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Diabetes & its Complications

Steroid-Induced Diabetes Mellitus in A Nephrotic Adolescent: Case Report

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ABSTRACT

Steroids are widely used in all sub-specialties of medicine as anti-inflammatory or immunosuppressive agents. Glucocorticoid-induced diabetes mellitus is a known but poorly reported complication of steroid therapy in developing countries.

We report a case of steroid-induced diabetes mellitus in a 14 year old female nephritic adolescent who developed diabetic ketoacidosis while on prednisolone therapy for nephrotic syndrome. She presented with two day history of abdominal pain, one day history of frequent loose stool and vomiting. She was restless on admission, had random blood glucose of 32mmol/L and 3+ of ketonuria. She was successfully managed with insulin therapy over a four month period while she was gradually weaned off prednisolone.

Diabetes mellitus is a common life-threatening complication of steroid therapy which requires good clinical surveillance and prompt management.

Keywords

Steroid, Prednisolone, Diabetes mellitus, Nephrotic syndrome.

Introduction

Therapeutic use of glucocorticoids as anti-inflammatory and immunosuppressive drugs dates back to the mid-20th century and are widely used currently in the treatment of many diseases [1]. Glucocorticoids are extensively used in almost every subspecialty of medicine [2] and they have many side effects which include hypertension, osteoporosis, and steroid-induced diabetes [3]. Steroid induced diabetes mellitus has been recognized as a complication of glucocorticoid use for more than six decades [4]. Reports of steroid induced diabetes are rare among children in the developing countries. Though a common problem, steroid induced diabetes is difficult to detect clinically which may explain why case reports and studies on it are rare. Screening for steroid induced diabetes should be considered in all patients treated with medium to high doses of steroids.

Case Report

We present AR a 14 year old known nephrotic syndrome patient on daily maintenance steroid therapy (Prednisolone at 80mg/m^2/day) who presented with generalized abdominal pain of six hour duration. She had two episode of watery non-bloody stool and an episode of vomiting a day before presentation. She had fever a week before presentation, but no urinary or respiratory symptoms. At presentation she was conscious, acutely ill-looking, in painful distress, had facial fullness, not pale, no sign of dehydration. There was generalized abdominal tenderness more around the peri-umbilical region. The pulse rate and blood pressure were within normal range (110bpm and 80/46mmHg respectively). Examination of the respiratory system was normal. Diagnosis of sub-acute peritonitis was initially made and she was commenced on intravenous antibiotics. She became restless on the second day of admission, abdominal pain persisted, there was frequent passage of urine and she had fast breathing. She had evidence of dehydration (i.e sunken eye, dry buccal mucosal, capillary refill time was > 4 seconds). Random blood glucose was 32mmol/L,

had acidosis (bicarbonate- 17mmol/L), ketonuria 3+. An assessment of steroid induced DM with ketoacidosis was made. She was rehydrated with 0.9% normal saline and commenced on intravenous insulin infusion at 0.1 unit/ kg/ hour. Random blood glucose was monitored every 30mins to 1 hour. Dextrose containing fluid was commenced when the blood glucose was <14 mmol/L. Prednisolone was reduced to half of the dose. Third day on admission the abdominal pain subsided and oral feed was commenced when RBG was 6.0mmol/L. Insulin infusion was discontinued 2 hours after commencement of subcutaneous Mixtard 70:30 given at a dose of 45 I.U in the morning and 25 I.U at night before food The random blood glucose was monitored before and 2hour after eating. Electrolyte, urea and creatinine were monitored daily and corrected until it became normal though creatinine was marginally elevated. Urinalysis showed proteinuria of 2+, ketonuria +, glucose negative other parameters were normal. RBG normalized on subcutaneous Mixtard 70:30, repeat urinalysis became normal. She was discharged home on subcute insulin, tabs prednisolone, and spironolactone after spending five days on admission. She was followed up at the Paediatric endocrinology and nephrology clinic where she was gradually weaned off prednisolone and subcutaneous insulin over a four-month period. She has since remained normoglycaemic without insulin.

Discussion

Glucocorticoid induced diabetes mellitus is defined as an abnormal increase in blood glucose associated with the use of glucocorticoids in a patient with or without a prior history of diabetes mellitus [5]. The exact prevalence of steroid induced diabetes is not known. The incidence of hyperglycaemia (defined as blood glucose > 200mg/dl) in hospitalized patients without a known history of diabetes who are treated with corticosteroids is over 50% [6]. Not all patients treated with glucocorticoids develop steroid induced diabetes; a number of predictors of onset of steroid induced diabetes which have been identified include: the dose and duration of steroid treatment, age, weight, previous glucose intolerance, reduced sensitivity to insulin or impaired insulin secretion stimulated by glucose, a family history of diabetes and race [1,3,7,8]. Early withdrawal from steroid is a protective factor and the gender of patient was not found to be a predictor [1,3,7,8].

Steroid is believed to negatively affect glucose metabolism in through different mechanism including: increased insulin resistance, increased glucose intolerance, β-cell dysfunction resulting in reduced β-cell mass, and impaired suppression of hepatic glucose production resulting from increased hepatic insulin resistance [9,10]. Steroid also reduces peripheral glucose uptake at the level of the muscle and adipose tissue [11,12]. Chronic exposure to steroid alters body composition, including the expansion of adipose tissue depots in the trunk, and impairs metabolism and insulin action, resulting in hyperglycaemia and dyslipidaemia [9]. The ability of steroid to induce hyperglycaemia depend on their dose and the duration of administration [9,13]. It is well known that glucocorticoid therapy may provoke new onset type 2 diabetes mellitus and invariably worsens hyperglycaemia in patients with preexisting diabetes mellitus [14]. Glucocorticoids are also known to cause of hyperosmolar hyperglycaemic nonketotic syndrome [15]. which require aggressive hydration and insulin therapy. Insulin therapy is generally the treatment of choice in glucocorticoid-induced diabetes mellitus because of its efficacy and safety compared to various types of oral hypoglycaemic agents. Insulin provides an immediate onset of action, can easily be titrated and has unlimited hypoglycaemic power. The different types of insulin available also allow clinician to prepare administration schedule appropriate for patients on different corticosteroid schedule [16,17]. Our patient was successfully managed with soluble insulin infusion at the critical stage and maintained on subcutaneous premixed short and intermediate acting insulin (Mixtard 70:30) over four months and she recovered completely.

Conclusion

Steroid induced diabetes mellitus is a common and life-threatening complication of glucocorticoid therapy in children. Clinicians should routinely screen for hyperglycaemia in patients placed on glucocorticoids and get the endocrinologist involved early in their patients' management.

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