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Case Report

PROGESTERONE INDUCED DIABETIC KETOACIDOSIS IN A YOUNG FEMALE A CASE REPORT

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Abstract
A 25year old recently married female presented to our emergency department with the sudden onset of breathlessness, pain abdomen and fever from past 1 hour. She was on tab progesterone 10 mg OD for past 10 days to postpone her regular menstruation for her marriage. She was never been diagnosed to have diabetics mellitus in the past. On arrival to emergency department she was tachypoenic, severely dehydrated and found to have elevated blood sugar levels. Arterial blood gas analysis showing severe wide anionic gap metabolic acidosis. She was diagnosed to have DKA. Managed as per standard DKA management protocol and was discharged from the hospital in euglcemic condition. Excess progesterone is known to impair both insulin sensitivity and secretion[1] and deteriorate glycemic control and resulted in complication like Diabetic ketoacidosis / Hyperglycemia in previously Diabetic patients.
Keywords: Progesterone, Diabetic mellitus, Arterial blood gas analysis, Diabetic keto Acidosis

Introduction

Diabetic ketoacidosis, a life threatening acute metabolic complication of diabetes mellitus, may occur in people previously known with diabetes or present as the first manifestation of diabetics mellitus. Diabetic ketoacidosis as the first manifestation of diabetes is most often associated with type 1 diabetes due to the rapid and marked beta cells destruction with resultant absolute insulinopenia. The presence of ketoacidosis at type 1 diabetes has been shown to be a marker of depleted beta cell reserve and poor metabolic state, type 2 diabetes is however, characterized by a chronic asymptomatic pre-clinical phase which progresses through a period of insulin independence to insulin dependence. Diabetic ketoacidosis which depicts a state of severe insulin deficiency is increasingly recognized as the initial manifestation of type 2 diabetes[2]. Progesterone has been reported to impair the insulin suppression of hepatic glucose production, sensitivity in muscle and adipose tissue and insulin secretion from the pancreas[3].

Case Report

On 07/12/2014 at 7 PM, a 25year female patient came to our emergency department with history of sudden onset of breathlessness, fever, body pain, abdomen pain since 6 pm. She had burning micturition and ongoing menstruation. She had no past medical or surgical history her family history was nothing significant

On examination she was conscious, oriented, obeying commands. Tongue dry, severe dehydration, tachycardia-140/min, and tachypnea-42/min, BP-140/80mmhg, spo2-100%RA.Temp-98.6f.Systemic examination revealed No abnormality

Laboratory investigation revealed RBS-530mg/dl, ABG showed severe wide anionic gap (=32.4) metabolic acidosis PH7.006 with base excess of -28.2, lactate 2.1.elevated WBC count – 20830, platelet-633100, hyperkalemia K^+-5.6. Microscopic examination of urine showed protein +, sugar 3+, urine ketones 3, puscells 1-2. Chest X-ray AP view normal study.

In emergency department, she was managed with standard DKA management protocol and shifted to MICU for further management. Patient recovered and got discharged after 72hrs with normal ABG and Euglycemic condition. Along with maintenance Insulin for diabetes management,

Discussison

A case of a 25 year female presented to our emergency department with diabetic ketoacidosis following progesterone treatment, which she had taken for the postponement of Menstruation cycle. She was investigated and found to have no other precipitating factor other than progesterone.

Progesterone is a steroid hormone involved in the female menstrual cycle, pregnancy and embryogenesis. Recent studies have shown a correlation between high progesterone levels and glucose intolerance. Exogenous progesterone decreases insulin sensitivity with subsequent development of hyperglycemia in rodents[1]. High glucose concentrations cause the development of insulin resistance in peripheral tissues, including of both insulin secretion and insulin sensitivity[1]. The biochemical basis for insulin resistance induced by hyperglycemia is still unclear, but we hypothesize that it may involve variation in concentration of IR and /or Glut – 4 protein as a result of the effects of oestrogen and progesterone. Progesterone administration reduces insulin sensitivity in rats[3].

In premenopausal women, the menstrual cycle represents a continuous state of change in terms of female sex steroid environment[5]. Impaired glucose metabolism during luteal phases of menstrual cycle and or decreased sensitivity during luteal phase may be the reason for the observed high fasting blood level in luteal phase of menstrual cycle compared to follicular phase[4]. Menstrual cycle variabity of insulin has been tested in previous studies. Insulin and a related measure of insulin resistance change over the menstrual cycle, with levels beginning to rise before ovulation and reaching maximum levels during the luteal phase[5]. These fluctuations across the cycle are most likely explained by the fact that insulin and insulin resistance by the homeostatsis model of insulin resistance were positively associated with estrogen and progesterone[3].

Therefore, increase in both exogenous and endogenous progesterone deteriorates glucose tolerance. Several studies have suggested possible mechanism by which glucose tolerance is deteriorated by progesterone. Acute administration of progesterone reduces the ability of insulin to suppress endogenous hepatic glucose production in rats. In addition female progesterone receptor knockout mice have larger pancreatic islets and show lower fasting glucose levels with higher insulin levels on glucose injection. These findings demonstrate that progesterone affects not only insulin sensitivity, but also insulin secretion[1].

In our patient the impaired glycemic control and DKA is secondary to the use of progesterone. Studies have shown diabetic ketoacidosis can occur in both type 1 and type2 diabetes mellitus. A significant proportion of DKA occurs in patients with type 2 diabetes[2].

Conclusion

Diabetes mellitus complications should be suspected in patients with undiagnosed/diagnosed diabetes mellitus who present with clinical features suggestive of Diabetic ketoacidosis / hyperosmolar hyperglycemic state and who is on drugs which affect regulation of normal glycemic control.

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