Nursing in the NICU: Common Neonatal Conditions

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Purpose and Objectives

The purpose of this course is to provide a review of common complications seen with neonatal intensive care patients. Risk factors, symptoms, and management of common conditions will be discussed.

After successful completion of this course, you will be able to:

1. Describe risk factors for the neonate to develop complications.
2. Identify at least four common conditions seen in the NICU.
3. Discuss intraventricular hemorrhage (IVH), including risk factors, symptoms, and management.
4. Review patent ductus arteriosus (PDA), including risk factors, symptoms, and management.
5. Review persistent pulmonary hypertension (PPHN), including risk factors, symptoms, and management.

6. Describe at least two respiratory disorders of the neonate, including risk factors, symptoms, and management.
7. Discuss development of sepsis and necrotizing enterocolitis (NEC) in the neonate, including risk factors, symptoms, and management.
8. Identify fluid replacement and management considerations.
9. Recognize common electrolyte imbalances in the neonate, including risk factors, symptoms, and management.

Introduction

The neonate is considered one of our most vulnerable patient populations. Knowledge, skills, caring, and compassion are all necessary competencies of the nurses working in the neonatal intensive care unit (NICU). Care is individualized to these tiny patients, but there are common conditions and complications that can be seen.

Risk Factors of the Neonate

A newborn, or neonate, is a child under 28 days of age. This time period is the most crucial, as the child is at the highest risk of death (World Health Organization, 2014).

The majority of neonates who are in the NICU are premature and/or low birth weight, which puts them at even higher risk. The systems of the premature infant have not fully developed, which can make adaptation to extrauterine life complicated (Kenner & Lott, 2013).

Infants who are post-term, have experienced complications of labor and delivery, have genetic disorders, or have other diseases or disorders can also be patients in the NICU.

Common Conditions in the NICU

There are common conditions seen with NICU patients, affecting various systems, which will be discussed further.

These include:

- Intraventricular hemorrhage (IVH).
- Patent ductus arteriosus (PDA).
- Persistent pulmonary hypertension (PPHN).
- Apnea and bradycardia.
- Respiratory distress syndrome (RDS).
- Transient tachypnea of the newborn (TTNB).
- Sepsis.
- Necrotizing enterocolitis (NEC).
- Fluid and electrolyte imbalances.

Intraventricular Hemorrhage (IVH)

Intraventricular hemorrhage (IVH) is the most common type of neonatal intracranial hemorrhage. There is an increasing risk with decreasing gestational age, due to the presence of the germinal matrix in the brain, and cerebral blood flow patterns.

The germinal matrix is the embryonic structure that is unique to premature babies, which provides vascular supply for 24-32 weeks gestation. This structure is primitive, and made of smooth endothelial cells. It is highly vascular, and susceptible to bleeding (Stiles & Jernigan, 2010; Kenner & Lott, 2013).

There can be intravascular fluctuations in blood flow and hyperperfusion with infants <32 weeks gestation. There can also be disturbances in cerebral autoregulation, which is the maintenance of cerebral blood flow to the brain. In the extravascular area around the germinal matrix, vascular support can be deficient, and there can be an increased fibrinolytic activity which can cause diffuse clots. All of these factors can contribute to the development of IVH (Kaiser, 2009).

An IVH that occurs with term infants is rare. If this occurs, the site of origin is the choroid plexus, which lines the ventricles.

Introduction

Diagnosis of IVH is most commonly done with a cranial ultrasound. The degree of the IVH is determined by grading I through IV:

Grade I: Bleeding is confined to subependymal germinal matrix. There is a 10% chance of neuro sequelae following the occurrence of a Grade I IVH. This means that the patient will have a 10% chance of developing other neurological disorders / complications following the Grade I IVH.

Grade II: Extension into ventricular system without acute hydrocephalus (20% neuro sequelae)

Grade III: Germinal matrix with extension into ventricles and hydrocephalus (ventricular dilation) (35% neuro sequelae)

Grade IV: As Grade III, with extension into brain parenchyma (white matter)- paraventricular hemorrhage infarct (80-100% neuro sequelae)

Signs and Symptoms of IVH

An IVH occurs most commonly in the first few days of life. 50% of IVH occurs by 24 hours of age, and 90% occurs by 72 hours of age.

Signs and symptoms of IVH include:

- Decreased motor activity.
- Hypotonia/flaccidity.
- Seizures.
- Apnea.
- Bulging fontanel.
- Bradycardia.
- Hypotension.
- Falling hematocrit.
- Metabolic acidosis.

