International Journal of Healthcare Sciences ISSN 2348-5728 (Online)
Vol. 3, Issue 2, pp: (161-164), Month: October 2015 - March 2016, Available at: www.researchpublish.com

HYPERGLYCEMIA IN THE NEWBORN

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Abstract: **Neonatal diabetes mellitus (NDM) is a rare form of insulin dependent diabetes mellitus. Presents within the first month of life with hyperglycaemia, severe dehydration; glycosuria and absent ketonuria; several causes have been implicated with variable outcome. The need to identify hyperglycaemic neonates early and initiate therapy so as to prevent complications/death is highlighted and can still be achieved in a resource limited settings.**

Keywords: **Neonate, Hyperglycaemia, Glycosuria.**

1. INTRODUCTION

Neonatal hyperglycaemia is defined as whole blood glucose concentrations greater than 6.6 to 6.9mmol/L (plasma glucose >= 7.3 – 7.6mmol/L) regardless of the neonate's gestational age, weight or postnatal age presenting within the first 28 days of life with signs specific to hyperglycaemia which may include dehydration due to an osmotic duiresis, weight loss, failure to thrive, fever, glycosuria and metabolic acidosis[1-4]. Neonatal diabetes mellitus is a rare disorder with an estimated incidence of 1 in 400,000 live births. Clinically[r], NDM is classified into two: the transient neonatal diabetes mellitus in which condition the insulin secretion is spontaneously recovered, and the permanent neonatal diabetes mellitus that requires lifelong insulin therapy[r]

Neonatal hyperglycaemia may also be a sign of serious illness such as infection and management of the underlying illness is much more important than just achieving glucose control[5-9]. We present report of 2 babies with hyperglycaemia that were managed in a resource limited special care baby unit.

Case reports:

Case 1:

A 4 days old term female baby delivered to a 19 year old Primiparous woman via spontaneous vertex delivery at home. Baby did not cry at delivery. Pregnancy was booked at a mission hospital in Baga town although mother was not regular with antenatal visits and medications. Had a febrile illness in the first trimester, no history of peripartum pyrexia or body rash; not a known hypertensive or diabetic.

Baby was admitted at age 48hours on account of difficulty in breathing noticed soon after birth. No cyanosis or apnoea. Baby has also been refusing to suck from breast which warranted mother to express breast milk and give with cup and spoon, though baby still not tolerating enough. No fever or excessive crying however. Developed tonic seizures at age 48hours, had several episodes each lasting 1-2 minutes prior to presentation.

The first child of parents in a monogamous family setting, both parents are secondary school certificate holders, mother; a full time house wife while father is a trader-sells provisions.

On admission, baby was dyspnoeic and tachypnoeic with grunting respiration, respiratory rate of 80cpm; was lethargic with depressed primitive reflexes.

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Diagnosis of a 4 days old term baby with severe perinatal asphyxia + hypoxic ischaemic encephalopathy stage II with risk for sepsis? Hypoglycemia and electrolyte imbalance was made.

Initial investigations revealed: plasma glucose of 1.5mmol/L, PCV=49%, serum sodium 144mmol/L, potassium 5mmol/L, chloride 110mmol/L, bicarbonate 18mmol/L, urea 28.6mmol/L, total calcium 2.3mmol/L and ing. Phosphate 1.6mmol/L

Baby had intravenous 10% dextrose at 2mls/kg as bolus and subsequently placed on intravenous fluid 1/5 saline in 8.4% dextrose at 90mls/kg/day, was also commenced on intravenous cefuroxime at 100mg/kg/day, intramuscular gentamycin at 5mg/kg/day in two divided doses, intramuscular phenobarbitone at a loading dose of 20mg/kg stat with maintenance dose of 8mg/kg/day in two divided doses.

Two hours into admission, plasma glucose was 7.7mmol/L and at 6hours into admission, it was further increased to 10mmol/L at which point the intravenous fluid was changed to 1/5 Normal saline in 5% dextrose. Despite this, subsequent review shows un – recordable blood glucose using glucometer in the ward with urinalysis reading glycosuria of +++, ketones however negative. Plasma glucose estimation in the hospital laboratory was 81.1mmol/L. At this point, intravenous insulin at 0.1iu/kg/hour was prescribed but the baby died prior to commencement of insulin therapy.

