



بسم الله الرحمن الرحيم

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**Research submitted for partial fulfillment
of master degree in pediatric nursing**



Assessment of nurses knowledge regarding neonatal sepsis

_ Sudan _ Khartoum _

neonatal intensive care unit _

soba teaching hospital

nov 2017 to apr 2018.

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university 2018

2016-2018

الاية

قال تعالى:-

(فبأي آلاء ربكما تكذبان)

صدق الله العظيم

Dedication

TO:-

our mother and father of lovely and and light of the life.

To :-

My second mother my sister OMIA for his imotional and fainatiol support.

To:-

Family members the candles which bright the science and success full way
for us...

Acknowledgment

Great thank to my god and all my thank and deepest respect for:-

Our brothers , sisters ,colleagues ;source of our greatness.

And our teachers who bright the science and knowledge way for us.

And every one who contribute and play role in this search.we present for ours this little work is a few form more.

Deepest thank to my supervisor DR **.Imyaeltaibalhady**

And my thank extend to all staff in NICU in soba university hospital.

Abstract

background: neonatal sepsis is invasive infection, usually bacterial occurring during the neonatal period, the highest rate occur in low birth weight infant, infant with depressed function at birth as manifested by low Apgar score and infant with maternal prenatal risk factor such as low socio-economic status and PROM.

the present study is descriptive cross sectional study was conducted at the neonatal intensive care unit at Soerabaya University Tashih Hospital to assess nurses knowledge regarding neonatal sepsis in period between the Nov 2017 to April 2018.

The sample includes 32 nurses (3 pilot), all nurses worked up on neonatal intensive care unit at the time of the study and some nurses worked up on the unit at previous time.

the data was collected by using questionnaire which designed by the researcher.

The questionnaire consist of 15 questions divided into three parts, which the first part of question about socio-demographic data, the second part of questions about the knowledge of nurses regard neonatal sepsis and last part of questions about the performance.

The data was analyzed by using computerized program statistical package for social science (**SPSS version 22**), and presented in form figures and tables and p-value was used to test the association between the years of experience and knowledge of study group regard sign and symptom about neonatal sepsis which be significant (p-value 0.05).

the result showed most of the study group had good knowledge about definition of neonatal sepsis, definition of late neonatal sepsis, sign and symptom and predisposing factor and had fair knowledge about early neonatal sepsis.

ملخص الأطروحة

الخلفية:التسمم الوليدي عدوي منتشرة عادتاً بكثيرة، يحدث بنسب عالية فى المواليد ذوي الأوزان الناقصة والمواليد ذوي معدل abgar score المنخفض وعوامل متعلقه بحاله الأم مثل الأسر ذات الدخل المنخفض ثقب الغشاء الأميوني قبل الولادة بفترة طويله.

اجريت هذه الدراسة الوصفية الوصفية فى مستشفى سوبا التعليمي الجامعي فى وحدة العناية الوليدية المركزة فى الفتره من نوفمبر 2017 إلى إبريل 2018 لتقييم معرفة وممارسة الممرضات عن التسمم الوليدي.

الدراسة تضمنت 32 ممرضة (3نسبة خطأ)، كل ممرضه تعمل بوحدة العناية الوليدية المركزة أثناء فترة الدراسة بالإضافة إلى الممرضات اللاتي عملن بها فى وقت سابق.

جمعت البيانات عن طريق الإستبيان الذى صمم من قبل الباحث، يحتوى الإستبيان على 15 سؤال؛ الجزئية الأولى عن بيانات عينة الدراسة الإجتماعية، الجزئية الثانية عن معرفة الممرضات حول التسمم الوليدي .

تم تحليل البيانات عن طريق برنامج الكمبيوتر الإحصائي لعلم الإجتماع (الطبعة الثانية والعشرون) وتم عرض النتائج على شكل جداول ورسومات هندسية، ايضاً تم استخدام p.value لفحص إختبار العلاقة بين سنين الخبرة بوحدة العناية الوليدية ومعرفة الممرضات للأعراض والعلامات عن التسمم الوليدي التي كانت كبيره.

قد وضحت الدراسة أن أغلبية الممرضات لديهن معرفة جيدة حول تعريف التسمم الوليدي، تعريف التسمم الوليدي المتأخر والأعراض والعلامات ولديهن معرفة متوسة حول التسمم الوليدي المبكر.

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List of abbreviations

Abbreviation	Words
EOS	Early onset sepsis
LOS	Late onset sepsis
VLBW	Very low birth weight
NRN	Neonatal research network
CNS	Central nerves system
NICU	Neonatal intensive care unit
GBS	Gram positive streptococcus
CONS	Coagulase negative staphylococcus
DIC	Disseminated intravascular coagulation
LP	Lumbar puncture
NEC	Necrotizing enterocolitis
CBC	Complete blood count
INR	International normalize ratio
PT	Prothrombin time
PTT	Partial thromboplastine time
CSF	Cerebro spinal fluid
GI	Gastro intestinal
RSV	Respiratory syndrome virus
TPN	Total paranal nutrition
CRP	C reactive protein
PTL	Prolonged threatened labor

Chapter one

1.1.Introduction

Bacterial sepsis and meningitis continue to be major causes of morbidity and mortality in newborns, particularly in premature infant .although improvements in neonatal intensive care have decreased the impact of early-onsetsepsis in term infants, preterm infants remain at high risk for both early onset sepsis and its sequelae. Very low birth weight infants are also at risk for late-onset(hospital-acquired) sepsis. Neonatal survivors of sepsis can have severe neurologicsequelae due to central nervous system infection, as well as from secondaryhypoxemia resulting from septic shock, persistent pulmonary hypertension, and parenchymal lung disease⁽¹⁾.

Incidence :1– 8 per 1000 live births. Higher in preterm babies.Mortality 5– 15%,Mortality for early onset > late onset.

Babies who present very soon after birth may have mortality up to 50%.⁽¹¹⁾When pathogenic bacteria gain access in the blood stream,they may cause an overwhelming infection .the systemic bacterial infections of neonates are termed as neonatal sepsis which incorporates septicemia , pneumonia and meningitis of the newborn .in most cases it is caused by klebsiella pneumonia, staphylococcus aureus, e.coli, pseudomonas aeruginosa,acinetobacter ,etc.

The predisposing factors of neonatal sepsis are intrauterine infections , premature and prolonged rupture of membrane ,meconium stained liquor , repeated vaginal examination , maternal infections , lack of aseptic practices ,birth asphyxia ,resuscitation without aseptic precautions ,low birth weight ,invasive procedures ,needle pricks ,superficial infections ,aspiration of feeds and lack of breast feeding.⁽³⁾

1.2.Justification

Many of the neonate admitted to the neonatal intensive care unit and high dependent unit develop neonatal sepsis that led to increase morbidity and mortality rate ;so the researcher have a desire to study the nurses knowledge regard neonatal sepsis.

1.3.Research objectives

1.3.1.General:-

-to assess knowledge of nurses regard neonatal sepsis.

1.3.2.Specific:-

-to determine nurses knowledge about the neonatal sepsis.

-to insure aseptic technique.

chapter tow

2.Literaturereview

2.1.Definition: -

Neonatal sepsis is a clinical syndrome of systemic illness accompanied by bacteremia occurring in the first month of life.

2.2.Incidence:

The overall incidence of primary sepsis is 1–5 per 1000 live births. The incidence is much higher for very low birth weight infants (birthweight <1500 g), with early-onset sepsis rate of 2% and late-onset nosocomial sepsis rate of 36% according to data from the National Institute of Child Health and Human Development Neonatal Research Network . The mortality rate is high (13–25%); higher rates are seen in premature infants and in those with early fulminant disease⁽²⁾

2.3. Pathophysiology:-

Neonatal sepsis can be classified into 2 relatively distinct syndromes based on the age of presentation: early-onset and late-onset sepsis.

