

BONE MINERAL CONTENT (BMC) IN CYSTIC FIBROTIC PATIENTS WITH NORMAL VITAMIN D STATUS.

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Poor mineralization in CF patients has been reported in vivo since last decade. A group of 7 well controlled CF patients (aged 8.1±2.5 y) with normal faeces was studied. They received Creon (5-11 capsules/day) and vitamin D (400-2400 IU/day). The Schwachman test ranged 55-90. An age matched control group of 7 healthy children was also studied. BMC, by dual-photon absorptiometry (153 Gd) of trabecular bone in lumbar vertebrae was used. Standard deviation score for height were less than 1 SD in both groups.

	Ca	Alk-P	25D	24,25D	1,25	BMC/BW
	mg/dL	u/L	ng/mL	ng/mL	pg/mL	g/cm ²
CF	9.6±.5	168±48	15.3±4.4	1.9±.8	243±41	.55±.04
Control	9.5±.4	220±117	18.8±3.2	1.8±.4	74±45	.71±.1

**p<0.001; *p<0.025; m±SD

Vit D nutritional status is in the low normal range and reasonable for winter. Using bone width (BW) concept, bone mineral content shows a significant reduction in CF group, similar to osteoporotic adults. This can be due to calcium malabsorption. The significant rise in 1,25 D reflects the body's calcium needs. Extra calcium and/or vit D supplements are probably required for those patients.

MINERAL CONTENT OF THE HUMAN SKULL DURING DEVELOPMENT.

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Measurements of mineral accumulation in infancy are usually referable to cartilaginous bones and yet the membranous skullbones of preterm infants are palpably soft and craniotabes is well recognised in preterm and fullterm infants. Hardening of the vault and increasing radiological density occur with growth and maturation. Calcium and magnesium contents were measured in post-mortem samples of frontal bone from 108 children aged from 20 weeks gestation to 10 months of life. Samples of bone were dried, ashed and the mineral content determined by atomic absorption spectrophotometry. A small progressive increase in calcium/100 g tissue was seen in stillbirths and early neonatal deaths in the second half of gestation, 21.7g/100 g at 28 weeks to 22.6 g/100 g bone at 40 weeks (r = 0.23, p < 0.02), but there was little subsequent change over the first year of life. The calcium content of many infants dying more than 7 days after birth was reduced. Calcium and magnesium contents were strongly correlated (r = 0.34, p < 0.001) over the whole range of gestations studied. (Magnesium content at 40 weeks gestation = 0.307 ± 0.030 (SD), n=19). Our results are similar to previous data reported on cartilaginous long bones (femur and humerus) and suggest that mineral accretion in cartilaginous and membranous bones is similar in early development.

Dual energy densitometry (DXD) measurement of bone mineral content (EMC) in preterm babies

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DXD was used to measure EMC in preterm infants < 30 weeks gestational age (GA) fed formula milk complying with recommended guidelines for calcium (Ca) and phosphorus (P) content and ratio. DXD images were obtained at 48 hours of age and at weekly or 2 weekly intervals until term. Results from 9 infants are available. Mean (± SD) EMC at birth was 2.7 (± 1.5) mg/mm and at 6 weeks postnatal age (PNA) was 2.0 (± 0.5) mg/mm. 5 babies studied at term had a mean (± SD) EMC of 3.7 (± 1.1) mg/mm. The expected EMC of newborn infants of this GA range is 3.2 (± 0.6) mg/mm and of newborn infants at term is 9.2 (± 1.8) mg/mm¹. One infant developed a fracture of the femur. His EMC fell from 2.3 mg/mm to 0.8 mg/mm in the first week of life and was 0.2 mg/mm at 8 weeks PNA. The fracture was detected at 14 weeks PNA at which time the EMC was 3.3 mg/mm.

We conclude that EMC did not increase in these babies as it does in utero and at term was well below the expected value. Risk of fracture may be predicted from very low EMC. Supplementation of formula milk with Ca and P may not be sufficient to improve mineralisation and other factors must be investigated.

¹Greer et al 1983, Ped Res 17: 259-262.

