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Case Report

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Cholestasis and neonatal hypoglycaemia

Abstract

Glucose is essential as the main brain energy source, hypoglycemia is dangerous and may cause neuronal damage as well as siezers and acidosis. The undetectable hypoglycemia has serious effects if passed unnoticed specially in infants who cannot explain their sensation and complain , any abnormal clinical or laboratory response should be addressed as a special entity and dealing with as if a complain that must be investigated.

Volume 10 Issue 4 - 2020

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Received: June 23, 2020 | **Published:** July 14, 2020

Case report

An infant 83 days old for Kassi operation, 4.5kg, normal growing parameters and reactive, without mother complain regarding feeding or suckling, normal birth history, progressive jaundice after birth and history of blood transfusion after Hb drop in rural pediatric department. Upper endoscopy showed no bile, liverbiopsy showed long standing biliary outflow obstruction and fibrosis for correlation .chest was free. Abdominal Ultrasound; non-visualized CBD, dysmorphic gall bladder, triangular cord sign, congenital inguinal and umbilical hernia, prominent porta hepatis lymph nodes. Echo good function with filamentous structure arising from the IVC at its entrance to the right atrium. In the pediatric ward his random blood sugar(RBS) was ranging between 100-125mg/dl .

Preoperative lab

BIL total 8mg/dl (normal<1mg/dl), BIL direct 4.8mg/dl. ALB; 4gm%, AST; 365, ALT; 230. ALK; 524. GGT; 1057. UREA;13mg/dl. Creatinine; 0.3mg/dl (Normal values:05-1.5 mg/dl) Hb; 11%. TLC 8cells/cumm of blood. Platelet count: $390x10^9$/L(Normal:$150-450x10^9$/L). INR; 0.9.Na; 137mmol/l.K; 4.6mmol/l. CRP-Negative. CT abdomen; showed no abnormality. No history of present illness of parents regarding endocrinopathy, hepatic or metabolic disease, and parents are not related.

Anesthetic events

The infant had his warmed maintenance fluid and fasting hours in department and before induction intraoperative maintenance was started, all standard monitors, preoxygenation, Fentanyl 2mic/kg and inhalational induction with gradual elevation of Sevoflurane and Esmeron was given/kg. Central line was deployed by U/S, warmed Bur-hugger blanket was under the infant with rapping hands and head. Just after induction and before skin incision RBS was measured by drop technique which was 68mg/dl and abolus of Glucose 10% -2.5ml/kg was given(short term hypoglycemia treatment) ,starting the operation and he was diagnosed as type 2 obstruction, the next hourly reading of RBS was 77mg/dl and again second bolus of Glucose 10% was given, then starting Glucose 10% infusion 5mg/kg/min, the operation lasted for 4hours including hernial repair and caudal analgesia. During the procedure the patient was on PRVC, TV 6ml/kg, peak 17, ETCo2 38, the saturation dropped once and was related to right endotracheal tube shift and pulse ranged from 115-125 with fentanyl maintenance and oral temperature readings were noticed to be 36 degree centigrade even with all precautions.

Extubation and Intensive care management

With full recovery extubation was done and maintenance fluids started as usual 4:1(0.9 Saline to Glucose 10%) with fluid maintenance 80 %. After an hour we changed Glucose concentration to 12.5% as RBS was 70mg/dl, after half an hour RBS was 65mg/dl consequently we changed Glucose concentration to 15% .The next reading 57mg/dl by follow up and with pediatric consultation, follow up of the RBS revealed the need to increase Glucose concentration till 17.5% then increased the rate of maintenance to 90% and by reaching this point RBS readings stabilized between 90-100mg/dl.

And readings were scheduled to be hourly instead of every half an hour. after 12 hours of operation before the next reading and because of sleeping and after sampling for lab the infant was lethargic and showed no response and progressive desaturation started till saturation reached 75%, endotracheal intubation was done and RBS was by drop technique 200mg/dl just after intubation, lactate was 90, VBG showed pH7.10, Pco_2 47, $HCO_3$14.9AG17.9, Cl; 105, K3.9, Ca++0.9 and BE -14.9. An hour VBG was pH 7.28, Pco_2 48, HCO3; 22.6, AG; 10 BE-4 and AG; 8.8 by the next hour was normal readings, and he was conscious and extubated, CT brain was done with normal findings, lactate 2 hours later dropped to normal level .Post-operative day 1 lab BIL total 6mg/dl(normal<1mg/dl), BIL direct 3.6mg/dl.

ALB; 2,7gm%, AST; 156 ,ALT;109ALK; 396, GGT; 603, UREA; 7mg/dl, Creatinine;0.35mg/dl (Normal values:05-1.5 mg/dl), Hb;9 %, TLC10cells/cumm of blood, Platelet count: 259x10^9/L (Normal: 150-450 x 10^9/L)INR; 1.1, Na; 137mmol/l, K; 4mmol/l .CRP-Negative.

Pediatric consultant asked forcortisol level at 9PM and AM, c-peptide, glucagon level, ACTH, TSH, T3, and insulin level in blood.

The infant stopped fluids by the third day postoperatively and started oral artificial feeding and while transit from injection to oral feeding some readings were low specially when sleeping so adding (potato starch)and corticosteroid (oral syrup) daily dose; 1mg/kg/daily after feeding.

