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Case Report

Recurrent Diabetic Ketoacidosis in Pregnancy: Sweet Success

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ABSTRACT

Diabetic pregnancy is a high risk pregnancy associated with poor feto-maternal outcome. When poorly controlled it leads to Diabetic Ketoacidosis (DKA). A pregnant woman is more prone to develop DKA even at lower glucose levels. DKA increases the maternal and fetal morbidity and mortality by manifolds. Immediate recognition and aggressive management are essential to improve the feto-maternal outcome. Treating the underlying cause is important to prevent recurrence. Here we present a case of recurrent DKA [9 episodes] in pregnancy fortunately with a good outcome.

Key words: Diabetes in pregnancy, diabetic ketoacidosis, lipohypertrophy.

INTRODUCTION

Diabetic pregnancy is a high risk pregnancy associated with poor feto-maternal outcome. When poorly controlled, it leads to Diabetic Ketoacidosis (DKA) which increases feto-maternal morbidity and mortality. DKA episodes in pregnancy may be caused by hyperemesis, infections, non-compliance, drugs etc. Here we report a case of recurrent DKA secondary to improper administration of insulin. The case is reported for its rarity [9 episodes of DKA in same pregnancy], good feto-maternal outcome by proper and aggressive management and for reiterating the importance of clinical examination in all patients.

CASE REPORT

A 22 year old Saudi female, $G_4P_2+_1$, live one and previous one IUFD , admitted through ER with 29 weeks pregnancy with the complaint of 5-6 episodes vomiting per day for 2 days. Patient was a known case of type 1 DM for past 12years, on insulin and had been admitted 9 times for DKA in the present pregnancy. The present pregnancy was a planned pregnancy. All admissions in this pregnancy were due to vomiting and pain abdomen under different consultants. The first admission was for hyperemesis and DKA at 7 weeks gestation and subsequently there were 7 more admissions at 2-3 weekly intervals. She was noncompliant, always presented to the ER, attended the endocrine clinic infrequently and was presumed to be

negligent regarding her diabetes control and antenatal care. However, on history she denied missing any of her insulin doses or any dietary indiscretion. The husband appeared to be supportive and both were extremely anxious for fetal wellbeing. There was no history suggestive of any infective focus in the body or fever.

Obstetric history: Her first pregnancy was a spontaneous abortion at 4 weeks. Second pregnancy was terminated by caesarean section at 36 weeks for fetal bradycardia, when a live girl, 2.5 kg was delivered. Third pregnancy was IUFD at 36 week due to DKA, delivered vaginally.

On admission her examination revealed that patient was dehydrated, had tachypnea 20/min, tachycardia 143 bpm and hypotension 90/61 mmHg. The systemic examination was normal and obstetric examination revealed a 28 weeks sized uterus, relaxed, FHS normal.

Hb-11gm%, TLC-8200/cumm, DLC-Neutrophils-76%, lymphocytes-20%, monocytes -04%, LFT –Normal, RFT-normal, blood glucose was 253 mg/dl , urine for sugar 3+, urine for ketones-3+. Urine for albumin was1+, Blood urea 19 mg/ dl, serum creatinine 0.9 mg\dl, sodium 132.3mmol/l, potassium 4.6 mmol/l, bicarbonate18.3mmol/l,O_2 saturation was 94%, ABG revealed pH 7.3, $PCO_2$19mmHg, PO_2 93.3 mmHg. HbA_1C was 9.The fundus examination, chest radiograph and ECG were normal, urine and blood cultures were negative. Cardiotocography was normal. Ultrasound revealed a live fetus, adequate liquor and gestation age corresponding to her LMP. There was no fetal anomaly. Thyroid function tests normal

She was hospitalized in the high dependency unit and resuscitation started with oxygen, aggressive intravenous fluids and insulin infusion. After maternal stabilisation and reversal of acidosis, patient was subjected to proper work up to explore the cause of repeated episodes of hyperemesis and DKA.

