Garth Hunt
Advanced Case Study
12/2/2012

Prematurity & Necrotizing Enterocolitis

Introduction:

Baby AW was a premature infant who was born at Intermountain Medical Center's (IMC) labor and delivery floor September 10, 2012. The patient was transferred to the newborn intensive care unit (NICU) for prematurity where multiple complications were experienced, including necrotizing enterocolitis (NEC). The following case study will discuss NEC in neonates, as well as the nutritional implications involved. The social and medical history, nutrition diagnosis, treatment, and progress while Baby AW was at IMC will also be discussed.

Patient Profile and Social History:

Baby AW was a premature infant born at 25 weeks and 3 days gestational age (GA). Since birth, the patient has been in the NICU at IMC hospital receiving treatment when concern for bowel loops occurred. The patient was transferred to Primary Children's Medical Center (PCMC) for further treatment of NEC and was transferred back to IMC. Family history was significant for obesity, hypothyroidism, depression, anxiety and attention deficit and hyperactivity disorder (ADHD).

Medical History:

Baby AW has an extensive medical history within the two months of life due to prematurity. Since September 10, 2012, the patient experienced various complications as a result of immature pulmonary function such as a left pneumothorax, pulmonary interstitial emphysema, and mild pulmonary hemorrhage. Baby AW also developed complications related to a large patent ductus arteriosus, thrombocytopenia, and sepsis. Poor feeding related to immaturity proved to complicate growth and required nutrition support to meet needs. The onset of bowel loops led to the development of NEC which required surgical resection and anastamosis of the colon.

Treatment and Progress

Baby AW presented to the NICU on September 10, 2012 post birth for required chest compressions and intubation because of underdeveloped lungs related to 25 week gestational age. Two days later, extubation was attempted and was not successful. Total parenteral nutrition (TPN) was started on September 12th per IMC protocol along with antibiotics. On day of life 4, Baby AW had improved pulmonary hypertension and resolved jaundice. On September 15, 2012 a left pneumothorax occurred in conjunction with a pulmonary hemorrhage. Baby AW was given trophic feeds of 9mls/kg/d, 6 kcals/kg/d, and 0.1g/kg/d through NJ tube. Trophic feeds were continued through September 25th until abdominal distention and bowel loops

were experienced. On October 6, 2012 Baby AW underwent PDA ligation and showed no signs of normal gastrointestinal function. On the 11th of October, Baby AW was transferred to PCMC where NEC was diagnosed and treated through a colon resection and anastamosis. While at PCMC, the patient received TPN and remained intubated. On October 21, 2012, enteral feeds via NJ tube were initiated and tolerance was assessed through the 24th when the TPN was discharged. Extubation was attempted again on November 4, 2012 and proved successful this time. Baby AW was transferred back to IMC's NICU where the patient was assessed by cardiology as a result of bradycardia. Baby Aw continued to tolerate full enteral feeds and infections have resolved.

Anthropometrics:

At birth, Baby AW was 29.5 centimeters (cm) in length, weighed 0.6 kilograms (kg), and had an occipital frontal circumference (OFC) that measured 21 cm. Per Fenton Growth Chart, Baby AW's weight has increased steadily following the curve at 3%. The patient's length has also increased consistently with minor plateaus at 28-29 weeks gestation and at 32-34 weeks gestation. The length remains below the 3% curve but continues to follow the curve. AW's OFC continued to increase each week and showed only minor plateaus. Although the OFC measurements were below the 3% curve, they continued to increase. All anthropometric measurements show that Baby AW was consistently gaining weight and achieving optimal growth.

