

NUTRITIONAL STATUS OF PRETERM INFANTS AT DISCHARGE: A STUDY AT TEHRAN VALIE-E-ASR HOSPITAL

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ABSTRACT

Background: Nutrients meeting recommended dietary intakes take time to establish and once established are rarely maintained throughout hospital stay in preterm infants.

Methods: Our purpose is to define the nutritional status and growth parameters of hospitalized patients at discharge in our hospital. Data were obtained concerning all 50 infants born weighing <1800 g and <34 weeks of gestation who survived until at least 21 days of age.

At weekly intervals, intakes of fluid, energy, and protein from all sources were determined and body weight, head circumference and length were recorded.

Results: 50 premature neonates with gestational age of 30.5 ± 2.35 weeks and birth weight of 1337 ± 232 g comprising 26 (52%) females and 24 (48%) males were entered into this study from April to July 2004.

The mean time of caloric intake of 120Kcal /kg/d was 18.41 ± 6.7 days of age. 37.8% of mothers provide breast milk, 51% preterm formula and the rest of the patients consumed both. Biochemical markers of nutritional status showed that 64% of neonates had blood urea nitrogen <5 mg/dL, 70% had alkaline phosphatase >450 IU/L, 73% had serum phosphorus <4.5 mg/dL, and 49% had serum albumin <3 g/dL at time of discharge. Except for serum albumin, comparisons of biochemical parameters between small for gestational age & appropriate for gestational age infants were not significant at discharge time.

Comparisons of weight and head circumference at birth and at discharge time showed that a significant number of neonates became small for gestational age at discharge ($p < 0.05$).

Conclusion: Availability of appropriate amino acid solutions for neonates, parenteral phosphorus, trace elements and human fortifier could reduce these problems.

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INTRODUCTION

Nutritional assessment is a continuous process for premature infants in neonatal units and growth compared with standard norms and growth rate remain the cornerstone for evaluation of nutritional adequacy.^{1,2}

Premature infants are at risk for nutrient deficiencies and toxicities for many reasons and because of these fac-

tors, the premature infant's nutrient needs are greater than those experienced by the healthy term neonate.¹

It is clear that the most prematurely born infants, even if appropriate for gestational age (AGA) at birth, become small for gestational age (SGA) soon after birth and remain SGA at discharge. The consequences of inadequate early nutrition with respect to growth and development of the central nervous system is of particular concern.

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Table I. Mean amount of caloric & protein intake during the first weeks of life.

Weeks	Caloric intake Kcal/kg/day	Protein intake g/kg/day
First	48.66 ± 7.65	0.91 ± 0.4
Second	52.94 ± 23.8	1.55 ± 0.72

Table II. Mean weight gain (g/d).

Weeks	Weight gain
First	-13.12 ± 6.8
Second	8.24 ± 4.43
Third	10.78 ± 503

This concern stems, in part, from data demonstrating that malnutrition during a critical period of central nervous system development, if not corrected during the critical period, results in irrecoupable deficits. The high incidence of subtle developmental and behavioral problems, poor attention, and specific learning problems observed in low birth weight infants during childhood and early adolescence may reflect inadequate nutrition during the finite critical period of a specific neurodevelopmental event.³

David Barker and his colleagues published a series of retrospective epidemiological articles that suggested that fetal malnutrition leads to a series of endocrine, metabolic, and physiological adaptations that allow the fetus to survive an adverse in utero nutritional environment.^{4,5} However, these changes may become programmed, and can be maladaptive in later life, when, for example, nutrients are more available. These maladaptive responses may increase the risk of insulin resistance, hypertension and cardiovascular disease. SGA infants clearly have abnormalities in the growth hormone/IGF/insulin pathways and in leptin metabolism. There is now good evidence that low birth weight; small head circumference, low ponderal index, or decreased lengths at birth (all suggestive of fetal malnutrition) are associated with increasing risk of cardiovascular disease in later life. These data present a dilemma to the neonatologist.⁶ The aim of nutrition in preterm infants is to re-establish as soon as possible and then to maintain growth rate which is qualitatively and quantitatively similar to the fetus at the same gestational age without inducing significant metabolic stress.⁷ These data highlight the need for better nutritional management of low birth weight infants.

