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Neonatal Jaundice: Its Myths and Facts

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Abstract:

Newborn jaundice is a yellow discoloration of the skin and eyes caused by an excess of bilirubin, a yellow-colored material produced by the liver or red blood cells, in the baby's blood. Jaundice can be caused by a variety of factors, but it is more common in premature neonates. Jaundice is easily diagnosable, but however require quick and on the spot treatment. If not treated properly, it leads to many complications. The best way to prevent jaundice is to breastfeed the baby early and often, as soon as possible after birth (ideally within the first hour, a cesarean section is done) and at least 8 to 12 times in each 24-hour period. Parents believing that their baby's jaundice could be treated by placing them in the sun, delays in readmitting a baby to hospital or admitting them to the neonatal intensive care unit (NICU), delays in starting treatment and failing to diagnose other related conditions during the pregnancy, such as Rhesus incompatibility (mismatch between the mother's and baby's blood group) are all common mistakes about neonatal jaundice. It is therefore recommended that midwives should educate mothers on causes, identification of probable signs and early transfer of the baby to the health facility for expert treatment.

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Introduction

Premature neonates are susceptible to blood disorders such as anemia and neonatal jaundice. Anemia is a condition in which the body's red blood cells are insufficient. While all newborns have a steady loss in red blood cell count over the first few months of life, preterm babies may have a faster drop. Newborn jaundice is a yellow discoloration of the skin and eyes caused by an excess of bilirubin, a yellow-colored material produced by the liver or red blood cells, in the baby's blood. Jaundice can be caused by a variety of factors, but it is more common in premature neonates.

Neonatal jaundice is a common newborn condition, affecting about 60 percent of full-term infants in the first week of life. It is also referred to as neonatal hyperbilirubinaemia. Jaundice is easily diagnosable, but however require quick and on the spot treatment. If not treated properly, it leads to many complications. Various myths and misconceptions about neonatal jaundice exist and this can lead to delay in seeking appropriate medical help when needed leading to lifelong complications or even loss of life.

Concept of Neonatal Jaundice

Neonatal jaundice is a yellowish discoloration of the white part of the eyes and skin in a newborn baby due to high bilirubin levels (Olusanya et al, 2015). It is also known as neonatal hyperbilirubinaemia.

Anne and Allison (2006) defined that jaundice is yellowish pigmentation of the tissues seen in the skin conjunctiva, caused by excess blood bilirubin and that jaundice is not a disease in itself , it is a sign of abnormal bilirubin, produced from the breakdown of haemoglobin is usually conjugated in the liver and excreted in the bile . Conjugation is the process of adding certain groups to the bilirubin molecules, makes it water soluble and greatly enhances its removal from the blood an essential step in excretion. Unconjugated bilirubin which is fat soluble, has a toxic to across brain cells.

However, is unable to cross blood-brain barrier until the plasma level rise above 340µmol/L, but when it does it may cause, neurological damage, fits and mental handicap. Serum bilirubin may rise to 340µmol before the yellow coloration of jaundice is evident in the skin and conjunctiva (normal 3 to 13µmol/L). Jaundice develops when there is an abnormality at some stage in the metabolic sequence caused by one or more factors like excess haemolysis of red blood cells with the production of more bilirubin than the liver can deal with, abnormal liver function that may cause incomplete uptake of unconjugated bilirubin by hepatocytes, ineffective conjugation of bilirubin or interference with bilirubin secretion into the bile from the liver to the duodenum.

Seeley, Stephens and Tate (2008) defined jaundice as a yellowish staining of the skin and sclera caused by a build-up of bile pigments in the circulation and interstitial spaces. These authors explained further that jaundice develops in the neonate because the liver is functionally immature. It lacks adequate amount of the enzyme required in the production of bilirubin. This enzyme system usually develops within two weeks after birth in a healthy neonate, as it is not fully developed at birth.

Parul (2010) defined jaundiced as the visible manifestation of hyperbiliubinaemia and that the clinical jaundice in neonates appear on the face at a serum bilirubin level of 5mg/dl, whereas in adult, it is diagnosed as little as 2mg/dl. The yellowish discoloration is first seen

on the skin of the face, nasolabial folds and tip of the nose in the neonate. It is detected by blanching the skin with digital pressure in the natural light. Neonatal jaundice is also termed as icterus neonatorum or as neonatal hyperbilirubinaemia. Amount 60% term neonate and about 80% preterm neonates have bilirubin level greater than 5mg/dl in the first week of life and about 6% of term babies will have bilirubin levels exceeding 15mg/dl.

