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Review article

DIABETIC KETOACIDOSIS: A REVIEW OF CURRENT TREATMENT OPTIONS

D.Durga Prasad*, N.L.Swathi, M.Mona Sree, Preethi S, P.Sirisha, M.Gayathri, S.Bhavani

Pharm.D, Sri Venkateswara College of Pharmacy, RVS Nagar, Tirupathi Road, Chittoor, Andhra Pradesh, India, 517 127.

ABSTRACT

The first presentation of T1DM in adults is becoming less prevalent, although it continues to be widespread in the pediatric population due to the increasing prevalence of DKA. If a patient presents with DKA for the first time, it is likely that the underlying diagnosis is T1DM. This is especially true if the patient has a short history of symptoms, is young, and has a normal BMI. The condition known as DKA, also known as ketosis-prone type 2 diabetes or 'Flatbush' diabetes. physiological aims for metabolic treatment include: > Reduction of blood ketone concentration by 0.5 mmol/L/hour; > Increase of venous bicarbonate by 3.0 mmol/L/hour; > Reduction of capillary blood glucose by 3.0 mmol/L/hour; and > Maintenance of potassium levels between 4.0 and 5.5 mmol/L.If these targets are not met, then the FRIII rate should be increased in order to compensate. Despite NICE recommendations, access to psychological treatments for patients with type 1 diabetes is unsatisfactory in many places. Diabetes specialist teams should be contacted within 24 hours of any hospitalization for DKA, and the patient should be reviewed by a specialist before being discharged. This education support should include the following elements in order to promote best practice: a review of the normal glycemic control procedures Discussion on sick day policies, assessment of the requirement for home ketone testing (blood or urinary) and education to enable this, as well as contact telephone numbers for the diabetes specialist team, including after-hours service.

Key Words:- DKA, DM, Review study.

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Corresponding Author

Dr.D.Durga Prasad
Sri Venkateswara College of Pharmacy, RVS Nagar, Tirupathi Road, Chittoor, Andhra Pradesh, India.

E-mail: drdurgaprasad1998@gmail.com

INTRODUCTION

Despite the fact that diabetic ketoacidosis (DKA) is a completely preventable illness, it is not a rare presentation to the hospital. DKA is caused by a decrease in effective circulating insulin, which is accompanied by an increase in counter-regulatory hormones in the bloodstream. This potentially life-threatening consequence of type 1 diabetes mellitus (T1DM) is frequently mismanaged, resulting in morbidity and an increased duration of stay in the hospital setting. Mortality rates have decreased dramatically over the previous 20 years, and now account for less than 1 percent of the population. Therapy while in the hospital. > Infection: most typically chest, urinary tract, and skin infections. > Acute coronary syndrome or via Advanced near-patient testing technology has enhanced patient care by enabling more rapid diagnosis and more frequent monitoring of therapy response, both of which are beneficial to patients. An alarming number of persons acquire diabetic ketoacidosis (DKA) while already in the hospital, according to the findings of the National Diabetes Inpatient Audits [2] Newly diagnosed type 1 diabetes > Poor compliance with insulin administration. > Inadequate insulin event.

Is it T1DM if you get a new diagnosis of diabetes that presents with DKA?

The first presentation of T1DM in adults is becoming less prevalent, although it continues to be widespread in the pediatric population due to the increasing prevalence of DKA. [3] If a patient presents with DKA for the first time, it is likely that the underlying diagnosis is T1DM. This is especially true if

the patient has a short history of symptoms, is young, and has a normal BMI. The condition known as ABSTRACT DKA, also known as ketosis-prone type 2 diabetes or 'Flatbush' diabetes, can occur in some people with type 2 diabetes mellitus (T2DM). A genetic component may be involved: it is more common in people of African-Caribbean descent, and triggers such as severe intercurrent illness or metabolic stress may also play a role. The absence of typical autoantibodies associated with a diagnosis of T1DM (glutamic acid decarboxylase, insulin autoantibody 2, zinc transporter 8) raises the suspicion of T2DM; however, results typically take more than a week to come back, making them ineffective for acute management of the condition. Initially, any patient presenting with DKA should be assumed to have type 1 diabetes and should be treated accordingly.