2.4.Tybe:-

2.4.1. Early-onset sepsis (EOS):-

Presents in the first 3–5 days of life and is usually a multisystem fulminant illness with prominent respiratory symptoms. Typically the infant has acquired the organism during the antepartum or intrapartum period from the maternal genital tract. Several infectious agents, notably treponemes, viruses, Listeria, and probably Candida, can be acquired transplacentally via hematogenous routes. Acquisition of other organisms is associated with the birth process. With rupture of membranes, vaginal flora

or various bacterial pathogens may ascend to reach the amniotic fluid and the fetus. Chorioamnionitis develops, leading to fetal colonization and infection. Aspiration of infected amniotic fluid by the fetus or neonate may play a role in the resultant respiratory symptoms. Finally, the infant may be exposed to vaginal flora as it passes through the birth canal. The primary sites of colonization tend to be the skin, nasopharynx, oropharynx, conjunctiva, and umbilical cord. Trauma to these mucosal surfaces may lead to infection.

Early-onset disease is characterized by a sudden onset and fulminant course that can progress rapidly to septic shock and death.⁽³⁾

2.4.2. late-onset sepsis (LOS):

May occur as early as 5 days of age. LOS is usually more insidious but it can be fulminant at times. It is usually not associated with early obstetric complications. In addition to bacteremia, these infants may have an identifiable focus, most often meningitis in addition to sepsis. Bacteria responsible for LOS and meningitis include those acquired after birth from the maternal genital tract (vertical transmission) as well as organisms acquired after birth from human contact or from contaminated equipment/environment (nosocomial). Therefore, horizontal transmission appears to play a significant role in late-onset disease. The reasons for the delay in development of clinical illness, the predilection for central nervous system disease, and the less severe systemic and cardiorespiratory symptoms are unclear. Transplacental transfer of maternal antibodies to the mother's own vaginal flora may play a role in determining which exposed infants become infected, especially in the case of group B streptococcal infections. In case of nosocomial spread, the pathogenesis is related to the underlying illness and debilitation of the infant, the flora in

the neonatal intensive care (NICU) environment, and invasive monitoring and other techniques used in the NICU. Breaks in the natural barrier function of the skin and intestine allow opportunistic organisms to invade and overwhelm the neonate. Infants, especially the premature, have an increased susceptibility to infection because of underlying illnesses and immature immune defenses that are less efficient at localizing and clearing bacterial invasion⁽³⁾

2.5. Microbiology:-

The principal pathogens involved in EOS have tended to change with time. Before 1965, *Staphylococcus aureus* and *Escherichia coli* used to be the most commonly isolated organisms. In the late 1960s, (GBS) emerged as the most common microorganism. Currently, most centers continue to report GBS as the most common microorganism, even though the incidence has decreased considerably after the widespread adoption of universal antenatal screening for GBS colonization at 35–37 weeks' gestation and intrapartum prophylaxis with penicillin or ampicillin for colonized women. The incidence of EOS secondary to GBS decreased from 1.7 per 1000 live births in 1993 to 0.28 per 1000 in 2008 (>80% reduction). The second most common bacteria are gram-negative enteric organisms, especially *E. coli*. An increase in the incidence of *E. coli* has been noted in EOS in VLBW infants to the extent that *E. coli* is currently the predominant microorganism in this group of patients. This increase was noted in late 1990s and early 2000s and appears to be stabilizing. Recent data from NICHD-NRN suggest that widespread use of intrapartum antibiotic prophylaxis to reduce vertical transmission of GBS has not resulted in a further increase in non-GBS EOS among the larger cohort of infants of all birthweights or among VLBW infants beyond what was noted previously. GBS and *E. coli* account for two-thirds of all cases of EOS.

Other pathogens causing EOS include *Listeria monocytogenes*, *Staphylococcus*, enterococci, anaerobes, *Haemophilus influenzae*, and *Streptococcus pneumoniae*. The pathogens that cause LOS or nosocomial sepsis tend to vary in each nursery; however CoNS, especially *Staphylococcus epidermidis*, are the most predominant⁽³⁾

Other microorganisms causing EOS include Gram-negative rods including:-

- *Pseudomonas*.
- Group B *Streptococcus* (GBS.)
- *Escherichia coli*.
- Coagulase-negative *Staphylococcus*.
- *Haemophilus influenzae*.
- *Listeria monocytogenes*.
- *Serratia*, and *Proteus*.
- *S. aureus*.
- GBS *Klebsiella* and fungal microorganisms

Trends in the epidemiology of early-onset sepsis show a decreasing incidence of GBS disease. This can be attributed to the implementation of a prenatal screening and treatment protocol for GBS. In a 2009 study involving 4696 women, prenatal cultures showed a GBS colonization rate of 24.5%, with a positive culture rate of 18.8% at the time of labor. As many as 10% of prenatally culture-negative women were found to have positive cultures at the time of labor. With intrapartum antibiotic prophylaxis rates of 93.3%, 0.36 of 1000 infants developed early-onset GBS disease^(4,5).

2.4.1.1.Risk factors for EOS:

-Maternal factors predictive of GBS disease include documented maternal GBS.intrapartum fever ($\geq 38^{\circ}\text{C}$) and other signs of chorioamnionitis.PROM more than 18 hours. Neonatal risk factors Include prematurity less than 37 weeks' gestation and LBW less than 2,500 g⁽¹⁾.

2.4.1.2.Clinical presentation of EOS:-

Early-onset disease can manifest as asymptomatic bacteremia, generalized sepsis, pneumonia, and/or meningitis. The clinical signs of EOS are usually apparent in the first hours of life; 90% of infants are symptomatic by 24 hours of age. Respiratory distress is the most common presenting symptom. Respiratory symptoms can range in severity from mild tachypnea and grunting, with or without a supplemental oxygen requirement, to respiratory failure. Persistent pulmonary hypertension of the newborn can also accompany sepsis. Other less specific signs of sepsis include irritability, lethargy, temperature instability, poor perfusion, and hypotension.(DIC) with purpura and petechiae can occur in more severe septic shock (GI) symptoms can include poor feeding, vomiting, and ileus. Meningitis may present with seizure activity, apnea, and depressed sensorium, but may complicate sepsis without specific neurologic symptoms, underscoring the importance of the (LP) in the evaluation of sepsis.

Other diagnoses to be considered in the immediate newborn period in the infant with signs of sepsis include transient tachypnea of the newborn, meconium aspiration syndrome,

intracranial hemorrhage, congenital viral disease, and congenital cyanotic heart disease. In infants presenting at more than 24 hours of age, closure of the ductus arteriosus in the setting of a ductal-dependent cardiac anomaly (such as critical coarctation of the aorta or hypoplastic left heart syndrome) can mimic sepsis. Other diagnoses that should be considered in the infant presenting beyond the first few hours of life with a sepsis-like picture include bowel obstruction, necrotizing enterocolitis (NEC), and inborn errors of metabolism.⁽¹⁾

2.4.1.3. Evaluation of the symptomatic infant for EOS:

Laboratory evaluation:

- CBC
- PT and PTT.
- RFT&LFT.
- INR.
- LP.
- chest radiograph.
- blood culture and sensitivity ⁽¹⁾

2.4.2. Late-onset sepsis (LOS):

Organisms that have been implicated in causing late-onset sepsis include the following:

- Coagulase-negative Staphylococcus.
- Staphylococcus aureus.
- E coli.

