low resources countries, such sub-Saharan Africa, with a significant morbimortality [5] [6] [7]. The incidence of severe neonatal jaundice in the African region in 2017 was estimated at 667 cases/10,000 live births (LB) compared with only 3.7 cases/10,000 LB for the European region [8]. Buthani in 2010 estimated that of 75.400 annual cases of BE worldwide, 35% occurred in sub-Saharan Africa, corresponding to a prevalence of 56 cases per 100.000 LB, compared to 37% in South Asia.

Despite the high frequency reported EB in the literature in sub-Saharan Africa, no previous works in Senegal have looked at it to our knowledge. Our study, conducted in the Neonatology Department of the Albert Royer National Children's Hospital (CHNEAR), aims to describe the epidemiological, diagnostic and prognostic aspects of EB.

2. Patients and Methods

Study framework: Study was conducted in the neonatology department of the pediatric hospital with a capacity of 30 beds, and which receives children from 0 to 2 months of life, all out born because there is no obstetrical unit in the hospital. Dosage of serum bilirubinemia is done at the hospital laboratory and for the management of jaundice, phototherapy and exsanguino transfusion are carried out.

Type, period and population of the study: This was a retrospective, descriptive study from January 1, 2015 to June 30, 2019 (4 years and 6 months). Included were newborns with BE and for whom bilirubinemia levels were available. BE was retained in any child with obvious jaundice, associated with one/or more of the following signs, not attributable to an intercurrent pathology: impaired archaic reflexes, axial/peripheral tonus disorders, coma, convulsions, abnormal movements like chorea and winding of limbs, central apnea.

Data collection and analysis: All data were collected from patient medical records and from hospital records. We looked at next parameters: epidemiological and socio-demographic, maternal and familial (age, parity, ABO rhesus group, previous anti-D serum administration ...); obstetrical (follow-up and obstetric complications ...); perinatal (term and delivery way, instrumental maneuver, neonatal adaptation, trophicity at birth); postnatal (delay of jaundice

onset, associated signs, signs of encephalopathy); therapeutics (phototherapy, exchange-transfusion, other therapeutics ...) and evolution (duration of jaundice, death, sequelae, complications ...). Data was analyzed with the statistical software SPSS version 2.0. The descriptive study was carried out by calculating variable frequencies and proportions for qualitative variables and by averages/standard deviations for quantitative variables.

3. Results

Socio-demographic and background data: We recorded 30 cases of EB, out of 2617 admissions (1.14%) with an average age of 6.7 days (6 hours to 25 days) and a sex-ratio of 1.3. Eleven cases (36.6%) occurred in the third trimester (**Figure 1**). History of early death was noted in siblings in 5 cases, jaundice in a single case. Parental consanguinity was noted in 3 cases. Average maternal parity was 2.4 and majority of women (24 cases, 80%) had done at least 4 prenatal consultations (PNCs).

Perinatal data: The majority of children were born full-term (29 cases; 96.7%), vaginally (24 cases; 80%) and 7 children (23.3%) had intrauterine growth retardation. They were all exclusively breastfed (27 cases; 90%) (**Table 1**).

Diagnostic, therapeutic and evolution: At admission, all children presented with franc jaundice and signs of encephalopathy. Fever was noted in 17 children (56.7%). (**Table 1**). The main signs of encephalopathy were diminution of archaic reflexes (23 cases; 76.7%), low suction (22 cases; 73.3%) and central apnea (12 cases, 40%) (**Table 1**). The majority of newborns (18 cases; 60%) had a total bilirubin level above 300 mg/l and the average bilirubinemia was 322.6 mg/litre. Anemia was found in more than one-third of newborns (12 cases; 40%), thrombopenia in 7 newborns (23.3%) (**Table 2**). Neonatal infection (10 cases; 33.3%) and incompatibility in the ABO system (10 cases; 33.3%) were the most found etiological factors. A case of Crigler-Najjar disease was confirmed (**Table 3**). All children received intensive phototherapy, along with the "Tunnel" device. Exchange-transfusion was performed only in 7 newborns (23.3%). The average duration of jaundice was 9.1 days (1 to 37 days). Nine patients died, representing a mortality rate of 30%. Of the eleven children followed up, all had psychomotor delay and four developed severe encephalopathy, with microcephaly, no motor acquisition (axial hypotonia, not sitting and walking beyond 2 years, language delay, abnormal movements with coils of the limbs or generalized tonic convulsions). It should be noted that the search for deafness was not done in these children.