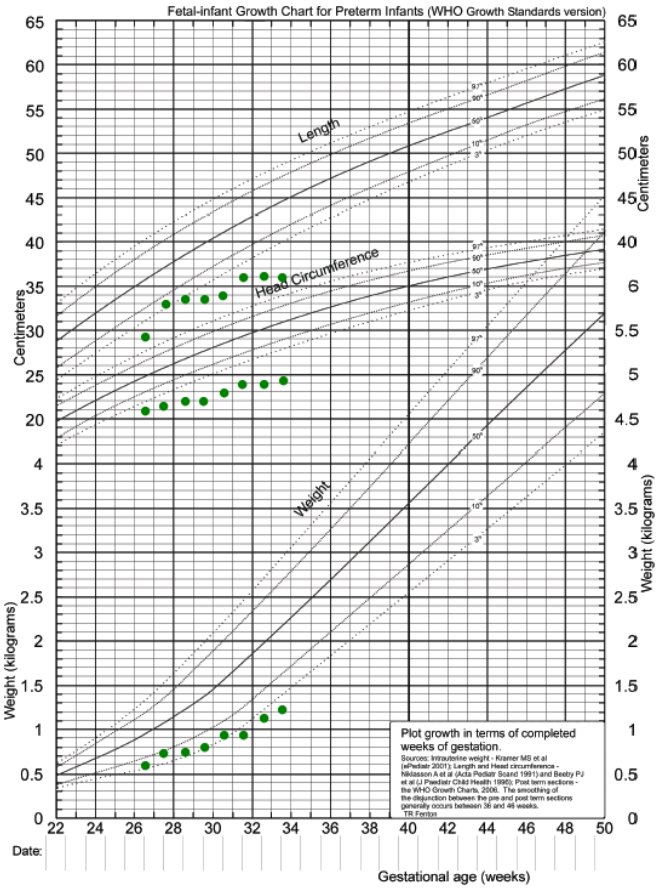

Figure 1 Fenton Growth Chart for weight, OFC, and length of Baby AW.

Baby AW had adequate weight gain throughout treatment at IMC. At birth, Baby AW weighed 600 g and gained 20 g within the first week making her 105% of her initial body weight (620 g/600 g = 1.03 x 100 = 103%) which is optimal to support development and linear growth. On average, IMC's recommended weight gain for premature infants is 15-20 g/kg/d. The patient's weight was assessed weekly to determine proper growth and weight gain. A weekly average weight gain was determined by using the following equation: [(current weight) – starting weight (g))/ current weight (kg)] / 7 days. This equation evaluates average weight gain per day and helps assess that adequate growth is achieved. The weights for each week of GA can be found in the table below.

During week 27, Baby AW had an optimal weight gain of 16g/kg/d (700g – 620g = 80g / 0.7 kg = 114.3g/kg / 7 days = 16g/kg/d). Average weight gain 7.7g/kg/d during week 28 was suboptimal (740g – 700g = 40g / 0.74kg = 54.4 g/kg / 7 days = 7.7g/kg/d). Optimal weight gain returned throughout weeks 29 and 30, of 14g/kg/d (820g – 740g = 80g / 0.82 kg = 97 g/kg / 7 days = 14g/kg/d) and 20 g/kg/d (950g – 820g = 130g / 0.95kg = 136.8 g/kg / 7 days = 20g/kg/d) respectively due to increased tolerance to enteral feeds via a NJ tube. During week 31, Baby AW developed NEC and showed no average weight gain. With nutrient feeds being met through TPN during weeks 31 and 32, optimal weight gain returned for an average of 25g/kg/d (1170g – 960g = 210g / 1.17kg = 179.5 g/kg / 7 days = 25g/kg/d). During week 33, Baby AW had an average weight gain of 11.5g/kg/d (1240g – 1140g = 100g / 1.24kg = 80.6 g/kg / 7 days = 11.5g/kg/d) on full enteral feeds.

