Journal of Intensive and Critical Care
ISSN:2471-8505

Open access Case Report

Diabetic Ketoacidosis Precipitated by Latrogenic Thyroxin Toxicosis: A Critical Care Admission

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ABSTRACT

We report a presentation of severe Diabetic Ketoacidosis (DKA) due to Thyroxin (T4) toxicosis admitted to our Intensive Care Unit. Excess ingestion of Thyroxin over a three month period, resulted from an inadvertent prescribing error. Accepting that there were failures of our safeguards across the primary and secondary care setting, we highlight and share our case and the importance on identifying an atypical precipitant of DKA.

Keywords: Intensive care; Diabetic ketoacidosis; Fetal outcomes

INTRODUCTION

A 59 year old female presented with acute confusion, hyper-glycaemia, progressive lethargy and vomiting to our emergency department. A history of 1 stone weight loss over three weeks was reported. Her medical history included Insulin Dependent Diabetes Mellitus, Hypertension, Hypothyroidism (recent diagnosis 3 months prior to admission), BMI 39.2 and alcohol excess. Diabetic control was poor, HbA1c 94 mmol/L, her last admission for DKA was in 2013.

CASE PRESENTATION

On assessment, the patient was notably confused with a Glasgow Coma Scale (GCS) of 14/15, Kussmaul breathing rate at 32 bpm, 100% oxygen saturation and a stable blood pressure of 152/62 mmHg. Sinus tachycardia of 125 bpm, confirmed on Electrocardiogram (ECG), persisted after fluid resuscitation. Chest auscultation was clear with no features of infection on the chest x-ray. Mottled skin was noted with cool peripheries and hypothermic at 33°C. In keeping with DKA, she was hyper-glycaemic 29.3 mmol/L and ketonemic 5.4 mmol/L, initial Arterial Blood Gas (ABG) which confirmed severe metabolic acidosis with a large base deficit, requiring critical care input (Table 1).

Table 1: Laboratory blood tests.

Thyroid	Result	Reference	Haematology	Result	Reference
TSH (mU/L)	0.01	0.27-4.5	Haemoglobin (g/L)	125	117-149
Free T4 (pmol/L)	33.2	11-23	White Cell Count (109/L)	20.3	4.3-11.2
	Diabetes		Platelets (109/L)	406	150-400
Glucose (mmol/L)	32.6	3.0-6.0		**Biochemistry**	
HbA1c (mmol/mol)	94	20-41	Sodium (mmol/L)	137	133-146
Urinary Ketones	4+	-	Potassium (mmol/L)	4.1	3.5-5.3
	ABG		Urea (mmol/L)	5.8	2.5-7.8
pH	6.9	7.35-7.45	Creatinine (µmol/L)	51	45-84

Received:	30-May-2022	**Manuscript No:**	ipjicc-22-13844
Editor assigned:	01-June-2022	**PreQC No:**	ipjicc-22-13844 (PQ)
Reviewed:	15-June-2022	**QC No:**	ipjicc-22-13844
Revised:	20-June-2022	**Manuscript No:**	ipjicc-22-13844(R)
Published:	27-June-2022	**DOI:**	10.35248/2471-8505-8.6.87

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Citation Samad F, Mohmed Z, Shirgaonkar A (2022) Diabetic Ketoacidosis Precipitated by Latrogenic Thyroxin Toxicosis: A Critical Care Admission. J Intensive Crit Care. 8:87.

pCO2 (kPa)	3.1	10-13.3	CRP (mg/L)	40	0-5
pO2 (kPa)	7.3	4.7-6.0	ALT (U/L)	55	0-33
Glucose (mmol/L)	29.3	9-22	ALP (U/L)	382	30-130
BE (mmol/L)	-27.3	0 ± 2	GGT (U/L)	389	6-42
HCO3- (mmol/L)	-4.4	23.0-28.0			

DKA treatment protocol was commenced, and empirical antibiotics started. Laboratory blood tests showed in confirmed a hyperthyroid state (Table 1), raised free Thyroxine 33.4 pmol/L and suppressed thyrotropin (TSH) 0.01 mU/L. The patients care was escalated to the Intensive Care Unit. A Fixed Rate Insulin Infusion commenced, IV fluid therapy and Levothyroxine withheld. Our patient swiftly recovered within 48 hours of treatment with full resolution of DKA and subsequently discharged to the ward.

Upon further collateral history and review of medical notes, found that there was an adverse prescribing error on the hospital discharge letter dated three months prior where a diagnosis of hypothyroidism and probable Poly-glandular Syndrome Type II. She was to be discharged to commence Levothyroxine 50 micrograms once daily with further follow up and the General Practitioner (GP) was advised to review in two weeks' time with thyroid function tests. However, the dose of Levothyroxine had been transcribed as 500 micrograms once daily instead, ten times higher than the prescribed dose.

RESULTS AND DISCUSSION

There have been reports of patients concomitantly presenting in hyperthyroid states with DKA [1-5]. Although this simultaneous presentation is unusual and uncommon, DKA may mask thyrotoxicosis or thyroid storm, identification can prevent a fatal outcome as such in reports leading up to a cardiac arrest [6,7]. This case highlights the importance to consider uncommon causes which may be masked. In our case we were able to identify the associated cause due to collateral history and medical notes, which may not be available in every case.

DKA is metabolic disorder and an endocrinological emergency. Characteristic of DKA is a triad of hyper-glycaemia, metabolic acidosis and ketonaemia secondary to insulin deficiency. Various causes such underlying infection or a concurrent illness, trauma, surgical stress, poor insulin compliance and uncommonly iatrogenic thyroxin toxicosis may precipitate DKA [8,9].

Glycaemic control is known to worsen in hyperthyroid states, glucose metabolism is modulated potentiating the development of DKA [1,10]. This occurs *via* several mechanisms. The activity of insulin is reduced and resistant, reducing the peripheral glucose uptake, glycogen stores are depleted, and gut absorption of glucose is increased [2,3,9,11]. There is an uptake of GLUT2 receptors at the plasma membrane of hepatocytes, subsequently contributing to increased hepatic glucose output, additionally, there is a high turnover of free fatty acids (FFA) due to increased lipolysis, aiding hepatic gluconeogenesis [3,11]. By these mechanisms diabetic patients with hyperthyroidism have the potential to precipitate DKA due to an overall impairment of their glycaemic control.

Point score using the Burch-Wartofksy scale would at least be 25 for this patient and therefore is unlikely to suggest that this patient was in a thyroid storm [12]. Overall identifying a precipitating factor for DKA should be sought and all possible causes considered in those patients whom the reason for presenting with DKA is unclear or there is resistance to treatment. Additional sources of information should be sought, forming an accurate history aids identifying and directing treatment. In our case, as the patient was confused, we were able to obtain collateral history and in addition to reviewing the medical notes, were able to identify this adverse drug event early.

CONCLUSION

We have shared our case of an adverse event as a consequence of iatrogenic Thyroxin Toxicosis which precipitated DKA. A life threatening event in which if the underlying cause had not been identified could have resulted to serious consequences. We have duly apologised to the patient and family for this adverse event, duty of candour has been given. Following submission of an incident report, subsequent discussions in our risk management have taken place and lessons learnt have been disseminated to ensure patient safety. We highlight the important of counselling in diabetic and thyroid patients of the potential serious complications that can occur.

ACKNOWLEDGMENT

No competing interest for any author

DECLARATION OF CONFLICTING INTERESTS

The authors declared no potential conflicts of interest for the research, authorship, and/or publication of this article.

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