

THE ASSOCIATION BETWEEN ASPHYXIA AND NEONATAL PATHOLOGICAL JAUNDICE INCIDENCE IN UMBU RARA MEHA HOSPITAL EAST SUMBA REGENCY

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ABSTRACT

Jaundice causes 70% morbidity and 10% infant mortality worldwide, approximately 1.1 million newborns are affected by jaundice every year. The purpose of this study was to analysis the association between asphyxia and the incidence of neonatal pathological jaundice at Umbu Rara Meha Hospital, East Sumba Regency. The research used a quantitative method with case and control approach using secondary collection data medical records from January to October 2023. The sample ratio was 1:1, the case group sample is 75 babies with jaundice and the control group sample is 75 babies who were born in the hospital. The sampling taken by consecutive sampling. The analysis used the Chi-square test. The data analysis showed there was an association between asphyxia and neonatal pathological jaundice incidence with $p=0.001$. The data result showed there was an association between asphyxia and neonatal pathological jaundice incidence at Umbu Rara Meha Hospital, East Sumba Regency.

Keywords: Jaundice, Neonatal, Neonatal Health, Asphyxia



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INTRODUCTION

Neonatal jaundice is a condition of yellow discoloration of the skin, mucosa, and sclera due to elevated blood serum bilirubin levels $> 5\text{mg/dl}$ (Ministry of Health, 2019). Approximately 10% of all newborns have elevated bilirubin levels, with around 1.1 million newborns every year affected by jaundice (WHO., 2018). Jaundice causes 70% morbidity and 10% mortality worldwide. The current incidence rate is 667.8 per 10,000 births in southern Asia and Africa and 3.7/10,000 births in Europe (Nayagi and Angel, 2023). Based on the results of the Indonesian

Demographic Health Survey (2018), The neonatal mortality rate in Indonesia is 32/1000 live births. and it is caused by asphyxia (37%), low birth weight babies (34%), sepsis (12%), hypothermia (7%), jaundice neonatorum (6%), and congenital abnormalities (1%).

According to East Nusa Tenggara Provincial Health Office data (2022), the incidence of neonatal jaundice has increased to 10.2%. And at Umbu Rara Meha Hospital 2020 there were 39 cases, in 2021 there were 61 cases and in 2022 there were 80 cases. In January-October 2023 there were 89 cases. In 2021 there were 61 neonatal jaundices treated and 1 baby died because of jaundice. In 2022 There were 80 neonatal jaundice cases, and one baby died with the main diagnosis and two babies died with comorbidities. It could be seen from the number of jaundice cases, there is an increase of neonatal jaundice every year (Perinatal Room Report of Umbu Rara Meha Hospital in 2023).

According to Sri Susanti (2022), several maternal, perinatal, and neonatal factors that are common causes of jaundice in newborns are asphyxia, LBW, sectio Caesarea, sepsis, congenital abnormalities, gestation period, type of delivery, ABO and Rh blood group incompatibility, G-6-PD enzyme deficiency, low birth weight, asphyxia, prematurity, polycythemia, gender, breast milk, drugs, race, and mothers with a history of DM. The impact that often occurs when jaundice is not quickly treated can cause kernicterus and encephalopathy, which are acute manifestations of bilirubin toxicity encountered in the first week after birth (Ministry of Health, 2019).

Neonatal jaundice is divided into two categories, namely physiological and pathological jaundice. Physiologic jaundice usually appears after 24 hours and resolves within two to three weeks. Physiologically higher total serum bilirubin (TSB) levels (Ansong-Assoku., 2023). According to the Ministry of Health (2022), pathological jaundice is seen within the first 24 hours of life, elevated bilirubin > 5 mg/dl within 24 hours, index bilirubin for full-term infants > 13 mg/dl and less-term infants > 10 mg/dl, rec bilirubin level > 2 mg/dl, and persists at more than two weeks of age.

