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EXPERIENCE REPORT

Diabetes and its chronic decompensation: an experience report

Diabetes y su descompensación crónica: relato de experiencia

Diabetes e sua descompensação crônica: um relato de experiência

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Abstract

Diabetic Ketoacidosis (DAC) is a complication that needs to be understood so that its warning signs are identified by health professionals and so that control measures can be taken, thus avoiding such a problem, so the aim was to understand the decompensation metabolic pathway that promotes CAD in patients with DM. This is an experience report elaborated from the analysis of intervention data and clinical evolution of a health service user who presented CAD due to decompensated DM and died even after the medical procedures. DKA is a hyperglycemic emergency that requires prompt medical intervention. In this sense, early diagnosis and treatment of DM is important because it prevents it from evolving to CAD. However, the study presented shows the opposite, since the patient was unaware that she had DM, so she did not undergo any treatment and died due to complications from DM.

Descriptors: Diabetic Ketoacidosis; Diabetes Complications; Hyperglycemia; Insulin; Intensive Care Units.

Resumén

La Cetoacidosis Diabética (CAD) es una complicación que requiere ser comprendida para que sus signos de alarma sean identificados por los profesionales de la salud y poder tomar medidas de control, evitando así dicho problema, por lo que el objetivo fue conocer la vía metabólica de descompensación que promueve EAC en pacientes con DM. Se trata de un relato de experiencia elaborado a partir del análisis de datos de intervención y evolución clínica de un usuario de un servicio de salud que presentó EAC por DM descompensada y falleció incluso después de los procedimientos médicos. La CAD es una emergencia hiperglucémica que requiere una intervención médica inmediata. En este sentido, el diagnóstico y tratamiento precoz de la DM es importante porque evita que evolucione a EAC. Sin embargo, el estudio presentado demuestra lo contrario, ya que la paciente desconocía que padecía DM, por lo que no realizó ningún tratamiento y falleció por complicaciones de la DM.

Descriptores: Cetoacidosis Diabética; Complicaciones de la Diabetes; Hiperglucemia; Insulina; Unidades de Cuidados Intensivos.

Resumo

A Cetoacidose Diabética (CAD) é uma complicação que necessita ser compreendida para que seus sinais de alerta sejam identificados por profissionais de saúde e para que medidas de controle possam ser tomadas, evitando assim, tal problema, por isso, objetivou-se compreender a descompensação metabólica que promove a CAD em pacientes com DM. Trata-se de um relato de experiência elaborado a partir da análise dos dados de intervenção e evolução clínica de usuário de serviço de saúde que apresentou CAD por conta da DM descompensada e faleceu mesmo após as condutas médicas. A CAD é uma emergência hiperglicêmica que necessita de intervenção médica rápida. Nesse sentido, o diagnóstico precoce e tratamento da DM é importante pois impede que evolua para CAD. No entanto, o estudo apresentado mostra o oposto, visto que a paciente desconhecia que era portadora de DM, por conta disso não fez nenhum tratamento e faleceu em razão das complicações da DM.

Descritores: Cetoacidose Diabética; Complicações do Diabetes; Hiperglicemia; Insulina; Unidades de Terapia Intensiva.

Introduction

Diabetes Mellitus (DM) is characterized by a set of metabolic diseases with problems in the metabolism of glycolysis. According to the Brazilian Society of Diabetes, DM is a chronic disease in which the body does not produce insulin or cannot properly use the insulin it produces. Such characteristics of the disease have a negative impact on diabetic health[1].

Diabetes Mellitus has two categories: type 1 and type 2. Type 1 DM is usually affected and diagnosed since childhood, but it can also be identified in adults. It is characterized by little or no production of insulin by the pancreas, through the immune system's attack on beta cells, which are responsible for secreting and synthesizing the hormone insulin, which regulate blood glucose levels. The main symptoms of this type are polydipsia, polyphagia and frequency. The incidence of type 1 DM is 5% of the cases of affected people and is more frequent in Asians and Afro-descendants[2].