Weight Gain According to Gestational Age (GA)							
GA	Date/Time	Weight (kg)	Length (cm)	GA	Date/Time	Weight (kg)	Length (cm)
33	11/07/12	1.24	36	31 cont.	10/24/12	0.98	-
	11/06/12	1.24	-		10/23/12	0.96	-
	11/05/12	1.2	36		10/22/12	0.92	36
32	11/04/12	1.17	-	30	10/21/12	0.93	-
	11/03/12	1.13	-		10/20/12	0.93	-
	11/02/12	1.15	-		10/19/12	0.92	-
	11/01/12	1.14	-		10/18/12	0.90	-
	10/31/12	1.17	-		10/17/12	0.91	-
	10/30/12	1.11	-		10/15/12	0.95	34
	10/29/12	1.18	-	29	10/8/12-10/14/12	0.82	-
31	10/28/12	1.18	36	28	10/1/12-10/7/12	0.77	33.5
	10/27/12	1.01	-	27	9/24/12-9/30/12	0.74	-
	10/26/12	1.04	-	26	9/17/12-9/23/12	0.66	33
	10/25/12	1.00	-	25	9/10/12-9/16/12	0.60	29.5

Medications:

While at IMC NICU, Baby AW has received multiple medications as treatment for the many complications that come with prematurity. The following table is a list of the patient's active medications along with common side effects.

Medication (dose)	**Use**	**Diet-related Side effects/restricitions**	**Signs/symp experienced by patient**
Hydrocortisone (0.9mg)	CORTICOSTEROID ANTI-INFLAMMATORY	↑appetite, esophagitis, N/V, dyspepsia, peptic ulcer, bloating, GI bleeding/perforation	bloating
Lorazepam (0.1mg)	ANTI-ANXIETY MUSCLE RELAX.	Anorexia, ↑appetite, ↑thirst, dry mouth, N/V, constipation, diarrhea.	constipation
Morphine (0.1mg)	ANALGESIC	Anorexia, dehydration, dry mouth, taste changes, dysphagia, dyspepsia, N/V, constipation, diarrhea, impaction, ↓gastric motility.	Impaction, constipation
Caffeine (6.5mg)	STIMULANT	N/V, GI distress, dyspepsia, ↑gastric acid, diarrhea,	None
TriViSol + FE	MULTIVITAMIN	None	None

Laboratory Data:

The following table is an average of Baby AW's laboratory history for each week of gestational age while receiving treatment at IMC and PCMC. Trends seen throughout treatment are represented.

Laboratory Data									
Test	Week 26	Week 27	Week 28	Week 29	Week 30	Week 31	Week 32	Week 33	Normal
Na	132L	144	140	143	135L	135L	138	134L	137-146 mmol/L
K	4.0L	4.2	3.9L	4.3	5.0	4.5	4.3	5.4H	4.1-5.3 mmol/L
Cl	109	110H	108	111H	99	99	105	101	98-109 mmol/L
CO2	26H	25H	22	24	28H	29H	22	28H	18-24 mmol/L
Glu	115H	184H	110H	77	86	82	93	100	60-108 mg/dL
BUN	20H	35H	25H	7	10	15	12	13	5-17 mg/dL
Crea	0.87H	0.69H	0.58H	0.37	0.34	0.46	0.32	0.27	0.14-0.52 mg/dL
WBC	26.1H	23H	19.7H	-	18.3	14.6	8.8	-	5-19.5 K/uL
RBC	4.4	4.3	3.9	-	4.2	3.52	4.11	-	3-5.4 M/uL
Hgb	9.4L	9.7L	9.7L	-	10.3	9.2L	12.0	-	10-18 g/dL
Hct	28L	30L	27L	-	38.4	31	36.8	-	31-55%
MCV	87.6	93.1	86.8	-	90.4	92.5	89.5	-	85-123 fl oz
MCH	30.4	30	29.2	-	29.3	29.0	29.2	-	28-40 pg
MCHC	35.4	33.6	34.3	-	32.3	33.5	32.6	-	32-36 g/dL