The ethology of neonatal jaundice is divided into unconjugated hyperbilirubinemia and conjugated hyperbilirubinemia. Unconjugated hyperbilirubinemia is a condition in which there is an increase in serum or plasma bilirubin levels that usually occurs due to dysregulation of bilirubin metabolism, which includes increased production, impaired absorption in the liver, and decreased conjugation of bilirubin. In newborns, unconjugated hyperbilirubinemia is very common, and elevated levels of unconjugated bilirubin can lead to life-threatening kernicterus (Singh., 2023). Conjugated hyperbilirubinemia is characterized by an increase in conjugated or direct serum bilirubin (>10 mg/dL) and is caused by impaired hepatobiliary function, which includes the liver, bile ducts, spleen, and pancreas. Conjugated jaundice is almost always pathologic and requires immediate evaluation and treatment (Joseph and Samant, 2023).

Metabolizing bilirubin utilizing albumin binding: after bilirubin is released into the plasma, albumin picks it up throughout the body as a transporter. Albumin

strongly binds bilirubin, and under ideal conditions, plasma does not contain free, unconjugated bilirubin (not bound to albumin). Following the hepatic transport mechanism, bilirubin is absorbed into the hepatocytes of the liver sinusoids through two different mechanisms: passive diffusion and receptor-mediated endocytosis. Most of the unconjugated bilirubin entering the hepatocytes is extracted. The conjugated bilirubin exiting the hepatocytes is excreted via urine.

According to Abbey (2019), the yellow colour that comes from jaundice may be more visible elsewhere, such as on the sclera, nails, mucous membranes, soles of the feet, and palms of the hands. Yellowing of the skin is usually more noticeable on the head and face. Symptoms like drowsiness, unwillingness to suckle or not suckling as usual, and unconsciousness. Dark yellow urination, which should be colorless; pale-colored stools, which should be yellow or orange; and malaise, anorexia, fatigue, dark urine, and stool color.

One of the risk factors that causes jaundice is asphyxia, because it can cause an increase in bilirubin. This is due to the reduction of glycogen produced in the liver due to insufficient oxygen intake to the organs of the body, so the function of the hepatic organs is not optimal. Infants who have had a history of asphyxia will tend to experience neonatal jaundice. In neonates, liver function is not yet fully functional, so the bilirubin glucuronidation process does not occur optimally. If there is a disturbance in liver function, it can cause hypoxia, acidosis, and a lack of glucose in the body, which can cause high levels of indirect bilirubin in the blood (Pratiwi., 2022). Therefore, it is necessary to analyze the association between asphyxia and the incidence of pathological neonatal jaundice at Umbu Rara Meha Hospital, East Sumba Regency.

RESEARCH METHOD

The research used a quantitative method with case and control approach using secondary collection data medical records from January to October 2023. The population in this study were all the babies admitted to Umbu Rara Meha Hospital from January to October 2023, namely 1,007 babies. The sample ratio was 1:1, the case group sample is 75 babies with jaundice and the control group sample is 75 babies who were born in the hospital. The sampling taken by consecutive sampling. The analysis used the Chi-square test.

RESULTS AND DISCUSSION

The results showed that the distribution of respondents based on asphyxia incidence variables and neonatal pathological neonatal jaundice incidence.

Frequency distribution of asphyxia incidence variables at Umbu Rara Meha Hospital, East Sumba Regency

Table 1 Frequency distribution of asphyxia incidence variables at Umbu Rara Meha Hospital, East Sumba Regency

	Neonatal Jaundice			
	Case		control	
Asphyxia	frequency	(%)	frequency	(%)
- Asphyxia	20	26,7	4	5,3
- Non-asphyxia	55	73,3	71	94,7
Total	75	100	75	100

Based on table 1, The majority of sample in the case group had no asphyxia (73.3%) and most of sample in the control group had no asphyxia (94.7%).