Case 2:

JJO was term; 7 days old male baby, delivered to a 36 year old P3 + 0, woman, pregnancy was supervised at the University of Maiduguri Teaching Hospital (UMTH), Maiduguri, period said to be uneventful. There were no perinatal risk factors for sepsis identified. Delivery was in UMTH by spontaneous vertex, baby said to have cried at delivery with Apgar scores of 7 and 9 at 1 & 5 minutes respectively. Birth weight of 4.2kg (Admitting weight was 2.75kg) = lost 32.2% of birth weight

Mother is not a known diabetic or hypertensive, the 3rd child in a monogamous family setting; mother a full time house wife, father a businessman

Baby was brought in at age 7 days on account of a day's history of refusal of feeds and 3 hours history of fever and convulsions with reduced urinary output.

Major findings at presentation were fever (t= 40^{0}C), severely dehydrated (very sunken eyes with markedly reduced skin turgor), dyspnoeic with subcostal recessions, respiratory rate of 70/minute; was lethargic with sunken eyes and depressed primitive reflexes.

Diagnosis of a 7 days old term male neonate with severe sepsis in shock; to rule out meningitis was made.

Initial investigations reveals: Random plasma glucose of 18.3mmol/L, Packed cell volume 39%, sodium 190mmol/L, Potassium 9.4mmol/L(? haemolysis), repeat was 4.4mmol/L, chloride 144mmol/L, bicarbonate 17mmol/L, urea 45.2mmol/L, creatinine 547mol/L

Urinalysis revealed: protein ++, glucose ++++, P^H 5, ketones –ve

Additional consideration of acute renal failure complicating severe sepsis was entertained.

Baby was commenced on intravenous fluid -Normal saline at 20ml/kg as anti-shock over an hour which had to be repeated because baby was still in shock, had oxygen administered via nasal catheter, intravenous ceftriazone at 100mg/kg/day in two divided doses, intramuscular gentamycin at 5mg/kg/day in two divided doses (which was subsequently discontinued because of renal impairment), intramuscular phenobarbitone at loading dose of 20mg/kg with maintenance of 8mg/kg/day in two divided doses and intravenous fluid 4.3 % dextrose in o.18 saline at 150ml/kg/day.

Repeat estimation of Random plasma glucose at 2 and 3hours into admission were 21.6mmol/L and 25.5mmol/L.

At this point, neonatal hyperglycaemia was also considered and baby was commenced on soluble subcutaneous insulin at 0.1iu/kg/dose 6 hourly while blood glucose is also monitored 6 hourly and insulin given if > 14 mmol/L, intravenous fluid was changed to 5% dextrose in 0.18 saline because of documented hyperglycaemia.

After 5 days of s/c soluble insulin administration, the blood sugar stabilized at 4 – 6 mmol/L. Insulin was discontinued and baby was discharged. Repeat E/U/C prior to discharge reveals Sodium of 138mmol/L, Potassium = 3.4 mmol/L, Chloride of 84mmol/L, Bicarbonate of 18mmol/L Urea = 7.2mmol/L and Creatinine of 89mol/L.

Baby was seen twice during follow up visits with normal plasma glucose levels of 5.3 & 4.3mmol/L and thriving well. Subsequent reviews at 3 months, 6 months, 12 months, 15 months and 24months reveals normal blood glucose levels with no recurrence of hyperglycaemia.

2. DISCUSSION

Hyperglycaemia, referred to plasma glucose greater than 126mg/dl is seen most frequently in very low birth weight infants receiving intravenous glucose infusion[1, 8]. Sepsis, asphyxia and stress can also lead to hyperglycaemia by catecholamines and cortisol influences on glycogenolysis, gluconeogenesis and insulin response[1, 2, 4, 9, 10]. On the other hand, endotoxins may have a direct effect on insulin actions in septic infants[3]. Transient and permanent diabetes mellitus has to be differentiated from the transient hyperglycaemia seen in neonates receiving parenteral glucose infusion[10-12]. In our cases, hyperglycaemia may be contributed by a number of factors, in case 1 though the mother booked for antenatal care, was not regular with antenatal visits and drugs, the mother delivered at home with baby refusing to feed for some days prior to presentation. These may be features of both perinatal asphyxia and sepsis. For the 2nd case, the baby presented with classic features of severe neonatal sepsis with acute renal failure, all these can predispose the baby to stress and severe hyperglycaemia.