- Klebsiella.
- Pseudomonas
- Enterobacter
- Candida
- GBS
- Serratia.
- Acinetobacter.
- Anaerobes⁽⁴⁾.

Trends in late-onset sepsis show an increase in coagulase-negative streptococcal sepsis; most of these isolates are susceptible to first-generation cephalosporins. The infant's skin, respiratory tract, conjunctivae, gastrointestinal (GI) tract, and umbilicus may become colonized from the environment, and such colonization to the possibility of late-onset sepsis from invasive microorganisms. Vectors for such colonization may include vascular or urinary catheters, other indwelling lines, or contact with caregivers who have bacterial colonization..

Pneumonia is more common in early-onset sepsis, whereas meningitis and bacteremia are more common in late-onset sepsis. Premature and ill infants are more susceptible to sepsis and subtle nonspecific initial presentations; considerable vigilance is therefore required in these patients so that sepsis can be effectively identified and treated.

When neonatal sepsis is suspected, treatment should be initiated immediately because of the neonate's relative immune suppression. Begin antibiotics as soon as diagnostic tests are performed .⁽⁴⁾

2.4.2.1 risk factors for late-onset sepsis:-

GBS disease colonization of the infant from maternal and community (or less commonly, hospital) sources.

gestational age and lack of maternally derived protective antibody. The use of IAP for GBS has had no significant impact on the rate of GBS LOS, remaining at 0.33 cases per 1,000 live births from 1999 to 2005. Preterm infants account for a disproportionate number of GBS late-onset infections; from 1999 to 2005 surveillance revealed that 52% of late-onset GBS cases occurred in infants born at <37 weeks' gestation with a median gestational age of 30 weeks among the preterm cases. GBS LOS is more often complicated by meningitis than early-onset disease and is predominantly caused by polysaccharide serotype III strains. Although mortality from GBS LOS is low (1%–5% in term and preterm infants, respectively), sequelae in survivors of GBS meningitis can be severe. A recent study of GBS meningitis occurring in infants born at <36 weeks' gestation from 1998 to 2006 revealed that a quarter of all infants died or survived with significant neurologic impairment. **Gram-negative bacteremia** is often associated with UTI. Different series report 20% to 30% of UTIs in infants under 1 month of age are complicated by bacteremia. Mortality is low if promptly treated, and sequelae are few unless meningitis occurs. *L. monocytogenes* can also cause late-onset disease, with onset commonly by 30 days of life, and can account for up to 20% of LOS in some centers. Late-onset listeriosis is frequently complicated by meningitis, but unlike late-onset GBS meningitis, the morbidity and long-term sequelae are infrequent if the disease is diagnosed and treated in a timely fashion. Term infants with LOS generally present with fever and/or poor feeding and lethargy to the private pediatrician or emergency department. Evaluation in the infant younger than 3 months old

in most centers includes at minimum a CBC, urine analysis, CSF cell count, glucose and protein, and cultures of blood, urine, and CSF. Infants under 1 month are generally hospitalized for empiric IV therapy that includes coverage for GBS, *Listeria*, and gram-negative organisms (commonly ampicillin and cefotaxime); for infants over 1 month, management varies in different centers.⁽³⁾

2.4.2.2.Symptoms and evaluation of LOS:

Lethargy, an increase in the number or severity of apneic spells, feeding intolerance, temperature instability, and/or an increase in ventilatory support all may be early signs of LOS—or may be part of the variability in the course of the VLBW infant. The difficulty in distinguishing between these two in part explains the frequency of evaluation for LOS; 62% of VLBW infants had at least one blood culture drawn after the third day of life. With mild symptoms and a low suspicion for the presence of sepsis, it is reasonable to draw a CBC with differential and a blood culture and wait for the results of the CBC (while monitoring the infant's symptoms closely) before beginning empiric antibiotic therapy. If the CBC is abnormal or the infant's status.

worsens, empiric antibiotic therapy should be started. If the suspicion for sepsis is still low, and/or the clinical impression is that a CONS infection is likely, it is not unreasonable to obtain a blood culture only. Ideally cultures of urine and CSF should also be obtained before antibiotic therapy, both to guide empiric therapy and to ensure proper follow-up (such as renal imaging if a UTI is present). A study of late-onset infection in VLBW infants underscores the importance of performing a LP in the evaluation of LOS in this population. Two-thirds of a cohort of over 9,000 infants had one or more blood cultures drawn after 72 hours of life; one-third had a LP. Culture-proven meningitis was diagnosed in 134

infants (5% of those on whom a LP was performed) and in 45 out of 134 cases, the coincident blood culture was negative. If a previously well, convalescing premature infant presents primarily with increased apnea with or without URI symptoms, consideration should be given to a viral source of infection as well. Tracheal or nasal aspirate should be sent for rapid analysis and culture to rule out respiratory syncytial virus (RSV), parainfluenzae, and influenza A and B if seasonally appropriate⁽³⁾.

2.6.BARRIERS OF INFECTION:

The physical and chemical barriers to infection in the human body are present in the newborn but are functionally deficient. Skin and mucous membranes are broken down easily in the premature infant. Neonates who are ill, premature, or both are at additional risk because of the invasive procedures that breach their physical barriers to infection. Because of the interdependence of the immune response, these individual deficiencies of the various components of immune activity in the neonate conspire to create a hazardous situation for the neonate exposed to infectious threats.

2.7.Organs response to sepsis:

2.7.1.Cardiopulmonary response to sepsis:

In overwhelming sepsis, there may be an initial early phase characterized by pulmonary hypertension, decreased cardiac output, and hypoxemia. These cardiopulmonary disturbances may be due to the activity of granulocyte-derived biochemical mediators, such as hydroxyl radicals and thromboxane B₂ (an arachidonic acid metabolite). These biochemical agents have vasoconstrictive actions that result in pulmonary hypertension when they are released in pulmonary tissue. A toxin derived from the polysaccharide capsule of type III *Streptococcus* has also been shown to cause pulmonary hypertension⁽¹⁾.

2.7.2. Gastrointestinal involvement in sepsis:

The intestines can be colonized by organisms in utero or at delivery through swallowing of infected amniotic fluid. The immunologic defenses of the GI tract are not mature, especially in the preterm infant. Lymphocytes proliferate in the intestines in response to mitogen stimulation; however, this proliferation is not fully effective in responding to a microorganism, because antibody response and cytokine formation are immature until approximately 46 weeks..

Necrotizing enterocolitis has been associated with the presence of a number of species of bacteria in the immature intestine. Overgrowth of these organisms in the neonatal lumen is a component of the multifactorial pathophysiology of necrotizing enterocolitis⁽¹⁾.

2.7.3. Ventriculitis:

Ventriculitis is the initiating event in meningitis, with inflammation of the ventricular surface. Exudative material usually appears at the choroid plexus and is external to the plexus. Ependymitis then occurs, with disruption of the ventricular lining and projections of glial tufts into the ventricular lumen. Glial bridges may develop by these tufts and cause obstruction, particularly at the aqueduct of Sylvius. The lateral ventricles may become multiloculated, a process that is similar to formation of abscesses. Multiloculated ventricles can isolate organisms in an area, making treatment more difficult. Meningitis is likely to arise at the choroid plexus and extend via the ventricles through aqueducts and into the subarachnoid space to affect the cerebral and cerebellar surfaces. The high glycogen content in the neonatal choroid plexus provides an excellent medium for the bacteria. When meningitis develops from ventriculitis, effective treatment is complicated because adequate antibiotic levels in the

cerebral ventricles are difficult to achieve. When ventricular obstruction is present, it causes additional problems⁽¹⁾..