4. Discussions

BE is a real concern in neonatology in sub-Saharan Africa. This first study identified 30 cases, but don't reflect the extent of the BE-related problem in Senegal. Many other cases are frequently observed in other pediatrics wards in Dakar and interior of Senegal. In addition, this was a retrospective work which only focused

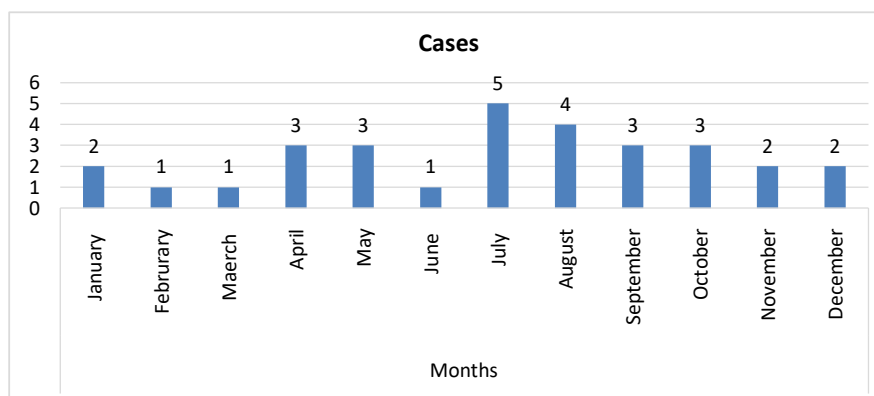


Figure 1. Repartition of cases by month.

Table 1. Anamnestic and perinatal data.

Settings	Numbers (n)	Percentage (%)
Perinatal data		
Prematurity	1	3.3
Term babies	29	96.7
Caesarean delivery	6	25
Instrumental delivery	0	0
APGAR < 7 at 1 minute	1	3.3
Neonatal resuscitation	5	16.7
Exclusive breastfeeding	27	90
Intra-Uterine Growth restriction (IUGR)	7	23.3
Clinical data		
Jaundice	30	100
Fever	17	56.7
Clinical anemia	3	10
Hepatomegaly	3	10
Encephalopathy	30	100
Signs of encephalopathy		
Weak suction	22	73.3
Abolition archaic reflexes	23	76.7
Hypotonia	14	46.7
Hypertonia	9	30
Lethargy	9	30.0
Abnormal movements	8	26.7
Convulsions	6	20.0
High-pitched scream	2	6.7
Apnea	12	40
Respiratory distress	10	33.3

Table 2. Biological data.

Parameters	Number	Percentage (%)
Total bilirubinemia (mg/liter)		
<200	6	20
200 - 300	6	20
>300	18	60
Anemia Hemoglobin < 12 g/dl)	12	40
Thrombopenia (<150.000/mm ³)	7	23.3
Leukopenia (<4000/mm ³)	1	3.3
Hyperleukocytosis (>20,000/mm ³)	6	20
C Reactive Protein positive (>6 mg/L)	6	20

Table 3. Etiological factors.

	Number of cases	Percentage
Neonatal infection	10	33.3
ABO incompatibility	10	33.3
Rhesus Incompatibility	6	20
Crigler-Najar type I	1	3.3
Unknown	3	10

on cases where clinical signs were evident during hospitalization, whereas many sensorineural disorders can occur later in follow-up [10].

Hospital frequency was 1.14%. A study in Nigeria found a higher frequency, between 2.3% and 3.4% of admissions [11]. However, in these works, detection for EB was more active especially during the follow-up, which may explain the difference. Another study in Egypt showed that up to 12% of icteric infants had EB [12]. In developed countries, reported population incidences appear to be much lower compared to those observed in our units, ranging from 0.86 to 1.3 cases/100,000 live births [13] [14] [15]. The majority of cases occurred in term infants (96.7%), but this reflects the fact that detection was not done systematically in premature infants, and sensorineural sequelae observed during their follow-up are not necessarily interpreted in light of history of jaundice. The risk of EB is higher in preterm and/or low birth weight children due to their increased vulnerability [16]. Morioka I. *et al.* in Japan reported an incidence of 0.18% in preterm infants [17]. Age of admission (6.7 days) was very late, considering that jaundice started within 72 hours in 63.3% of cases. In comparison, the reported age of admission in Nigeria was lower, between 3 and 6 days [11]. All children arrived after the clinical signs of encephalopathy were installed. Diagnosis delay of neonatal jaundice is frequent in our practice, as the symptom is unknown by many parents. It is therefore imperative that jaundice be taught to mothers as a sign of danger, which should lead them to seek urgent medical attention. Such a

policy could significantly reduce the risk of EB [18] [19]. However, recognizing jaundice may be difficult in neonatal periods, especially on black skin and even experienced professionals may have difficulty in appreciating its severity [20].