Treatment of IVH

Treatment is generally supportive for neonates with IVH, and depends upon the severity of the bleeding. Correction of underlying disturbances that may contribute to the IVH may be indicated. Fluids, transfusions, correction of acid-base imbalances, and cardiorespiratory support may be performed. Medication aimed at lowering the incidence of hydrocephalus, such as the use of acetazolamide (Diamox) to lower production of cerebral spinal fluid (CSF) is controversial (Annibale, 2014).

Developmental care principles, including minimal handling, is important when working with premature infants. This can lower the incidence of an IVH, and decrease potential effects following an IVH occurrence. Handling, and care to avoid, includes: lifting the lower extremities above midline with diaper changes, rapid fluid boluses, and high oxygen and ventilation, as these can all increase the chance of an IVH occurring (Verklan & Walden, 2010).

Test Yourself:

In the premature neonate, the site for an IVH is:

Choroid plexus

Germinal matrix - Correct

Hypothalamus

Patent Ductus Arteriosus (PDA)

To briefly review fetal circulation, oxygenated blood is carried from the placenta to the fetus through the umbilical vein. The umbilical vein has a small branch that carries blood to the liver, then to the inferior vena cava; the larger branch, the ductus venosus, empties directly into the inferior vena cava.

The inferior vena cava enters the right atrium, and the majority of blood from right atrium crosses the foramen ovale into the left atrium. There is a small amount of blood that enters the pulmonary artery to the lungs, but more blood from the pulmonary artery is diverted to the descending aorta through the ductus arteriosus (Kenner & Lott, 2013).

The ductus arteriosus diverts blood from the pulmonary artery to the aorta during fetal circulation (right to left direction in utero). This ductus begins to constrict and functionally close within hours of birth, and normally has a full anatomical closure in weeks.

The closure of smooth muscle with the ductus arteriosus is influenced by hypoxia and acidosis. These factors can inhibit the closure of this ductus, resulting in a patent ductus arteriosus (PDA). The amount and direction of the blood shunting through PDA is dependent of peripheral vascular resistance (PVR), systemic vascular resistance (SVR), and the size of the PDA. 40-50% of infants <29 weeks gestation have a PDA (with a left to right shunt) (Kenner & Lott, 2013).

Did You Know?

PDA is an important factor in determining the presentation of many other congenital heart anomalies, due to "duct dependence." With some forms of congenital heart defects, a PDA is the only manner in which oxygenated blood can be circulated. If the PDA closes before surgical intervention, it is a medical emergency.

Signs and Symptoms of PDA

Diagnosis of PDA is most commonly done through an echocardiogram. This can determine the presence and size of the PDA, as well as the direction of the blood shunting.

Signs and symptoms of PDA include:

- An audible murmur in (loudest at the left upper sternal border).
- A widened pulse pressure (the difference between the systolic and diastolic blood pressures).
- Swinging oxygenation requirements to maintain oxygen saturation.
- Apnea and bradycardia.
- Pulmonary congestion and/or edema.
- Bounding or full pulses.
- Difficulty feeding.
- Tachypnea.
- The patient may be asymptomatic.

(Kenner & Lott, 2013; Kim, 2012)

Treatment of PDA

If a patient is asymptomatic, or the PDA is small and appears to be closing, there may be no treatment. In these circumstances, the size of the PDA is monitored through echocardiograms to ensure that the ductus is closing.

The aim of PDA treatment is to reduce pulmonary over-circulation and improved organ perfusion. Treatment includes fluid and nutrition management, ventilation, cardiotropic support (such as digoxin), indomethacin or ibuprofen, or surgical ligation. Fluid restriction can be a common management strategy. Digoxin or diuretics may be used with patients who develop congestive heart failure. Indomethacin or ibuprofen are used to close the PDA. Surgical ligation is indicated following failed medical attempts at PDA closure (Kenner & Lott, 2013; Kim, 2012).

Persistent Pulmonary Hypertension of the Newborn (PPHN)

Persistent pulmonary hypertension (PPHN or PPH) occurs when the neonate's circulatory system does not have a normal transition after birth. Normally, the systemic vascular resistance increases and pulmonary vascular resistance falls after birth, which results in a left to right shunting through PDA and increase in pulmonary blood flow. PPHN is a result of elevated pulmonary vascular resistance to the point that venous blood is diverted to some degree through fetal shunts (i.e. the ductus arteriosus and foramen ovale) into the systemic circulation and bypassing the lungs, resulting in systemic arterial hypoxemia.

Conditions Associated with PPHN

Asphyxia, hypoxia, acidosis or other complications cause an increase in the pulmonary vascular resistance, which causes a right to left shunt through the PDA, with a subsequent decrease in pulmonary blood flow. This condition is most often associated with perinatal asphyxia in 50-70% of reported cases. Other conditions associated with this syndrome include hypoglycemia, hypocalcemia, hyperviscosity syndrome and sepsis. PPHN is usually seen in term and post-term infants (Kenner & Lott, 2013; Sallaam, 2012).