Management of DKA :

The Joint Diabetes Societies issued a national guideline for managing diabetic ketoacidosis (DKA) in 2010 (which was modified in 2013), in an effort to eliminate diversity in DKA management standards between hospitals, and so perhaps improve the overall standard of care. [4]

The most important elements of DKA management:

The most critical initial therapeutic intervention in DKA is adequate fluid replenishment, which is followed by insulin infusions. The primary goals of fluid replacement are as follows: > restoration of circulatory volume, > clearance of ketones, and > correction of electrolyte abnormalities, insulin should be supplied intravenously at a constant rate based on body weight using the following formula: 0.1 units per kilogram of body weight each hour for every kilogram of body weight. It may be essential to estimate the patient's weight; nevertheless, treatment should not be postponed until a precise weight has been obtained. Intravenous insulin infusion at a fixed rate (FRIII) not only lowers blood glucose levels, but it also has the additional benefit of suppressing further ketogenesis while also restoring electrolyte balance. It is necessary to monitor biochemical parameters on a regular basis, including glucose, capillary ketones, venous pH, and serum potassium. When assessing acid-base status, it is not necessary to draw arterial blood; instead, venous sampling is sufficient because the difference between arterial and venous pH/HCO3 is not large enough to alter the diagnosis or management of DKA.[1,2,3]

Physiological aims for metabolic treatment include: > Reduction of blood ketone concentration by 0.5 mmol/L/hour; > Increase of venous bicarbonate by 3.0 mmol/L/hour; > Reduction of capillary blood glucose by 3.0 mmol/L/hour; and > Maintenance of potassium levels between 4.0 and 5.5 mmol/L. If these targets are not met, then the FRIII rate should be increased in order to compensate. Considering that clearing ketones is equally important as normalizing blood glucose, it is frequently necessary to administer intravenous 10 percent dextrose in order to avoid hypoglycemia and allow continued FRIII to suppress ketogenesis; 10 percent dextrose should be administered when the blood glucose falls below 14.0 mmol/L. It is critical to maintain the 0.9 percent sodium chloride solution in order to maintain proper circulatory volume, which means that it is frequently required to infuse both solutions at the same time . DKA is resolved by a number of means. pH greater than 7.3 units > Bicarbonate greater than 15.0 mmol/L Ketone levels in the blood are less than 0.6 mmol/L.[4,5]

Troubleshooting:

If the DKA does not resolve and the treatment goals are not being met, the cannula's patency and location should be evaluated. Check to see that the correct rate of intravenous infusions (FRIII and fluids) has been provided to the patient. Examine the patient again for signs of concurrent pathology, such as intra-abdominal infection or myocardial infarction. Consider whether insulin resistance is likely due to factors such as obesity, concurrent steroid medication, or an increase in the rate of FRIII. Reevaluate hydration status and consider increasing the rate at which intravenous (IV) fluids are administered. Subcutaneous insulin with a lengthy half-life should be continued. When IV treatment is discontinued, the patient's basal (long acting) analogue insulin (e.g., Levemir/detemir, Lantus/glargine, Tresiba/degludec) should be continued in conjunction with the FRIII to prevent rebound hyperglycemia. If a new diagnosis of T1DM is made in a patient who presents with DKA, basal insulin should be started as soon as possible, and IV insulin should be continued until there is some basal subcutaneous (SC) insulin available. In the case of newly diagnosed individuals requiring conversion to SC insulin, consult with a professional. It is appropriate to just restart the SC insulin regimen that was in place prior to DKA. However, if the patient's glycemic management was inadequate before to admission (high HbA1c, recurrent hypoglycemia), a medication reassessment is recommended.[6,7]

The patient can resume SC insulin therapy if the DKA has cleared and he or she is able to eat and drink normally again. It is critical that the intravenous insulin infusion is not stopped until at least 30–60 minutes after the delivery of the SC insulin dose taken with a meal has taken place. The patient's basal insulin should have been continued at the same time that the FRIII was administered. It is important to note that if the basal insulin was accidentally interrupted, the insulin infusion should not be terminated until some kind of background insulin has been administered, such as a stat dose of Insulin equal to half the patient's regular daily dose of basal insulin. If the patient was previously on twice-daily

fixed-mix insulin (e.g. NovoMix 30), they should resume their normal SC insulin either before breakfast or before dinner, as needed. Allow 30–60 minutes after the SC insulin was administered before stopping the IV insulin infusion. It is recommended that decisions on SC insulin treatment (such as which regimen to use, which insulin to use, and what doses to use)[8,9,10] in newly diagnosed patients be made with the assistance of the diabetes specialist team. The patient should be given a variable rate insulin infusion and IV fluids appropriate to their fluid status if their DKA has subsided but they have not yet been given the opportunity to eat and drink normally. In specific cases, DKA may be necessary.

