

Neonatal Malaria with Hyperglycemia and Hyperlipidemia: A Case Report

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Abstract

Neonatal Malaria manifests most commonly as fever, anemia, hepatosplenomegaly, jaundice, loose stools and poor feeding however hyperglycemia and hyperlipidemia associated with malaria is not mentioned in literature.

Case Presentation

A full term neonate was admitted in December 2014, in Neonatal Intensive-care Unit (NICU) as fever and anemia with splenohepatomegaly, his peripheral smear was positive for falciparum malaria with negative sepsis screen however there was hyperglycemia, glucosuria and hyperlipidemia associated with it, malaria was and hyperglycemia was treated with antimalarials and insulin respectively although hyperlipidemia persisted on discharge.

Key Words: Hyperglycemia, Hyperlipidemia, Neonatal Malaria.

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Introduction

Congenital or Neonatal Malaria is a rare disease and is associated with significant mortality(1,2). Malaria involves multisystem infection. Haematological, renal and central nervous system adds to the mortality (3). However association of hyperglycemia and or hyperlipidemia with malaria are rarely encountered.

Case Report

A 18 days old full term, normally delivered male newborn, weighing 2.5 kg (birth weight 2.4 Kg) on breast feeding was admitted in December 2014, in NICU with history of high fever since five days. Mother was 24 year old, second pregnancy (PARA) with normal antenatal history, but had a postpartum complaints of fever since 5 days. Her peripheral smear was positive for Falciparum malaria parasite⁺⁺⁺ with platelet count 39,000, his (T)oxoplasmosis, (O)ther Agents, (R)ubella (also known as German Measles), (C)ytomegalovirus, and (H)erpes Simplex (TORCH) infections was negative. On clinical examination the neonate was active, feeding well but had icterus and hepato-splenomegaly (liver 3cm and spleen 4cm), congenital anomalies, other systems and fundus were also normal. His initial investigations were Hemoglobin (Hb) 6.1 gm, Total Leukocyte Count (TLC) 10,700, platelets 18000, blood group O Positive O^(+ve), corrected reticulocyte count 4% and peripheral smear positive for falciparum + 4 while sepsis screen and lumbar puncture, was normal, random blood sugar was 711 mg% with high glycosuria but keto acids were absent, however, blood sample was reported to be highly lipemic. Ultrasonography (USG) and Computed Tomography (CT) of abdomen showed

hepatosplenomegaly, but X-ray chest was normal. Other blood investigations such as C-reactive Protein (CRP), Glucose-6-phosphate Dehydrogenase deficiency(G-6-PD),Thyroid Stimulating Hormone (TSH), Thyroxine (T4) and Triiodothyronine (T3), Blood Urea creatinine and electrolytes were normal, urine was positive for reducing substance, Serum Glutamic Oxaloacetic Transaminase (SGOT) 350 u/l, SGPT 384 u/l, total bilirubin 8.0 mg/dl, direct and indirect fraction being 6.3mg/dl, and 1.7 mg/dl and Alkaline Phosphatase (ALP) 1950 u/l.

TORCH titre, only Cytomegalovirus Immunoglobulin G (CMV-IgG) was positive Immunoglobulin M(IgM) negative HSV-I (IgM) was positive and HSV-II (IgM) was equivocal, but these tests were carried out on a sample which was highly lipemic. A second fasting blood sample on 3rd December was also reported to be highly lipemic, lipid profile being cholesterol 305 mg/dl, triglycerides 1691mg/dl HDL 13mg/dl (low) and Low-density Lipoprotein (LDL) 72mg/dl (low), Random Blood Glucose (RBG) was 892 mg%. Insulin infusion was started initially as Blood glucose was very high at the rate of 0.02U/kg/hour and later it was increased up to 0.1 U/Kg/hour but on 6thDec, it was stopped as the levels approached towards normoglycemia, a comparatively low Cholesterol and Triglycerides were reported but they were still above normal levels (Cholesterol 285mg/dl Triglycerides 989mg/dl, HDL 17mg/dl (low), LDL 70mg/dl (low). Mother's RBS and fasting lipid profile was Cholesterol 195mg/dl, triglycerides 81mg/dl, HDL 59mg/dl, LDL 94mg/dl. Neonate was not given any intravenous fluids (except insulin infusion) neither any parenteral nutrition during the stay in hospital. Post prandial C-peptide levels was low 0.52

ng/ml (range 1.2- 3.4ng/ml). Patient was treated with Inj. Ampicillin and Inj. Gentamycin initially but as the sepsis screen was negative it was stopped, Inj. Artesunate and Clindamycin, was completed. Packed RBC transfusion was given. Patient responded to intravenous insulin infusion as the blood sugar levels came to normal prior to one day of discharge, however surprisingly there was no polyuria, no dehydration in spite of blood sugar levels remained high for few days. Patient remained active and continued on breast feeding during period of admission. On Dec^{8th} patient was discharged against medical advice due to economic reasons.