As she presented with vomiting we were looking for a cause of hyperemesis and recurrent DKA but could not get any satisfactory explanation. Meanwhile, we were joined by a new team member- an intern who was asked to work up the case. During examination of upper limbs he discovered bilateral lumps measuring 5×4 centimeters, firm, non tender in the deltoid muscles at site of insulin injections in both the arms. The patient had been self-administering insulin at the same site continuously since her diagnosis of DM 12 years ago. Subsequently, the injection site was changed to abdomen and she was kept hospitalized for close feto-maternal monitoring. She received a course of steroids for fetal lung maturity. At 33 weeks she developed mild hypertension with increasing albuminuria but did not have any new episode of DKA.

Elective caesarean was done at 34 weeks and a live male baby was delivered. The baby weight was 1.9kg, placenta weighed 300 grams and no congenital anomaly was present. Baby had an episode of mild respiratory distress and was admitted in NICU for two days. Mother and baby were doing well and discharged on request on 4th post-operative day.

DISCUSSION

The incidence of DKA has been reported as 1% to 10% from different studies. [1] While Cousins [2] reported an incidence of 9.3% during 1965-85, recent and larger case series [3] reported it to be 1.2%.A pregnant diabetic is more prone to develop DKA as compared to a non-pregnant diabetic as normal metabolic changes during pregnancy predispose to DKA. Majority of cases in pregnancy occur in 2^{nd} or 3^{rd}trimester, develop rapidly, at a lower blood glucose level and may not

always manifest with the classic symptoms or laboratory findings. Maternal mortality from DKA has been reported between 4%–15% from different studies. [4-6]

Fetal loss rates may be as high as 10–25% for a single episode of DKA despite substantial improvements in perinatal and neonatal care. [7]

Hyperemesis, infection, non-compliance, insulin pump failure, β-sympatheto-mimetic and corticosteroids for preterm labor alongwith poor management may be the precipitating factors. [8]

Symptoms of DKA are polyuria, polydipsia, nausea, vomiting, abdominal pain, weakness, weight loss. Signs include hyperventilation, ketotic breath, tachycardia, hypotension, disorientation or coma. [7-9]

Prompt recognition and management of this medical emergency is essential in order to optimize feto-maternal outcome. Finding the underlying cause is important. A careful history and physical examination may elicit the precipitating cause or may identify non-compliance with medical therapy. [9]

Our case of DKA presented as nausea and vomiting as early as 7 weeks and was labelled as hyperemesis gravidarum [HG]. Each time she was admitted we were looking for a cause of HG investigating her for the same even in later pregnancy though HG rarely persists beyond 20 weeks. [10]

The differential diagnosis of hyperemesis gravidarum includes urinary tract infection, uremia, thyrotoxicosis, diabetic ketoacidosis, Addison disease, hypercalcemia, gastritis, peptic ulcer disease, pancreatitis, bowel obstruction, hepatitis drug-induced vomiting, central nervous system (CNS) disease, and vestibular disease. [11]

Inspite of all investigations and USG we could not find a cause which was later elicited by history and examination.

Correct injection technique is important for insulin to be effective in achieving glycemic control. Non-rotation of injection sites may result in localized lipohypertrophy, degeneration or atrophy causing unpredictable and delayed absorption. Patient counseling regarding injection technique at first visit and audit at each subsequent visit is essential. [12,13]
Insulin injection site amyloidosis and zinc granulomas have also been reported affecting absorption kinetics. [14-16]

Making a diagnosis is a step wise process involving information gathering and generating a hypothesis. Data acquisition begins with history, clinical examination and investigations. [16]

However in recent times we have forgotten this stepwise approach –the investigations are ordered without examination and many a times the diagnosis is missed or delayed because of this.

We were able to find the underlying cause of recurrent DKA as lipohypertrophy on examination. This reiterates the importance of physical examination in making a diagnosis.
Importance of physical diagnosis has been emphasized right from the time of Hippocrates [460-370 B.C] to Laennec [1816] to Osler. [17]

A study found that 90% of physicians believed that physical examination is important in patients of hypertension and rheumatology but only laboratory tests were enough for diagnosis and management of diabetes mellitus and hyper-cholesterolemia. [18] However cases like ours prove that physical exam is and will remain important for diagnosing disease and developing the patient-doctor relationship. [19]

In Osler's words “Learn to see, learn to hear, learn to feel, learn to smell and know that by practice alone can you become experts”. [20] The combination of a good

history, thorough physical examination along with the correct use of investigations can help us become better diagnosticians to provide the best care to our patients.

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