Pregnancy DKA is a serious concern to both the mother and the foetus, with a risk of foetal death due to the toxicity of ketones to the foetus in pregnancy. Female patients should be treated in the birth suite or on the high dependency unit, where they will receive collaborative medical and obstetric care. It's important to remember that in women, DKA can manifest as stomach pain and can develop with only a very slight spike in glucose levels.[11,12] Maintain conventional DKA protocol, with early obstetrics evaluation and foetal monitoring, if necessary, depending on the stage of pregnancy in which the patient is.[5] When calculating the initial FRIII dose, use the booking weight;[13] however, because pregnancy is a condition of insulin resistance, the insulin infusion rate may need to be increased to compensate. Pregnancy can occasionally be the cause of DKA, and all women of reproductive age who present with DKA should be evaluated for pregnancy.

Uses of the pump:

Insulin pump therapy, also known as CSII (continuous subcutaneous insulin infusion), is used by people who do not need to take any long-acting insulin supplements. In this case, if there is any interruption in insulin delivery (for example, if the cannula is blocked or dislodged), hyperglycemia and then ketoacidosis can develop very quickly unless the problem is identified and corrected,[14] for example, by re-positioning the cannula, changing the tubing, or initiating alternative insulin such as an intravenous infusion, within minutes. When a pump user gets DKA, the CSII becomes unreliable due to decreased tissue perfusion, which affects insulin absorption and glucose tolerance. DKA treatment should be initiated as soon as possible once the pump therapy has been temporarily ceased.[15, 16] As soon as the DKA has subsided, CSII can be restarted at the patient's usual basal rate; however, intravenous insulin infusion should be continued until the meal bolus has been administered.

CONCLUSION:

Preventing recurrent DKA is important. An unusually high proportion of acute hospitalizations for DKA occur in a limited group of persons with type 1 diabetes. These are frequently young persons who are experiencing psychosocial difficulties, and poor compliance with insulin medication is typical. When it comes to diabetes control, support and education are more important than having to switch to a different insulin regimen. Despite NICE recommendations, access to psychological treatments for patients with type 1 diabetes is unsatisfactory in many places. Diabetes specialist teams should be contacted within 24 hours of any hospitalization for DKA, and the patient should be reviewed by a specialist before being discharged. This education support should include the following elements in order to promote best practice: a review of the normal glycemic control procedures Discussion on sick day policies, assessment of the requirement for home ketone testing (blood or urinary) and education to enable this, as well as contact telephone numbers for the diabetes specialist team, including after-hours service.

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REFERENCES

Evans K. Diabetic ketoacidosis: update on management. Clin Med (Lond). 2019 Sep;19(5):396-398. doi: 10.7861/clinmed.2019-0284. PMID: 31530688; PMCID: PMC6771342.

Thiem U, Heinze G, Segel R, Perkmann T, Kainberger F, Mühlbacher F, Hörl W, Borchhardt K. VITA-D: cholecalciferol substitution in vitamin D deficient kidney transplant recipients: a randomized, placebo-controlled study to evaluate the post-transplant outcome. Trials. 2009 May 29;10:36. doi: 10.1186/1745-6215-10-36. PMID: 19480654; PMCID: PMC2701431.

Banerjee D, Chitalia N, Ster IC, Appelbaum E, Thadhani R, Kaski JC, Goldsmith D. Impact of vitamin D on cardiac structure and function in chronic kidney disease patients with hypovitaminosis D: a randomized controlled trial and meta-analysis. Eur Heart J Cardiovasc Pharmacother. 2021 Jul 23;7(4):302-311. doi: 10.1093/ehjcvp/pvz080. PMID: 31830258; PMCID: PMC8302255.

Cianciolo G, Cappuccilli M, Tondolo F, Gasperoni L, Zappulo F, Barbuto S, Iacovella F, Conte D, Capelli I, La Manna G. Vitamin D Effects on Bone Homeostasis and Cardiovascular System in Patients with Chronic Kidney Disease and Renal Transplant Recipients. Nutrients. 2021 Apr 25;13(5):1453. doi: 10.3390/nu13051453. PMID: 33922902; PMCID: PMC8145016.

Kalantar-Zadeh K, Ganz T, Trumbo H, Seid MH, Goodnough LT, Levine MA. Parenteral iron therapy and phosphorus homeostasis: A review. Am J Hematol. 2021 May 1;96(5):606-616. doi: 10.1002/ajh.26100. Epub 2021 Feb 9. PMID: 33471363; PMCID: PMC8248123.

Ahmed B, Nasir K, Mehmood A, Abid MA, Zehra NA, Khan AH, Hussain SA, Kashif WU, Iqbal R. Effect of physical activity and vitamin D compared with vitamin D alone on muscle strength, back flexibility and aerobic activity in patients with chronic kidney disease: A comparative study from Pakistan. Asia Pac J Clin Nutr. 2021 Dec;30(4):566-572. doi: 10.6133/apjcn.202112_30(4).0002. PMID: 34967184.

