Euglycemic Diabetic Ketoacidosis A Dreaded Complication in Post-Operative Patients In ICU

Dr. Wasim Feroz[1], Dr. Sandeep Nale[2], Dr. H. S. Rawat[3]

[1]Resident, [2]Assistant Professor, [3]Professor & Head, Department of Anaesthesia, DVVPFs Medical College & Hospital, Ahmednagar-414111. (MS) India

Corresponding Author : Dr. Wasim Feroz

E-mail : wasimferoz69@gmail.com

Address : Department of Anaesthesia, DVVPFs Medical College & Hospital, Ahmednagar-414111. (MS) India

Abstract :

Diabetic ketoacidosis (DKA) is a life threatening metabolic disorder and a known complication of Diabetes mellitus, caused by insulin deficiency or insulin resistance, which allows the catabolism of free fatty acids into ketone bodies, with high blood sugar levels. A rare variant of it is, euglycemic diabetic ketoacidosis (EKDA). We report a case of 60 year old female who was posted for Posterior lumbar interbody fusion (PLIF) and had this complication postoperatively. We believe that this case would serve as a reminder to all practitioners and intensivist to consider ketosis in a diabetic patient despite their serum glucose levels being within the normal range and all anaesthetist to consider the use of insulin during the intra op period even the blood sugar level is normal in peri operative period. This case report summarizes, in brief, the etiology, pathophysiology and treatment of EDKA.

Keywords : Euglycemic diabetic ketoacidosis, metabolic acidosis, anion gap, ketonemia, ketonuria

Introduction:

EKDA is variant of DKA with clinical triad of increased anion gap metabolic acidosis, ketonemia or ketonuria and normal blood glucose levels <200 mg/dL.[1,3] It is usually seen in patients who are on insulin therapy or with the triggering factors like starvation or dehydration.[4] Diagnosis of EDKA is challenging because of normal blood sugar level. Early diagnosis can lead to favourable outcome or else can turn fatal.

Case report:

A 50 year old female, weight 55kg k/c/o hypertension since 5 years on Tablet. Telmisartan 40mg once day, diagnosed with Prolapse intervertebral disc (PID) posted for PLIF came for pre anaesthetic check-up. During pre-anaesthesia check-up, she complains of breathlessness on exertion and relived on rest (NYHA II). Rest her preanesthetic, evaluation was unremarkable. All relevant investigations including electrocardiogram (ECG) and two-dimensional echocardiography were normal except blood sugar level which was above the normal level, F-240mg/dl, PP-334 mg/dl. Patient was advised for physician opinion to control blood sugar level and HbA1C. Patient was started on insulin therapy Human actrapid insulin 10-10-8 units and after 5 days of optimisation, she was posted for the surgery under American Society of Anaesthesiology (ASA) II.

On the day of surgery, before induction patient was vitally stable with BSL of 116 mg/dl. Patient was induced with combined spinal and general anaesthesia. Premedication was with Injection. Ondensetron 4 mg, Injection. Ranitidine 50mg, Injection, Glycopyrolate 0.2 mg, Injection. Nalbuphine 5 mg, Intravenous Fluid (IVF) – Ringer Lactate (RL). Spinal anaesthesia was given by Bupivacaine 0.5 % Heavy 2.8 ml + 15 μg Fenatanyl & General Anaesthesia was given by Injection. Propofol 150 mg + Injection. Rocuronium 50 mg, Intubated with Endotracheal Tube 7.0↓ Direct laryngoscopic Vision after triple manoeuvre and preoxygenation for 3 minutes. Maintenance was with O2 (2) + N2O (2) + Isoflurane (0.8-1) Vol %, (To give Hypotensive Anaesthesia) & Injection. Atracurium 5 mg every half an hour and 4 RL. Intraoperatively her Pulse Rate (PR) was monitored with pulse oximetry along with oxygen saturation (SpO2), non-invasive blood pressure (BP), EtCo2 and ECG. During the surgery, her hemodynamic parameters were stable with Pulse Rate varying between 68/min and 84/min, systolic BP 90-100 mmHg, diastolic BP 50-60 mmHg and SpO2 100%.

Hourly reading of BSL was between 120mg/dl – 140 mg/dl with a urine output of 100ml/hr.