On the other hand, type 2 DM most often affects adults, but the diagnosis in children is still possible. Type 2 is characterized by the body's inability to properly use the insulin it produces, or the body does not produce enough insulin to establish control over the body's glycemic rate. Contrary to DM 1, the family history is common, even with the complex hereditary pattern. The symptoms are very similar to type 1, but in some cases, there may be no symptoms. The vast majority of people who have DM are type 2, about 85% to 90% of cases, and 7.6% is the estimated prevalence rate of cases in Brazil[1-3].

DM can be diagnosed in two ways: clinically and laboratory. The clinical diagnosis consists of the identification of classic symptoms such as polydipsia, polyuria and polyphagia, in addition to these symptoms, metabolic decompensation is another identified factor that will be taken as a diagnostic criterion, associated with blood glucose greater than or equal to 200 mg/dL. The laboratory diagnosis will assess the conditions of fasting blood glucose (if it presents values equal to or greater than 126 mg/dL in two tests, it closes the result), oral glucose tolerance test (if it presents a value equal to or greater than 200 mg/dL, it concludes diagnosis) and glycated hemoglobin (above 7% is associated with greater risks)[1].

The treatment of DM consists of being medicated, through insulin and other antidiabetic drugs, and non-medicated, through the practice of physical exercises and a new food plan[1]. Such a strategy of changing habits for the treatment of DM has been gaining strength and encouragement over the years, with a positive index throughout the therapeutic process.

One of the frequent complications in patients with Diabetes Mellitus is diabetic ketoacidosis (DKA) which consists of the following clinical picture: hyperglycemia, metabolic acidosis, dehydration and ketosis. This scenario is promoted due to the depletion of insulin, mainly due to infections, inappropriate use of insulin or lack of diagnosis of diabetes[4]. Ketoacidosis is an emergency clinical condition that requires professional attention and knowledge of those involved in the practice of care.

Metabolic decompensation in diabetic ketoacidosis (Figure 1) occurs when there is significant insulin deficiency associated with excessive excretion of counterregulatory hormones (glucagon, catecholamines, cortisol, and growth hormone). Thus, there is hyperglycemia due to glycogenolysis (degradation of glycogen), gluconeogenesis (production of glucose from substrates other than glucose), reduced renal excretion, and decreased uptake by insulin-sensitive peripheral tissues, which results in extracellular and hyperosmolarity. , consequently, cellular dehydration[5].

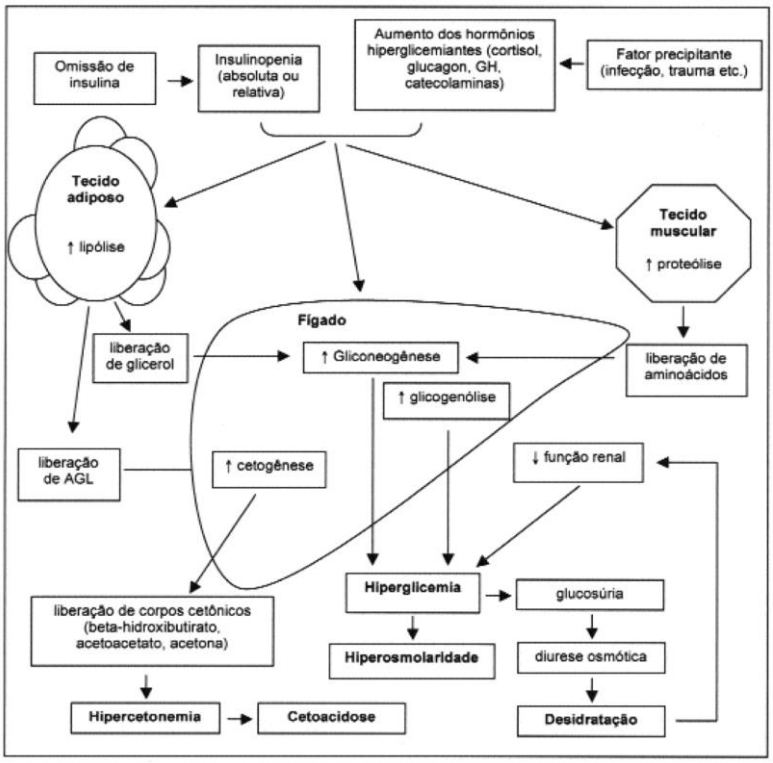
It is extremely important to emphasize that when hyperglycemia exceeds the renal threshold, it promotes glucosuria and osmotic diuresis, accentuating the loss of extracellular volume. Furthermore, the breakdown of fatty acids from adipose tissue (lipolysis) for oxidation to ketone bodies in the liver promotes ketonemia and metabolic acidosis[4,5].