Bover J, Gunnarsson J, Csomor P, Kaiser E, Cianciolo G, Lauppe R. Impact of nutritional vitamin D supplementation on parathyroid hormone and 25-hydroxyvitamin D levels in non-dialysis chronic kidney disease: a meta-analysis. Clin Kidney J. 2021 Feb 5;14(10):2177-2186. doi: 10.1093/ckj/sfab035. PMID: 34603696; PMCID: PMC8483691.

Arenas Jimenez MD, González-Parra E, Riera M, Rincón Bello A, López-Herradón A, Cao H, Hurtado S, Collado S, Ribera L, Barbosa F, Dapena F, Torregrosa V, Broseta JJ, Soto Montañez C, Navarro-González JF, Ramos R, Bover J, Nogués-Solan X, Crespo M, Dusso AS, Pascual J. Mortality in Hemodialysis Patients with COVID-19, the Effect of Paricalcitol or Calcimimetics. Nutrients. 2021 Jul 26;13(8):2559. doi: 10.3390/nu13082559. PMID: 34444716; PMCID: PMC8401800.

Al-Hashimi N, Abraham S. Cholecalciferol. 2021 Aug 27. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan–. PMID: 31747175.

Drugs and Lactation Database (LactMed) [Internet]. Bethesda (MD): National Library of Medicine (US); 2006–. Vitamin D. 2021 Oct 18. PMID: 29999973.

1Giannini S, Passeri G, Tripepi G, Sella S, Fusaro M, Arcidiacono G, Torres MO, Michielin A, Prandini T, Baffa V, Aghi A, Egan CG, Brigo M, Zaninotto M, Plebani M, Vettor R, Fioretto P, Rossini M, Vignali A, Fabris F, Bertoldo F. Effectiveness of In-Hospital Cholecalciferol Use on Clinical Outcomes in Comorbid COVID-19 Patients: A Hypothesis-Generating Study. Nutrients. 2021 Jan 14;13(1):219. doi: 10.3390/nu13010219. PMID: 33466642; PMCID: PMC7828675.

Lebwohl M, Kircik L, Lacour JP, Liljedahl M, Lynde C, Mørch MH, Papp KA, Perrot JL, Gold LS, Takhar A, Thaçi D, Warren RB, Wollenberg A. Twice-weekly topical calcipotriene/betamethasone dipropionate foam as proactive management of plaque psoriasis increases time in remission and is well tolerated over 52 weeks (PSO-LONG trial). J Am Acad Dermatol. 2021 May;84(5):1269-1277. doi: 10.1016/j.jaad.2020.09.037. Epub 2020 Sep 18. PMID: 32950546.

Fassio A, Gatti D, Rossini M, Benini C, Fracassi E, Bertoldo E, Viapiana O, Milleri S, Gatti M, Adami G. Pharmacodynamics of Oral Cholecalciferol in Healthy Individuals with Vitamin D Deficiency: A Randomized Open-Label Study. Nutrients. 2021 Jul 2;13(7):2293. doi: 10.3390/nu13072293. PMID: 34371803; PMCID: PMC8308331.

Banerjee D, Chitalia N, Ster IC, Appelbaum E, Thadhani R, Kaski JC, Goldsmith D. Impact of vitamin D on cardiac structure and function in chronic kidney disease patients with hypovitaminosis D: a randomized controlled trial and meta-analysis. Eur Heart J Cardiovasc Pharmacother. 2021 Jul 23;7(4):302-311. doi: 10.1093/ehjcvp/pvz080. PMID: 31830258; PMCID: PMC8302255.

Cianciolo G, Cappuccilli M, Tondolo F, Gasperoni L, Zappulo F, Barbuto S, Iacovella F, Conte D, Capelli I, La Manna G. Vitamin D Effects on Bone Homeostasis and Cardiovascular System in Patients with Chronic Kidney Disease and Renal Transplant Recipients. Nutrients. 2021 Apr 25;13(5):1453. doi: 10.3390/nu13051453. PMID: 33922902; PMCID: PMC8145016.

Kalantar-Zadeh K, Ganz T, Trumbo H, Seid MH, Goodnough LT, Levine MA. Parenteral iron therapy and phosphorus homeostasis: A review. Am J Hematol. 2021 May 1;96(5):606-616. doi: 10.1002/ajh.26100. Epub 2021 Feb 9. PMID: 33471363; PMCID: PMC8248123...

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