2.7.4.Arachnoiditis:

Arachnoiditis is the next phase of the process and is the hallmark of meningitis. The arachnoid is infiltrated by inflammatory cells producing an exudate that is thick over the base of the brain and more uniform over the rest of the brain. Early in the infection, the exudate primarily contains PMNs, bacteria, and macrophages. It is prominent around the blood vessels and extends into the brain parenchyma. In the second and third weeks of infection, the proportion of PMNs decreases; the dominant cells are histiocytes, macrophages, and some lymphocytes and plasma cells. Exudate infiltration of cranial roots 3-8 occurs. After this period, the exudate decreases. Thick strands of collagen form, and arachnoid fibrosis occurs, which is responsible for obstruction. Hydrocephalus results. Early-onset GBS meningitis is characterized by much less arachnoiditis than late-onset⁽¹⁾.

2.7.5.Vasculitis:

Vasculitis extends the inflammation of the arachnoid and ventricles to the blood vessels surrounding the brain. Occlusion of the arteries rarely occurs; however, venous involvement is more severe. Phlebitis may be accompanied by thrombosis and complete occlusion. Multiple fibrin thrombi are especially associated with hemorrhagic infarction. This vascular involvement is apparent within the first days of meningitis and becomes more prominent during the second and third weeks⁽¹⁾.

2.6.6.Cerebral edema:

Cerebral edema may occur during the acute state of meningitis and may be severe enough to diminish the ventricular lumen substantially. The cause is unknown but is likely to be related to vasculitis and the increased permeability of blood vessels. It may also be related to cytotoxins of microbial origin. Herniation of edematous supratentorial structures does not generally occur in neonates, because of the cranium's distensibility..

2.6.7.Infarction:-

Infarction is a prominent and serious feature of neonatal meningitis, occurring in 30% of infants who die. Lesions occur because of multiple venous occlusions, which are frequently hemorrhagic. The loci of infarcts are most often in the cerebral cortex and underlying white matter but may also be subependymal within the deep white matter. Neuronal loss occurs, especially in the cerebral cortex, and periventricular leukomalacia may subsequently appear in areas of neuronal cell death .⁽¹⁾

2.7. VIRAL INFECTIONS:

Viruses are very small particles that infect cells. They cannot multiply on their own and require a living host, such as humans, animals, or plants. They can reproduce only by invading and taking over the host cells. Young children and exposure is high. Viruses are hard to destroy without damaging or killing the living cells they infect. Therefore, drugs are not used to control them. However, many viral diseases can be prevented by immunization, such as measles, rubella, varicella, mumps, and poliomyelitis⁽²⁾.

2.8.FUNGAL INFECTIONS:

2.8.1.type:

2.8.1.1.Mucocutaneous candidiasis:

Fungal infections in the well term infant are generally limited to mucocutaneous disease involving *C. albicans*. *Candida* species are normal commensal flora beyond the neonatal period and rarely cause serious disease in the immunocompetent host. Immaturity of host defenses and colonization with *Candida* before complete establishment of normal intestinal flora probably contribute to the pathogenicity of *Candida* in the neonate. Oral and GI colonization with *Candida* occurs before the development of oral candidiasis (thrush) or diaper dermatitis. *Candida* can be acquired through the birth canal, or through the hands or breast of the mother. Nosocomial transmission in the nursery setting has been documented, as has transmission from feeding bottles and pacifiers⁽³⁾.

2.8.1.2.Oral candidiasis:

in the young infant is treated with a nonabsorbable oral antifungal medication, which has the advantages of little systemic toxicity and concomitant treatment of the intestinal tract. Nystatin oral suspension (100,000 U/mL) is standard treatment (1 mL is applied to each side of the mouth every 6 hours, for a minimum of 10 to 14 days). Ideally, treatment is continued for several days after lesions resolve. Gentian violet (1%, applied once or twice) is an effective treatment for thrush, but it does not eliminate intestinal fungal colonization. This topical dye has fallen out of favor in the United States: it stains skin and clothing, can irritate the mucosa with prolonged use, and has been shown to be mutagenic in vitro. Miconazole oral gel (20 mg/g) is also effective, but is only approved for use in the United States in patients 16 years of age and older. Systemic fluconazole is highly effective in treating chronic

mucocutaneous candidiasis in the immunocompromised host. A 2002 pilot study demonstrated the superiority of oral fluconazole over nystatin suspension in curing thrush in otherwise healthy infants, but fluconazole is not currently approved for this use. Infants with chronic, severe thrush refractory to treatment should be evaluated for an underlying congenital or acquired immunodeficiency. Oral candidiasis in the breastfed infant is often associated with superficial or ductal candidiasis in the mother's breast. Concurrent treatment of both mother and infant is necessary to eliminate continual cross-infection. Breastfeeding of term infants can continue during treatment. Mothers with breast ductal candidiasis who are providing expressed breast milk for VLBW infants should be advised to withhold expressed milk until treatment has been instituted. Candida can be difficult to detect in breast milk as lactoferrin inhibits the growth of Candida in culture. Freezing does not eliminate Candida from expressed breast milk.⁽³⁾

2.8.1.3. Candidal diaper dermatitis:

is effectively treated with topical agents such as 2% nystatin ointment, 2% miconazole ointment, or 1% clotrimazole cream. Concomitant treatment with oral nystatin to eliminate intestinal colonization is often recommended, but not well studied. It is reasonable to use simultaneous oral and topical therapy for refractory candidal diaper dermatitis.

2.8.1.4. Systemic candidiasis:

Systemic candidiasis is a serious form of nosocomial infection in VLBW infants. Recent data on late-onset candidal sepsis from the NICHD NRN showed that 9% of a cohort of 1,515 infants with birth weights of less than 1,000 g developed candidal sepsis or meningitis, primarily caused by *C. albicans* and *C. parapsilosis*. One-third of these infants died. Invasive candidiasis is associated with overall poor neurodevelopmental outcomes and higher rates of threshold retinopathy of

prematurity, compared to matched VLBW control infants. GI tract colonization of the LBW infants often precedes invasive infection, and risk factors for colonization and invasive disease are similar. The most significant epidemiologic factors specific to candidal LOS in the NICHD cohort studies were birth weights of $\leq 1,000$ g, presence of a central catheter, delay in enteral feeding, and days of broad-spectrum antibiotic exposure. Other clinical factors included in a recent clinical predictive model for invasive candidiasis in the population with birth weights of $\leq 1,000$ g include the presence of candidal diaper dermatitis, vaginal delivery, lower gestational age, and significant hypoglycemia and thrombocytopenia. The use of H₂ blockers or systemic steroids has also been identified as independent risk factors for the development of invasive fungal infection⁽³⁾.

2.8.4. Diagnosis of fungal infection:

Candida can be cultured from standard pediatric blood culture systems; the time to identification of a positive culture is usually by 48 hours, although late identification (beyond 72 hours) does occur more frequently than with bacterial species. Specialized fungal isolator tubes can aid in the identification of fungal infection if it is suspected by allowing for direct culture on selective media. Both fungal culture and fungal staining (KOH preparation) of urine obtained by suprapubic aspiration can be helpful in making the diagnosis of systemic candidiasis. Specimens obtained by bag urine collection or bladder catheterization are difficult to interpret as they can be readily contaminated with colonizing species. We have obtained urine by SPA from VLBW infants under bedside ultrasound guidance for maximal safety. Before the initiation of antifungal therapy, CSF should be obtained for cell count and fungal culture.⁽³⁾

2.9.treatment of neonatal sepsis :

Isolation precautions for all infectious diseases, including maternal and neonatal precautions, breast-feeding.