Saladin (2004) viewed jaundice as a yellowing of the skin and sclera resulting from high levels of bilirubin in the blood. He explained that bilirubin is produced by the liver and jaundice may therefore occur whenever there is a rapid rate of erythrocyte destruction, disease such as hepatitis, liver cirrhosis and cancer interfere with liver functions and ability to dispose bilirubin effectively in preterm infants where the liver is not well developed.

Marilyn and David (2007) stated that the term "hyperbilirubinaemia" refers to an excessive level of accumulated bilirubin in the blood and is characterized by jaundice or icterus, a yellowish discoloration of the skin and other organs. Hyperbilirubinaemia is a common finding in the new-born and in most instances is relatively benign. However, in extreme cases, it can indicate a pathologic state. Hyperbilirubinemia may result from increased unconjugated or conjugated bilirubin. The unconjugated form is the most commonly seen in newborns.

Jaundice occurs because your baby's body has more bilirubin than it can get rid of. Bilirubin is made when the body breaks down old red blood cells. It leaves the body through urine and stool. During pregnancy, your body removes bilirubin from your baby through the placenta. After birth, your baby's body must get rid of the bilirubin on its own. Breast-fed newborns can become dehydrated easily if feedings are spaced too far apart. This lack of enough milk in the body makes it harder for your baby to get rid of wastes such as bilirubin. Also, some of the things that make up breast milk can change the way the body removes bilirubin. In rare cases, too much bilirubin may be caused by infections, a problem with the baby's digestive system, or a problem with the mom's and baby's blood types (Rh incompatibility). Your baby may have one of these problems if jaundice appears less than a day after birth.

Types of Neonatal Jaundice

There are four types of jaundice in neonates namely; physiologic, pathologic, breastfeeding and breast milk jaundice.

Physiological Jaundice

This is the most abundant type of newborn hyperbilirubinemia, having no serious consequences. It usually appears between 24–72 h of age and between 4th and -5th days can be considered as its peak in term neonates and in preterm at 7th day, it disappears by 10–14 days of life. Unconjugated bilirubin is the predominant form and usually its serum level is less than 15 mg/dl (Puppalwar et al, 2012).

Causes of Physiological Jaundice

As the name implies, it is due to normal physiological changes in newborn. When babies are inside the uterus they need extra red blood cells to meet their oxygen needs. After birth, these extra cells break down, releasing a substance called bilirubin. The liver filters bilirubin from the blood and excretes it in stool. It takes several days or weeks (even longer in preterm babies) before a newborn's liver functions fully, (Maisels et al, 2012). In the meantime, it can be hard for babies to get rid of the bilirubin that collects after birth. Low levels of bilirubin are

safe, but prolonged high levels may cause brain damage if left untreated. So, babies with jaundice should be carefully monitored and treated if their bilirubin levels get too high.

The baby's first stools consist of meconium, a black, sticky substance that contains, on average, 450 mg of bilirubin (which is quite a lot). Colostrum, the first milk a mother's body makes, acts as a natural laxative to cause the passage of meconium. Frequent breastfeeding results in frequent stools (infant formula lacks this special laxative). Practices that interfere with breastfeeding, separation of mother and baby, rigid feeding schedules (as opposed to 8-12 or more feedings in each 24 hours), lack of skin-to-skin contact, early use of pacifiers, and poor positioning or latch (limiting milk transfer), result in fewer breast feedings, fewer stools, and a greater risk for worsening jaundice.

Diagnosis of Neonatal Jaundice

Neonatal jaundice diagnosis is made clinically by checking for yellowish discoloration of the skin and sclera and in the laboratory by checking blood sample for serum bilirubin level.

Treatment of Physiologic Jaundice

The best treatment for physiologic jaundice is frequent and effective breastfeeding, at least 8-12 or more times in each 24-hour period. Giving water, glucose water, or formula supplements won't help because they lack the laxative effect of colostrum. Instead, they can interrupt development of the mother's milk supply and increase the risk of weaning. Photo therapy might be needed if the value exceeds 12mg/dl (Okwundu et al, 2012).

