Red sea University
Faculty of Medicine
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The incidence, causes and outcome of Neonatal admission
From January to February
In P.P.T.H

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February 2010.
الآية الحكيمة

(وَمَا نَعَايْنَاهُ مِنْ أَيْنَاءٍ رَبِّيَّةٍ شَهِيدٍ) 

آية (68) سورة هود
Dedication
To:
all who lightened our ways of life
(mother & father)

Their help pushed us to be independent &
encouraged us.

The other members of our families.

Every one who teaches us award.

Our friends who shared with us our long way.

To you....
Acknowledgement

Praise to Allah the merciful for giving us health to achieve this goal.
To our big home Red Sea University.
Appreciated Dr. Afaf Mahmoud Abdullah for her guidance, advice and encouragement.
Special thanks to Dr. Mohamed Mohamed, the head of community department,

Red Sea University for his help.

Last but least deep gratitude and special thanks to our beloved friends and to any one who helped us in way or another as well as to any who has been, now or ever will read this research.
ABSTRACT

Prospective hospital based study was conducted in port Sudan pediatric teaching hospital, at duration between January to February 2010. The objective of this study is to know the incidence and causes and the outcome of the neonate admitted to the hospital. A base study was done using questionnaire regarding the causes of admission. The study revealed that the commonest cause of neonatal admission was prematurity (50%).
ملخص الدراسة

دراسة توقعيَّة أجريت في مستشفى بور تسودان التعليمي في الفترة ما بين يناير حتى فبراير 2005م.

الهدف من الدراسة الاطلاع على معدل وأسباب ونتائج دخول الأطفال حديثي الولادة (من عمر ساعة إلى 28 يوم) إلى المستشفى.

أجريت الدراسة باستخدام الاستبيان والذي تضمن أسئلة تتعلق بأسباب دخول المستشفى.

وقد كشفت الدراسة أن السبب الرئيسي هو الأطفال ناقصي النمو.
ABBREVIATION

P.P.T.H: port Sudan pediatric teaching hospital.
R.D.S: respiratory distress syndrome.
I.V.H: inter ventricular hemorrhage.
C.V.S: cardiovascular system.
C.N.S: central nervous system.
R.S.S: red sea state.
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chapter (1)
1-1 introduction

A study conducted by Plan Sudan 1997 revealed that RSS stand as the second poorest in North Sudan. 
enclosing all Sudanese coast 760 KM.
Total population of the state 3,746,000 (World Bank 2003).
Low population density in rural area 3.3 per square kilometer.
Under five of age around 100,000.
Infant mortality rate 112/1000 live births.
Annual growth rate in 2005 = 0.52.
Under nutrition among mothers is 22.5% (OXFAM 2002) above 16.9% consider as national norm.
Around 69.4%of the mother had no easy access to specialized neonatal health care.
Total annual births in Port Sudan maternal hospital around 3500.
Around 10% form total births need urgent medical help.
There is only one neonatal intensive care unit level one in the state.
1-2 justification:

1. Neonatal admission is important study because themortality and morbidity rate of neonate increases especially in developing countries.
2. Different cases of neonatal disease causes severe illness such as cardiac arrest, brain damage, haemorrhage, severe chest infection, skeletal deformity and all of these may fatal and cause death.
3. As consequence of these may impact occur:

   (1) socially very neonate unresponsive with surrounding even.

   (2) Physically the baby growth is mostly affected and become disturbed.
1-3 objectives:

general objective:

to study the incidence, causes and outcome of neonatal admission in port Sudan teaching hospital.

Specific objective:

To identify the predisposing factors, nursing staying pattern, care toward them, and the outcome after admission.
Literature review:-

Definition:
the neonatal period define as first 28 days of life,its highly vulnerable period during which many of physiological adjustment required for extra utine existence are complete.

Preterm birth:
In humans, preterm birth refers to the birth of a baby of less than 37 weeks gestational age. It is the major cause of neonatal mortality in developed countries. Premature infants are at greater risk for short and long term complications, including disabilities and impediments in growth and mental development. Significant progress has been made in the care of premature infants, but not in reducing the prevalence of preterm birth. The cause for preterm birth is in many situations elusive and unknown; many factors appear to be associated with the development of preterm birth, making the reduction of preterm birth a challenging proposition.

Causes

Maternal background
A number of factors have been identified that are linked to a higher risk of a preterm birth: low socio-economic or educational standing and single motherhood, as well as age at the upper and lower end of the reproductive years be it more than 35 or less than 18 years of age.

Pregnancy interval makes a difference as women with a 6 months span or less between pregnancies have a two-fold increase in preterm birth, stressful conditions, hard labor, and long hours are probably linked to preterm birth. Patients who had undergone previous induced abortions have been shown to have a higher risk of preterm birth only if the termination was performed surgically but not medically. Adequate maternal nutrition is important.

Factors during pregnancy:
Multiple pregnancies (twins, triplets, etc.) are a significant factor in preterm birth. The March of Dimes Multicenter Prematurity and Prevention Study found that 54% of twins were delivered preterm vs. 9.6% of singleton births. Triplets and more are even more endangered. The use of fertility medication that stimulates the ovary to release multiple eggs and of IVF with embryo transfer of multiple embryos has been implicated as an important factor in preterm birth. Maternal medical conditions
increase the risk of preterm birth, and often labor has to be induced for medical reasons; such conditions include high blood pressure, pre-eclampsia, maternal diabetes, asthma, thyroid disease, and heart disease.