To define the nutritional status and growth parameters of hospitalized patients at discharge, we tried to detect and correct the possible etiologies involving the problems in our hospital.

MATERIAL AND METHODS

This observational study was conducted on 50 infants from April to July 2004.

Preterm infants with gestational age of <34 weeks and a birth weight of 1800 g were considered eligible. Only those surviving beyond 21 days of age were enrolled in this study. Gestational age was assessed using Ballard

score, considering maternal dates and fetal ultrasound. Body weight was measured using standard unit scales. Growth weight (daily), head circumference and length (weekly), type of diseases, amount and type of parenteral nutrition and energy in kcal/kg (daily) besides biochemical nutritional parameters such as serum phosphorus, calcium, alkaline phosphatase, and blood urea nitrogen (BUN) were measured at discharge time.

All infants were fed according to a standard protocol that was uniformly applied. The aims of this protocol are to establish an energy intake of >50 kcal/kg/day on day 1, commence total parenteral nutrition (TPN; 10-12.5% dextrose. 0.5 g protein/kg) on day 1, and intravenous lipids (0.5-3 g/kg) and enteral feeds on day 3 when permitted. Recommended protein intakes from all sources up to 3.0 g/kg/day were considered acceptable. Enteral feeds with human or a term infant formula (20cal/oz, 2.0 g protein/100 kcal) were started and increased at a rate of 20mL/kg/day. Infants were fed by continuous infusion until ~ 32-33 weeks` corrected age, at which point bolus feeds were introduced. Intake data were collected on a daily basis by a single observer.

An enteral intake of 120 kcal/kg/ day was assumed to be adequate. The data were analyzed using Chi-square and t-test. Results were considered significant at $p < 0.05$.

RESULTS

Gestational age (GA) and birth weight (BW) of studied infants were 30.5 ± 2.35 and 1337 ± 232 respectively, comprising 26 (52%) female and 24 (48%) male.

The mean duration of assisted ventilation and being NPO were 4.5 ± 4 (1-15 days) and 6 ± 4.4 days respectively. 17 patients had been resuscitated at birth and a 5 minute Apgar score < 6 was detected in 3 patients.

37.8% of mothers provided breast milk, 51% preterm formula and the rest of the patients consumed both. The mean amount of protein, caloric intake and weight gain during the first weeks of life are illustrated in Tables I, II.

For 35 neonates, lipid infusion was started, out of whom 20 were SGA at discharge time. The mean amount of lipid infusion between SGA (0.85 ± 0.5 g/kg) and AGA (1.2 ± 2 g/kg) infants was not significant ($p > 0.05$).

Comparisons of weight (W) and head circumference (HC) of < 10% of standard norms at birth and at the discharge time were significant ($p < 0.05$), (Table III). The mean time of caloric intake of 120Kcal /kg/d was 18.41 ± 6.7d; 6.5% of patients tolerated this amount until 10 days, 69.5% until 20 days and 97.8% until 30 days of their life.

64% of neonates had BUN <5 mg/dL, 70% had alkaline phosphatase >450 IU/L, 73% had serum phosphorus (P) <4.5 mg/dL, and 49% had serum albumin (Alb) <3g/dL at the end of the second week of their life. Except for serum albumin comparisons, biochemical parameters between SGA & AGA infants did not vary significantly at discharge time (Table IV).

12 exclusively breast fed neonates had BUN <5 mg/dL and 10 out of 17 of these infants were SGA at discharge time.

Table III. Comparison of weight and head circumference according to percentile curve at birth and at discharge.

	Weight				Head circumference			
	Birth	7d	14d	Discharge	Birth	7d	14d	Discharge
<10%	11	16	16	28	6	7	10	20
>10%	39	34	34	22	44	43	40	30
Total	50	50	50	50	50	50	50	50

DISCUSSION

The results of this simple study are important. Data from this study suggest that a more realistic picture can be obtained by expressing the data.