A. Prevention:

1. GBS prophylaxis:

Because of the widespread use of intrapartum antibiotic prophylaxis, EOS secondary to GBS has been reduced by 80%. Approximately 10–30% of pregnant women are colonized with GBS in the vaginal or rectal area. Consensus guidelines regarding management of GBS were published by the Centers for Disease Control and Prevention in 1996 and were later revised in 2002 and in 2010. These guidelines are supported by the AAP and the American College of Obstetricians and Gynecologists. The guidelines recommended that all pregnant women should be screened at 35–37 weeks' gestation for vaginal and rectal GBS colonization. At the time of labor or rupture of membranes, intrapartum chemoprophylaxis should be given to all pregnant women identified as GBS carriers. Women with GBS isolated from the urine ($>10,000$ colony-forming U/mL) during their current pregnancy should receive intrapartum chemoprophylaxis because such women usually are heavily colonized with GBS and are at increased risk of delivering an infant with EOS. Women who have previously given birth to an infant with invasive GBS disease should receive intrapartum chemoprophylaxis as well. Penicillin is the drug of choice, but ampicillin is an acceptable alternative. Cefazolin, and less commonly vancomycin, may be used for penicillin-allergic women. The risk-based approach is no longer acceptable except for circumstances in which screening results are not available before labor and delivery. In these circumstances, intrapartum antibiotic prophylaxis should be given to women <37 weeks' gestation, those with PROM ≥ 18 hours, and women

who have a fever $\geq 38^{\circ}$ C (100.4° F). The new guidelines recognize the availability of commercial nucleic acid amplification tests (NAAT) such as polymerase chain reaction for rapid detection of GBS.

Antibiotic prophylaxis should be given if the NAAT testing returns positive or an intrapartum risk factor develops regardless of NAAT results. In addition, the guidelines specifically addressed threatened preterm labor and preterm premature rupture of membranes with detailed algorithms. Briefly, women with threatened PTL or PROM should be screened for GBS colonization on admission unless a GBS culture was obtained within the preceding 5 weeks. In both of these situations, women should receive GBS prophylaxis (typically for 48 hours) unless the screening results are negative. The new recommendations also provided clarification on optimal GBS culturing methods. Finally, the guidelines provided specific recommendations for management of neonates born to mothers who are GBS colonized, have risk factors for sepsis, or were exposed to chorioamnionitis⁽⁶⁾.

2. Prevention of nosocomial sepsis in premature infants in NICU:

A subset of nosocomial sepsis is central line-associated bloodstream infections). Although primary prevention of CLABSI relies on minimizing the use of central lines, novel technologies such as antiseptic and antimicrobial impregnated catheters in addition to meticulous care during PICC insertion and maintenance are key factors in preventing CLABSIs. Hand hygiene is the single most important measure. Maternal milk contains a number of substances responsible for innate immune and humoral responses against pathogens; therefore, promotion of breast-feeding is a key step in the prevention of NICU infections. Medical stewardship of antibiotics, steroids, and H2 blockers is mandatory; indiscriminate use of these agents has been associated with increased nosocomial sepsis. Enhancement of the enteric microbiome composition with the possible use of probiotics may restore gut

immune function and help prevent necrotizing enterocolitis and sepsis. Use of bioactive substances with known anti-infective properties such as lactoferrin may be helpful. A recently published multicenter study conducted in Italy showed that oral bovine lactoferrin was beneficial in preventing LOS in VLBW infants during their stay in NICU, regardless of the type of nutrition. Finally, specific and targeted pharmacologic prophylactic interventions have been used with some success. For example, specific antifungal prophylaxis with fluconazole has been associated with 85% reduction in invasive fungal infection. However, the use of pagibaximab, a recombinant monoclonal antibody targeting staphylococcal species, does not appear to offer protection against gram-positive CLABSIs in NICU ⁽⁶⁾.

B. Empiric antibiotic therapy:

Treatment is most often begun before a definite causative agent is identified. For EOS, it usually consists of ampicillin and gentamicin. This empirical regimen covers the most commonly encountered microorganism; namely GBS and *E. coli*, and has proved to be efficacious over the years. In nosocomial sepsis, the flora of the NICU must be considered; however, staphylococcal coverage with vancomycin plus an aminoglycoside such as gentamicin or amikacin is usually begun. Third-generation cephalosporins should be avoided as an empirical therapy for EOS or nosocomial sepsis because they are associated with increased risk for antibiotic resistance and invasive fungal infections. The empirical treatment for suspected LOS in a neonate admitted from the community is ampicillin and gentamicin; cefotaxime can be added only when there is a concern for meningitis.

C. Continuing therapy:

Based on culture and sensitivity results, clinical course, and other serial laboratory studies (eg, CRP). Monitoring for antibiotic toxicity is important, as well as monitoring levels of aminoglycosides and vancomycin. When GBS is documented as the causative agent, penicillin G is the drug of choice; however, an aminoglycoside is often added because of documented synergism in vitro ⁽⁶⁾.

D. Complications and supportive therapy :

1. Respiratory. Ensure adequate oxygenation with blood gas monitoring, and initiate oxygen therapy or ventilator support if needed.
2. Cardiovascular. Support blood pressure and perfusion to prevent shock. Use volume expanders such as normal saline, and monitor the intake and output of fluids. Inotropes such as dopamine or dobutamine may be needed.
3. Hematologic:
 - a. Disseminated intravascular coagulation With DIC, one may observe generalized bleeding at puncture sites, the gastrointestinal tract, or CNS. In the skin, large-vessel thrombosis may cause gangrene. Laboratory parameters consistent with DIC include thrombocytopenia, increased prothrombin time, and increased partial thromboplastin time. There is an increase in fibrin split products or d-dimers. Treatment options include fresh-frozen plasma, 10 mL/kg; vitamin K ; platelet infusion; and possible exchange transfusion .
 - b. Neutropenia. Multiple factors contribute to the increased susceptibility of neonates to infection, including developmental quantitative and qualitative neutrophil defects. Colony-stimulating factors (CSFs) comprise a group of cytokines that are central to the hematopoiesis of blood cells, as well as to the maintenance of homeostasis and overall

immune competences Granulocyte CSF and granulocyte-macrophage-CSF.

have been used in neonates with established sepsis associated with neutropenia, in neutropenic infants without sepsis, and prophylactically in neonates at risk for sepsis. Limited data suggest that CSFs administration may reduce mortality when systemic infection is accompanied by severe neutropenia. A recent randomized control trial that enrolled 280 small for gestational age extremely preterm infants and used early GM-CSF prophylactically showed no reduction in sepsis or improvement in survival in the treated group. Intravenous immunoglobulin does not appear useful either as a prophylactic or as an adjunct to antibiotic therapy in serious neonatal infection.

4. Central nervous system. Implement seizure control measures using phenobarbital, and monitor for the syndrome of inappropriate antidiuretic hormone (decreased urine output, hyponatremia, decreased serum osmolarity, and increased urine specific gravity and osmolarity).
5. Metabolic. Monitor for and treat hypoglycemia or hyperglycemia.

Metabolic

acidosis may accompany sepsis and is treated with bicarbonate and fluid replacement⁽⁶⁾.