**Common neonatal disease:**

**2-1 Neonatal jaundice:**

**Background**

Jaundice is the most common condition that requires medical attention in newborns. The yellow coloration of the skin and sclera in newborns with jaundice is the result of accumulation of unconjugated bilirubin. In most infants, unconjugated hyperbilirubinemia reflects a normal transitional phenomenon. However, in some infants, serum bilirubin levels may excessively rise, which can be cause for concern because unconjugated bilirubin is neurotoxic and can cause death in newborns and lifelong neurologic sequelae in infants who survive. For these reasons, the presence of neonatal jaundice frequently results in diagnostic evaluation.

**Pathophysiology**

Neonatal physiologic jaundice results from simultaneous occurrence of the following 2 phenomena:

- Bilirubin production is elevated because of increased breakdown of fetal erythrocytes. This is the result of the shortened lifespan of fetal erythrocytes and the higher erythrocyte mass in neonates.
- Hepatic excretory capacity is low both because of low concentrations of the binding protein ligandin in the hepatocytes and because of low activity of glucuronyl transferase, the enzyme responsible for binding bilirubin to glucuronic acid, thus making bilirubin water soluble (conjugation).

Neonatal jaundice, although a normal transitional phenomenon in most infants, can occasionally become more pronounced. Blood group incompatibilities (e.g., Rh, ABO) may increase bilirubin production through increased hemolysis. Historically, Rh isoimmunization was an important cause of severe jaundice, often resulting in the development of kernicterus. Although this condition has become relatively rare in industrialized countries following the use of Rh prophylaxis in Rh-negative women, Rh isoimmunization remains common in developing countries.
age

The risk of significant neonatal jaundice is inversely proportional to gestational age.

Sex

Risk of developing significant neonatal jaundice is higher in male infants. This does not appear to be related to bilirubin production rates, which are similar to those in female infants.

History

- Presentation and duration of neonatal jaundice
  - Typically, presentation is on the second or third day of life.
  - Jaundice that is visible during the first 24 hours of life is likely to be nonphysiologic; further evaluation is suggested.
  - Infants who present with jaundice after 3-4 days of life may also require closer scrutiny and monitoring.
  - In infants with severe jaundice or jaundice that continues beyond the first 1-2 weeks of life, the results of the newborn metabolic screen should be checked for galactosemia and congenital hypothyroidism, further family history should be explored, the infant's weight curve should be evaluated, the mother's impression as far as adequacy of breastfeeding should be elicited, and the stool color should be assessed.

- Family history
  - Previous sibling with jaundice in the neonatal period, particularly if the jaundice required treatment
  - Other family members with jaundice or known family history of Gilbert syndrome
  - Anemia, splenectomy, or bile stones in family members or known heredity for hemolytic disorders
  - Liver disease

- History of pregnancy and delivery
  - Maternal illness suggestive of viral or other infection
  - Maternal drug intake
  - Delayed cord clamping
  - Birth trauma with bruising
• **Postnatal history**
  - Loss of stool color
  - Breastfeeding
  - Greater than average weight loss
  - Symptoms or signs of hypothyroidism
  - Symptoms or signs of metabolic disease (eg, galactosemia)

**Causes**

- Physiologic jaundice is caused by a combination of increased bilirubin production secondary to accelerated destruction of erythrocytes, decreased excretory capacity secondary to low levels of ligand in hepatocytes, and low activity of the bilirubin-conjugating enzyme uridine diphosphoglucuronoyltransferase (UDPGT).
- Pathologic neonatal jaundice occurs when additional factors accompany the basic mechanisms described above. Examples include immune or nonimmune hemolytic anemia, polycythemia, and the presence of bruising or other extravasation of blood.
- Decreased clearance of bilirubin may play a role in breast feeding jaundice, breast milk jaundice, and in several metabolic and endocrine disorders.
- Risk factors include the following:
  - Race: Incidence is higher in East Asians and American Indians and is lower in African Americans.
  - Geography: Incidence is higher in populations living at high altitudes. Greeks living in Greece have a higher incidence than those living outside of Greece.
  - Genetics and familial risk: Incidence is higher in infants with siblings who had significant neonatal jaundice and particularly in infants whose older siblings were treated for neonatal jaundice. Incidence is also higher in infants with mutations/polymorphisms in the genes that code for enzymes and proteins involved in bilirubin metabolism, and in infants with homozygous or heterozygous glucose-6-phosphatase dehydrogenase (G-6-PD) deficiency and other hereditary hemolytic anemias. Combinations of such genetic variants appear to exacerbate neonatal jaundice.
  - Nutrition: Incidence is higher in infants who are breastfed or who receive inadequate nutrition. Data suggest that the difference between breastfed and formula-fed infants may be less pronounced with some modern formulas. However, formulas containing protein hydrolysates have been shown to promote bilirubin excretion.
- Maternal factors: Infants of mothers with diabetes have higher incidence. Use of some drugs may increase the incidence, whereas others decrease the incidence.
- Birthweight and gestational age: Incidence is higher in premature infants and in infants with low birthweight.
- Congenital infection.