The intra-operative period was for 3 hours, & during the end of surgery and just before extubation, there was a sudden Drop in BP- 57/37 mm/Hg, 60/40 mm/Hg , and the respiratory effort was not good. We planned not to extubate and shift her to ICU for keep her on mechanical ventilator support. Ionotropic support was started (Dopamine @ 20 mg/hr) & adequate fluid was administered. After 1 hour Patient was semiconscious, drowsy & hypotensive. BSL was 149mg/dl. On examination patient was having crepts and wheeze in the lower zones of chest with no peripheral edema, with accessory use of respiratory muscle. Later, she was started on Nor Adrenaline infusion (0.4 mg/ml), colloid (Voluven) & to find the cause of hypotension and cardiac status, central line was introduced, and Central Venous Pressure (CVP) was 7 cm of H2O & Arterial Blood Gas (ABG) was done. In spite of all the resuscitative measures patient was vitally, hemodynamically and clinical unstable.

Diagnosis, Intervention & Outcome: Patient's blood glucose levels were checked, which was within normal range. An arterial blood gas analysis revealed high anion gap metabolic acidosis and hypokalemia. Therefore we decided to check for urine ketones, which was strongly positive & diagnosis of EDKA was made. She was treated with 4L bolus of Intra Venous (IV) normal saline and an insulin drip as per the protocol. The basic metabolic profile was monitored every 4 hour, and serum glucose levels were checked every hour. She was weaned off the ventilator over next 24 hours. Subsequent course was uneventful & she was discharged after 3 days.

Discussion

DKA is defined as having a combination of hyperglycemia (serum glucose >250 mg/ dL), acidosis (arterial pH <7.3 and bicarbonate <15 mEq/L) and ketosis (moderate ketonuria or ketonemia).[1] Glycemic control is achieved in our human body using a balance between the insulin levels and the levels of counter-regulatory hormones like glucagon, growth hormone, glucocorticoids and epinephrine. DKA occurs when there is either a decrease in insulin or when there is an excess of counter-regulatory hormones both of which causes hyperglycemia. Though there is hyperglycemia, the end organs are unable to utilize the available glucose due to the comparative lack of insulin, and this leads to lipolysis thereby leading to excessive production of ketone bodies.[2] The underlying mechanism of EDKA is either due to decreased hepatic production of glucose during fasting state or enhanced urinary excretion of glucose induced by an excess of counter-regulatory hormones. Thus, when a diabetic patient is exposed to any triggering factor for DKA and is fasting or starving while continuing the insulin treatment regularly, the liver will be in a state of glycogen depletion, thereby producing a lesser amount of glucose. On the other hand, there will be lipolysis and fatty acid production, which finally leads to excessive ketone body production.[3] Burge et al had reported in their study that short-term fasting is a well-known mechanism of developing euglycemic ketoacidosis when there is insulin deficiency.[4] Once diagnosed, management of EDKA is simple & is almost similar to the management of DKA, i.e. rapid correction of dehydration using intravenous fluids and the use of insulin drip along with a dextrose containing solution until the anion gap, and bicarbonate levels normalize. If diagnosed early and management aggressively with fluids and insulin drip, EDKA may be easily reversed, thus minimizing morbidity and mortality.

Conclusion:

High clinical suspicion is required to diagnose EDKA and should be considered as one of the diagnosis by an intensivist because normal blood sugar levels masquerade the underlying DKA and cause a diagnostic and therapeutic dilemma. It is also advisable for anaesthetist to start the insulin drip as per the protocol for the patients who are on regular insulin therapy during the intra op period.

References:

1. Nyenwe EA & Kitabchi AE 2016 The evolution of diabetic ketoacidosis: an update of its etiology, pathogenesis and management. Metabolism 65 507–521. (doi:10.1016/j. metabol.2015.12.007).
2. Laffel L 1999 Ketone bodies: a review of physiology, pathophysiology and application of monitoring to diabetes. Diabetes/Metabolism Research and Reviews 15 412–426. (doi:10.1002/(SICI)1520-7560(199911/12)15:6<412::AID-DMRR72>3.0.CO;2-8).
3. Kitabchi AE, Umpierrez GE, Miles JM & Fisher JN 2009 Hyperglycemic crises in adult patients with diabetes. Diabetes Care 32 1335–1343. (doi:10.2337/dc09-9032).
4. Burge MR, Hardy KJ & Schade DS 1993 Short-term fasting is a mechanism for the development of euglycemic ketoacidosis during periods of insulin deficiency. Journal of Clinical Endocrinology and Metabolism 76 1192–1198. (doi:10.1210/jc.76.5.1192).