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Case Report Open Access

Steroid Induced Diabetic Ketoacidosis (DKA) in a 13 year Old Female with Renal Disorder

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Abstract

Background: Diabetic ketoacidosis (DKA) is a common complication of poorly controlled diabetes mellitus in children and a rare complication of steroid therapy. Patients on steroid therapy may develop hyperglycemia as a complication, but presentation with DKA is rather unusual.

Aim: To highlight a rare clinical entity of DKA induced by prednisolone in a 13 year old female on treatment for nephrotic syndrome.

Case report: NC was a 13 year old female who presented with first episode of generalized body swelling, oliguria, massive proteinuria and hypercholesterolenaemia with normal renal function. She was not a known diabetic and had no family history of diabetes mellitus. She was started on prednisolone at 20 mg three times daily for nephrotic syndrome. Two weeks after commencement of prednisolone, she developed DKA with blood glucose of 31.1 mmol/l, glycosuria and ketonuria. She received intravenous insulin, fluids and was discharged on mixtard insulin with withdrawal of prednisolone. Her fasting blood sugar gradually normalized to between 3.1-4.5 mmo/L and insulin stopped after 4 months of treatment. She has remained normoglycaemic on follow up.

Conclusion: The possibility of hyperglycaemia and DKA should be anticipated on every adolescent on steroid therapy for nephrotic syndrome. We therefore recommend routine blood glucose monitoring for early identification of DM in order to avoid DKA in such patients.

Keywords: Diabetic Ketoacidosis; Steroid Induced; Prednisolone; Renal Disorder

Introduction

Glucocorticoids have profound effect on carbohydrate metabolism, stimulating the production of glucose from amino acids and glycerol in the liver [1]. The pharmacological action of glucocorticoids include opposing insulin action, decreasing glucose utilization, increase protein breakdown and activation of lipolysis thereby providing amino acids and glycerol for gluconeogenesis [1,2]. The production of extra insulin occurs in most people on steroids to counteract the hyperglycemic effect of steroids, however those who lack this ability, develop hyper glycaemia [2].

Glucocorticoids are frequently prescribed drugs in various disease conditions [3]. Nearly half of the patients treated with Glucocorticoids for more than two weeks developed a deranged glucose metabolism and those with risk factors for diabetes mellitus present with frank diabetes mellitus which is equivalent of an unmasked type 2 diabetes mellitus (T2DM) [4]. The increased endogenous productions of glucocorticoids in adiposities have been presumed to play a role in development of type 2 diabetes [5]. The risk of T2DM is higher with older age and those with increased body mass index [4,6].

In a study of patients with renal transplant who received high dose steroids for more than one year, some developed steroid –induced diabetes mellitus [2,7]. In about 50% of cases of steroid induced hyperglycaemia, the hyperglycaemia may persist despite reduction or even discontinuation of the steroid, this is especially so in patients with risk factors for diabetes mellitus [8,9].

Although steroid induced diabetes mellitus have been documented, presentation as DKA is unusual. We therefore, report a case of 13year old female who developed DKA while on tablet prednisolone for treatment of nephrotic syndrome.

Case Report

NC was a 13 year old female, who presented with 4months history of recurrent generalized body swelling and oliguria. No previous history of body swelling was identified. She did not have haematuria, joint pains or swelling. There was no family history of diabetes mellitus or kidney disease. She was not a known diabetic and had no clinical features of diabetes mellitus. On examination she had anasarca, mildly pale, not dysphonic, weight 45 kg (BMI= 20 kg/m^2). She was conscious and alert. The examinations of the respiratory and cardiovascular systems were normal. Her blood pressure was normal at 110/70 mm Hg. Investigations revealed a normal renal function with sodium of 130 mmol/l, potassium 3.1 mmol/l, bicarbonate 20 mmol/l, Urea 2.8 mmol/l, Creatinine 40 mmol/l. There was also hypoalbuminaemia of 17 g/l and hypercholesterolemia of 8.29 mmol/l. Urinalysis showed massive proteinuria and no haematuria. Packed cell volume was 25%. She was diagnosed as idiopathic nephrotic syndrome and was commenced on tablet prednisolone 20 mg three times daily. Two weeks after commencement of prednisolone, she was rushed into the Children's Emergency Ward with complaints of weakness, excessive micturition, polydyspia and vomiting. On examination, she was lethargic, in