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Baby AW had weeks of hyponatremia, hypokalemia, and elevated chlorine levels throughout hospital stay related to use of TPN and saline solution for medicine administration. Electrolyte levels were monitored daily and adjusted through TPN and fluids to correct of suboptimal and elevated levels. Hyperkalemia was falsely elevated in week 33 of gestational age related to hemolysis during the biochemical analysis. Carbon dioxide levels were elevated during weeks 26-27, 30-31, and 32 related to respiratory distress and not being able to breathe efficiently without support. Glucose levels were initially elevated r/t to stress, hydrocortisone medication, and use of TPN for nutrition support. They have returned within normal limits as TPN was adjusted. BUN and creatinine levels were elevated between the weeks of 26-28 related to renal insufficiency as a result of prematurity. As gestational age increased, renal efficiency increased and labs returned within normal limits. White blood cell count remained elevated through week 28 due to sepsis and lung infections but has since decreased as infections have resolved. Hgb and Hct were suboptimal during weeks 26-28 due to multiple blood draws for analysis. Pack red blood cell (PRBC) transfusions were administered throughout these weeks to help elevate suboptimal levels.

Clinical Evaluation:

Baby AW had jaundice at birth, which is common in most infants of prematurity due hyperbilirubinemia. With phototherapy, jaundice resolved within the first week of life. The patient's prematurity can decrease the ability to latch onto the breast and produce a suck. The patient showed no signs of permanent physical handicaps at this time, but it is too early to determine this at this time. Baby AW has difficulty swallowing due to prematurity but the ability is expected to develop as gestational age increases.

Dietary:

Initially, Baby AW was placed on TPN due to prematurity and received trophic feeds of breast milk (BrM) 20 kcals/oz. Because the patient was receiving trophic feeds, nutrient needs were calculated for neonates in transition to tube feeds. According to IMC protocol for prematurity, total fluid goals were 120-200 mL/kg, kcal requirements were 90-120 kcals/kg, and protein needs were estimated between 3-4 g/kg. As Baby AW received full enteral feeds, fluid needs were adjusted to 150-200 mL/kg, kcals to 110-130 kcals/kg, and protein to 3-4 g/kg. During week 28 when bowel loops were diagnosed, Baby AW was switched back

	Kcals (kcals/kg)	**Protein** (g/kg)	**Fluids** (mL/kg)
TPN	90-95	3-4	120-150
TPN to Tube Feed	90-120	3-4	120-150
Tube Feed	110-130	3-4	150-200
Term Infants	108	2.2	140-160

TPN until normal bowel function was achieved post surgery. Baby AW quickly transitioned back to receive full enteral feeds with optimal tolerance.

In week 26, Baby AW tolerated TPN better than enteral feeds because of prematurity. BrM 20 kcals/oz was introduced through NG tube as trophic feeds which accounts for the low kcals/kg intake average in the table below. Baby AW increased tolerance to enteral feeds during week 27 and 28 and TPN was discontinued in week 29 due to tolerance to full enteral feeds. Feeds were not charted during weeks 29-30, but it is assumed that feeds continued to be tolerated until abdominal distention and bowel loops were diagnosed in the middle of 29. Baby AW was transferred to PCMC for surgical treatment that same week and was placed on TPN until full function of intestines was achieved and enteral feeds were tolerated in week 33. No breast feeds or bottle feeds have been attempted due to prematurity and the inability to suck and swallow at this time.

Average Weekly Intake							
Week of GA	BrM or Formula (kcal/oz)	Avg Fluid (mL/kg)	Avg Kcals (kcal/kg)	Avg Protein (g/kg)	TPN? (%, Pro/Fat)	Tolerance to diet	Adequate to needs?
26	BrM 20	135	47	2.8	TPN 10%, 3g/3g	GOOD	YES
27	BrM 20	139	76	3.5	TPN 10%, 3g/3g	GOOD	YES
28	BrM 20	143	88	3.0	TPN 10%, 3g/3.5g	GOOD	YES
32	-		110	2.9	TPN 10%	GOOD	YES
33	BrM 22	135	105	3.3	D/C	GOOD	YES

Nutrition Screening:

MM was assessed at a high nutrition risk based on the nutritional standards at IMC due to symptoms of prematurity, NEC, and the inability to suck and swallow at this time.

Nutrition Note:

Nutrition Assessment(s) and Follow-Up Note(s) based on IMC format.