Physical

- Neonatal jaundice first becomes visible in the face and forehead. Identification is aided by pressure on the skin, since blanching reveals the underlying color. Jaundice then gradually becomes visible on the trunk and extremities. This cephalocaudal progression is well described, even in 19th-century medical texts. Jaundice disappears in the opposite direction. This phenomenon is clinically useful because, independent of other factors, visible jaundice is an indication to check the bilirubin level, either in the serum or noninvasively via transcutaneous bilirubinometry.
- In most infants, yellow color is the only finding on physical examination. More intense jaundice may be associated with drowsiness. Brainstem auditory-evoked potentials performed at this time may reveal prolongation of latencies, decreased amplitudes, or both.
- Overt neurologic findings, such as changes in muscle tone, seizures, or altered cry characteristics, in a significantly jaundiced infant are danger signs and require immediate attention to prevent kernicterus. In the presence of such symptoms or signs, effective phototherapy should commence immediately without waiting for the laboratory test results. The potential need for exchange transfusion should not preclude the immediate initiation of phototherapy.
- Hepatosplenomegaly, petechiae, and microcephaly may be associated with hemolytic anaemia, sepsis, and congenital infections and should trigger a diagnostic evaluation directed towards these diagnoses. Neonatal jaundice may be exacerbated in these situations.
Management:

**Phototherapy** is an effective and safe method for reducing indirect bilirubin levels, phototherapy is begun when indirect bilirubin levels are between 15 and 18 mg/dL. Phototherapy is initiated in premature infants when bilirubin is at lower levels, to prevent bilirubin from reaching the high concentrations necessitating exchange transfusion. Blue lights and white lights are effective in reducing bilirubin levels.

Complications of phototherapy include an increased insensible water loss, diarrhea, and dehydration. Additional problems are macular-papular red skin rash, lethargy, masking of cyanosis, nasal obstruction by eye pads, and potential for retinal damage.

**Exchange transfusion** usually is reserved for infants with dangerously high indirect bilirubin levels who are at risk for kernicterus. As a rule of thumb, a level of 20 mg/dL for indirect-reading bilirubin is the "exchange number" for infants with hemolysis who weigh more than 2000 g. Asymptomatic infants with physiologic or breast milk jaundice may not require exchange transfusion, unless the indirect bilirubin level exceeds 25 mg/dL.

Small infusions of whole blood crossmatched with that of the mother and infant are alternated with withdrawals of an equivalent quantity of the infant's blood, which is discarded. Depending on the size of the infant, aliquots of 5 to 20 mL per cycle are withdrawn and infused, with the total procedure lasting 45 to 90 minutes. The total amount of blood exchanged is equal to twice the infant's blood volume.

Complications of exchange transfusion include problems related to the blood (transfusion reaction, metabolic instability, or infection), the catheter (vessel perforation or hemorrhage), or the procedure (hypotension or necrotizing enterocolitis). Unusual complications include thrombocytopenia and graft-versus-host disease. Continuation of phototherapy may reduce the necessity for subsequent exchange transfusions.
Management:

Phototherapy is an effective and safe method for reducing indirect bilirubin levels, phototherapy is begun when indirect bilirubin levels are between 16 and 18 mg/dL. Phototherapy is initiated in premature infants when bilirubin is at lower levels, to prevent bilirubin from reaching the high concentrations necessitating exchange transfusion. Blue lights and white lights are effective in reducing bilirubin levels.

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Small infusions of whole blood crossmatched with that of the mother and infant are alternated with withdrawals of an equivalent quantity of the infant's blood, which is discarded. Depending on the size of the infant, aliquots of 5 to 20 mL per cycle are withdrawn and infused, with the total procedure lasting 45 to 90 minutes. The total amount of blood exchanged is equal to twice the infant's blood volume.

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2-2 Neonatal seizures

Seizures during the neonatal period may be the result of multiple causes, with characteristic historical and clinical manifestations. Seizures caused by hypoxic-ischemic encephalopathy (postasphyxial seizures), a common cause of seizures in the full-term infant, usually occur 12 to 24 hours after a history of birth asphyxia and often are refractory to conventional doses of anticonvulsant medications. Postasphyxial seizures also may be caused by metabolic disorders associated with neonatal asphyxia, such as hypoglycemia and hypocalcemia. IVH is a common cause of seizures in premature infants and often occurs between 1 and 3 days of age. Seizures with IVH are associated with a bulging fontanel, hemorrhagic spinal fluid, anemia, lethargy, and coma. Seizures caused by hypoglycemia often occur when blood glucose levels decline to the lowest postnatal value (at 1 to 2 hours of age or after 24 to 48 hours of poor nutritional intake). Seizures caused by hypocalcemia and hypomagnesemia develop in high-risk infants and respond well to therapy with calcium, magnesium, or both.

