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Original Research Article

Acute bilirubin encephalopathy in term neonates: a hospital-based study

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ABSTRACT

Background: Acute Bilirubin Encephalopathy and kernicterus is an important cause of cerebral palsy, developmental delay and hearing impairment in low-middle income countries. Interventions such as universal screening for neonatal jaundice, Rhesus immunoglobulins, intensive phototherapy and exchange transfusion have made kernicterus rare in high income countries, but in our set up such cases continue to be reported.
Methods: Retrospective observational study where case records of term neonates brought to the neonatal ICU with signs and symptoms of acute bilirubin encephalopathy during the years 2016 and 2017 were sought and analysed.
Results: A total of ten term babies reported to the neonatal unit with severe hyperbilirubinemia along with signs and symptoms of bilirubin encephalopathy of which 60% were females. 90% had a birth weight of more than 2.5 kg and mean birth weight was 2.7±0.25 kgs. All the babies were out born. A 4 babies were born at home of which 3 pregnancies were completely unsupervised during the antenatal period. 90% of the babies were from the rural areas, 6 of the cases were from the districts Rajouri, Poonch and Reasi where the terrain is hilly, 2 from rural areas of Jammu and 1 from Kathua. Only 1 was from the Jammu city. The age at admission ranged from 3-9 days and serum bilirubin from 24 to 43.3 mg %. A 5 babies had ABO incompatibility, 1 Rh incompatibility, 1 sepsis, while no cause could be found in 3.
Conclusions: Neonatal jaundice is often not easily appreciated by mothers and caregivers in the home setting until it becomes severe enough, at which point neurological damage may have already occurred. There is an urgent need to train the primary health care personnel in assessment and early identification of risk factors for severe neonatal hyperbilirubinemia. They can help the families to seek prompt treatment for this preventable cause of cerebral palsy and mental retardation.

Keywords: Acute bilirubin encephalopathy (ABE), Kernicterus, Neonatal hyperbilirubinemia

INTRODUCTION

Neonatal hyperbilirubinemia, unrecognized and untreated in a timely manner, can cause serious and often irreversible post-ictal outcomes.[1] In 2004, American Academy of Pediatrics in an attempt to clarify the use of terminologies, recommended that the term acute bilirubin encephalopathy(ABE) be used to describe the acute bilirubin induced neurological symptoms manifesting in the first few weeks of life, while kernicterus should be reserved to describe the more chronic sequelae of ABE.[2,3] Kernicterus can have varied presentation. At the milder

end, children may have movement disorder or isolated auditory neuropathy and hearing loss.

Children with severe manifestations will have a permanent incapacitating condition characterized by dystonia, choreoathetosis, severe neurological hearing impairment, paralysis of upward gaze and dental enamel dysplasia.[4]

Kernicterus has become rare in developed countries. In United States least number of cases were reported from 1973 and 1983.

During this time liberal phototherapy was readily adopted and implemented by pediatricians who relied extensively on exchange transfusion for total serum bilirubin levels more than 20mg %. In 1990's however a resurgence of kernicterus was reported in infants that were being cared for in home environment often with limited medical supervision during the first week after birth.[5-7]

ABE remains a significant cause of morbidity and mortality throughout the world especially in low and middle income countries where it can account for up to 15% of the neonatal deaths.[8] However a comprehensive approach to diagnosis and a set of simple clinical interventions may help reduce the incidence of this largely preventable disease.[8] The surest way to prevent kernicterus is to prevent the occurrence of severe hyperbilirubinemia. Delayed recognition of severe hyperbilirubinemia, therefore, requires the urgent attention of pediatricians as well as other members of the health care team.[6]

Due to the absence of formal reporting it has been a challenge to gauge the magnitude of kernicterus in our country. No previous study has been conducted in our setting.

METHODS

A retrospective hospital record based observational study. Newborns with severe hyperbilirubinemia commonly report to the neonatal ICU of Pediatrics Department SMGS hospital Jammu.

It is a tertiary care facility that offers specialized management including intensive phototherapy, exchange transfusions, IVIG for such babies, and is manned by resident doctors who are specialists in Paediatrics and a consultant neonatologist.

SMGS hospital is the only referral hospital for such babies in whole of Jammu region. The hospital serves as a referral centre to private nursing homes, district hospitals and community health centres of the Jammu province. Authors decided to carry out retrospective hospital record-based study for the year 2016 and 2017.

Inclusion criteria

- The case records from the record section of SMGS Hospital for all the babies who reported to our neonatal unit with severe hyperbilirubinemia were analysed and only term babies who had signs and symptoms of acute bilirubin encephalopathy at the time of presentation were included in the study.

Exclusion criteria

- Preterm babies and those with meningitis were excluded from the study.

The clinical profile of such babies including residence, age of initial concern, clinical symptoms and signs, time interval between onset of symptoms and hospitalization, and outcome were recorded.

Individual patient information collected included date of birth, gender, gestational age, admission weight, place of birth, signs of bilirubin encephalopathy, serum bilirubin at the time of admission, blood group of the mother and baby, G6PD status, phototherapy and exchange transfusions provided and the outcome. An attempt was made to identify the risk factors.