The specific abnormality which produces persistent pulmonary hypertension is not known. Possible pathogenetic mechanisms include:

- Repeated intrauterine closure of the ductus.
- Abnormal responsiveness of the pulmonary vasculature to hypoxia with an inability to relax.
- Repeated intrauterine hypoxia, which stimulates the hypertrophy of medial smooth muscle which surrounds pulmonary arterioles, enabling vessels to constrict to an extreme degree for long periods of time.
- Regional alveolar hypoxia due to poor ventilation which is not apparent radiographically, even though other parts of the lung are ventilating normally.
- Undergrowth of the pulmonary vascular tree. This is frequently the cause in infants with congenital diaphragmatic hernia or other causes of pulmonary hypoplasia.
- Alterations in vasoactive mediator levels.
- Microthrombus formation in the pulmonary vascular bed.

Signs and Symptoms of PPHN

Diagnosis of PPHN is most commonly done through an echocardiogram. This can determine the presence and size of the PDA, and Doppler studies can determine the direction of the blood shunting.

Signs and symptoms of PPHN include:

- Murmur.
- Respiratory distress.
- Decreased pulmonary blood flow (oligemic lung fields).
- Hypoxia.
- Hypercarbia.
- Hypoglycemia (from anaerobic metabolism).
- Cyanosis.
- Metabolic acidosis.
- Difference between preductal and postductal PaO2.
- Hypotension (usually a late sign).

Treatment of PPHN

The goal of management is to reduce pulmonary vascular resistance, improve acidosis and improve perfusion.

Treatment of PPHN includes:

- Improve alveolar oxygenation (FiO2).
- Ventilation, to decrease pulmonary vascular resistance.
- Minimize pulmonary vasoconstriction.
- Maintain systemic blood pressure and perfusion (dopamine, dobutamine).
- Low stimulus, sedation; paralytics may be used. This can help decrease oxygen demands.
- Maintain hemoglobin levels, monitor fluid and electrolytes.
- Consider induction of an alkalotic state.
- Consider a trial of vasodilatation (including nitric oxide, prostacyclin).
- Consider extracorporeal membrane oxygenation (ECMO) support.

(Kenner & Lott, 2013; Sallaam, 2012)

Apnea and Bradycardia

Apnea, and resulting bradycardia, is a common occurrence with neonatal patients. Apnea is described as a pause in breathing that lasts 15 to 20 seconds or greater, usually associated with bradycardia, oxygen desaturation, and/or cyanosis (CPM Resource Center, 2010a; Gardner, Carter, Enzman-Hines, & Hernandez, 2011).

There are three forms of apnea:

Central Apnea: This type of apnea occurs when there is a disruption of the signal from the central nervous system to the respiratory muscles, also known as the "respiratory drive". This is the most common type of apnea seen with NICU patients.

Obstructive Apnea: This occurs as a result of an obstructive process, especially at the level of the pharynx.

Mixed Apnea: This is a combination of both central and obstructive apnea.

Causes of Apnea

The most common cause of apnea with neonates is apnea of prematurity. The immature central nervous system of the premature infant places them at high risk for central apnea. There is also an immature and often overactive response to vagal stimulation, which causes a reflex central apnea. Premature infants less than 34 weeks gestation and low birth weight infants less than 1800 g are at the highest risk for apnea of prematurity, and should be monitored closely (CPM Resource Center, 2010a; Gardner et al., 2011).

Other causes of apnea with a NICU patient may include:

- Sepsis.
- Pulmonary impairment, including lack of surfactant, meconium aspiration, transient tachypnea of the newborn.
- Cardiovascular impairment, including a PDA or congenital heart disease.
- Neurological impairment, including IVH, seizures, hydrocephalus, or perinatal asphyxia.
- Anemia.
- Metabolic imbalances, such as acidosis, hypocalcemia, hypoglycemia.
- Necrotizing enterocolitis.
- Temperature imbalance, including hypothermia and hyperthermia.
- Medications, including narcotics, sedatives, or exposure to maternal medications.

Treatment of Apnea

Prior to treatment and management of apnea, the cause must be identified. Although apnea of prematurity is the most common cause, other potential factors must be considered. Treatment of apnea includes:

- Tactile stimulation. If the infant does not respond to tactile stimulation; suctioning, positioning, or bag and mask ventilation may be needed.
- Pharmacological treatment may include methylxanthines, such as caffeine or theophylline. Methylxanthines work on the central nervous system by blocking adenosine receptors, which can stimulate the respiratory drive.
- Treating the underlying cause, such as correcting metabolic imbalances, maintaining thermoregulation, etc.