Our study like other available data suggests that as a population, preterm infants are in a state of sub-optimal nutrition at the time of discharge from hospital and beyond. It is likely that improving this situation would be beneficial both in the short-term, and also potentially for longer-term health and development.⁸ Under-nutrition is due to delayed commencement of TPN and intralipids, intolerance to parenteral glucose and lipids, initial small enteral feeding volumes, withholding of feeds for feeding intolerance, and the use of unfortified breast milk. This situation is of importance in infants weighing < 1000 g at birth where 80-90% of infants are SGA at hospital discharge.⁷ In one study 22.4% of VLBW became SGA at discharge,⁹ compared with 30.4% in our study where early TPN was limited due to glucose and lipid intolerance and concerns regarding aminoacid metabolism. Although we tried to introduce enteral feeding early, full enteral feeds were not established by day 20 in 30% of our infants. None of the infants developed necrotizing enterocolitis.

Besides nutrient intake and growth parameters, biochemical indices were considered to optimize nutrition management. It is recommended that the growth and nutritional status of preterm infants born at less than 34 weeks' gestation or 1800 g birth weight be assessed clinically and biochemically before hospital discharge. Serum phosphorus <4.5 mg/dL, alkaline phosphatase >450 IU/L, and blood urea nitrogen <5 mg/dL should be considered early indicators of malnutrition. Serum phosphorus concentra-

tions are the best indicator of calcium and phosphorus status in infants.² Low serum phosphorus concentrations presumed osteopenia and may be associated with rickets.¹⁰

About seventy percent of our patients had low serum phosphorus and high alkaline phosphatase concentrations and with respect to non-availability of parenteral phosphorus that should be used early in TPN solutions, it is expected that osteopenia of prematurity occurs more in our patients.

Serum proteins are often monitored to determine the adequacy of protein and energy intake. From birth to 3 months of age, premature infants have lower serum albumin, prealbumin, retinol binding protein, and ceruloplasmin levels than term infants. Serum albumin has a longer half life than serum prealbumin so serum concentrations reflect different time periods and nutritional intakes. Some investigators identified a strong correlation between serum and urinary urea and protein intake and growth.¹ 64% and 50% of our patients had low BUN and serum albumin respectively.

Our study showed that serum albumin concentration is a good indicator of protein status especially in SGA infants. With respect to unavailability of trophamine as an appropriate amino acid for neonates and the existing concern for using aminoplasma which has a high concentration of some toxic aminoacids we maximally used 1.5 g/kg/day of this aminoacid for our neonates.

37% of our mothers provided breast milk as the sole source of feeding. Approximately 50% of solely breastfed infants with mean birth weight of less than 1800 g have been reported to have hypophosphatemia 4-8 weeks after initial hospital discharge although these infants received fortified human milk during hospitalization and had normal phosphorus values at discharge.² Carlson and Ziegler examining growth in preterm infants noted that poorer gain was more marked in infants fed fortified human milk than preterm formula.¹² With respect to high cost of human milk fortifier most of our patients did not receive fortified breast milk that could have worsened their nutritional problems.

Nutritional requirements and intake vary depending on patient population; the smaller the infant, the more complicated the perinatal course, the greater the variation in requirements and intake. Feeding practices also vary further thus the nature and amount of the nutritional deficit will differ between infants and nurseries and what is rate-limiting in one situation may not be rate-limiting in another.

In this study poorer growth was primarily related to inadequate energy, protein and phosphorus and probably other trace element intake.

Table IV. Comparison of biochemical parameters, predisposing diseases and sex of infants between SGA and AGA infants at discharge time.

	SGA	AGA	Total	P-value
Anemia	2	2	4	NS****
Low P	15	21	36	NS
Low Alk-p	15	20	35	NS
Low Alb.	14	10	24	0.01
Low BUN	11	21	32	NS
Male	13	13	26	NS
IVH*	15	9	24	NS
PDA**	13	13	26	NS
Assisted				
Vent***	6	5	11	NS

IVH* = Intraventricular hemorrhage, PDA**= Patent ductus arteriosus, Assisted vent***=Assisted ventilation, NS****=Not significant

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Undernutrition in this study is due to intolerance of parental glucose and lipids, initial small enteral feeding volumes, withholding of feeds for feeding intolerance, and the use of unfortified breast milk. Availability of trophamine, parenteral phosphorus, trace elements and human fortifier could reduce these problems. More aggressive enteral feeding might also have reduced the deficit.

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