Statistical analysis

Data was analysed using Microsoft Excel.

RESULTS

A total of ten term babies reported to the neonatal unit with severe hyperbilirubinemia along with signs and symptoms of acute bilirubin encephalopathy. All the babies were out born and the age at admission ranged from 3-9 days with a mean age of 3.9±2.2 days. 9 out of 10 babies were from the rural areas, 6 from the districts Rajouri, Poonch and Reasi where the terrain is hilly, 2 from rural areas of Jammu and 1 from Kathua.

Only 1 was from the Jammu city. 60% were females. 90% had a birth weight of more than 2.5kg and mean birth weight was 2.7±.25kgs. A 90% were born per vaginally and 30% of the pregnancies and deliveries were unsupervised.

Babies had serum bilirubin ranging from 24 to 43.3mg% with mean serum bilirubin of 33±0.6mg% and had moderate to severe ABE. 5 babies had ABO incompatibility, 1 Rh incompatibility, 1 sepsis, while in 3 no cause could be found.

All the babies were treated with double surface phototherapy and 9 underwent double volume exchange transfusion. 1 baby required 2 exchange transfusion while in 1 blood could not be arranged. All the babies survived.

Table 1: Demographic characteristics of term newborns with acute bilirubin encephalopathy.

Characteristic	Variable	Number	Percentage
Gender	Male	4	40
	Female	6	60
Birthweight	>2.5 kg	9	90
	<2.5 kg	1	10
Place of Birth	Inborn	0	0
	Out born	10	100
Place of delivery	Home	4	40
	Govt Health facility	5	50
	Private Clinic	1	10
Mode of delivery	PV	9	90
	LSCS	1	10
Pregnancy supervised	Yes	7	7
	No	3	30
Cause of jaundice	ABO incompatibility	5	50
	Rhesus incompatibility	1	10
	G6PD deficiency	0	
	Sepsis	1	10
	No aetiology	3	30
Treatment	Double surface phototherapy	10	100
	Exchange transfusion		
	Once	8	80
	Twice	1	10

DISCUSSION

Severe NNJ remains a life-threatening condition in many areas of the world, though the true dimension of the problem is largely unknown. Severe NNJ has different etiologies, dependent on variable genetic backgrounds and geographical location, even within regions of the same country. The identification of needs and a concerted effort to improve management at different levels of the health system can significantly reduce ABE and improve opportunities for thousands of newborns around the world.[9] In the present study all the babies were out born, mostly from far off areas and were brought to the hospital 3 to 9 days after births and serum bilirubin at the time of admission ranged from 24 mg% to 43.3 mg%. The delay in the seeking medical assistance could be due to hilly topography of Jammu province, low literacy, ignorance, non-availability of facilities of phototherapy and exchange transfusion in nearby hospitals and belief among the mothers that neonatal jaundice is a trivial disease that disappears with exposure to sunlight. Arnold G et al, in their study of 104 neonates with acute bilirubin encephalopathy identified home birth, self-referral, and G6PD screening status as important risk factors for presentation with acute bilirubin encephalopathy.[10]

In the present study 50% of the neonates had ABO incompatibility, 10% Rh incompatibility, 10% sepsis and 30% had no obvious cause. Bao et al, in a study of clinical features of 116 term and near term infants from China also reported that the most common cause of acute bilirubin encephalopathy was ABO incompatibility followed by sepsis and infection.[11] Ogunlesi TA et al, in a review of the hospital records of 115 babies managed for bilirubin encephalopathy in southwest Nigeria found that G6PD deficiency, ABO incompatibility and septicaemia were commonly associated causes. They also observed that prematurity, low birth weight, severe anaemia and inability to do exchange blood transfusion were significant risk factors for mortality among babies with bilirubin encephalopathy.[12]

Mukri S et al, in a study of risk factors for kernicterus concluded that risk of kernicterus is high with serum bilirubin >18 mg% even in term babies with non-hemolytic jaundice. They further stated that asphyxia and maximum serum bilirubin and free bilirubin are important risk factors for occurrence of kernicterus in term babies with non-hemolytic jaundice.[13] Targeted preventive strategies are essential at each health care level especially the primary health level, which include documentation of mother's blood group, use of Rh immunoglobulins, meticulous risk assessment and providing parents with written and oral information about jaundice.[14]

CONCLUSION

Neonatal jaundice is often not easily appreciated by mothers and caregivers in the home setting until it

becomes severe enough to deeply discolor the sclera and the skin, at which point neurological damage may have already occurred .A considerable number of infants arrive at the hospital or health care facility only after irreversible bilirubin encephalopathy has occurred training the primary health care personnel for early identification of neonatal hyperbilirubinemia can help the families seek prompt treatment for this preventable cause of cerebral palsy and mental retardation. Increasing the number of regular check-ups of babies born to mothers with O and Rh-negative blood groups should be strongly recommended as a preventive measure.

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