Discussion

Hyperglycemia is inversely related to gestational age and weight in neonates, it is more common in premature and very low birth babies as compare to term neonates. Etiology of hyperglycemia may be, due to antenatal history of drugs such as theophylline, dexamethasone and glucose infusions especially in preterm newborns, total parenteral nutrition, stress, infections, transient neonatal diabetes mellitus. In our case, the child was full term, weighing normally, on breast feeding, with normal antenatal history, and no sepsis with normal maternal lipid profile, peripheral smear positive for falciparum malaria hence the diagnosis was Full term appropriate for gestational age with severe anemia, complicated falciparum malaria, hyperlipidemia with hyperglycemia. Transient Diabetes of newborn is accompanied by hyperglycemia which responds to insulin, remains for few weeks or months and then it resolves, however few cases may have abnormal glucose tolerance test or recurrence of diabetes in older childhood; in some cases there is no remission and is known as permanent

neonatal diabetes. The newborns in such cases are usually IUGR and have dehydration, it is thought to be due to either inadequate secretion or some degree of insulin insensitivity, in our case the newborn was fullterm normal with no such manifestations, had hyperlipidemia, although C-peptide level was low but insulin levels could not be done (10). Usually Hypoglycemia is a complication in severe malaria. however in our case it was hyperglycemia, which may be because of associated hyperlipidemia, although a few cases of malaria associated with hyperglycemia in older children have been reported (4,5). Neonatal Hyperglycemia due to Hyperlipidemia is attributed to an increase in free fatty acid levels which decrease peripheral glucose utilization primarily by substituting carbon and altering enzymatic activity that preferentially leads to fatty acid carbon oxidation rather than glucose oxidation. Fatty acids also inhibit the effect of insulin to suppress hepatic glucose production. These two conditions increase glucose concentrations in the plasma⁶. Hyperlipidemia as a complication of congenital or neonatal malaria may be rare, as we could not find literature on malaria associated with both hyperglycemia with hyperlipidemia in neonates, although dyslipidemia has been reported with the malaria in adults⁷⁻⁹. Other possible diagnosis might have been Familial hyperlipidemia, but as father and other family members lipid profile was not available diagnosis of familial hyperlipidemia was not confirmed. Though presentation of familial hyperlipidemia is usually late in childhood and is associated with obesity and increased insulin levels but it is not associated with hyperglycemia. But exact cause of hyperlipidemia could not be confirmed that is, whether it was due to malaria or some other condition. Limitations: As the samples

were highly lipemic values of certain investigations might have been altered. Father's Lipid profile, Newborn insulin and free fatty acid levels and a repeat C-peptide level, could not be done as the facility was not available in our Institute, in addition there was economic restrictions ,newborn was not available for follow up.

Conclusion

Newborns presenting with hyperglycemia with hyperlipidemia are rare with possible differential diagnoses of Transient Neonatal Diabetes Mellitus (TNDM), Familial Dyslipidemia (FH) and probably Neonatal Malaria in such cases. However in our case was difficult to conclude that, whether hyperglycemia with hyperlipidemia was per se or it was due to associated Malaria.

Conflict of interests: None.

References

1. Wilson WR, Sande MA, Drew WL. Current Diagnosis and Treatment in Infectious Diseases. New York: Lange Medical Books/McGraw Hill; 2001: 798.
2. Desai M, Kuile FO, Nosten F, McGready R, Asamo K, Brabin B, et al. Epidemiology and burden of malaria in pregnancy. *Lancet Infect Dis* 2007; 7(2): 93–104.
3. Dughat M, Dughat P: Thrombocytopenia in Patients of Malaria – Correlation with type of Malaria and it's Clinical Significance. Online International Interdisciplinary Research Journal 2013; 3(2): 22.
4. Dass R, Barman H, Duwarah SG, Deka NM, Jain P, Choudhury V. Unusual presentation of malaria in children: An experience from a tertiary care centre in North East India. *Indian J Pediatr* 2010; 77 (6): 655-60.
5. Tombe M, Bhatt K, Obel AO. Clinical surprises and challenges of severe malaria at Kenyatta National Hospital, Kenya. *East Afr Med J* 1993; 70(2): 117-19.
6. Hemchandra A, Cowett R. Neonatal Hyperglycemia. *Pediatrics Review American Academy of Pediatrics* 1999. Available at: <http://pedsinreview.aappublications.org/content/20/7/e16>. Accessed December 5, 2013.
7. George G, Holz JR. Lipids and Malarial Parasites : Bulletin of the world Health Organization 1977; 55(2-3):237-48.
8. Visser BJ, Wieten RW, Nagel IM, Grobusch MP. Serum Lipids and lipoproteins in Malaria a systematic Review and Meta Analysis. *Malaria Journal* 2013; 12(1):442.
9. Krishna AP, Chandrika, Kumar S, Acharya M, Patil SL. Variation in common Lipid parameters in malaria infected patients. *Indian J Physiol Pharmacol* 2009;53(3): 271-4.
10. Polak M, Cave H. Neonatal Diabetes Mellitus: a disease linked to multiple mechanisms. *Orphanet Journal of Rare Diseases* 2007; 2:12.