Prevention of Physiologic Jaundice

The best way to prevent jaundice is to breastfeed the baby early and often, as soon as possible after birth (ideally within the first hour, a cesarean section is done) and at least 8 to 12 times in each 24-hour period. Responding to baby's early signs of hunger; not waiting for baby to cry, as that is a late sign of hunger and may actually interfere with effective feeding.

Avoid supplementation with water, glucose water, or infant formula. Do not supplement with expressed milk, donor milk, or formula unless there is a medical reason (such as weight loss of more than 10 percent after attempts to correct breastfeeding problems, failure to produce or transfer breast milk, evidence of dehydration). It will be known if a baby is getting enough to eat if there are three or more stools a day by day 3. The baby's stool will be black (meconium) on days 1 and 2; green (transitional) on days 3 and 4; and yellow (normal) by day 5. If it continues to be black stools on day 3 or green stools on day 5, there might be need to contact the baby's health care provider right away. This could be a sign that the baby isn't getting enough to eat.

Pathological Jaundice

Pathologic jaundice is the most serious type of jaundice. It occurs within 24 hours after birth, and is characterized by a rapid rise in a baby's bilirubin level. The most likely cause is blood incompatibility or liver disease. Prompt medical attention is necessary, and blood transfusions may be required. Breastfeeding can continue during treatment. Bilirubin levels with a deviation from the normal range and requiring intervention would be described as pathological jaundice. Appearance of jaundice within 24 h due to increase in serum bilirubin beyond 5 mg/dl/day, peak levels higher than the expected normal range, presence of clinical jaundice more than 2 weeks and conjugated bilirubin (dark urine staining the clothes) would be categorized under this type of jaundice.

Causes of Pathologic Jaundice

The most common causes of pathologic jaundice are haemolysis arising from (a) Rh hemolytic disease, (b) ABO incompatibility and (c) Glucose-6-phosphate dehydrogenase (G-6-PD) deficiency and minor blood group incompatibility and breakdown of blood clots arising from birth injuries and bleeding into spaces like hematoma, etc.

1. Rh Factor Hemolytic Disease: Rhesus hemolytic disease of the newborns (RHDN) results from maternal red-cell alloimmunization. Maternal antibodies are produced against the fetal red blood cells, when fetal red blood cells are positive for a certain antigen, usually at what time a baby having Rh positive born to a Rh-negative mother (and Rh-positive father), then maternal immunoglobulin (IgG) antibodies might cross the placenta into the fetal circulation and cause a wide variety of symptoms in the fetus, ranging from mild to severe hemolytic anaemia and fetal hydrops. To facilitate early treatment in neonates who are dubitable to have Rh factor, a blood group and Rh typing, DCT, PCV (packed cell volume) and serum bilirubin on cord blood should be performed. A reticulocyte count should be sent before the first exchange transfusion (ET). Vigorous phototherapy is required immediately after the birth and it should be continued until a level, which is 5 mg/dl less than the level estimated for exchange blood transfusion. In preterm babies, lower values of intervention for treatment of Rh hemolytic disease have been demonstrated. Phototherapy and exchange blood transfusion are recommended when a level is greater than 0.5% and 1% birth weight (kg) respectively. Eight intravenous immunoglobulin (IVIG) can be used in a dose of 500 mg/kg 12 hourly × 2 doses after the first ET. After the first ET starting of Phenobarbitone 5 mg/kg/day × 5 may be recommended.
2. ABO Incompatibility: The incidence of the incompatibility of the ABO blood groups of the mother and fetus, when the mother has the blood group O and the newborn has the A or B blood group, is 15–20% of all pregnancies. Babies with O-blood group mothers should be closely checked for and discharged after 72 h. Routine cord blood screening is not recommended for newborns with O-group mothers. Jaundice owing to ABO incompatibility usually appears 24 h after the birth. In the presence of significant jaundice or jaundice appearing within 24 h, the work up for pathological jaundice should be done. Intensive phototherapy is advised at SB 12–17 mg/dl depending upon postnatal age of the baby. Exchange blood transfusion is indicated at TSB. The weight at birth as a criterion for phototherapy and ET may be used for preterm newborns.
3. Glucose-6-Phospate Dehydrogenase (G6PD) Deficiency: G6PD, most common enzymopathy, is the deficiency of an enzyme in RBCs (Moiz et al, 2012). It is the most vital disease of the pathway of hexose monophosphate. Investigations for G6PD deficiency should be considered in infants with severe jaundice in a family with a history of significant jaundice or in a geographic origin associated with G-6-PD deficiency. Decreased bilirubin conjugation resulted from variation in the UGT1A1 and OATP2 genes play an important role in the progression of hyperbilirubinemia in G6PD deficient newborns (D'Silva et al, 2014).