The association between asphyxia and neonatal pathological jaundice incidence at Umbu Rara Meha Hospital, East Sumba Regency

Table 2 The association between asphyxia and neonatal pathological jaundice incidence at Umbu Rara Meha Hospital, East Sumba Regency

Asphyxia	Neonatal Jaundice							
	Case		Control		Total			
	frequency	(%)	frequency	(%)	N	%	p	OR
- Asphyxia	20	26,7	4	5,3	24	16,0	0,001	6,455
- Non-asphyxia	55	73,3	71	94,7	84,0	84,0		
Total	75	100	75	100	150	100		

Based on Table 2, the chi-square analysis test showed that there was an association between asphyxia and neonatal pathological jaundice incidence at Umbu Rara Meha Hospital, East Sumba Regency, with a p-value = 0.001 and an OR of 6.455, which means that asphyxia babies are at a risk of 6.4 times experiencing neonatal jaundice

DISCUSSION

This study aimed to determine the association between asphyxia and the neonatal jaundice incidence. there was an association between asphyxia and neonatal pathological jaundice incidence at Umbu Rara Meha Hospital, East Sumba Regency, with a p-value = 0.001 and an OR of 6.455, which means that asphyxia babies are at a risk of 6.4 times experiencing pathological neonatal jaundice.

This research is in line with research conducted by Suprihatini (2023) at Tanjungpinang City Hospital, which showed an association between asphyxia and the incidence of neonatal jaundice. This is because the liver and other organs can have a negative impact due to the severity of hypoxemia in asphyxiated neonates. This research is also supported by research conducted by Rohani (2017), which found a association between asphyxia and the incidence of neonatal jaundice. A

hepatic physiological function can cause hepatic shock (severe hepatic disorders) caused by asphyxia. The reduction of glycogen produced by the body in the liver is caused by insufficient oxygen intake to the organs of the body, so the function of the hepatic organs is not optimal. Babies who already have a history of asphyxia will tend to experience neonatal jaundice. In neonates, the liver function does not function fully, so the bilirubin glucuronidation process does not occur optimally (Pratiwi., 2022).

This study is supported by research conducted by Widiawati (2017), showing there is an association between asphyxia and neonatal jaundice. This is because asphyxia in infants causes a lack of oxygen distribution to the organs of the neonate's body and can cause the work function of the organs to not be maximized, resulting in changes in function and perfusion to the liver. Glycogen produced by the liver will be reduced, causing jaundice in the long term and death in the short term (Saptanto., 2017). This study is also supported by Rozilina (2023) at NTB Provincial Hospital. Stating that asphyxia is associated with the incidence of neonatal jaundice. This occurs due to hypoperfusion in the liver due to a lack of oxygen, which will disrupt the uptake and metabolism of bilirubin by hepatocyte cells as well as the production of unconjugated bilirubin, which increases beyond the ability to excrete it.

Neonatal who experiencing asphyxia with an Apgar score of less than 7 within 1 minute are classified as having severe birth asphyxia according to the classification of diseases (WHO., 2022). According to the Ministry of Health (2019), neonatal asphyxia is the failure of a baby to breathe spontaneously and regularly at birth or shortly after birth, characterized by hypoxemia and acidosis. This is due to a lack of oxygen to the organs, so organ function is not optimal and glycogen produced by the liver is reduced. Asphyxia can cause hepatic hypoperfusion, which will then interfere with hepatocyte bilirubin metabolism (Wardhani, 2023).

The low oxygen intake to the organs of neonates causes the organs to not function optimally. As a result, the glycogen produced by the body in the liver decreases, leading to jaundice and death in the short term. In neonates with asphyxia, jaundice can be prevented by monitoring the pregnancy to prevent fetal distress or asphyxia in the fetus and providing rapid and appropriate resuscitation care to asphyxied neonates, thus reducing neonate mortality. Oxygen deprivation in the fetus reduces oxygen intake to organs and tissues, causing jaundice neonatum. These results also show that infants with a history of asphyxia are more likely to experience neonatal jaundice because hepatic function is not yet functioning properly and the process of bilirubin glucuronidation is inhibited (Widiawati, 2017).