- Clinical Characteristics of Neonatal Seizures:

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<td>Focal clonic</td>
<td>Repetitive, rhythmic contractions of muscle groups of the limbs, face, or trunk</td>
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<td>May be unilateral or multifocal</td>
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<td>May appear synchronously or asynchronously in various body regions</td>
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<td>Cannot be suppressed by restraint</td>
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<tr>
<td>Focal tonic</td>
<td>Sustained posturing of single limbs</td>
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<td>Sustained asymmetric posturing of the trunk</td>
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<td></td>
<td>Sustained eye deviation</td>
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<tr>
<td></td>
<td>Cannot be provoked by stimulation or suppressed by restraint</td>
</tr>
<tr>
<td>Myoclonic</td>
<td>Anhythmic contractions of muscle groups of the limbs, face, or trunk</td>
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</table>
Typically not repetitive or may recur at a slow rate
May be generalized, focal, or fragmentary
May be provoked by stimulation

Generalized tonic
Sustained symmetric posturing of limbs, trunk, and neck
May be flexor, extensor, or mixed extensor/flexor
May be provoked by stimulation
May be suppressed by restraint or repositioning

Ocular signs
Random and roving eye movements or nystagmus
Distinct from tonic eye deviation

- The diagnostic evaluation of infants with seizures should involve an immediate determination of capillary blood glucose levels with a Chemstrip. In addition, blood concentrations of sodium, calcium, glucose, and bilirubin should be determined. When infection is suspected, CSF and blood specimens should be obtained for culture.

The treatment of neonatal seizures may be specific, such as treatment of meningitis or the correction of hypoglycemia, hypocalcemia, hypomagnesaemia, hyponatremia, or vitamin B6 deficiency or dependency. In the absence of an identifiable cause, therapy should involve an anticonvulsant agent, such as 20 to 40 mg/kg of phenobarbital, 10 to 20 mg/kg of phenytoin (Dilantin), or 0.1 to 0.3 mg/kg of diazepam (Valium), followed by one of the two longer acting drugs. Treatment of status epilepticus requires repeated doses of phenobarbital and may require diazepam or midazolam, titrated to clinical signs.

- The long-term outcome for neonatal seizures usually is related to the underlying cause and to the primary pathology, such as hypoxic-ischemic encephalopathy, meningitis, drug withdrawal, stroke, or hemorrhage.
2-3 Respiratory Distress Syndrome:-
RDS occurs after the onset of breathing and is associated with an insufficiency of pulmonary surfactant.

Lung Development
The lining of the alveoli consists of 90% type I cells and 10% type II cells. After 20 weeks of gestation, the type II cells contain vacuolated, osmophilic, lamellar inclusion bodies, which are packages of surface-active material. This lipoprotein surfactant is 90% lipid and is composed predominantly of saturated phosphatidylcholine (lecithin), but also contains phosphatidylglycerol, other phospholipids, and neutral lipids. Surfactant prevents atelectasis by reducing surface tension at low lung volumes and contributes to lung recoil by increasing surface tension at larger lung volumes when it is diluted during inspiration as the alveolar radius increases.

The timing of surfactant (lecithin) production is begins between 32 and 34 weeks of gestation. By 34 to 36 weeks, sufficient surface-active material is produced by the type II cells in the lung, is secreted into the alveolar lumen, and is excreted into the amniotic fluid. The concentration of lecithin in amniotic fluid indicates fetal pulmonary maturity.

Incidence:
Infants at greatest risk for RDS are premature and have an immature L/S ratio. The incidence of RDS increases with decreasing gestational age. RDS develops in 30% to 60% of infants between 28 and 32 weeks of gestation. Other risk factors include delivery of a previous preterm infant with RDS, maternal diabetes, hypothermia, fetal distress, asphyxia, male sex, white race, being the second born of twins, and delivery by cesarean section without labor.

Clinical Manifestations
Manifestations of RDS include cyanosis, tachypnea, nasal flaring, intercostal and sternal retractions, and grunting. Grunting is caused by closure of the glottis during expiration, the effect of which is to maintain lung volume (decreasing atelectasis) and gas exchange during exhalation. Atelectasis is well documented by radiographic examination of the chest, which shows a ground-glass haze in the lung surrounding air-filled bronchi (the air bronchogram
During the first 72 hours, infants with RDS have increasing distress and hypoxemia. In infants with severe RDS, the development of edema, apnea, and respiratory failure necessitates assisted ventilation. Thereafter, uncomplicated cases show a spontaneous improvement that often is heralded by diuresis and a marked resolution of edema. Complications include the development of a pneumothorax, a PDA, and bronchopulmonary dysplasia (BPD).

**Prevention and Treatment:**
Strategies to prevent preterm birth include maternal cervical cerclage, bed rest, treatment of infections, and administration of tocolytic medications. Additionally, prevention of neonatal cold stress, birth asphyxia, and hypovolemia reduces the risk of RDS. If premature delivery is unavoidable, the antenatal administration of corticosteroids (e.g., betamethasone) to the mother (and thus to the fetus) stimulates fetal lung production of surfactant; this approach requires multiple doses for at least 48 hours.

**COMPLICATIONS OF RESPIRATORY DISTRESS SYNDROME**
- Pulmonary Air Leaks
- Patent Ductus Arteriosus
- Bronchopulmonary Dysplasia (Chronic Lung Disease)
- Retinopathy of Prematurity (Retrolental Fibroplasia)
- Transient Tachypnea of the Newborn
- Meconium Aspiration Syndrome
- Primary Pulmonary Hypertension of the Newborn (Persistent Fetal Circulation)
- Apnea of Prematurity

**2-4 neonatal infection:-**

Neonatal infection can be classified into congenital and acquired.