(CPM Resource Center, 2010a; Gardner et al., 2011)

Test Yourself:

Neonates who are highest risk of apnea and bradycardia are those that:

Have a PDA

Are at a birthweight less than 2500 grams

Are born at a gestation less than 34 weeks – Correct

Respiratory Distress Syndrome (RDS)

Respiratory Distress Syndrome (RDS) is also known as neonatal RDS, Respiratory distress syndrome of the newborn, or formerly known as Hyaline Membrane Disease (HMD). To briefly review, surfactant is composed of lipids, proteins, and phospholipids, which decreases surface tension, increases lung compliance, and helps recruit alveoli to open with respirations. Without surfactant, the alveoli will collapse at the end of each expiration.

RDS occurs as a result of insufficient surfactant production, primarily related to prematurity. Surfactant levels are usually not sufficient until 30-35 weeks gestation. In older infants surfactant deficiency can arise when they develop asphyxia, shock and acidosis, such as infants with meconium aspiration (Kenner & Lott, 2013).

Diagnosis of RDS

Diagnosis of RDS is done by arterial blood gases (ABGs), which can show hypoxemia and acidosis, and chest x-ray (CXR). Radiographic changes usually appear shortly after birth, but can be delayed for 12-24 hours.

The classic "ground glass" appearance consists of a bilaterally symmetrical homogeneously stippled pattern of fine lucencies (air bronchograms) interspersed with linear densities that causes the heart and diaphragm contours to become obscured.

The patient usually has a decreased lung volume, and vertically oriented ribs leading to a bell shaped thorax.

Signs and Symptoms of RDS

Signs and symptoms of RDS include:

- Tachypnea.
- Retractions.
- Grunting.
- Shallow respirations.
- Nasal flaring.
- Apnea.

- Color changes.
- Edema.

(Kenner & Lott, 2013)

Treatment of RDS

Treatment of RDS includes:

- Effective initial resuscitation.
- Oxygen- used to treat hypoxemia; must be closely monitored to keep oxygen saturation levels within department guidelines.
- Respiratory support, including the use of continuous positive airway pressure (CPAP). Ventilator assistance may be needed.
- Administration of surfactant.

(Kenner & Lott, 2013)

Did You Know?

Following surfactant administration, the patient must be closely monitored, and preparation for changes in oxygenation and/or ventilation needs to be anticipated. When the alveoli open and surface tension decreases, oxygenation can quickly increase (therefore decreasing the need for supplemental oxygen), and the need for assisted ventilation can rapidly decrease.

Transient Tachypnea of the Newborn (TTNB)

Transient tachypnea of the newborn (TTNB or TTN) was also formerly known as "wet lung."

This disorder results from the delayed absorption of fetal lung fluid following delivery, and is temporary in nature.

Risk factors for developing TTNB include:

- Prematurity.
- Low birth weight infants.
- Precipitous delivery.
- Cesarean sections.
- Inadequate initial resuscitation.

Diagnosis of TTNB

ABGs may be done with TTNB, which can show mild hypoxemia and acidosis. The main test for diagnosing TTNB is the CXR. Typical radiologic features are ill-defined but can include increased central vascular markings ("star-burst" appearance), hyper-aeration, evidence of interstitial and pleural fluid, prominent interlobar fissures, and cardiomegaly.

Signs and Symptoms of TTNB

Signs and symptoms of TTNB include:

- Tachypnea.

- Retractions.
- Grunting.
- Nasal flaring.
- Color changes (in severe cases).
- Crackles on auscultation.

Treatment of TTNB

TTNB generally resolves by 72 hours of age, and thus, treatment is supportive. If symptoms progress beyond 72 hours further investigation into another cause should be considered.

Treatment includes:

- Oxygen.
- Fluids.
- Minimal handling.
- Neutral thermal environment.
- Rarely does TTNB progress to where CPAP or mechanical ventilation is required. However, an infant may fatigue which can lead to the need of assisted ventilation.

(Kenner & Lott, 2013)

Test Yourself:

Baby Bee has a CXR done, and the report describes a ground glass appearance in the lung fields. This would be consistent with:

TTNB

RDS - Correct

Apnea of prematurity

Sepsis

Sepsis is a toxic condition caused by the spread of invading organisms, or their by-products, through the bloodstream or in other tissue in the body. It is also known as septicemia. The definition of sepsis has evolved beyond that of an infection. There is now a spectrum of symptoms of systemic inflammatory response syndrome (SIRS) to septic shock (Caple, 2011). Causes of sepsis include viral, fungal, bacterial or parasitic in nature. Neonatal sepsis differs from adult sepsis in terms of symptoms, diagnosis and treatment, and continues to have a high rate of morbidity and mortality for infants. Neonatal sepsis occurs within the first 30 days of life, although some late-onset sepsis has been described within the first 90 days of life, particularly when related to prematurity (Gardner, 2009).