Longstanding research proceeds so as to characterize clinical hypoglycemia. This can be characterized as a plasma glucose level underneath 68mg/dL, the cutoff glucose level for roughly at or beneath 50mg/dL has been viewed as adequate to undergo further testing to characterize an etiology of hypoglycemia, as many counter-regulatory responses occur at this level.[1,2]

Plasma glucose levels are maintained at ~55–60mg/dL after birth, and then increase to 70mg/d Lat approximately 2days of life, by abrupt cutting of umbilical cord the new born adapts by physiological changes to keep glucose homeostasis. Increased catecholamine concentration immediately which stimulates glucagon with subsequent decrease in insulin: glucagon ratio.[3]

In response to feeding, glucose is absorbed through the gastrointestinal tract through glucose transporters, Regulatory physiologic processes are then activated, including increased plasma insulin levels, glycogen synthesis, and triglyceride synthesis. Gluconeogenesis, ketogenesis and lipolysis are inhibited, and there is relative increase in peripheral glucose uptake. Conversely, as declining plasma glucose levels occur, insulin levels start to decrease at plasma glucose levels of approximately 80–85mg/dL. Counter-regulatory glucagon and epinephrine pathways are elicited, which can stimulate hepatic glycogenolysis and gluconeogenesis, lipolysis, and ketogenesis; this may occur at plasma glucose levels as early as 68mg/dL. Cortisol and growth hormone (GH) release then occur at even lower plasma glucose levels.[4]

Sepsis

At the beginning pediatric team suspected sepsis as the primary cause of life threatening hypoglycemia and starting from first daypost surgical antibiotic ,sending lactate which was normal and observing vital signs which were within normal and TLC which was within normal and during first 48 hours cultures revealed negative regarding drain and urine.[5]

Hyperinsulinemia

Hypoglycemia in infancy can also be caused by many disorders that result in hyperinsulinemia. Insulin levels are inappropriately elevated at the time of hypoglycemia. In addition, there is an absence of ketonemia and acidosis, which was excluded by laboratory findings for the infant (Random insulin level 0.2Uu/ml),C- peptide was 0.52ng/ml while normal fasting of c-peptide level; (0.8-3.9ng/dl) and after 2 hours of meal;(1.2-8.6ng/dl), this was for sure not corresponding with insulinoma specially CT abdomen which was free regarding pancreas.[6] The Glycemic response to glucagon at the time of hypoglycemia reveals a brisk rise in glucose, indicating glucose mobilization has been restrained by insulin and that glycogenolytic mechanisms are intact.[7,8]

Anterior-hypopituitarism

We began by gross exclusion by evidence from present laboratory results hypopituitarism may introduce in neonates with intermittent hypoglycemia and cholestatic jaundice. Patients may have physical signs that would propose hypopituitarism, for example, Clift- palate, microcephaly or ophthalmic irregularities seen with septo-optic dysplasia. Inborn syphilis can cause hypopituitarism, in addition to the well-recognized manifestation of hepatitis which were not complete as the cause of elevated liver enzymes was diagnosed as Kasi case. Inanterior hypopituitarism cases besides the defect in growth hormone the need for thyroid hormone and corticosteroids supply is mandatory. One of the reasons why hydrocortisone and L-thyroxine should be increased in cholestasis is because supplements of fat-soluble vitamins are needed. Insufficiency of bile acids secretion in the intestine causes difficulty for the absorption of fat-soluble substances. Serum bile acid levels, an established marker for cholestasis.[9] In this infant it should be noted that there was no anomalies in ECHO or CT ,the cause of hyperbilirubinemia was surgical and corrected and triglycerides were 98 and elevated after operation to 203mg/dl ,no need to supplement L-thyroxine (not anterior hypopituitarism).

Adrenal insufficiency

Essential adrenal deficiency can likewise give hypoglycemia and cholestasis. At the point when adrenal sickness is serious, as in cortisol manufactured catalyst abandons, adrenal drain or innate nonattendance of adrenals, hypernatremia, hyperkalemia, or questionable genitalia may give analytic insights which were (-ve).

Glycogen storage diseases usually present with hepato- megaly and tendency for hypoglycemia but biopsy is diagnostic and it was also negative.[10]

Postoperative stress response

As the patient was in stress, measuring cortisol level at 9AM:3.9ng/dl which was low and cortisol level 9 PM:6.1ng/dl which was normal, and TSH, free T3 and T4 which revealed normal free T3, T4 but lower normal TSH:1.67ulU/ML while normal TSH should be (1.7-9) and this was the major clue to suggest poor postoperative stress response in an ill child. The pediatric hormonal stress response has its maximum peak during surgery and resolves by the end of surgical stress or maximally 6 hours later then returns to normal which was found in the lab 48 hours after operation.[11,12]

Conflicts of interest

The authors have no conflicts of interest to declare.

Funding

None.

Acknowledgment

None.

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Citation: Gaballa NK, Bassiouny H, Hameed TA, et al. Cholestasis and neonatal hypoglycaemia. *MOJ Clin Med Case Rep*. 2020;10(4):85–87.
DOI: 10.15406/mojcr.2020.10.00350