**1-CONGENITAL INFECTION (TORCHS):**

**TOXOPLASMOSIS**

Toxoplasma gondii is an intracellular parasite (protozoa) capable of crossing the placenta and causing fetal infection. Victims of toxoplasmosis have a latent infection that can reactivate later in life. The primary form of toxoplasmosis in pregnancy is seen in the placenta, but it can also affect the fetus. The classic triad of congenital toxoplasmosis includes hydrocephalus, cataracts, and chorioretinitis. Other symptoms include hearing loss, mental retardation, microcephaly, and failure to thrive. Toxoplasmosis can also cause encephalitis, meningitis, and seizures in the newborn. The diagnosis is usually made through serology or by identifying the organism in tissue samples. Treatment with antibiotics and antiparasitic drugs may be necessary to prevent serious complications in the fetus.

Result only when maternal occur during pregnancy at birth the majority of affected newborn infant (70%) are asymptomatic, the characteristic manifestation chorioretinitis, cerebral calcification,
Psychomotor retardation, hydrocephalus or microcephaly and convulsions may appear later in infancy.

Childhood

CYTOMEGALOVIRUS:
Maternal infection is usually inapparent, neonates with severe transplacental infection are usually SGA and may suffer from or sequentially develop deep jaundice, hepatosplenomegaly, purpura, respiratory distress, microcephaly and retinopathy.

CONGENITAL RUBELLA:
During maternal infection the rubella virus can cross the placenta, and result in the abortion of the first of an infant with the congenital rubella infection. The risk to the fetus is much greater during the first 8 weeks of gestation. The affected infant might show: purpura, hepatosplenomegaly, jaundice, congenital heart disease, respiratory distress, or neurological abnormalities. Those infants who survive will suffer from one or more of the following (congenital rubella syndrome): cataract, glaucoma, microphthalmia, retinopathy, deafness, congenital heart disease, cerebral palsy, microcephaly.

NEONATAL HERPES SIMPLEX:
Herpes simplex virus mostly type B may cause a severe generalized disease in the neonate with high mortality. The majority of cases are thought to be acquired during passage through an infected birth canal.
The virus, however can cross the placenta and result in serious congenital
malformation.

The clinical picture ranges from few vesicular lesions affecting the skin, eye, or mouth
to a generalized severe disease involving
Many organ, resembling bacterial septicemia.

NEONATAL SEPTICEMIA AND NEONATAL MENINGITIS:

DEFINITION:

Generalized bacterial infection during the first 4 weeks of life associated with general
systemic manifestation
And positive blood culture.

In 40% of cases, it is associated with meningitis, and in 20-25% of cases with
pneumonia or urinary tract infection.

Septicemia and meningitis are considered together since the etiology and clinical
picture are closely similar and
Frequently associated.

PREDISPOSING FACTOR

Mother infected before delivery e.g bacterial endocarditis, septicemia, urinary tract
infection.

Premature rupture of membranes.

Prematurity and low birth weight or infant who require hospitalization and
manipulations

As infant of diabetic mother.

Babies with congenital defect leading to exposure of the internal organs as
meningoceles

Or omphaloceles.

Mother with no antenatal care, poor hygiene, low socioeconomic status, cervicitis not
treated well.
Etiologic organism:
E.coli, pseudomonas, proteus, klebsiella, group B streptococci, listeria monocytogenes, staphylococcus aureus.

PORTAL OF ENTRY
Skin, mucous membranes, umbilical cord, nasopharynx, lungs, GIT, and urinary tract.

Clinicle manifestation:-

Onset:
Early onset sepsisemia manifestation are present at birth or any time during the first week (usually less than 3 days).
Late onset sepsisemia manifestation appear 8-28 days after delivery.

Early manifestations:

Clinicle picture:-
symptoms and signs are subtle, non specific vague.
- poor suckling.
- lethargic, hypoonic.
- tendency to hypothermia.
- looks bad, not doing well, and off colour.

manifestation in an established case:-
GIT: vomiting, constipation, diarrhea, abdominal distension, hepatomegaly.
Jaundice.
LUNG: tachypnea, resoritory distress, apnea, cough.
CVS: tachycardia, bradycardia, heart failure, hypotension, pallor or cyanosis and shock.
CNS: tremor, convulsion, apnea, bulging anterior
Blood: jaundice, anemia, purpura, ecchymosis, splenomegaly.
late manifestation:
- organomegaly
- direct hyperbilirubinemia
- DIC

Purpura
Convulsion.
Diagnosis:
1) history:
A predisposing factor as prematurity, low birth weight, premature rupture of membrane (<24 hours), infected birth canal, septic manipulation during delivery.
2) clinical manifestation: as refuse of feeding, diarrhea, convulsion, tachycardia, etc.
3) A sepsis work up which include:
- blood culture.
- CSF analysis
- urine analysis.

Gastric aspirate gram stain.
Buffy coat smear.
Culture from any septic focus.

TREATMENT:
General
(1) feeding by tube or I.V alimentation may be needed.
(2) anti convulsant (phenobarbitone) for convulsion
(3) warming for hypothermia.
(4) fresh blood transfusion may be given antibodies, nutrients, correct anemia and DIC.
(5) care of the umbilical cord (by ethyl alcohol).
(6) maintenance of fluid and electrolyte balance, ventilatory assistance and support of blood pressure with intronic agents.