The Hyperosmolar Hyperglycemic State (HHH), formerly known as hyperosmolar hyperglycemic non-ketotic coma (CNHH), is an acute and metabolic complication of type II diabetes mellitus caused by physiological stress, which mainly affects older ages (>50 years), has as characteristic plasma hyperosmolarity, severe hyperglycemia, altered level of consciousness, absence of ketoacidosis and profound dehydration. It usually develops after a period of symptomatic hyperglycemia, when fluid intake is insufficient to prevent extreme dehydration from hyperglycemia-induced osmotic diuresis.

The precipitating factors include serious infections; Brain stroke; myocardial infarction; pancreatitis; burns; liver transplantation; non-adherence to diabetes treatment and drugs that impair glucose tolerance such as glucocorticoids, or that increase fluid loss such as diuretics or that decrease insulin secretion such as immunosuppressive therapy. The pathophysiology resembles that of diabetic ketoacidosis. In the clinical manifestations, we initially have polydipsia, asthenia, polyuria; marked dehydration; arterial hypotension; tachypnea; tachycardia; Diffuse neurological manifestations such as lethargy, mental confusion, convulsions, delirium and coma. Focal neurological manifestations such as aphasia, nystagmus, hemiparesthesia, hemianopia and hemiparesis. The diagnosis is by severe hyperglycemia collected from a capillary blood glucose sample obtained in the assessment of an altered mental status. Serum osmolarity, plasma hyperosmolarity and absence of significant ketosis (ketones should be tested in urine). Treatment is aimed at correcting metabolic disorders and triggering factors and maintaining tissue perfusion[6,7].

Knowing that diabetic ketoacidosis is a relevant clinical complication, in which its knowledge is relevant for the health service, this study aims to understand the metabolic decompensation that promotes diabetic ketoacidosis in patients diagnosed with Diabetes Mellitus by case study.

Figure 1. Pathophysiology of diabetic ketoacidosis. Matipó, MG, Brazil, 2022



Source: Baroni, et al[4:1436].

Methodology

This is an exploratory study of the experience report type, with a descriptive nature and a qualitative approach. The case in question arises from objective observations that occurred, in the period from April 06, 2022 to April 15, 2022, from medical assistance to the reduction of visual acuity, dyspnea and severe metabolic decompensation due to the evolution of Diabetes Mellitus not previously diagnosed and treated. Data were compiled according to the clinical history of the user and the actions taken in the care proposal established by the medical team. This case report was carried out in a public hospital in the Zona da Mata of the State of Minas Gerais-Brazil.

Experience Report

Patient M.J.C., female, 55 years old, resident of the Zona da Mata of the State of Minas Gerais-Brazil, came to the hospital with a chief complaint of reduced visual acuity and dyspnea. He arrived at the hospital presenting manifestations of dyspnea, disorientation and hypoactivity, which could have been caused by dehydration. During their care, they performed the HGT (hemoglycotest) resulting: HI (capillary blood glucose above the device's detection capacity). Then, the blood glucose test was performed, which resulted in a value of 482 mg/dL, indicating that the patient could be a carrier of type 2 DM, since she claimed not to have knowledge of her possible pathologies and not to do medical follow-up. In addition, he also had low hemoglobin and hematocrit, onset of anemia and high segments. After this analysis, intravenous insulin and hydration were administered in order to stabilize the patient's condition. Soon after, the patient presented hypokalemia, a fact that may have been a consequence of the insulin therapy that can cause such a change, thus, it was necessary to use potassium ampoules before starting the next insulin applications to raise the blood glucose level, thus decreasing major consequences, such as arrhythmia and possible cardiorespiratory arrest. The next day, the patient was in severe hypoxia with oxygen saturation at 82%. It was necessary to use a nasal catheter to improve oxygenation, raising the saturation to 92%. Then, a small dose of corticosteroid was administered to improve saturation and replacement of the nasal catheter with the mask in order to increase saturation. Creatinine dosage of 1.6 mg/dL was also identified. As a result, a decrease in urinary volume was detected, requiring the use of a probe to assess diuresis. The patient was hypokalemic, had hypoxia and bradycardia, so 1 intravenous potassium ampoule was administered even before starting hydration and insulin application in order to avoid hypokalemia, but there was no compensation. The hemogram still showed alterations suggestive of an inflammatory condition, indicating leukocytosis of 15,000.