Risk Factors and Causes of Sepsis

Sepsis that is found in neonates less than 72 hours of age is classified as early-onset sepsis, and after 72 hours is considered late-onset sepsis. Sepsis can be caused by bacteria, viral, or fungal infections. The most serious cause of neonatal sepsis is from Group B Strep (GBS) (Gardner, 2009).

Risk factors for developing sepsis including maternal, neonatal, and environmental factors.

Maternal risk factors contributing to neonatal sepsis include:

- No prenatal care
- Malnutrition
- Low socioeconomic status
- Substance abuse
- Fever
- Active urinary tract infection (UTI)
- Chorioamnionitis
- Positive or unknown Group B Strep
- Premature rupture of membranes (ROM) or premature labor <37 weeks
- Prolonged ROM >24 hours
- Prolonged or difficult labor, and multiple pregnancy

Risk Factors and Causes of Sepsis

Neonatal risk factors include:

- Prematurity
- Low birth weight
- Congenital anomalies (especially ones that disrupt first line of defense, such as gastroschisis)
- Male gender
- Inborn errors of metabolism
- Asphyxia/fetal distress
- Meconium aspiration

Environmental risk factors include:

- Resuscitation
- Invasive procedures
- Length of hospitalization
- Use of antibiotics

(Gardner, 2009; Gardner et al., 2011)

Diagnosis of Sepsis

Diagnostic workup is required with suspected sepsis, or infants at risk. Not one specific test can definitively rule out or confirm sepsis with 100% certainty. Therefore, it can take a combination of laboratory tests, imaging, and clinical symptoms to diagnose neonatal sepsis. Common tests include complete blood count (CBC), blood culture, ABG, C-reactive protein (CRP), lumbar puncture, suprapubic tap or urinalysis, and CXR (Gardner, 2009; Gardner et al., 2011).

Signs and Symptoms of Sepsis

Signs and symptoms of sepsis are categorized as early and late. Often, symptoms in the early stage are non-specific, making it difficult to diagnose (Gardner, 2009; Gardner et al., 2011). The early stage of sepsis

development is also known as "warm shock." The main symptoms are from a decrease in systemic vascular resistance, due to vasodilation. These symptoms include:

Gastrointestinal: Feeding intolerance, abdominal distention

Respiratory: Tachypnea, apnea, grunting, nasal flaring, retractions, increased oxygen requirements

Cardiovascular: Widened pulse pressure (the difference between systolic and diastolic pressure), bradycardia or tachycardia, pink flushing, mottling, full pulses

Neurologic: Lethargy, hypotonia, temperature instability (hypothermic or hyperthermic), behavioral changes

Metabolic: Acidosis, glucose instability (hypoglycemia or hyperglycemia)

Complete blood count (CBC) changes: Increase or decrease in white blood cells (WBC), more immature WBC (increased I:T ratio), decreased platelets

Late Stage Sepsis

The late stage of sepsis development is caused from the body being unable to meet the oxygen demands of tissues. Tissue damage and lactic acidosis can occur, and this is now a hypovolemic state (Gardner, 2009; Gardner et al., 2011). These include:

Cardiovascular: Further decrease in systemic vascular resistance produces a significant decrease in systolic pressure, causing a narrowed pulse pressure.

Neurologic: Seizures.

Metabolic: Decreased perfusion to kidneys (increased BUN, creatinine, renal failure), acidosis, enlarged spleen/liver.

Multiple organ dysfunction syndrome (causes death).

Treatment of Sepsis

Treatment of sepsis includes:

- Anti-infective therapy (based on type of organism).
- Fluid and electrolyte maintenance.
- Hand hygiene and aseptic technique.
- Neutral thermal environment.
- Oxygenation and ventilation.
- Maintaining perfusion.
- Monitoring and treating seizures or other complications.

For a more in-depth look at neonatal sepsis, see RN.com's course Neonatal sepsis: Assessment and care.

Necrotizing Enterocolitis (NEC)

Necrotizing enterocolitis (NEC) is an inflammatory disease of the bowel which can cause ischemic and necrotic injury to the bowel. The progression of the disease varies with each patient, from a slow to a rapid advancement, and can have a very sudden onset. The damage can range from inflammation and injury to the mucosa to full thickness necrosis.

NEC has a very high morbidity and mortality with NICU patients, with as high as 50% mortality rate, depending on severity (Springer, 2014). Infants weighing less than 1500g have a 10% chance of developing NEC, and 1-8% of all NICU patients are at risk for NEC (CPM Resource Center, 2010b; Gregory, DeForge, Natale, Phillips, & Van Marter, 2011; Springer, 2014).