HYDROGEN ION [H]⁺ LOAD AND CALCIUM-PHOSPHATE SOLUBILITY IN IV NUTRITION AND THE PRETERM INFANT (PTI)

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Significant osteopenia occurs in circa 30% of PTI. Inadequate mineral substrate intake to match intrauterine accretion rates is thought to be a major cause. Calcium (Ca) and Phosphate (iP) intake in IV feeding is restricted by solution solubility to less than half the desired intake. 17 PTI (median wt 1.20kg, gest 28w) were entered into a cross-over study comparing Addiphos (KabiVitrum) and K₂HPO₄ as the iP source, while keeping amino acid intake standard. Results: (i) An increase in Ca - iP solubility and increased Ca and iP intake (P<0.001) can be achieved by lowering solution pH using Addiphos instead of K₂HPO₄. (ii) Plasma Ca and iP is significantly increased with Addiphos (P<0.01). (iii) To match intrauterine Ca and iP accretion there was a 2x increase in [H]⁺ load. (iv) With (iii) there was no significant change in blood acid-base parameters, but there was an increase in urine titratable acidity excretion. Conclusion: Intravenous Ca and iP concentration of infusion can be increased by lowering pH of parenteral solution using Addiphos. This increased [H]⁺ load is well tolerated by PTI.

MAGNESIUM METABOLISM IN PRETERM INFANTS: THE EFFECT OF CALCIUM AND PHOSPHORUS.

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Studies on rickets of prematurity have focussed on the calcium, phosphorus and vitamin D requirements of infants, but skeletal development may be stressed if magnesium is deficient. The present study examines the effects of increasing calcium and phosphorus on magnesium retention. Five groups of very low birthweight infants were fed milk with magnesium content 5 mg/100 ml. Calcium and phosphorus were supplemented to the following concentrations (Ca:P mg/100 ml); Group A 44:33, Group B 84:33, Group C 125:33, Group D 125:50 and Group E 125:64. Three-day balance studies were performed starting at 10, 20, 30 and 40 days. Increasing both calcium and phosphorus contents decreased magnesium retention to lower than in utero accretion rates. Doubling magnesium content of milk to 10 mg/100 ml did not prevent negative magnesium balance in very preterm infants at 10 days, although magnesium retention doubled in older (20 days) or more mature (32-34 weeks gestation) infants. We conclude that increasing calcium and phosphorus content of milks to prevent rickets of prematurity could produce a magnesium deficiency in very low birthweight infants, with possible compromise of bone formation.

EFFECT OF SUPPLEMENTAL PHOSPHORUS ON RED CELL PHOSPHATE IN PRETERM AND FULL TERM NEWBORNS (Preliminary report).

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Phosphate changes in plasma and erythrocytes were studied in preterm and fullterm infants during the first two weeks of life. Group I : three preterm infants with no problems, mean gestational age (GA) 33.5±1.5(±SD) weeks and mean birth weight (B.W) 2010±250gm (±SD), group II : six preterm infants with problems (3 respiratory distress syndrome, 2 septicemia, 1 necrotizing enterocolitis) mean GA 34±1.7 weeks, mean B.W 2260±490 gm; group III : four full term infants with problems (2 with birth asphyxia and 2 with meconium aspiration) with mean BW 3550±490gm. Blood samples were drawn on 1st, 4th, 7th 10th and 14th day of life. All babies with problems received glucose 10% and calcium gluconate 10% (40mg/Kg/day) for the first 48 hours of life and TPW or infant formula thereafter. The mean phosphate intake in mg/Kg/day from day 4 to day 14 ranged in group I from 49±9 to 60±17 (mean±SD) in group II from 44±20 to 63±4.1 and in group III from 24±14 to 41±5. Plasma and red cell (RBC) Pi in both groups of preterms showed a steady increase during the study. There was no difference between normal and problematic preterm infants. In the full term newborns with problems Pi remained unchanged extra and intracellularly.

Days	1st			4th			14th		
	RBC Pi	Plasma Pi	RBC Pi	Plasma Pi	RBC Pi	Plasma Pi			
Group I	1.5±0.6	5.0±0.4	1.8±0.7	6.1±1.3	3.0±1.0	7.6±0.6			
Group II	1.8±0.8	5.0±0.7	2.3±0.6	6.0±1.3	2.9±0.9	7.6±0.6			
Group III	1.5±0.3	5.0±0.3	2.0±0.7	6.2±0.7	2.0±0.2	6.0±0.7			

Values are expressed as mean±SD in mg/dl.

The red cell organic phosphate concentrations of ATP and 2,3DPG seem to follow inorganic phosphate changes. The renal phosphate reabsorption index (TMP/GFR) increased progressively in all three groups of babies over the first 2 weeks of life. Sick preterm and fullterm neonates increased their ability to retain Pi during this study period. The lower Pi values in problematic fullterm infants were probably due to the lower Pi intake. This study indicates that by altering supplementation of phosphate we can alter phosphate retention in neonates.