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respiratory distress and severely dehydrated. Urgent bedside random blood sugar was unrecordably high and a concurrent venous blood sample sent to the laboratory was 31.1 mmol/l. Urinalysis showed glycosuria and ketonuria. Arterial blood gases and blood ketones was not done due to lack of facilities. Serum bicarbonate showed acidosis of 12 mmol/l. She was admitted as a case of diabetic ketoacidosis and commenced on intravenous normal saline, intravenous insulin at 0.1 iu/kg (4.5 IU) stat and subsequently continuous insulin drip of 0.1 u/kg/hour. Insulin therapy was later changed to subcutaneous soluble insulin after 48 hours at the dose of 10 units given 30 minutes before each meal. Urinalysis showed one plus glucose and no ketonuria. She continued on thrice daily dose of subcutaneous insulin and blood sugar monitored. Her fasting blood glucose (FBG) gradually normalized at levels between 3.1-3.3 mmol/l. Steroid therapy was discontinued temporarily. The insulin therapy was changed to Mixtard insulin at a dose of 20 IU in the morning and 8 IU in the evening and she was discharged home after one week on Mixtard. Mother discontinued subcutaneous insulin at home four months after diagnosis of DKA as blood glucose remained normal. She was seen in clinic one month after discontinuation of insulin with a FBG level of 2.9 mmol/l. She was gradually re-introduced recommenced on prednisolone tablets at 10 mg three times daily (low dose) and she has remained normoglycaemic with FBG levels between 3.1-4.5 mmol/l.

Discussion

Glucocorticoids are the commonest treatment option for idiopathic nephrotic syndrome with its mechanism of action as an immunosuppressive agent [10]. Glucocorticoids use is associated with many side effects including hyperglycemia, hypertension, pancreatitis, peptic ulcer etc. [11]. Glucocorticoids induced hyperglycaemia is not uncommon, but presenting with diabetic ketoacidosis is rather unusual [12,13]. Diabetic ketoacidosis is rare in patients with non-insulin dependent diabetes mellitus and also in drug induced diabetes mellitus. Our patient was commenced on prednisolone at 20 mg thrice daily for treatment of idiopathic nephrotic syndrome. She developed features of diabetic ketoacidosis after two weeks of steroid. She was not a known diabetic and had no family history suggestive of diabetes mellitus. The presentation of excessive micturition and polydipsia with findings of severe lethargy, dehydration, respiratory distress with marked hyperglycaemia and ketonuria was diagnostic of DKA. The glucocorticoid induced insulin resistance, lipolysis and ketogenesis were likely to have precipitated the diabetic ketoacidosis which have been reported in other studies [3,12,14].

Steroid induced hyperglycaemia represents a form of type 2 diabetes mellitus which is now seen commonly in obese children. However, our patient was not obese (BMI= 20 kg/m^2) and islet cell antibody (ICA) due to autoimmune destruction of the pancreatic islet cell which is an evidence of type 1 diabetes mellitus was not done for financial reasons. The age of our patient supports the findings that puberty is a risk factor for diabetes mellitus [15,16].

The result from a clamp study of 357 children by Morgan et al. [15] reported insulin resistance during puberty. Children with early puberty have an increased risk of type 2 diabetes mellitus because the period of lowered insulin sensitivity is longer in these children [17]. The effect of puberty probably explains the increase sensitivity of our patient to steroid leading to glucose intolerance and diabetes mellitus.

Diabetes induced by steroids is usually transient and easily controlled [18,19]. Forty percent of children with primary renal diseases on glucocorticoids develop deranged glucose metabolism with older age and increased body mass index as risk factors [4,6]. In about 50% of cases of steroid induced hyperglycaemia, the hyperglycaemia may persist despite reduction or even discontinuation of the steroid, this is especially so in patients with risk factors for diabetes mellitus [8,9].

The hyperglycaemic effect of prednisolone usually remits within 48 hours after discontinuation of the drug [9]. In a study of 79 children who received kidney transplant and were placed on steroids, 7 developed steroid induced diabetes mellitus. The diabetes was transient and easily controlled and no patient developed permanent insulin dependent diabetes mellitus [18]. Similarly, during the treatment of glucocorticoids induced DKA in a child with acute rheumatic fever, the insulin need of the patient progressively reduced with reduction of the glucocorticoid dose. There are limited literatures on the experience and effects of steroids on blood glucose in children with nephrotic syndrome. In our patient, withdrawal of steroid led to gradual reduction of blood glucose, with normalization of FBG r over 4 months after presentation with DKA and withdrawal of steroid.

Conclusion

DKA is a risk in all children on prolonged glucocorticoid therapy, especially in adolescents. There is therefore need for routine blood glucose estimation in all patients on prolonged steroids for two weeks or more, especially those who are pubertal and in those with a family history of diabetes mellitus to avoid development of hyperglycaemia and possibly DKA.

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