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Case Report

Prolonged neuroglycopenia in a type 1 diabetic pregnancy: a case report

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ABSTRACT

Hypoglycemia in pregnancy is a metabolic abnormality associated with substantial morbidity. Here we report the adverse events triggered during pregnancy in a primi with type 1 diabetes. A 19-year-old primi, type 1 diabetic since 13 years of age and history of recurrent seizures since fourth month of pregnancy presented at 28 weeks with severe hypoglycemia. Clinico-radiological impression of a metabolic encephalopathy secondary to prolonged neuroglycopenia was kept. She underwent preterm caesarean section and delivered a 1.86 kg baby. She was weaned off the ventilatory support and discharged on POD 38 on tracheostomy T piece. After that she continued treatment from local hospital, and expired 2 weeks later. presently her child is 4 years old, healthy with age-appropriate milestones.

Keywords: Neuroglycopenia, Type 1 diabetic pregnancy, Hypoglycemia, Metabolic encephalopathy, Coma, Seizure

INTRODUCTION

Hypoglycemia is a metabolic abnormality associated with substantial morbidity. The risk of severe hypoglycemic episodes increases with the duration of the disease. Hypoglycemia triggers a series of physiological, psychological and behavioural responses. Neuroglycopenia is a serious life-threatening condition caused by deficiency of glucose in central nervous system. It is of two types-acute and chronic. In acute neuroglycopenia the symptoms result by activation of sympathetic nervous system, and it occurs when there is rapid decrease in blood glucose.[1] Chronic neuroglycopenia is rare and it occurs in patients with insulin secreting tumors. Here we discuss a case of severe neuroglycopenia in a type 1 diabetic primigravida.

CASE REPORT

A 19-year female presented with unresponsiveness and abnormal movements of upper and lower limbs since morning with history of involutary micturition and defecation around 11 PM on November 21, 2019. She was 28 weeks pregnant (Primigravida). She was a known case of type 1 diabetes mellitus for 13 years of age on insulin (Injection HM 30/70 30-0-26) with history of seizures since second trimester of pregnancy (on T. levetiracetam 250 mg BD. At the time of arrival patient was unconscious, not oriented with abnormal upper and lower limb movements with PR: 130/min BP: 120/80 mmHg, SpO_2: 77% with O_2, GRBS: 27 mg/dl, chest: bilateral crepitations, pupils: sluggishly reacting to light. Obstetric examination: uterus corresponds to 26 weeks, fetal pole

present. In view of poor GCS (E2VTM3) she was intubated; which was later converted to tracheostomy.

She had recurrent episodes of unresponsiveness mostly in morning hours one month prior to present episode, for which neurology consultation was done, EEG done showed focal epileptic discharges from bilateral temporal region suggestive of nocturnal hypoglycemia or seizure disorder. Her Hb:10.9, TC: 33400 (N92M3L4E1), Na/K: 142/3.6. Peripheral smear: neutrophilic leukocytosis with left shift and toxic granules. Noradrenaline support was started. $MgSO_4$ loading dose given. Bedside ECHO: Good LV function, IVC collapsing, no RWMA. CECT head: hypodensities in bilateral basal ganglia with relative hyperdensity in bilateral thalamus s/o hypoglycemic/hypoxic insults. Blood culture and sensitivity: MSSA, endotracheal culture and sensitivity: MDR Acinetobacter. CSF Autoimmune encephalitis panel negative. She was started on injection meropenem in view of MDR Acinetobacter. She was given appropriate treatment but continued to have poor GCS, Quadriparesis and aspiration pneumonia.

Magnesium sulphate regimen was given for neuroprotection at 32 weeks gestation and dexamethasone was administered. Obstetric USG and Doppler done showed an estimated fetal weight of 1.2 kg, with normal Doppler. She was followed up daily with fetal heart auscultation and it was decided to terminate the pregnancy by 34 weeks after a multi-departmental board review. She underwent elective LSCS on 27/12/19 at 34 week 2 days and delivered a female baby of 1.86 kg. She was gradually weaned off from ventilator, IV antibiotics (Polymyxin and meropenem infusion) stopped.

EEG was done which showed ictal rhythms from right temporal area and generalised epileptiform discharges with severe diffuse electrophysiological dysfunction. MRI done was suggestive of hypoglycemia / hypoxic insult. She developed bilateral posturing of both upper and lower limbs. Patient maintained on O_2 with T piece with GCS E4VTM3. She was discharged on postoperative day 38 with tracheostomy and Ryles tube. After that she continued treatment from local hospital, and expired 2 weeks later. present her child is 4 years old, healthy with age-appropriate milestones.

DISCUSSION

Hypoglycemia is a medical emergency and their manifestations include neuroglycopenic symptoms such as inability to concentrate, drowsiness, confusion, speech abnormality and in coordination due to brain fuel deprivation and neurogenic or autonomic symptoms such as palpitations, tachycardia, diffuse weakness, anxiety and hunger.[2,3] Prolonged hypoglycemia can result in neuronal dysfunction and death, with deficits ranging from measurable cognitive impairments to aberrant behaviour, seizures and coma.[2,3] In sustained hypoglycemia, the integrity of cerebral neurons is not preserved.[4]

Sleep impairs the counter regulatory hormone responses to hypoglycemia even in normal subjects.[3] Seventy percent of the patients were sleeping immediately before presentation with severe neuroglycopenia. In another study it was seen that increases in catecholamine, ACTH, cortisol and growth hormone in response to hypoglycaemia were distinctly weaker during the later part of nocturnal sleep.[5] It has also been seen that plasma glucagon levels decrease significantly during night time sleep in healthy individuals.[5] It is possible that the decreased adrenomedullary response and the glucagon response with a decreased arousal during sleep contributed further to the loss of autonomic response.

Severe hypoglycemia is three times as frequent in early pregnancy compared with the period before pregnancy, and the incidence is highest in gestational week 8-16 and lower in the second part of pregnancy.[6] Hypoglycemia can have possible effects on birth weight, placental weight, or placental ratio. The most common finding is that maternal hypoglycemia is associated with an increased risk of intrauterine growth retardation (IUGR) or a small for gestational age (SGA) newborn. In addition, they are at increased risk for hypoglycemia, sepsis, seizure, stillbirth, respiratory distress, and meconium aspiration, SGA newborns are at risk for long-term outcomes such as poor school performance, hyperactivity, hypertension, and cardiovascular disease.[7] In women with recurrent episodes of hypoglycemia, the clear benefits of strict glycemic control must be weighed against the hazards of hypoglycemia.[8]

CONCLUSION

Our case highlights the importance of preventing severe hypoglycemic events by early identification of women at increased risk of severe hypoglycemia in pregnancy. By doing so, special education and individual modification of glucose monitoring, diet, and insulin treatment could be provided to those identified as high-risk subjects. They may benefit from intensified glycemic analysis in terms of continuous glucose monitoring or by adoption of alternative treatment modalities. This case is presented for its rarity since we could save the fetus even after the mother required ventilation for 6 weeks.

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