Treatment Options for Pathologic Jaundice

The treatment options for jaundice include phototherapy further subdivided to conventional, intensive and exchange transfusion, and pharmacological treatment subdivided to phenobarbitone, intravenous immunoglobulins (IVIG), (Smith- Wintjens et al, 2011), metalloporphyrins and follow up remedies.

1. Phototherapy: Hyperbilirubinemia can be treated easily without or with a minimal adverse effect with phototherapy. The efficacy of phototherapy depends on surface area exposed to phototherapy: Double surface phototherapy may be more effective than single surface phototherapy). Spectrum of light source: Special blue tubes with the mark F20T12/BB should be used rather than F20T12/B lights and Irradiance or energy output may be increased in a phototherapy unit by lowering the distance of the neonate to within 15–20 cm. Continuous phototherapy is better than intermittent phototherapy. Phototherapy should not be interrupted except during breast-feeding or nappy change.
    a. Conventional Phototherapy One can use conventional or fiber-optic phototherapy units provided jaundice is non-hemolytic or its progression is slow.
    b. Intensive Phototherapy In the circumstances including hemolytic jaundice, rapidly increasing bilirubin, or ineffectiveness of a conventional unit, using of intensive phototherapy is warranted. Placing the baby on the bili-blanket and using additional overhead phototherapy units contain blue lights and then lowering the phototherapy units to within a distance of 15–20 cm are two significant remedies.
2. Exchange Transfusion: Through exchange transfusion bilirubin and hemolytic antibodies are removed.
3. Pharmacological Treatment: Pharmacological treatment of neonatal jaundice can further be categorized into different subheadings such as phenobarbitone, Intravenous immunoglobulins and Metalloporphyrins etc. (Kaseem et al, 2013).
    a. Phenobarbitone: Bilirubin processing including hepatic uptake, conjugation and its excretion are ameliorated by this agent thus helps in decreasing level of bilirubin. However, the effect of phenobarbitone is not rapid and takes time to show. When used for 3–5 days in a dose of 5 mg/kg after birth prophylactically, it has shown to be effective in babies with hemolytic disease, extravasated blood and in pre-term without any significant side effects. There is a huge literature documenting efficacy and mechanism of action and complications of treatment for Phenobarbital.
    b. Intravenous Immunoglobulin (IVIG): High dose IVIG (0.5–1 gr/kg) has shown to be effective in decreasing the needs of exchange transfusion and phototherapy in babies with Rh hemolytic disease (Cortey et al, 2014).
4. Follow-up: Babies having roughly 20 mg/dl serum bilirubin and that requiring exchange transfusion should be kept under follow-up in the high risk clinic for neurodevelopmental outcome. Hearing assessment (Brainstem Evoked Response Audiometry (BAER)) should be done at 3 months of corrected age.

Breast Feeding Jaundice

This is now known as Sub-optimal intake jaundice. Formerly called "breastfeeding jaundice" or "lack of breastfeeding jaundice" or even "starvation jaundice" this is caused by infrequent or ineffective breastfeeding. The Academy of Breastfeeding Medicine (ABM) says it is the result of too little breastfeeding (from breastfeeding difficulties or a delay in milk production) and therefore low caloric intake, combined with limitations in bilirubin metabolism and transport. All of this can cause bilirubin levels to be higher in the baby's blood. Formula feeding is no "cure-all" for this kind of jaundice; the key is to make sure your child is taking in enough calories. Often, this resolves once the mother's milk supply improves when her baby is around 2-5 days of age. Skin-to-skin care and frequent breastfeeding can help, as the ABM explains, "with milk supply and makes mother's milk easily available to the infant" To ensure that a breastfeeding baby is transferring milk, there is need to listen for sounds of suckling and swallowing (Kaseem, et al., 2013).