E. Future developments:

Intensive research continues in the development of vaccines (especially for GBS) as well as synthetic monoclonal antibodies to the specific pathogens causing neonatal sepsis (ie, antistaphylococcal antibodies). Research is also ongoing into blocking some of the body's own inflammatory mediators that result in significant tissue injury, including endotoxin inhibitors, cytokine inhibitors, nitric oxide synthetase inhibitors, and neutrophil adhesion inhibitors. Finally, recent trials are showing probiotics and

lactoferrin to be promising agents in the prevention of LOS and necrotizing enterocolitis.⁽⁶⁾

2.10.Prognosis:-

With early diagnosis and treatment, term infants are not likely to experience long-term health problems associated with neonatal sepsis; however, if early signs or risk factors are missed, mortality increases. Residual neurologic damage occurs in 15-30% of neonates with septic meningitis. Mortality from neonatal sepsis may be as high as 50% for infants who are not treated. Infection is a major cause of fatality during the first month of life, contributing to 13-15% of all neonatal deaths. Low birth weight and gram-negative infection are associated with adverse outcomes.⁽⁷⁾

Neonatal meningitis occurs in 2-4 cases per 10,000 live births and contributes significantly to mortality from neonatal sepsis; it is responsible for 4% of all neonatal deaths. In preterm infants who have had sepsis, impaired neurodevelopment is a concern.⁽⁸⁾

Proinflammatory molecules may negatively affect brain development in this patient population. In a large study of about 6000 premature infants who weighed less than 1000 g at birth, preterm infants with sepsis who did not have meningitis had higher rates of cognitive deficits, cerebral palsy, and other neurodevelopmental disabilities than infants⁽⁹⁾.

who did not have sepsis. Infants with meningitis may acquire hydrocephalus or periventricular leukomalacia. They may also have complications associated with the use of aminoglycosides, such as hearing loss or nephrotoxicity.⁽¹⁰⁾

2.11.Nursing Assessment:

Diagnosis of neonatal infections is challenging. Most infants will have some risk factors and the presenting symptoms are many and nonspecific, including poor feeding, breathing difficulty, apnea and bradycardia, gastrointestinal problems, increased oxygen requirement or ventilator support needs, lethargy or hypotension, decreased or elevated temperature, unusual skin rash or color change, persistent crying, or irritability. Adding to the challenge of correctly identifying the infection, the list of conditions to consider in the differential diagnosis is extensive, including metabolic and congenital abnormalities.

Nursing assessment focuses on early identification of a newborn at risk for infection to allow for prompt treatment, thus reducing mortality and morbidity. Be aware of the myriad risk factors associated with newborn sepsis ⁽⁴⁾.

among the factors that contribute to the newborn's overall vulnerability to infection are poor skin integrity, invasive procedures, exposure to numerous caregivers, and an environment conducive to bacterial colonization. Few newborn infections are easy to recognize because manifestations usually are nonspecific. Early symptoms can be vague because of the newborn's inability to mount an inflammatory response. Often, the observation is that the newborn does not "look right." Assess the newborn for common nonspecific signs of infection, including:

- Hypothermia.
- Pallor or duskiness.
- Hypotonia.
- Cyanosis.
- Poor weight gain.
- Irritability.

- Seizures.
- Jaundice.
- Grunting.
- Nasal flaring.
- Apnea and bradycardia.
- Lethargy.
- Hypoglycemia.
- Poor feeding (lack of interest in feeding).
- Abdominal distention⁽⁴⁾.

Since infection can be confused with other newborn conditions, laboratory and radiographic tests are needed to confirm the presence of infection. Be prepared to coordinate the timing of the various tests and assist as necessary. Evaluate the complete blood count with a differential to identify anemia, leukocytosis, or leukopenia. Elevated C-reactive protein levels may indicate inflammation. As ordered, obtain x-rays of the chest and abdomen, which may reveal infectious processes located there. Blood, cerebrospinal fluid, and urine cultures are indicated to identify the location and type of infection present. Positive cultures confirm that the newborn has an infection⁽⁴⁾.

2.12.Nursing Management:

To enhance the newborn's chance of survival, early recognition and diagnosis are key. Often the diagnosis of sepsis is based on a suspicious clinical picture. Antibiotic therapy is usually started before the laboratory results identify the infecting pathogen. Along with antibiotic therapy, circulatory, respiratory, nutritional, and developmental support is important. Antibiotic therapy is continued for 7 to 21 days if cultures are positive, or it is discontinued within 72 hours if cultures are negative. With the use of antibiotics along with early recognition and supportive care, mortality and morbidity rates have been reduced greatly. Nurses possess the

education and assessment tools to decrease the incidence of and reduce the impact of infections on women and their newborns. Implement measures for prevention and early recognition, including:

- Formulating a sepsis prevention plan that includes education of all members of the health care team on identification and treatment of sepsis.
- Screening all newborns daily for signs of sepsis.
- Monitoring sepsis cases and outcomes to reinforce continued quality-improvement measures or to modify current practices.
- Outlining and carrying out measures to prevent nosocomial infections, such as:
 - Thorough handwashing hygiene for all staff.
 - Frequent oral care and inspections of mucous membranes.
 - Proper positioning and turning to prevent skin breakdown.
 - use of a septic technique for all process.
 - Frequent monitoring of invasive catheter sites for signs of infection.
- Identifying newborns at risk for sepsis by reviewing risk factors
- Monitoring vital sign changes and observing for subtle signs of infection
- Monitoring for signs of organ system dysfunction:
 - Cardiovascular compromise—tachycardia and hypotension
 - Respiratory compromise—respiratory distress and tachypnea
 - Renal compromise—oliguria or anuria
 - Systemic compromise—abnormal blood values
- Providing comprehensive sepsis treatment:
 - Circulatory support with fluids and vasopressors
 - Supplemental oxygen and mechanical ventilation
 - Obtaining culture samples as requested
 - Antibiotic administration as ordered, observing for side effects
 - Promoting newborn comfort

Assessing the family's educational needs and providing instructions as necessary. Perinatal infections continue to be a public health problem, with severe consequences for those affected. By promoting a better understanding of newborn infections and appropriate use of therapies, nurses can lower the mortality rates associated with severe sepsis, especially with appropriate timing of interventions. The potential for nursing interventions to identify, prevent, and minimize the risk for sepsis is significant. Primary disease prevention must be a major focus for nurses. Family education plays a key role in the prevention of perinatal infections, in addition to following accepted practices in immunization ⁽⁴⁾.

2.2.Previous study

2.2.1.previous study no (1):

The study reveals that more than half of the sample (62.7%) has accepted knowledge concerning neonatal sepsis; while more than one third (37.3%) has unaccepted knowledge.

These results supported by a similar study in Baghdad for nurses' knowledge about universal precautions in NICU which found that most of them (71.4%) has accepted knowledge were used universal precaution to prevent infection in NICU ⁽¹⁷⁾. Another study in Baghdad for nurses' knowledge about nosocomial infection in NICU showed that more than half of the have accepted knowledge ⁽¹⁸⁾. The results of this study contrast with study done by İlhan, and Toruner (2015) ⁽¹⁹⁾.

-The experience years in NICU shows a significant association with the knowledge about sepsis. Most of the nurses (71.9%) have less than six years of expert in NICU, which can increase their knowledge in dealing with sepsis, this result agree with studies about nurses and their experience in specialties' area found that there were a strong relation ^(18, 20, 21).

2.4.2.previous study (2):

the present study found in adequate nurses knowledge and practice related to nosocomial infection, principle of disinfectant, sterilization. This result is congruent with SABAH et al⁽²²⁾ which found The present study showed that few of nurses have competent level of performance about infection control.