This research contradicts the research conducted by Auliasari (2019). Stating that there is no association between asphyxia and neonatal jaundice. This is because almost all neonates do not experience asphyxia. For neonates with a history of asphyxia, resuscitation was performed immediately after birth and early adequate breast milk intake. With the results of this study, it can be concluded that asphyxia is a risk factor for neonatal jaundice. This is due to the lack of oxygen intake to the organs, so that organ function is not optimal and the glycogen produced by the liver is reduced, which causes jaundice.

This research contradicts the research conducted by Primasdika (2023), who stated that there is no association between asphyxia and neonatal jaundice. This is caused by the fact that one way to prevent asphyxia is to identify the risk of asphyxia and provide appropriate labor assistance. To prevent oxygen deprivation and the accumulation of unconjugated bilirubin, appropriate and rapid initial treatment in asphyxia cases is carried out.

The impact that occurs when the risk of neonatal jaundice is not treated immediately, namely acute bilirubin encephalopathy,” refers to the acute symptoms of bilirubin toxicity that appear within the first week after birth. Infants with severe jaundice will show poor suction reflexes, weak cry movements, and hypotonia in the early phase of acute bilirubin encephalopathy. Impaired consciousness, irritability, and hypertonia are signs of the intermediate phase. These babies may have a fever and loud cries, which later turn into lethargy and hypotonia. Retrocollis, meaning the neck arches backwards, and opisthotonus, meaning the torso arches backwards, are manifestations of hypertonia. The chronic and permanent sequelae of bilirubin toxicity are referred to as kernicterus. Infants who survive chronic bilirubin encephalopathy may develop severe conditions such as athetoid cerebral palsy, hearing loss, dental-enamel dysplasia, upward eye paralysis, or, in rarer cases, intellectual disability. In the acute phase of bilirubin encephalopathy, most infants with kernicterus show some or all of the signs mentioned above. However, infants occasionally have very high elevations in bilirubin levels, and then, if present, signs of kernicterus appear only partially before clinical signs of acute bilirubin encephalopathy appear (Ministry of Health, 2019).

The management of unconjugated hyperbilirubin involves phototherapy, exchange transfusion, intra-venous immunoglobulin (IVIG), and sunlight therapy. Phototherapy remains the main treatment for treating unconjugated hyperbilirubin, where blood bilirubin levels are usually more than 15 mg/dl. Phototherapy is very effective in reducing total serum bilirubin, as bilirubin absorbs light in the blue-green range (460–490 nm). The newborn's eyes should be closed during phototherapy so that the retina is not damaged. As most bilirubin is excreted through

urine, it is important to maintain adequate hydration and ensure normal urine output. The management of conjugated hyperbilirubin is tailored to its specific etiology. Patients diagnosed with biliary atresia require Kasai surgery (hepatic portoenterostomy) (Ansong-Assoku., 2023).

According to the Ministry of Health (2019), ursodeoxycholic acid (UDCA) is used as a therapy for cholestasis because it can improve bile flow and reduce TSB levels. However, there is little data on the safety and efficacy of UDCA as a therapy compared to phototherapy, so UDCA is not recommended for regular use. The use of phenobarbital after birth is controversial and generally not recommended due to the side effects of phenobarbital administration, such as sedation in newborns. It can take several days before results are seen, but prophylactic use to reduce phototherapy or exchange transfusions in infants with G6PD deficiency has been found to be ineffective.

CONCLUSION

The results of the research that has been done indicate that there was an association between asphyxia and the neonatal jaundice incidence. there was an association between asphyxia and neonatal pathological jaundice incidence at Umbu Rara Meha Hospital, East Sumba Regency. After it became known that asphyxia was associated to incidence of neonatal jaundice, it was hoped that health workers, especially midwives in charge, will continue and improve services in the form of collaboration with health centers, clinics, and the private sector in preconception health promotion and screening activities in nutritional preparation, pregnancy distance, physical and mental preparation. For hospital services to be able to prevent and minimize babies born with asphyxia, it was hoped that these findings could be used as a reference. Future researchers are expected to investigate the additional variability associated with the incidence of neonatal jaundice.

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