**Specific:**

A combination of antibiotic are given till the result of culture sensitivity are available. A combination of ampicillin and gentamicin or cefotaxime and amikacin are given by I.V. route. The treatment is modified according to culture and sensitivity.

Therapy is continued for 10-14 days and in case of meningitis, it should be maintained for at least 3 weeks.
3-1 methodology

3-1 study area:-
This study was conducted in port-sudan teaching hospital, department of paediatric, port sudan city, red sea state, at duration between January to February 2010.

3-2 study population:-
Mothers of neonate in paediatric teaching hospital of port sudan.

3-3 study design:-
Hospital based prospective study.

3-4 sample size:-
Mother of neonate admitted to the hospital during period of data collection were interviewed.

3-5 method of data collection:-
The data have been collected using questionnaire which design according to the information need to fulfill the objective of study.

3-6 method of data analysis:-
The data was analysis using master sheet and computer.
chapter (4)
## 4-1 Result

Table (1): show sex distribution:

<table>
<thead>
<tr>
<th>sex</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>male</td>
<td>16</td>
<td>53.3</td>
</tr>
<tr>
<td>female</td>
<td>14</td>
<td>47.7</td>
</tr>
<tr>
<td>total</td>
<td>30</td>
<td>100</td>
</tr>
</tbody>
</table>

Figure (1)

![Bar chart showing sex distribution]

Table (2): show Age distribution:

<table>
<thead>
<tr>
<th>Age</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>1hour-4days</td>
<td>10</td>
<td>33.3</td>
</tr>
<tr>
<td>4days-2weeks</td>
<td>13</td>
<td>43.3</td>
</tr>
<tr>
<td>above</td>
<td>7</td>
<td>23.4</td>
</tr>
<tr>
<td>total</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Figure (2)

![Bar chart showing age distribution]
Table (3): Show tripe distribution:

<table>
<thead>
<tr>
<th>Tribe</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>بنى عامر</td>
<td>13</td>
<td>43.3</td>
</tr>
<tr>
<td>هندوة</td>
<td>5</td>
<td>16.7</td>
</tr>
<tr>
<td>آخرى</td>
<td>12</td>
<td>40</td>
</tr>
<tr>
<td>total</td>
<td>30</td>
<td>100</td>
</tr>
</tbody>
</table>

Figure (3):
Table (4): show presenting symptom:

<table>
<thead>
<tr>
<th>symptom</th>
<th>count</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>preterm</td>
<td>15</td>
<td>50</td>
</tr>
<tr>
<td>Yellow discoloration</td>
<td>6</td>
<td>20</td>
</tr>
<tr>
<td>Refuse feeding</td>
<td>4</td>
<td>13.3</td>
</tr>
<tr>
<td>convulsion</td>
<td>4</td>
<td>13.3</td>
</tr>
<tr>
<td>vomiting</td>
<td>3</td>
<td>10</td>
</tr>
<tr>
<td>fever</td>
<td>5</td>
<td>16.6</td>
</tr>
<tr>
<td>SOB</td>
<td>4</td>
<td>13.3</td>
</tr>
<tr>
<td>other</td>
<td>2</td>
<td>6.7</td>
</tr>
<tr>
<td>total</td>
<td>30</td>
<td>100</td>
</tr>
</tbody>
</table>

Figure (4):
Table (5): show the eventful of pregnancy:

<table>
<thead>
<tr>
<th>pregnancy</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>eventful</td>
<td>5</td>
<td>16.7</td>
</tr>
<tr>
<td>Not eventful</td>
<td>25</td>
<td>83.3</td>
</tr>
<tr>
<td>total</td>
<td>30</td>
<td>100</td>
</tr>
</tbody>
</table>

Figure (5):
Table (6): show antenatal care of mother:

<table>
<thead>
<tr>
<th>ANC</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>regular</td>
<td>24</td>
<td>80</td>
</tr>
<tr>
<td>Not</td>
<td>6</td>
<td>20</td>
</tr>
<tr>
<td>total</td>
<td>30</td>
<td>100</td>
</tr>
</tbody>
</table>

Figure (6):
Table (7): show mother vaccination:

<table>
<thead>
<tr>
<th>vaccination</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>yes</td>
<td>17</td>
<td>56.7</td>
</tr>
<tr>
<td>no</td>
<td>13</td>
<td>43.3</td>
</tr>
<tr>
<td>total</td>
<td>30</td>
<td>100</td>
</tr>
</tbody>
</table>

Figure (7):

Table (8): show maturity of the neonate:

<table>
<thead>
<tr>
<th>maturity</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>terme</td>
<td>11</td>
<td>36.7</td>
</tr>
<tr>
<td>preterm</td>
<td>19</td>
<td>63.3</td>
</tr>
<tr>
<td>Total</td>
<td>30</td>
<td>100</td>
</tr>
</tbody>
</table>

Figure (8):
Table (9): show mode of delivery:

<table>
<thead>
<tr>
<th>mode</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>normal</td>
<td>25</td>
<td>83.3</td>
</tr>
<tr>
<td>forceps</td>
<td>2</td>
<td>6.7</td>
</tr>
<tr>
<td>cesarean</td>
<td>3</td>
<td>10</td>
</tr>
<tr>
<td>total</td>
<td>30</td>
<td>100</td>
</tr>
</tbody>
</table>