Assessment:

Baby AW is now 33 5/7 weeks adjusted gestational age and day of life 58. Weight is 1240g with suboptimal average weight gain of 8.1g/kg/d over the past 7 days. Average intake over the last 7 days have been 105 kcals/kg and 3.3g/kg protein. Per Fenton growth chart, weight continues trending downward <3%ile, OFC following the curve <3%ile, and length also with downward trend <3%ile. Baby AW is now working back up on feedings of breastmilk 22 kcal/oz fortified with HMF. She remains on TPN for nutrition support while working up on

feedings with total fluids at 135mL/kg/d. TPN is concentrated at 80 mL/kg/d to improve protein, vitamin, and mineral intake. Feedings are currently given on a pump over 60 minutes due to concerns for reflux. Labs significant for low sodium and low phosphorous levels. Calcium and phosphorous have been increased in her TPN to optimize bone mineralization.

Diagnosis:

Inadequate oral intake related to prematurity as evidenced by total nutrition needs provided via IV nutrition and tube feeds at this time.

Interventions:

TPN: D15%, AA3.5 g/kg/d, IL 2g/kg/d at 80 mL/kg this provides: 70 kcals/kg
BrM 22kcal/oz fortified with HMF at 80 mL/kg/d. This provides 58 kcals/kg/d and 1.3 g/kg/d protein.

Monitor/Evaluation:

Meet the protein and micronutrient needs of a very low birth weight infant
Provide adequate nutrition for growth and weight gain of 15-20 g/kg/d

Patient Follow-Up

No ADIME Follow-Up note was written for Baby AW. Continued tolerance was seen with full enteral feeds when they were restarted after NEC was treated. Weight gain was seen and breast milk was fortified to provide additional nutrients for optimal weight gain and growth. Prognosis is good due to tolerance to fortified feeds via NJ tube and continued daily weight gain. Baby AW will benefit from further fortified feeds adjusted for growth and transition to breastfeeding and fortified bottle feeds.

Necrotizing Enterocolitis

Necrotizing Enterocolitis

Necrotizing Enterocolitis (NEC) is an infection found in more than 85% of all premature infants and is the leading cause of death in infants each year (1, 2). Prematurity is often seen as the most common risk factor for NEC because of the immaturity and sterile environment of the gastrointestinal system (2). For this reason, breast milk has been found to be the best source of nutrients for preterm infants and the best defense against bacteria and other infection causing

pathogens (1). This paper will discuss the risk factors, etiology, and signs and symptoms of NEC along with the importance of breast milk for the prevention and/or treatment of NEC.

Necrotizing Enterocolitis continues to be found in premature infants than term infants because of their low body weight and gestational age at birth. Researchers reported that 45% of all neonates with NEC weighed less than 1000g and were born before 34 weeks gestational age (2). Infants born early and with low body weight required more ventilation, medicine, and medical support to help sustain life leaving them more susceptible to infection and death (3). Since the gastrointestinal system is sterile at birth and begins maturing at the end of the third trimester, premature infants are born with a sterile and immature gastrointestinal immune system that lacks any micro flora to help fight the variety of bacteria that they will be exposed to while receiving treatment in the NICU (4).

Infants with immature gastrointestinal and immune systems are at high risk for bacterial exposure and infections (1). Many complications during pregnancy and post pregnancy can occur putting the infant at risk for developing NEC. Some of these complications include but not limited to amnionitis, membrane ruptures, birth asphyxia, small gestational age, very low birth weight, congenital heart disease, and exchange transfusions (1). Each one of these risk factors are different from one another, and supports the hypothesis that the cause of NEC comes from a variety of risk factors that leads to sepsis, vital organ damage, and ultimately death if left untreated (1, 5).

Researchers have had a hard time identifying the exact cause of NEC and decided that it is a result of three main pathways (4). First, the intestinal lining becomes damaged as a result of

poor intestinal motility, digestive ability, circulating regulation, barrier function, immune defense, and abnormal colonization of bacteria (1, 2). Second, bacteria infiltration and colonization of the intestinal lumen occur due to the lack of appropriate micro flora and immune response (1). And third, enteral feeds provide food for the colonized bacteria aiding their proliferation and colonization on the intestinal epithelial cells (1). Each one of these will be discussed in detail and how it contributes to the development of NEC.