2.4.3.previous study (3):

The majority of the studied sample were in the age group from 20-30 years old. Most of them have Diploma of Secondary Nursing School, current job experience from 5-10 years. Additionally, about 10% of the studied sample had previous attendance training courses about infection

control of invasive procedure. This indicates that a considerable proportion of the nurses in the present study had no long experience, especially in current job. Added to this is their qualification, which was mostly at the diploma level. These factors might have their repercussion on the levels of their knowledge and practice⁽¹⁶⁾.

2.4.4. previous study (4):

Concerning assessment of nurses' practice regarding infection controls, the current study demonstrates that, the majority of the studied sample had good practice level of infection control. Practice was found to be higher than what was reported in (Agaral and Thomas, 2003; Talaat and Shamia, 2010)⁽²²⁾.

Chapter three

3.Methodology

3.1.Study design:

Descriptive cross sectional study aimed to assess knowledge of nurses regard neonatal sepsis in period extended from nov 2017 to april 2018.

3.2.study aria:

the study was done on soba university teaching hospital which initiate on 1975;located in Khartoum state near to mystoma center northern madni street, it is provide many services for population such as medical, surgical, pediatric and obstetrical services and anther services.

3.3.Study setting:

The study was done on a septic neonatal intensive care unit which contain two section A&B ;the section A for preterm infant (containing to 4 radiant wormer and 4 incubator and capsule phototherapy machine) while section B for term and post term babes and surgical cases (which containing of 6 radiant wormer and 20 beds and 8 suberlet phototherapy machines),the numbers of all staff that werk on the unit is 20 nurse but the rotation system make a lot of nurses have an experiences in neonatal nursing.

3.4.Study population:

All nurses werk on neonatal intensive care unit at the time of study and some nurses werk on the unit at previous time.

3.5.Sampling technique:

Simple random sampling was used.

3.6.Sample size:

32 nurses.

3.7.Study variable:

-knowledge.

3.8.Data collection tools:

The data was collected by questionnaire which designed by the researcher depend on information on literature review.

The questionnaire content of two parts; part one collected information about section demographic data (4 questions),while part tow collected information about knowledge of nurses regard neonatal sepsis(6 questions).

3.9.Data collection technique:

The questionnaire felled by the nurses under study and every one toked time range in 5-10 minutes.

3 questionnaires pilot.-

3 questionnaire missed.

3.10.Data analysis tools:

The researcher interred the information in using SPSS (specific package for social science) version 22.

The data was organized and presented in forms of table and figures .

3.11.Ethicalconsedrations:

Approval letter which is taken from the university of shandi to the soba university teaching hospital manager and consent were taken to the nurses to inform them about the aim of the study.

Chapter four

4.result

Questionnaire analysis

4.1.Figures

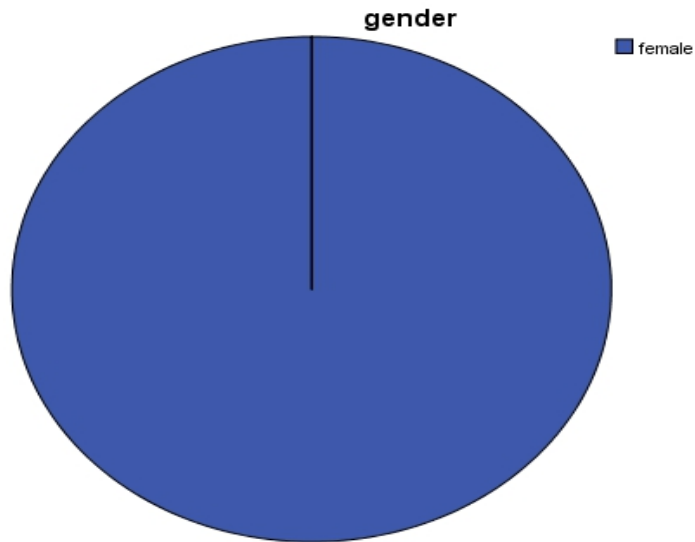


Figure no (1) distribution of gender among nurses in NICU unit in soba hospital.

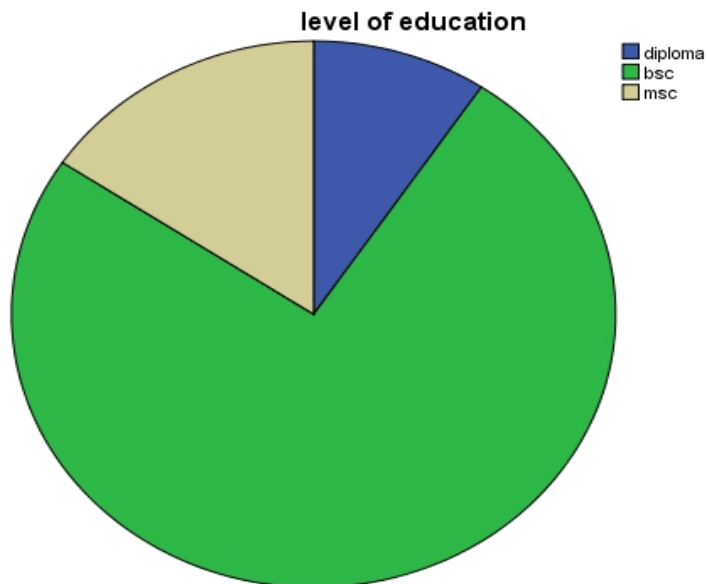


Figure no (2) level of education of nurses in NICU unit in soba hospital.

4.2.tables

Table no (1):distribution of study group in relation to their socio-demographic data:-

(n=32)

Variable	Frequency	Percentage
Age		
20-25)years(18	56.3%
(26-30)years	1	3.1%
(31-35)years	8	25.0%
Above 35years	5	15.6%
Gender		
Female	32	100.0%
Male	0	0.0%
Level of education		
Diploma	3	9.4%
Bsc	24	75.0%
Msc	5	15.6%
Phd	0	0.0%

the above table showed that the majority of the study group his age ranging between (20-25)years old and no male gender in the nursing staff and majority of them is BSC education level.

Table no (2) :

-distribution of the study group in relation to their year of experiences:

(n=32)

variable	Frequency	Percentage
Less than one year	2	6.3%
1-2years	11	34.4%
More than 2-4years	10	31.3%
More than 4 years	9	28.1%

-above table showed just 6.3% their experiences less than one year.

Table no(3) :-

-distribution of the study group in relation to their knowledge about the definition of neonatal sepsis.

(n=32)

Variable	Frequency	Percentage
Bacterial infection	9	28.1%
Fungal infection	1	3.1%
Viral infection	3	9.4%
All of them	19	59.4%

-the above table showed more than half of study group were good knowledgabout the definition of neonatal sepsis.

Table no (4) :-

-distribution of study group in relation to their knowledge about early neonatal sepsis:

(n=32)

Variable	Frequency	Percentage
3-5 days	23	71.9%
Usually acquired from mother	4	12.5%
A&b	1	3.1%
Not of the above	4	12.5%

-above table showed majority of the study group were fair knowledge about early neonatal sepsis.

Table no (5):

-Distribution of group study in relation to their knowledge about late neonatal sepsis:-

(n=32)

Variable	Frequency	Percentage
More than 5 days	5	15.6%
Usually nosocomial	1	3.1%
Neonatal with central line risk for	2	6.3%
All of them	24	75.0%

-above table showed more than tow third of the study group (75%) were good knowledge about late neonatal sepsis.

Table no (6):-

-Distribution of study group in relation to their knowledge regard the predisposing factor:-

(n=32)

Variable	Frequency	Percentage
Lack of a septic practice	3	9.4%
Invasive procedure	1	3.1%
Aspirations	2	6.3%
All of them	26	81.3%

-the above table showed most of the study group(81.3%) were good knowledge about predisposing factor of neonatal sepsis.