Risk Factors of NEC

The etiology and causes of NEC are still unknown, but it is thought to be multifactorial in nature. Contributory factors include development of inflammation and ischemia, enteral feeding, and abnormal bacterial colonization (such as infection).

Risk factors for developing NEC include:

- Prematurity.
- Low birth weight.
- Perinatal stress and asphyxia.
- Polycythemia.
- Cardiopulmonary disease, including PDA.
- Acute hypoxic event.
- Medications that decrease intestinal motility (including indomethacin and theophylline).
- Hyperosmolar enteral feedings (including breast milk that has human milk fortifier added).
- Enteral feedings with formula.
- Hypovolemia.
- Hypothermia.

(CPM Resource Center, 2010b; Gregory, et al., 2011; Springer, 2014).

Diagnosis

Tests used to diagnose NEC include CBC (to assess counts of white and red blood cells, and platelets), ABGs (to assess for metabolic acidosis), a blood culture, and electrolyte panel. Although an abdominal ultrasound may be helpful, the main test for diagnosing NEC is an abdominal x-ray. Taking views both anteroposterior (AP) and left lateral decubitis (left side of the infant is down) can reveal characteristic findings of NEC. Radiography can demonstrate dilated bowel loops, abnormal gas pattern, and thickening of bowel walls. Pneumatosis intestinalis is a definitive sign of NEC, showing intramural air bubbles that occur from bacteria (Gregory, et al., 2011; Springer, 2014).

Signs and Symptoms of NEC

Signs and symptoms of NEC can begin as early as 24 hours through 90 days of age, but most commonly are seen between 3 and 12 days of age.

These include:

- Abdominal distension.
- Bilious aspirate or emesis.
- Blood in the stools (occult or frank blood).
- Feeding intolerance.
- Decreased or absent bowel sounds.

- Apnea.
- Respiratory distress.
- Lethargy.
- Hypotonia.
- Thermal instability.
- Abdominal tenderness.
- Metabolic acidosis.
- Thrombocytopenia.
- Hypotension.

NEC Staging

The severity of NEC can be classified into stages according to the Bell Staging criteria. Staging is important in determining the management of the disease.

These stages include:

IA: Suspected NEC- signs include apnea, bradycardia, temperature instability, lethargy, residuals prior to gavage feedings, emesis, mild abdominal distension, positive occult blood in stools. Radiology may be normal, or show mild ileus or intestinal dilation.

IB: Suspected NEC- signs are the same as above, but also include bright red blood from the rectum. Radiology is the same as IB.

IIA: Proven NEC/mildly ill- signs are the same as above, but also include absent bowel sounds and may include abdominal tenderness. Radiological findings include ileus, intestinal dilation, and pneumatosis.

IIB: Proven NEC/moderately ill- signs are the same as above, but also include mild metabolic acidosis, mild thrombocytopenia, definite abdominal tenderness, and may have a right lower quadrant mass or abdominal cellulitis. Radiology is the same as IIA, plus portal venous gas, and ascites may be seen.

IIIA: Advanced NEC/severely ill, bowel intact- signs are the same as IIB, and also include apnea, bradycardia, disseminated intravascular coagulation (DIC), neutropenia, hypotension, respiratory and metabolic acidosis, marked abdominal tenderness and distension. Radiological findings are the same as IIB, but include definite ascites.

IIIB: Advanced NEC/severely ill, bowel perforated- signs are the same as IIIA. Radiology is the same as IIIA, and also include pneumoperitoneum.

Treatment of NEC

Treatment of NEC is considered in relation to the Bell Staging criteria.

Stages IA and IB: NPO, antibiotics, TPN and IV fluids. A minimum of three days treatment is considered.

Stages IIA and IIB: NPO, IV fluids and TPN, antibiotics, fluid resuscitation, correction of metabolic imbalances, respiratory and cardiac support. A minimum of 14 days treatment is considered. A surgical consult may be necessary.

Stage IIIA: Treatment is the same as above, as well as inotropic and ventilatory support. A surgical consult is indicated.

Stage IIIB: Surgical intervention is required; a bowel resection is performed.

Did You Know?

The routine use of probiotics has been studied in neonatal patients to prevent NEC, particularly very low birth weight (VLBW) infants. Guidelines have been established, and many academic centers are using this practice for VLBW at high risk, in facilities which have a high incidence of NEC. Many studies, including clinical trials, have concluded that the use of probiotics with very low birth weight infants can reduce the incidence of NEC, but there is insufficient evidence to establish the safety and efficacy of this treatment (Parker, 2014).

NEC Management

Other management strategies of an infant with NEC include:

- Minimal handling.
- Bowel decompression with an orogastric tube.
- Neutral thermal environment.
- Pain management.
- Decreased environmental stimulation.

(CPM Resource Center, 2010b; Gregory, et al., 2011; Springer, 2014).