The first main pathway is a multi-factoral one that includes many different physiological and structural problems commonly found in underdeveloped neonates. The processes of motility, digestion, circulation, structural and immune defense, and abnormal colonization all contribute to the damage that occurs in preterm infants during the development of NEC (2). For example, intestinal motility does not begin until after 34 weeks gestational age due to immaturity (2). Although feeding has shown to help intestinal maturity, prevention isn't guaranteed, especially with other complications involving poor blood circulation to the intestines or an underdeveloped line of defense, or mucosal layer, on the intestinal lining (2). With the immature structures and physiological processes of the gastrointestinal system, bacteria, pathogens and toxins can come in contact with the unprotected intestinal wall initiating inflammatory pathways creating intestinal damage (2).

In the second pathway, research suggests that bacteria infiltration and colonization of the intestinal lumen occur due to the lack of appropriate micro flora and a decreased immune response (2). In utero, the gut is sterile and requires bacteria from the mother and the environment to colonize appropriate intestinal flora (2). Premature infants are at a

disadvantage when it comes to this because of increased exposure to bacteria as a result of medical tests, tubes, and frequent use of antibiotics in the NICU and delayed enteral feeding shortly after birth (2, 5). Gastric acid production, protective mucus on epithelial cells, and the ability to breakdown toxins and bacteria present in the gastrointestinal tract is already decreased due to prematurity and this gives bacteria the advantage to adhere to the intestinal mucosa leading to a high risk of infections (3). Because of the environment, optimal pH, and decreased intestinal motility pathogenic bacteria is able to grow and develop (4).

Another factor important in the development of NEC is the introduction of enteral feeds comprising the third pathway in the multi-factoral cause of NEC. At birth, premature infants are unable to eat and therefore typically receive total parenteral nutrition (TPN) within the first days of life to allow for the gut to mature (4). During this time, treatments in the NICU are given which expose the infant to a variety of bacteria and give it the opportunity to colonize within the gut in place of the appropriate micro flora (2, 4). When gut maturation and motility have strengthened to the point of being able to handle enteral feeds without causing harm, abnormal micro flora are able to use the food as an energy source and multiply within the intestinal wall resulting in damage and sepsis (1).

Necrosis of the intestinal wall, as a result of sepsis, is one of the major symptoms of NEC (1). In fact, 33% of all infants with NEC develop sepsis due to abnormal colonization in the intestines (1). Most cases of sepsis result in the perforation of the terminal ileum and have a 20%-30% chance of death if not caught in time (1). In addition to sepsis, neonates often present to hospitals with other complications that help healthcare professionals identify the presence

of NEC. Some of the most common signs and symptoms include ileus, abdominal distention, bilious gastric residuals, hematochezia, lethargy, variance of body temperature, apnea, and metabolic acidosis (1). By recognizing these symptoms, proper treatment can be given to help limit the development of sepsis and lower the chance of death (1).

Proper medical treatment in the NICU involves both non-surgical and surgical techniques. Non surgical therapies are used most often and are considered sufficient for at least 75% of all patients with NEC (1). At the first appearance of symptoms, common practice is to discontinue all enteral feedings and provide bowel rest and recovery for intestinal injury (1, 2). Infants receive nutritional supplementation from TPN for approximately 14-21 days to help meet nutrient requirements and aid in growth while dealing with complications from NEC (1). Antibiotics are initiated to help remove abnormal bacterial colonies and other sources of infections. X-rays, blood draws, platelet counts, and blood gases are common biochemical tests used to assess the severity and progress of NEC and determine further treatment options (1). According to Guthrie et al, 25% of the time non-surgical methods do not work as a result of sepsis and peritonitis from a perforated ileum (1). Surgical resection, ostomy, and reanastamosis interventions are used to help remove damaged sections of the intestine and eventually restore proper bowel function after sepsis and peritonitis have subsided (1). Enteral feeds using breast milk are highly suggested once bowel function is normalized to help treat and further prevent NEC due its protective qualities (1, 2, 4).