Neonatal diabetes can also cause neonatal hyperglycaemia, which is defined as hyperglycaemia occurring in the first month of life, lasting for more than 2 weeks and requiring insulin therapy[2]. It is distinct from autoimmune type I diabetes mellitus which manifests after the first 3 to 6months of life[2, 3]. The condition is characterized by hyperglycaemia, glycosuria; hypoinsulinaemia and absent or minimal ketonuria. Treatment consists of rapid correction of dehydration and insulin therapy with normalization of growth parameters. In both cases highlighted, further investigations to evaluate possible causes of hyperglycaemia such as serum insulin assay could not be done due to our resource limitation. The first case though presented with hypoglycaemia, developed hyper glycaemia within hours of admission and commencement of therapy, the hyperglycaemia persist and baby died while efforts are being made at starting insulin therapy. This may be related to the administration of 10% dextrose as has been documented by other authors[2, 3, 9]. For the second case, the baby presented with classic features of neonatal hyperglycaemia probably predisposed by severe sepsis and acute renal failure that required antishock therapy twice to expand intravascular volume. Baby also had intravenous ceftriaxone as treatment of sepsis, probably the underlying cause in addition to insulin therapy; with prompt response and a good outcome. Subcutaneous insulin was used in our second case rather than intravenous insulin because of our limitation in resources (infusion pump and personnel) with a good outcome.

Neonatal hyperglycaemia has been associated with a wide spectrum of complications[2, 3, 13] such as dehydration which may be severe as in the second case highlighted or to death as in the first case. Some infants may end – up with abnormal neurodevelopmental outcome[3]. Further monitoring and follow - up 3 months, 6 months, 12 months, 15 months and at 24 months reveals normal plasma glucose levels with no recurrence of hyperglycaemia and no evidence of neurodevelopment outcome.

3. CONCLUSION

Neonatal hyperglycaemia, though not so common, does occur and can lead to adverse neurologic outcome and mortality. Identifying babies with hyperglycaemia, causative factors early and instituting prompt management; directed at both aetiologic factors and glycaemic control using insulin will go a long way in controlling both morbidity and mortality.

ACKNOWLEDGEMENT

We wish to thank the Nursing staff of the Special Care Baby Unit, UMTH for their support and dedication during the management of these babies. We also thank the management of the University of Maiduguri Teaching Hospital for allowing us use the case notes for this case report.

REFERENCES

[1] Richard E. Wilker. Hypoglycemia and Hyperglycemia In Spiral Manual of Neonatal Care, sixth Edition, Wolters Kluwer/Lippincott Williams & Wilkis, 2008; 540 – 549

[2] Kallapur SG,Kothare SV, Rathore AS, Gangal PS, Prabhu SB, Irani SF. Transient hyperglycemia in neonate(a case report and review of literature) J Postgrad Med 1987; 33:222

[3] Anusha H. Hemachandra, Richard M. Cowett. Neonatal hyperglycemia. http:// pedsinreview.aappublications.org 1999; 20; e16

[4] V R Kairamkonda and M Khashu. Controversies in the Management of Hyperglycemia in the ELBW Infant. Indian Pediatrics, vol 45; 2008, 29-38

[5] Kupper A. Wintergerst, Bruce Buckingham, Laura Gandrud, Becky J. Wong, Saraswati Kache and Darrel M. Wilson. Association of Hypoglycemia, Hyperglycemia and Glucose Variability With Morbidity and Death in the Pediatric Intensive Care Unit. Pediatrics.aappublications.org 2006; 118;173

[6] Anglina Kataria, Resmy Palliyil Gopi, Pradeep Mally, Bina Shah. Neonatal diabetes mellitus: current perspective. Research and Reports in Neonatology 2014:4 55-64

[7] Hays SP, Smith EO, Sunehag AL. Hyperglycemia are a risk factor for early death and morbidity in extremely low birth-weight infants. Pediatrics 2006; 118: 1811 – 1818.

[8] Michel Polak and Helene Cave. Neonatal diabetes mellitus: a disease linked to multiple mechanisms. Orphanet Journal of Rare Diseases2007, 2:12. http://www.OJRD.com/content/2/1/12

[9] Obasa TO, Adesiyun OO, Mokuolu OA. Transient neonatal diabetes or neonatal hyperglycaemia: A case report Niger J Paed 2013; 40 (2): 192 – 194

[10] Ferda Ozlu,Fylyz Tyker, Bylgyn Yuksel. Neonatal Diabetes Mellitus. Indian Pediatrics, 43; 2006

[11] Hye Young Jin, M.D, Jin-Ho Choi, M.D, Gu-Hwan Kim, PhD., and Han-Wook Yoo, M.D. Transient neonatal diabetes mellitus with macroglossia diagnosed by methylation specific PCR(MS-PCR). Korean Journal of Pediatrics vol. 53, No 3, 2010

[12] A Saini, V Kumar, S Kumar. Neonatal Diabetes Mellitus: Genetic Defect and Management Issues. https://isub.com/ IJPN/9/1/6895

[13] Mc. Salerno, N. Gasparini, M. L. Sandomenico, A. Franzese, A. Tenore. Two Interesting Cases of Transient Diabetes Mellitus:Journal of Pediatric Endocrinology and Metabolism. Vol 7, issue1,47-52