Exclusively infants with breastfeeding have a different physiological pattern for jaundice compared with artificially feed babies. Jaundice in breast fed babies usually appears between 24–72 h of age (Kubol, et al, 2013), peaks by 5–15 days of life and disappears by the third week of life. Higher bilirubin levels have been reported in these infants. In case of breastfed newborns, mild jaundice may take 10–14 days after birth or may reoccur during the breastfeeding period. Very large amounts of bilirubin rarely accumulate in the blood and cause cerebral lesions, a situation known as nuclear jaundice. These cuts may be followed by hearing loss, mental retardation, and behavioral disorders. A mild clinical jaundice has been observed in one third of all breastfed babies in the third week of life, which may persist for 2 to 3 months after birth in a few babies Decreased frequency of breastfeeding is associated with exaggeration of physiological jaundice. One of the significant procedures to manage the jaundice in a term healthy baby is the mothers' encouragement to breastfeed their babies at least 10–12 times per day (D'Silva, et al., 2014).

Breast Milk Jaundice

Hyperbilirubinemia is also associated with breast milk of mother in neonates. About 2%–4% of exclusively breastfed babies have jaundice in excess of 10 mg/deal in the third week of life. These babies in the third week of life with bilirubin serum levels higher than 10mg/dl should be considered for prolonged jaundice. A diagnosis of breast milk jaundice should be investigated if the serum bilirubin is predominantly unconjugated, other causes of prolonged jaundice have been eliminated and the infant is in good health, vigorous and feeding well and gaining weight adequately. Mothers should be advised to continue breastfeeding at more frequent intervals and bilirubin levels usually diminish gradually. Discontinuity of breastfeeding is not recommended unless levels exceed 20 mg/dl (Colletti, et al., 2007).

<u>Causes</u>

Although breast milk jaundice is quite rare, it often causes concern in part because why it happens is unclear. There may be differences in the infant's reabsorption of the bilirubin, or in the mother's milk. Breast milk jaundice can appear 2-5 days after birth. Bilirubin levels peak around 10–14 days, but they may remain high for several weeks, even as much as 3 months.

Management

If the bilirubin level continues to climb, the baby's health care provider may suggest supplementing breastfeeding with mother's milk or formula until jaundice resolves. In rare cases, breastfeeding may be interrupted for 24 hours, in an effort to reduce the bilirubin level. If this course of treatment is followed, then, there is need for mothers to continue expressing breast milk to maintain milk supply (Gilmour, 2004).

Myths and Misconception about Neonatal Jaundice

Parents believing that their baby's jaundice could be treated by placing them in the sun, delays in readmitting a baby to hospital or admitting them to the neonatal intensive care unit (NICU), delays in starting treatment and failing to diagnose other related conditions during the pregnancy, such as Rhesus incompatibility (mismatch between the mother's and baby's blood group) are all common mistakes about neonatal jaundice. Some of the myths includes:

1. All newborn babies have jaundice – it is normal.
2. Neonatal jaundice was passed from mother to child
3. Neonatal jaundice is harmless
4. Neonatal jaundice will resolve with time
5. Any kind of light will treat jaundice
6. Kernicterus is a rare complication of jaundice, so can't claim compensation for the child's brain injury

All newborn babies have jaundice – it is normal.

Neonatal jaundice is very common in newborn babies, particularly those that are premature, but not all babies develop the condition. 60% of babies born at full term and 80% of premature babies show signs of jaundice during the first few days of life and the signs includes: Yellow discolouration of the baby's skin and whites of their eyes, and dark staining of their urine which is visible on their nappy (Bhutani, 2012). Breastfed babies are at greater risk of developing neonatal jaundice. Babies with signs of jaundice must be diagnosed, monitored and treated quickly if it doesn't resolve to avoid serious, permanent injury (Cortey, et al., 2014).

Neonatal jaundice was passed from mother to child

Neonatal jaundice is caused by excessive bilirubin levels in the baby's blood. It is not caused by the mother's diet or behaviour in pregnancy. Some family-related or hereditary conditions can lead to jaundice, such as Rhesus incompatibility, where the mother and baby have incompatible blood groups. This should be detected in pregnancy and appropriate treatment given to safeguard the baby. Other hereditary conditions which can cause jaundice include sickle cell anaemia or cystic fibrosis. It can also be caused by infection. Where the mother's previous child (the baby's older sibling) needed phototherapy for neonatal jaundice, the current baby is at greater risk of jaundice (Kaseem, et al., 2013).

