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Short Communication

Significance of Strategies to Avoid Neonatal Hypoglycemia in Both Transient and Persistent and Management and Prophylaxis to Avoid Long Term Sequelae

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Neonatal hypoglycemia [NH]-avoidable cause of brain injury, common-influences 5 - 15% babies and 50% of at risk babies-linked with range of side effects [1]. Ideal time of screening, threshold at which treatment avoids brain damage not clear. Glucose primary metabolic fuel for fetus. Glucose got from mother via carrier-modulated diffusion down a concentration gradient via the placenta [2]. Fetal glucose amounts 80% of maternal amounts vary with alterations in maternal glucose amounts. In fetus function of insulin is like GH instead of controlling glucose amounts and liberation of insulin-takes place at lower insulin in fetus as compared to postnatal life [3].

Pathophysiology-Maternal and thus fetal glucose amounts escalate at labour time and delivery- secondary to liberation of maternal stress hormones-like catecholamines and glucocorticoid [4]. Tying umbilical cord glucose amounts supply interrupted reduction in glucose amounts in neonatal glucose low peak decreased -low point of glucose amounts reached 1 - 2hr following birth insulin liberation decreases and counter controlling hormones-glucagon and catecholamines escalate-------) stmn of gluconeogenesis and glycogenolysis gradual escalation of glucose amounts but don't reach adult amounts till 72h age [5]. Delay of postnatal adaptation NH. Glucose-Necessary fuel-brain and in newborn proportionately large brain explains total tissue needs [6]. Hence low glucose amounts not enough brain energy provision. Though can use alternative metabolic substrates limited supply. Lactate serves as alternative fuel, but in 1st 48h, ketones probably on d3-4 although each very little total brain energy demands met. At risk factors (for transient NH-preterm birth, SGA. LFD, Infant of DM mother, perinatal stress (birth asphyxia, hypothermia, resp distress, sepsis, poor feeding) [For continuous NH-Congenital hyperinsulinism, hypopituitarism, (ACTH deficiency, GH deficiency), Cortisol deficiency, Glycogen storage dis, gluconeogenesis disorder (FBP deficiency, PEPCK deficiency, PC deficiency, FA oxidative defects definition contradictory major goal find a threshold avoids brain damage. Commonest used is glucose amounts < 47 mg/dl [2.6 mmol/l) [7,8] main source 2 studies-66 preterm babies (BW < 1850 gm) -- glucose amounts -- < 47 mg/dl [2.6 mmol/l) [on 3 or ≥ 3 with enhanced risk of developmental delay at 18 month [6]. Follow Up-decreased motor &mathematical function continued till 8yrs [6]. 2nd study recorded brain stem or somatosensory evoked potentials in 17 infants 5 were newborn [6]. Neither demonstrated flattening of evoked potentials with glucose a amounts > 47 mg/dl [2.6 mmol/l), despite some with glucose a amounts -- < 47 mg/dl [2.6 mmol/l) [also possessed normal evoked potentials-both conclusions glucose amounts > 47 mg/dl [2.6 mmol/l) safe amounts incidence risk factors incidence differs study type, diagnostic threshold glucose screening method-but incidence of transient NH 5 - 15% of newborns [9], in at risk newborns-50%-Babies with a lot of factors might not have >incidence but gave robust hypoglycemia.

Management Screening Clinical signs-cyanosis, apnea, changed consciousness levels, convulsions, poor feeding [6] not only these many nonspecific most babies with low a amounts no clinical signs-advocate all babies with at risk factors regular screening of glucose-maximum advocate 1 - 4h following birth and then 3 or 4h till euglycemia sustained over 2 - 3 consecutive glucose amounts American Academy of Paediatrics (AAP) - advocate continuous monitoring till 12h following birth for diabetic mothers and large for date (LFD) babies for 24h late preterm or small for gestational

age (SGA) [6]. No proof to point cerebral glucose amounts differ in at risk groups [6]. Blood glucose amounts monitor, various methods [6]. Continuous interstitial glucose amounts monitor-tedious [6]. Treatment of NH-Goal-avoid or minimize brain damage-sustain glucose amounts above acceptable threshold. Initially usually feed baby with formula feed/breast feed if glucose amounts < 18 - 25 mg/dl (1 - 1.4 mmol/l) iv dextrose (bolus 200 mg/kg followed by infusion of approximately 4 - 8 mg/kg/min-usually needed [6]. But iv dextrose needs NICU admission-costly, invasive, separates mother from baby increased maternal anxiety and interfere breast feeding establishment. Severe or prolonged hypoglycemia persistent high or ongoing ≥ 3 days iv glucose needs point underlying metabolic or endocrine pathology. Escalated insulin-hyper insulins-suppresses alternate fuel generation thus sustain glucose > 3.5 mmol/l [6]. Extra treatment glucagon, diazoxide or glucocorticoids might needed [6]. Oral dextrose gel 200 mg/kg of 40% dextrose along with feeding advocated-1st line treatment in asymptomatic NH [10]. A RCT of 237 late preterm and term babies with NH (< 47mg/dl [2.6 mmol/l)) documented in contrast to feeding only, 40% oral dextrose gel 200 mg/kg feeding lesser treatment failures (dec NICU admission advocated in various national guidelines. Prophylaxis-Some proof even transient and undetected NH side effects, study of 1395 babies in centre with glucose screening demonstrated single episode TNH (< 35 mg/dl was correlated with lower 4th grade literacy and numeral proficiency at 10yrs [11]. Children with hypoglycemia and their later development (CHYLD) study showed clinically not detected low interstitial glucose amounts was correlated with escalated executive function impairment at 4.5yrs age [12]. Currently advocated-early feeding, ensure babies warm and dry with early skin contact. Further oral dextrose gel being tested in at risk babies with early BF outcomes-MRI studies NH can brain damage. Commonest ABI located in parietal and occipital areas implicated in visual processing still inconsistent if later visual problems [13]. Adequately powered RCT s required to see for both prophylactic and treatment interventions at several glucose thresholds with neuro developmental dysfunction evaluated at least till school age. Significance of this lies in encountering of a newborn term infant who developed TN H without any at risk factors presenting as listless, cold in hot environment with mother receiving epidural anaesthesia with single reading of 35 mg-recovered with immediate 10% dextrose and feeding although later sugars all above 58 mg.

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