Test Yourself

Apnea, bradycardia, and metabolic acidosis are all symptoms of which disease?

Sepsis

NEC

Both – Correct

Fluid and Electrolyte Imbalances

Infants characteristically have 80% water composition in their bodies. Preterm infants may have as high as 85-90% total body water. Therefore, fluid requirements are high, due to this high proportion of body water. After birth, this excess water must be mobilized and excreted. Diuresis is a physiologic process during the first days of life, typically after the first 24 hours.

The surface area of the newborn is relatively large, and increases with decreasing size. Therefore, insensible water losses will be greatest with very low birth weight infants and decreased gestational ages. Neonates also have a high metabolic rate, which affects fluid requirements.

The kidneys of the neonate are less efficient at excreting sodium or water load than an adult's kidney. Thus, babies have a limited ability to concentrate urine (Kenner & Lott, 2013; Polin & Spitzer, 2013).

Fluid Loss

As a review, there are three types of fluid loss: sensible, insensible, and abnormal losses.

Sensible: Examples include fluid loss through urine, stool, and sweat.

Insensible: Examples include fluid loss through the skin and lungs.

Abnormal: Examples include fluid loss through diarrhea, ostomy drainage, massive skin breakdown/wounds, aspirates, frequent blood sampling/bleeding, chest tube drainage, gastrointestinal (GI) anomalies (such as gastroschisis and oomphalocele).

Other Factors that Increase Fluid Loss Include:

- Phototherapy (can increase fluid loss two to three times normal).
- Non-humidified air or oxygen.
- Increased respiratory rate and effort; RDS.
- Increased prematurity (as a result of thin skin, vessels near the skin surface).
- Increased activity (can increase losses by 2-6%).
- Radiant heat (can increase losses by 2-3%).
- Increased abnormal losses.
- Cold stress/hypothermia (can increase losses by up to 10%).
- Further increase in metabolic rate (such as that associated with hyperthermia, disease processes).

(Kenner & Lott, 2013; Polin & Spitzer, 2013)

Electrolyte Review

Sodium (Na+):

- Major component of extracellular fluid (ECF)
- Normal values are 136-145 mmol/L
- Preterm infants require higher levels than term infants, as sodium is excreted more, due to the immaturity of the renal system
- Sodium takes part in regulation of acid-base balance, tissue osmolality and enzyme activity
- Sodium is essential for retention of body water

Potassium (K+):

- Major component of Potassium (K+)
- Normal values are 3.5-5 mmol/L
- Potassium also participates in enzyme activity, central nervous system function, regulation of tissue osmolality and glycogen use
- Potassium is essential for cardiac function

Chloride (Cl-):

- Another component of ECF
- Normal values are 99-111 mmol/L
- Chloride participates in tissue and cell osmolality
- Chloride is necessary for potassium retention, transport of carbon dioxide, and formation of hydrochloric acid in the GI tract

Calcium (Ca2+):

- Also a component of ECF
- Normal values 7-12 mg/dL
- Calcium is essential in blood coagulation, and neuromuscular function

Electrolyte Imbalances

Hypernatremia: Although preterm infants have increased excretion of sodium, they are also unable to rapidly increase their sodium excretion, in response to high sodium levels.

Causes of hypernatremia include dehydration (can be a result of increased insensible water loss or diuresis), over-administration of sodium supplementation, osmotic imbalances.

Signs and symptoms of hypernatremia include irritability, lethargy, vomiting, and seizures. Treatment is focused on the underlying cause (Kenner & Lott, 2013; Polin & Spitzer, 2013).

Hyponatremia: Hyponatremia is caused by overhydration, water retention, and hypovolemia. Water retention causes a dilution of serum sodium, but the total body sodium is within normal limits. This can be seen with renal failure, hepatic failure, congestive heart failure, etc. Hypovolemia can also be seen with third spacing and renal losses (such as with the use of diuretics).

Signs and symptoms of hyponatremia include vomiting, lethargy, poor perfusion, edema, and seizures. Treatment is based on the underlying cause, and replacement sodium must be done slowly (Kenner & Lott, 2013; Polin & Spitzer, 2013).

Hyperkalemia: Hyperkalemia is caused by acidosis, renal failure, trauma, bruising, and bleeding. Renal failure causes impaired potassium excretion. Metabolic acidosis forces potassium to leave the cells, as hydrogen is entering. IVH causes an endogenous release of potassium. With transfusions, if the baby receives blood that has been stored over 24 hours, it can have cellular rupture, which increases potassium in the blood.