Clinically, we found the classic signs of encephalopathy, dominated by neurological and respiratory impairment, including respiratory distress and apnea. Apnea is recognized as a fairly specific sign of BE [21]. Fever and anemia were the main associated signs. The association of anemia is due of hemolysis. Fever was often associated with thrombopenia (23.3%), leukopenia (3.3%), hyperleukocytosis (20%), in context of neonatal infection. The search for neonatal infection must be systematic in cases of neonatal jaundice.

Average serum bilirubinemia was very high (322.6 mg/L), further attesting the severity of the cases. Average serum bilirubinemia was more than 300 mg/l for up to 80% of children in one study in United States [22].

There are many factors that contribute to the occurrence of EB. We found classic causes such as ABO or rhesus incompatibilities and infections. Materno-fetal erythrocytes incompatibility jaundice is the most common cause of EB [9]. ABO incompatibility was found in 10 cases (33.3%). Rhesus incompatibility was found in 6 cases in infants whose rhesus-negative multiparous mothers did not receive Anti-D Serum in previous deliveries. The systematic use of Anti-D serum in rhesus-negative women has reduced the incidence of severe jaundice by rhesus alloimmunizations and thus that of EB [9]. But its use is not systematic in our context, principally because of the cost. We confirmed a case of Crigler-Najar with molecular biology. We did not specifically look for some classic causes, such as the G6PD deficit, cited as the leading cause in some African series and in the United States [22] [23] [24]. Dosage of G6PD enzyme is not routinely available in Senegal, but a study in 2005 found an incidence of 12% in the general population, which should require that in neonatology, all children with hemolytic jaundice be screened for G6PD [25]. Exclusive breastfeeding, which was mode of feeding of the majority of children, is associated with a higher risk of severe jaundice and BE, due to difficulties that may accentuate the entero-hepatic cycle of bilirubin [26].

Phototherapy is the main treatment of free bilirubin jaundice, except in very severe cases where exchange-transfusion should be considered [27]. Both therapies should help to avoid BE. They were available and feasible in our center, but all the children had already reached an advanced stage with an installed encephalopathy. Access to phototherapy is not always assured in sub-Saharan Africa, especially at the peripheral centers, which contributes significantly to the risk of EB. Even in centers with phototherapy, fluorescent lamps are rarely renewed, even beyond the recommended 2000 hours of use. As a result, only few devices deliver sufficient irradiation intensity for intensive phototherapy [28] [29]. As alternative, we should consider use of phototherapy techniques with filtered natural sun light, which can offer the same effectiveness without deleterious effects [30]. Exchange-transfusion is very often performed Subsaharan Africa, partly because of the unavailability of intensive phototherapy but also because

newborns are seen at an advanced stage with severe hyperbilirubinemia [24]. In our study, it was only performed for seven newborns; for the others we gave it up either because the neurological damage was already too severe, or because the jaundice had rapidly regressed under phototherapy tunnel.

The mortality rate in our series was 30%, comparable rates with 1/3 of children dying are reported in Nigeria and India [11] [31]. Brooks in the United States reported lower death rate (10%) between 1999 and 2006.

Our study has some limitations. As a retrospective work, we were not able to have a good evaluation of the prevalence, and our population is composed only by term infants with severe sign of bilirubin encephalopathy. In addition, it is a one center study that did not take into account all other cases occurring in other hospital wards of Dakar and all Senegal, so that we can have a population prevalence. It would be desirable to establish a national of BE. In the work, it was not possible to evaluate the risk factors of BE, because we did not compare with other babies with indirect hyperbilirubinemia.

Further children with BE were not followed up during a long period to estimate the exact long-term implication of BE in their neurodevelopment and quality of life. Evaluation of neurosensory problems was not done either.

5. Conclusion

EB is really concerning sub-Saharan Africa and Senegal. Only a better organization of perinatal care could reduce its incidence. Strengthening postnatal follow-up is necessary, in the context of early discharge from maternity, approximately 6 hours after delivery. Training and awareness campaigns for families and community health workers on early recognition of neonatal jaundice should be carried out, in order to promote the early use of health facilities prior to the installation of EB.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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