According to Lin et al, breast milk plays an important role in the development, maturity, and defense of the intestines of preterm infants compared to formula fed infants (2). Due to

the sterility of the gut at birth, neonates have no micro flora present in the intestinal tract and are more susceptible to pathogenic colonization. When neonates begin feeds with breast milk, they ingest organisms such as bifidobacterium, lactobacillus, and streptococcus that establish proper micro flora in the gut, reducing the possibility of a pathogenic colonization (4, 6). Because of this, premature infants who solely rely upon breast milk instead of formula have a lower incidence of NEC (4).

Breast milk contains other contents that help improve gastrointestinal function and reduce the development of and support the treatment of NEC. Amino acids (glutamine and arginine), erythropoietin, immunoglobulin, and epidermal growth factors are such contents that mature epithelial cells in turn reducing the risk of bacterial translocation through low adherence to the intestinal wall (2, 4). The amino acids glutamine and arginine are responsible for the integrity of the intestinal lining while also promoting cell growth along with epidermal growth factors (2). Erythropoietin plays a role in the intestinal development, cell migration and intestinal restitution. Current formulas have been fortified with the amino acids, erythropoietin, immunoglobulin, and epidermal growth factors to help prevent the onset of NEC for those infants who are unable to receive breast milk feedings for whatever reason (2). Although breast milk and formula have been accepted by the medical community as methods to feeding infants, breast is the best option for its effects it has on improving intestinal motility, maturity, defense, and normal bacterial colonization (2, 4).

Some premature infants are unable to receive anything by mouth or enterally and do not benefit from the added protection and maturation the breast milk offers to infants. Those

who are a few days post partum or recently diagnosed with NEC rely on TPN to receive adequate nutrition for growth and development (7). Lin et al, states that prolonged treatment where the infant is receiving nutrition orally or enterally with an immature intestinal system worsens intestinal function and causes gut atrophy and increased inflammatory pathways (2). The result is weakened epithelial cells in the intestine and welcoming abnormal colonization. For this reason, daily trophic feeds are recommended daily to increase digestive enzyme activity, digestive hormone release, intestinal blood flow and motility, feeding tolerance, growth, and a decreased development of sepsis and reduced length of hospital stay (2). Trophic feeds should continue until TPN is discontinued and the infant can tolerate full enteral feeds (2).

When full feeds are tolerated and proper intestinal motility has begun, it is important to adjust feeding to help maintain proper growth and development. No feeding protocols exist within the medical community as a gold standard and are specific to each facility. Intermountain Medical Center's enteral recommendations for premature infants suggest 110-130 kcals/kilogram, 3-4 grams/kilogram of protein, and 150-200 milliliters/kilogram of total fluids to help meet the infant's needs and promote adequate weight gain. When on TPN, infants should meet a weekly goal of 90-95 kcals/kilogram, 3-4 grams/kilogram of protein, and 120-150 milliliters/kilogram of total fluid. According to Groh-Wargo et al, enteral nutrition is the preferred method but alimentation is dependent upon the development of the gastrointestinal system, oxygen levels, and other morbidities (7). Transitions to human milk from TPN should be slow and frequent assessments should be performed to analyze tolerance (7). If inadequate

weight loss is seen, liquid fortifiers and infant formulas can be added to breast milk to help increase the calorie, protein, and mineral content for the growing neonate.

Human milk is highly suggested for feeds in preterm infants due to its high digestibility, immune enhancing effects, and nutrient content (7). It is vital for the preterm infant to receive breast milk shortly after birth to help increase intestinal motility, defense, and overall health. This in turn will help reduce the incidence of NEC in premature infants. Because NEC is one of the leading causes of death in infants around the world, it is important to understand the risk factors, signs and symptoms to be able to either prevent or receive proper treatment as soon as possible. This will help decrease the mortality rate and provide fuller and richer lives to infants and families around the world.

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