Table no (7):-

-distribution of study group in relation to their knowledge about commonest micro organism that cause neonatal sepsis:

(n=32)

Variable	Frequency	Percentage
Kelebsilla	8	25.0%
GBS	5	15.6%
Plasmodium	2	6.3%
A & b	17	53.1%

-the above table showed more than half (53.1%) study group were good knowledge about common microorganism cause neonatal sepsis.

Table no (8) :

-distribution of study group in relation to their knowledge regard sign and symptom of neonatal sepsis:

(n=32)

Variable	Frequency	Percentage
Fever	4	12.5%
Refuse feeding	4	12.5%
Vomiting	2	6.3%
All of them	22	68.8%

-the above table showed more than two third of study group (68.8%) were good knowledge regard sign and symptom of neonatal sepsis.

Chapter five

5.1. Discussion

nurses in neonatal intensive care unit are the key elements in sepsis care; because the nurses have vital role in neonatology and take the big responsibility in observing the first signs and symptom of the infection which make her important person for early diagnosis and intervention, through advocating early diagnosis ,complication and empirical antibiotic therapy⁽¹²⁾

the current study was conducted in soba teaching hospital in neonatal intensive care unit to assess knowledge and performance of nurses regard neonatal sepsis in period extend from dec 2017 to march 2018 and the result reflected that :

More than half of study group (59.4%) were a good knowledge about definition of neonatal sepsis and (40.6%) were a boor .this agree (approximation) with a similar study (13) which revel more than half of the study sample (62.7%)were accepted knowledge concerning neonatal sepsis.

regarding knowledge about definition of early neonatal sepsis (3.1%) were a good, (84.4%) were a fair and (12.5%) were a boor of the study group .this agree with (3)which states early onset neonatal sepsis may occur as early as days of age.EOS usually multisystem fulminate illness with prominent respiratory symptom Typically the infant has acquired the organism during the ante partum or intrapartum period from the maternal genital tract. Several infectious agents, notably treponemesviruses, Listeria, and probably Candida, can be acquired transplacentally via hematogenous routes. Acquisition of other organisms is associated with the birth process. With rupture of membranes, vaginal flora or various bacterial pathogens may ascend to reach the amniotic fluid and the fetus. Chorioamnionitis develops, leading to fetal colonization and infection. Aspiration of infected amniotic fluid by the fetus or neonate may

play a role in the resultant respiratory symptoms. Finally, the infant may be exposed to vaginal flora as it passes through the birth canal. The primary sites of colonization tend to be the skin, nasopharynx, oropharynx, conjunctiva, and umbilical cord. Trauma to these mucosal surfaces may lead to infection. Early-onset disease is characterized by a sudden onset and fulminate course that can progress rapidly to shock and death.

regarding knowledge regard late onset neonatal sepsis (usually nosocomial) more than two third of study group (75%) were a good and 25% were poor knowledge. this disagree with ABD-ALLA (14) which found in adequate nurses knowledge and practice related to nosocomial infection, principle of disinfection, sterilizations and slandered infection control precaution.

regarding knowledge about sign and symptom of neonatal sepsis showed significant association with years of experience in NICU state (p.value 0.05) , most of nurses (72%) were less than 4 years expert in NICU; which can increase knowledge and dealing with sepsis. this disagree with study (13) which state most of nurses (71.9%) were less than 6 years expert in NICU.

regarding knowledge about the risk factor, majority of study group (81.3%) were a good knowledge. this agree with (1) which state Maternal factors predictive of GBS disease include documented maternal GBS. intrapartum fever ($\geq 38^{\circ}\text{C}$) and other signs of chorioamnionitis. PROM more than 18 hours. Neonatal risk factors include prematurity less than 37 weeks' gestation and LBW less than 2,500 g.

regarding socio-demographic data the majority of the study group were in the age from 20-25 years old. most of them were BSC degree in nursing scenes , years of experience ranged between 1-2 years. this is disagree with NAGAT et al (16) which state the majority of studied sample

were in the age group from 20=30 years old, most of them were a diploma of secondary nursing scenes school.

also regarding the socio-demographic data all the nursing staff in the study aria were female gender because most of the nursing student in nursing science college were a female.

5.2. Conclusion

Neonatal sepsis still a significant burden due to its impact on neonatal mortality and long-term morbidity. Early diagnosis, treatment, and prevention remain an enigmatic area for nurses due to changes in epidemiology. Maintaining and improving neonatal care requires active involvement of everyone in health care system, nurses is essential part in health care system. All nurses work with neonatal need to improve their knowledge about caring of sepsis and how to prevent it.

the current study was conducted in soba teaching hospital in neonatal intensive care unit to assess knowledge and performance of nurses regard neonatal sepsis in period extend from nov 2017 to april 2018.

the study reflected the following:

the result showed most of the study group had good knowledge about definition of neonatal sepsis, definition of late neonatal sepsis, sign and symptom and predisposing factor and had fair knowledge about early neonatal sepsis. -p.value was used to test the association between the years of experience and knowledge of study group regard sign and symptom about neonatal sepsis which be significant (p.value 0.05).

also reflected their performance of the most of the study group had good regard hand washing and isolation precaution and infection control but poor performance regard notifying their colleagues to prevent infections.

5.3. Recommendation

the current study was conducted in soba teaching hospital in neonatal intensive care unit to assess knowledge of nurses regard neonatal sepsis in period extend from nov 2017 to April 2018. from the result and discussion the researcher was recommended the following:-

5.3.1. for the nurses:-

- improve their knowledge about the neonatal sepsis by education and training make researches and surveillance.
- importance of work shop and refreshment of knowledge from period to another. Increase knowledge about isolation technique.-
- encourages self learning.

5.3.2. For the head manger:-

- orientation program for all nurses about neonatal sepsis.
- select the nurse with high level of education to work in such area.
- encourage the expert nurse to teach new nurses and still keep on.
- Nursing etiquette lap to teach the staff how to nodding among them in way to infection control.

Chapter six

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6.2. Annex
questionnaire

Shandiuniversity

Post graduate collage

Faculty of nursing science

Pediatric nursing department

to assess nurses knowledge regard neonatal sepsis in soba
teaching hospital ,neonatal intensive care unit from nov 2017
to april 2018.

Part one socio-demo-graphic data:-

1-age:

(a)20-25years. (b)26-30y. (c)31-35y (d)above35y.

2-gender:-

(a)male. (b)female.

3-level of education:-

(a)diploma. (b) bsc (c)m.sc. (d)PhD

4-years of experiences in NICU:-

(a)less than one year.

(b)1-2years.

(c)more than 2-4 years.

(d)more than 4years.

Part two:-about knowledge of nurses regard neonatal sepsis:-

1-definition of neonatal sepsis:-

- (a)bacterial infection. (b)fungal infection (c)viral infection.
(d)all them.

2-early neonatal sepsis :-

- (a)3-5days after birth.
(b)usually acquired from the mother.
(c)a and b.
(d)not of the above.

3- late neonatal sepsis occurs within:-

- (a)more than 5 days.
(b)usually nosocomial (acquired from atmosphere around the neonate).
(c)neonate with central line risk for.
(d)all of them.

4-the predisposing factors of neonatal sepsis:-

- (a)lack of a septic practice.
(b)invasive procedure.
(c)aspirations.
(d)all of them.

5-the common organisms cause of neonatal sepsis:-

- (a)klebsilla.
(b)group b streptococcus.
(c)plasmodium.
(d)a and b.

6-sign and symptom of neonatal sepsis:-

- (a)fever. (b)refuse feeding. (c)vomiting. (d)all of them.