Figure (9):
Table (10): show neonate cry immediately or not:

<table>
<thead>
<tr>
<th>Cry immediately</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>yes</td>
<td>25</td>
<td>83.3</td>
</tr>
<tr>
<td>no</td>
<td>5</td>
<td>16.7</td>
</tr>
<tr>
<td>total</td>
<td>30</td>
<td>100</td>
</tr>
</tbody>
</table>

Figure (10):

Table (11): show neonate receive resuscitation:

<table>
<thead>
<tr>
<th>resuscitation</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>yes</td>
<td>5</td>
<td>83.3</td>
</tr>
<tr>
<td>no</td>
<td>25</td>
<td>16.7</td>
</tr>
<tr>
<td>total</td>
<td>30</td>
<td>100</td>
</tr>
</tbody>
</table>

Figure (11):
Table (12) shows the mother age:

<table>
<thead>
<tr>
<th>Mother age</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Below 20 years</td>
<td>5</td>
<td>16.7</td>
</tr>
<tr>
<td>20 - 35</td>
<td>23</td>
<td>76.6</td>
</tr>
<tr>
<td>Above 35</td>
<td>2</td>
<td>6.7</td>
</tr>
<tr>
<td>total</td>
<td>30</td>
<td>100</td>
</tr>
</tbody>
</table>

Figure (12):
Table (13) shows the consanguinity:

<table>
<thead>
<tr>
<th>The consanguinity</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Second degree</td>
<td>17</td>
<td>6.7</td>
</tr>
<tr>
<td>3rd degree</td>
<td>5</td>
<td>16.7</td>
</tr>
<tr>
<td>no</td>
<td>8</td>
<td>26.6</td>
</tr>
<tr>
<td>total</td>
<td>30</td>
<td>100</td>
</tr>
</tbody>
</table>

Figure (13):
Table (14): show the socioeconomic status:

<table>
<thead>
<tr>
<th>Status</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>low</td>
<td>24</td>
<td>80</td>
</tr>
<tr>
<td>moderate</td>
<td>5</td>
<td>16.7</td>
</tr>
<tr>
<td>high</td>
<td>1</td>
<td>3.3</td>
</tr>
<tr>
<td>total</td>
<td>30</td>
<td>100</td>
</tr>
</tbody>
</table>

Figure (14):
Table (15) show neonatal weight:

<table>
<thead>
<tr>
<th>weight</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 - 2.5 kg</td>
<td>17</td>
<td>56.7</td>
</tr>
<tr>
<td>2.5 - 3.5 kg</td>
<td>8</td>
<td>26.6</td>
</tr>
<tr>
<td>Above 3.5 kg</td>
<td>5</td>
<td>16.6</td>
</tr>
<tr>
<td>total</td>
<td>30</td>
<td>100</td>
</tr>
</tbody>
</table>

Figure (15):
Table (16) shows the diagnosis:

<table>
<thead>
<tr>
<th>The diagnosis</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>preterm</td>
<td>15</td>
<td>50</td>
</tr>
<tr>
<td>convulsion</td>
<td>3</td>
<td>10</td>
</tr>
<tr>
<td>jaundis</td>
<td>4</td>
<td>13.3</td>
</tr>
<tr>
<td>pneumonia</td>
<td>4</td>
<td>13.3</td>
</tr>
<tr>
<td>sepsis</td>
<td>4</td>
<td>13.3</td>
</tr>
<tr>
<td>total</td>
<td>30</td>
<td>100</td>
</tr>
</tbody>
</table>

Figure (16):
Table (17): show the outcome:

<table>
<thead>
<tr>
<th>The outcome</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Discharge well</td>
<td>24</td>
<td>80</td>
</tr>
<tr>
<td>death</td>
<td>6</td>
<td>20</td>
</tr>
<tr>
<td>total</td>
<td>30</td>
<td>100</td>
</tr>
</tbody>
</table>

Figure (17):
Table (18) shows the incidence of neonatal admission:

<table>
<thead>
<tr>
<th>The admission</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>neonate</td>
<td>30</td>
<td>6.5</td>
</tr>
<tr>
<td>other</td>
<td>462</td>
<td>93.5</td>
</tr>
<tr>
<td>total</td>
<td>492</td>
<td>100</td>
</tr>
</tbody>
</table>

Figure (18):
4-2 interpretation of result:

1) Table (1) show sex distribution of neonate in PTH: 53.3% of sample was male while 46.7% was female.

2) Table (2) show age of neonate at of admission:
   from 1 hours - 4 day was 33.3%, from 2 day - 2 week was 43.3%, above 2 weeks was 23.4%

3) Table (3) show the tripe of the mother
   43.3% was belong to bniamer tripe, 16.7 was belong to handawa trice, 40% belong to other tripe.

4) Table (4) show the presenting symptoms:
   50% of the cases was perterm, 20% was yellowish dis colouration, 13.3% was refuse feeding, 13.3 was convulsion
   10% was vomiting, 16.6 was fever, 13.3 was SOB, 6.7 was other complain.

5) Table (5) show the history of pregnancy
   83.3% was eventfull, 16.7 was evenfull.

6) Table (6) show antenatal care of the mother
   80% was regular, 20% was not.

7) Table (7) show the vaccination of the mother
   56% was vaccinated, 43 was not.

