Neonatal Malaria with Hyperglycemia and Hyperlipidemia: A Case Report

*Gurudutt Joshi1

1Surat Municipal Institute of Medical Education Research (S.M.I.M.E.R.) Affiliated to Veer Narmad South Gujarat University, India.

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.

*Corresponding Author:
Dr. Gurudutt Joshi MD Deptt of Pediatrics Surat Municipal Institute of Medical Education and Research (S.M.I.M.E.R.) Surat Gujarat India
Received date: Feb 10, 2015 ; Accepted date: Feb 22, 2015
A Case of Neonatal Malaria

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 hepatosplenomegaly (liver 3cm and spleen 4cm), congenital anomalies, other systems and fundus were also normal. Her 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 lumber 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 Transaminin (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 6th Dec, 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 parental nutrition during the stay in hospital. Post prandial C-peptide levels was low 0.52
ng/ml (range 1.2-3.4 ng/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 remaining 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, hyperlipedemia 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 be either inadequate secretion or some degree of insulin insensitivity, in our case the newborn was full term normal with no such manifestations, had hyperlipedemia, 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 hyperlipedemia, although a few cases of malaria associated with hyperglycemia in older children have been reported(4,5). Neonatal Hyperglycemia due to Hyperlipedemia 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 plasma(6). Hyperlipedemia as a complication of congenital or neonatal malaria may be rare, as we could not find literature on malaria associated with both hyperglycemia with hyperlipedemia in neonates, although dyslipedemia has been reported with the malaria in adults(7,9). Other possible diagnosis might have been Familial hyperlipedemia, but as father and other family members lipid profile was not available diagnosis of familial hyperlipedemia was not confirmed. Though presentation of familial hyperlipedemia 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 hyperlipedemia 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 hyperlipedemia 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 hyperlipedemia was per se or it was due to associated Malaria.

**Conflict of interests:** None.

**References**