Signs and symptoms of hyperkalemia include ECG changes (such as widened QRS, peaked T waves, lengthening of PR interval, or a P wave that is difficult to identify, ventricular fibrillation), decreased urine output, lethargy, and decreased muscle tone. Treatment includes correcting the underlying cause, as well as Kayexalate, administration of calcium, or an insulin/glucose drip (Kenner & Lott, 2013; Polin & Spitzer, 2013).

Hypokalemia: Hypokalemia is caused by potassium losses through the GI tract, insufficient potassium intake, and renal losses. GI losses include vomiting, diarrhea, gastric drainage, or loss through an ostomy. Renal losses may be as a result of diuretic therapy, renal disease, aminoglycosides, or amphotericin.

Signs and symptoms of hypokalemia include ECG changes (such as a shortened ST segment, flattened or inverted T-waves, or appearance of "U" wave), intestinal ileus, and gastric dilation. Treatment includes

determining and managing the underlying cause, correcting acid-base imbalances, use of potassium sparing diuretics, slow potassium supplementation, and correction of other electrolyte imbalances (Kenner & Lott, 2013; Polin & Spitzer, 2013).

Hypocalcemia: Causes of hypocalcemia include asphyxia, prematurity, reaction to stressors, infants of a diabetic mother, hypoparathyroidism, vitamin D metabolism defects, and phosphate imbalance. Prematurity and infants born to a diabetic mother are at a high risk for decreased calcium stores. High phosphate, such as those found in some formulas, can lower calcium levels.

Signs and symptoms of hypocalcemia include irritability, muscle twitching, jitteriness, tremors, poor feeding, lethargy, and seizures. Treatment of underlying cause is required, as well as supplementation of calcium (Kenner & Lott, 2013; Polin & Spitzer, 2013).

Did You Know?

Total parenteral nutrition (TPN) is commonly used with neonates to meet fluid, electrolyte, and energy requirements. Components of TPN include admixtures of amino acids, glucose, and electrolytes, along with lipids. Although many times the use of TPN is an essential treatment, there are potential complications. Development of infections and sepsis can occur as a result of the TPN components and with the type of line that is used for administration. There is a particular risk for late-onset sepsis (Zingg, Tomaske, & Martin, 2012).

Long-term use of TPN can cause liver damage, cholestasis, and liver failure. These serious complications can occur due to multiple factors, although the exact etiology is unknown. Risk factors for developing liver complications from TPN include low birth weight, prematurity, duration of and composition of TPN therapy, sepsis, absence of enteral feeding, male gender, and perinatal depression or shock (Jolin-Dahel et al., 2011).

Test Yourself:

Premature infants have immature kidneys, which can affect:

Fluid balance and sodium excretion - Correct

Potassium excretion and fluid retention

Chloride metabolism and fluid excretion

Case Study

Baby Joe was born at 26 weeks gestation, with a birthweight of 827 g. He was delivered via caesarean section for fetal distress. He required initial resuscitation with a bag and mask, and then began spontaneous breathing. His APGAR scores were 2 at one minute, and 7 at five minutes.

Initial assessment in the NICU includes:

HR 132, RR 32, BP 42/24 (mean=28), oxygen saturation 87% on room air. Baby Joe has intercostal, subcostal, and substernal retractions, and respirations are shallow.

What might be anticipated during the first few hours in the NICU?

Immediate Management:

Baby Joe is within oxygen saturation limits at the moment, but his respirations are shallow, labored, and he is tachypneic. His gestational age and condition warrants administration of surfactant. He will also most likely require ventilatory support.

Placement of umbilical catheters, both venous and arterial, would also be anticipated for monitoring and administration of fluids.

At Risk:

Due to Baby Joe's gestational age and birthweight, what would he be at risk for?

Answer:

Baby Joe is at risk for developing apnea and bradycardia, IVH, PDA, TTNB and RDS, NEC, and fluid and electrolyte imbalances. He will need to be closely monitored for signs and symptoms of those complications. General management strategies include a neutral thermal environment, minimal handling, and reducing environmental stimulation. Parental support is crucial.

Conclusion

Neonatal patients are at risk for developing many complications in the NICU. It is important for the NICU nurse to be able to anticipate, identify, and manage these complications.

Resources

National Association of Neonatal Nurses (NANN): www.nann.org

> Professional organization. Provides information about neonatal nursing, including education, guidelines, and position statements.

American Academy of Pediatrics (AAP): www.aap.org

> Professional organization. Provides information about neonatal and pediatric care, including education, guidelines, and links to the Neonatal Resuscitation Program (NRP).

Agency for Healthcare Research and Quality (AHRQ): www.ahrq.gov

> A branch of the U.S. Department of Health and Human Services. Provides tools on evidence-based practice and quality of care.

National Guideline Clearinghouse: www.guideline.gov

> A branch of the U.S. Department of Health and Human Services and the AHRQ. Provides a search for clinical care guidelines.

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