8) Table (10) show maturity of the neonate:
   36.7% of baby was term, 63.3 was preterm.

9) Table (8) show the mode of delivery of baby
   by normal vaginal delivery was 83.3%, by ceseason was 10%, 6.7 was forceps.

10) Table (9) show the cming of the baby
    83.3% was cry immediatly, 16.7% was not.

11) Table (11) show recived resustation:
YES was 16.7%, NO was 83.3%.

12) table show the mother age:
16.7% was below 20 years, 76% was between 20-35, 6.7% was above 35 years old.

13) table show the consanguinity
56.7% was first degree, 16.7% was second degree, 26.6% non.

14) table show socioeconomic status of the family,
80% was low, 16.7% was moderate, 3.3% was high.

15) table show the weight of the baby,
56% was from 1-2 KG, 26.6% from 2-3 KG, 16.6% was from 3.5-4 KG.

16) table show the diagnosis of neonate
50% was prematurity, 10% was convulsion, 13.3% sepsis, 13.3% jaundice, 13.3% pneumonia.

17) table show the outcome
90% was discharged well, 10% was dead.

18) table (18) show the incidences of neonatal admission:
6.5% was neonate, 93.5% of other child.
chapter (5)
5-1 discussion

This descriptive study was conducted in incidence, causes and outcome of neonatal admission in PTH, 30 neonate admitted to port sudan hospital between January to February were studied.

- From the study we found that the incidence of neonate admission was only small percentage (6.5%). so it’s low incidence.
- The study found that the commonest age admitted to hospital was between 4 days -2 weeks present 43.3%, and other percentage 33.3% in age (1 hour - 4 days), 23.4% in age above 2 weeks.
- Regarding the sex we found that the male are more affected than female.
- We found that the most trips affected is baniamir and hadandawa trip more than any other trip.
- Regarding to the causes of admission we found that the most of the neonate is premature which present 50% of the cases, which is may be due to immature lung or enter of fluid to the lung, also high percentage present by yellow discoloration of the skin and mucous membrane, the remaining are present by convulsion, fever, and shorting of breathing.
- We found that most of the mother having uneventful pregnancy (83.3%) and good antenatal care (80%) only 16.7% having eventful. but just about half of them was vaccinated.
- Regarding the mode of delivery we found that about two third of the neonate was delivered by normal vaginal delivery and the rest by cesarean section.
- We found that 83% of the neonate were cry immediately after delivery and only 16% were not and receive resuscitation.
- Most of the mother age was between the age of 20 -35 years old and the remaining was above or below that.

- From this study we found that half of the parent having second degree consanguinity, and one third having no consanguinity, and the rest is 3ed degree.
- We found that most of the affected neonate were of low socioeconomic status and the rest were moderate status.
- Regarding the weight we found half of the neonate of 1_2.5KG and one third was 2.4_3.5KG but only 16.6%is above 3.5KG.
- From the study its clear that the causes of neonatal admission was premature which present about 50%of the cases, convulsion, jaundice, pneumonia, and neonatal sepsis.
- We found that 80% of the neonates were discharged well and 20%of cases unfortunately were dead, 4 is preterm and 2 is due to sepsis.
chapter (6)
6-1 conclusion

This study was carried out in one month (January to February) in PTH in order to identify the incidence, causes, and outcome of neonatal admission in pediatric department.

The incidence of admission is very low only (6.5%).

The highest percentage causes of admission was the preterm neonate (50%) of all cases, and male more than female. And the bacteriomer and hadandawa trips are the most common affected groups.

Most of the neonate were discharged well (80%).
6-2 recommendation

1) prevent preterm birth include maternal bed rest, treatment of infections, and administration of tocolytic medications.
2) Additionally, prevention of neonatal cold stress, birth asphyxia, and hypovolemia reduces the risk of RDS.
3) If premature delivery is unavoidable, the antenatal administration of corticosteroids to the mother (and thus to the fetus) stimulates fetal lung production of surfactant.
4) health education to the mother.
Chapter (7)
7-1 reference:

- INTERNET:
  www.google.com

  Elsevier Inc/ 833-840

- Nasser Gammal – manual of pediatrics- publisher: dar
The incidence, causes and outcome of neonatal admission

- NO [ ] SEX [ ]
- Age: 1 hour 4 days [ ] 5 days 2 weeks [ ] above [ ]
- Tripe: [ ] Residence [ ]
- Complain of: fever [ ] yellowish discoloration [ ] vomiting [ ]
- Refuse feeding [ ] convulsion [ ] SOB [ ]
- Preterm [ ] other [ ]
- Pregnancy: evenfull [ ] un eventfull [ ]
- Antinatal care: regular [ ] not [ ]
- Mother vaccination: yes [ ] no [ ]
- Mode of delivery: normal [ ] foreceps [ ] cesarean [ ]
- The maturity: term [ ] preterm [ ]
- Cry immediately: yes [ ] no [ ]
- Resuscitate: yes [ ] no [ ]
- Mother age: below 20 [ ] 20-35 [ ] above [ ]
- Cosangunity: second degree [ ] third degree [ ] non [ ]
- Socioeconomic: low [ ] moderate [ ] high [ ]
- Weight: 1_2KG [ ] 2_3.5KG [ ] above [ ]
- Diagnosis: [ ]
- The outcome: discharged well [ ] death [ ]