Neonatal jaundice is harmless

Neonatal jaundice is usually harmless but it must be diagnosed, monitored and treated with phototherapy or an exchange transfusion if it doesn't resolve early. This is because prolonged or severe untreated jaundice can develop into more serious conditions. There have beem several legal actions taken for many clients who developed a pattern of brain damage known

as kernicterus when their neonatal jaundice was not properly treated leading to lifelong disability from cerebral palsy, learning disability and hearing loss (Kuboi, et al., 2013).

Neonatal jaundice is caused when the baby has excessive levels of bilirubin in their blood (known as hyperbilirubinaemia). Bilirubin is a yellow pigment that is released into the bloodstream during the body's normal process of breaking down old red blood cells. Bilirubin is processed in the liver and then stored in the bile duct and gallbladder. It helps the body digest fats in the small intestine (as bile), and is then excreted with the rest of the body's waste. If the baby's liver is struggling to process all the bilirubin in the baby's bloodstream, the excess, unprocessed bilirubin causes irreversible damage to the brain and spinal cord, causing permanent neurological disability. This is known as bilirubin encephalopathy. The pattern of damage and its characteristic yellow staining of the brain is known as kernicterus (Maisels, et al., 2012).

Kernicterus brain damage is preventable. It occurs when the baby's bilirubin levels have been allowed to rise to dangerous levels through inadequate monitoring or delayed treatment of hyperbilirubinaemia. Signs of neonatal jaundice may also indicate that the baby is suffering from other conditions which may need treatment, such as: Infection or sepsis; Liver disease; Incompatibility between the mother's and the baby's blood groups (Rhesus incompatibility); Bruising and metabolic disorders; Sickle cell anaemia; Enzyme deficiencies (Olusanya, et al., 2018).

Neonatal jaundice will resolve with time

Neonatal jaundice usually resolves within two weeks and may take a little longer if the baby is breastfed, but it is essential that the condition is recognised, diagnosed, properly monitored, and treated if the baby's bilirubin levels reach the threshold level for treatment or are rising at a rapid rate towards the threshold (Parul, 2010).

Any kind of light will treat jaundice

NHS Resolution's review of neonatal jaundice-related claims gave examples of midwives incorrectly advising parents to put their jaundiced baby in the sunlight to treat their condition. The national guideline, NICE Guideline CG98, which sets out the standard of care expected for the treatment of jaundice in newborn babies under 28 days, specifically states that sunlight should not be used as a treatment for neonatal jaundice.

Babies with high levels of bilirubin need phototherapy in hospital with appropriate monitoring. Phototherapy involves exposing the baby to a special type of light which helps break down and remove the excess bilirubin. The baby's eyes are protected and parents can still enjoy cuddles with their baby during the treatment. Phototherapy is usually effective but if the baby's bilirubin remains dangerously high, they may need an exchange blood transfusion to avoid damage to their brain. Sunlight is not an effective treatment and also carries the risk of dehydration (making the condition worse) and sunburn to the newborn baby's sensitive skin (Puppalwar, et al., 2012).

Kernicterus is a rare complication of jaundice, so can't claim compensation for the child's brain injury

Failure to diagnose jaundice or properly monitor bilirubin levels, delays in admitting or readmitting a discharged baby to hospital/NICU and delays in phototherapy or exchange transfusion treatment are all negligent care. So is falsely reassuring parents or failing to

advise them to seek urgent medical help if the baby's condition doesn't resolve or deteriorates. Mistakes such as these can lead to delays in essential treatment and severe brain injury and permanent disability from kernicterus (Shah, et al., 2003).

Where this occurs, the child's family can seek help from specialist solicitors with expertise in kernicterus brain injury as the child is entitled to claim significant compensation. Legal practitioners have helped children affected by kernicterus recover early interim payments and substantial compensation including costs of lifelong care and case management, adapted accommodation, education, therapies and medical costs, specialist equipment, assistive technology and transport, along with loss of earnings and other financial losses (Shah, et al., 2003).

Conclusion

Hyperbilirubinemia is more severe in newborns. Therefore, precautionary measure should be adopted by both parents, and clinicians to diagnose and treat the disease properly. Government and public health organizations should arrange seminars, workshops and trainings for mothers regarding neonatal jaundice. Medical scientists should search for new treatments and preventive measures having no side effects and capable of recovering babies more speedily. Partners should screen their ABO blood groups as well as Rh factor before marriage. Consanguineous marriages should be avoided.

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