With the critical condition where the patient was and without significant improvements, it was necessary to refer her through the easy SUS to the CTI and after four days, she was referred to a vacancy in a regional reference hospital. After two days of admission to the hospital, the test was performed again and the results were blood glucose of 440 mg/dL and glycated hemoglobin of 14.1%, with no improvement in the patient's decompensation, requiring another intravenous insulin application. In a new dosage, leukocytosis of 17,000 was identified, which demonstrates an inflammatory reaction without a specific focus. The antibiotic ceftriaxone was administered. There was an improvement in hemoglobin, but it did not reach the levels considered normal. His hematocrit remained low,

leukocytosis increased and segmented remained high. In addition, he presented with thrombocytopenia and uremia. He presented with abdominal pain at dawn, with significant leukocytosis, which may indicate a diagnosis of polymicrobial infection, so the administration of the antibiotic metronidazole was included in the prescription, which has a broad spectrum therapy in the treatment. However, the patient remained decompensated.

The patient presented with acidosis and bicarbonate was used to try to reverse this situation. Finally, it is noted that the patient had decompensated ketoacidosis. After 22 days of admission to the hospital, the patient died.

Discussion

According to the American Diabetes Society, patients with type 2 DM are 40% more likely to suffer from an eye disease. In addition, the longer the blood sugar levels are out of control, the greater the chances of developing more than one of these diseases[8]. Excess glucose in the bloodstream causes a kind of swelling in the lens, which acts like the lens of our eyes, which ends up modifying its shape, flexibility and consequently decreasing the ability to focus, which occurs in cases of decompensated diabetes, as in the case of the patient in question.

With regard to hypokalemia, insulin plays an important role in increasing its entry into the intracellular environment, thus, the increase in insulin has a strong action in hypokalemia, being particularly observed in patients recovering from diabetic ketoacidosis and/or severe hyperglycemia.

Diabetic ketoacidosis (DKA) is an acute complication of diabetes mellitus, affecting mainly patients with type 1 DM. DKA results from insulin deprivation and excess counterregulatory hormones. Insulin is an anabolic hormone and its lack favors catabolic processes, that is, the breakdown of molecules, such as lipolysis, which results in the release of fatty acids which will be oxidized and converted into acetyl-CoA, this substance in large quantities will be converted to ketone bodies and their retention in plasma causes metabolic acidosis[4]. The patient addressed in this case study did not use insulin, since she was not aware of her condition, she had type 2 DM. However, this fact may have caused the CAD, as the omission of insulin therapy is an important cause factor of it. In addition, we have that CAD presents complications/symptoms characterized by hyperglycemia, metabolic acidosis, dehydration and ketosis, in the involvement of profound insulin deficiency.

Hyperosmolar hyperglycemic state (HHE) is a complication of diabetes mellitus, usually caused by physiological stress and commonly in DM 2. The estimated number of deaths is up to 20%, which is higher than the number of deaths from DKA (currently < 1%). Its development usually occurs after a period of symptomatic hyperglycemia, where fluid intake is inadequate to avoid extreme dehydration resulting from hyperglycemia-induced osmotic diuresis. The first symptom of HHE is an altered level of consciousness ranging from disorientation or confusion to coma, which are usually due to extreme dehydration with or without prerenal azotemia, hyperosmolality and hyperglycemia[6]. On the other hand, in cases of CAD there may be focal or generalized seizures and transient hemiplegia. DKA and HHE can be confused, as they present similar symptoms, such as polyuria, polydipsia, altered mental status, dehydration, and in some cases, the omission of insulin. We can check the parameters for diagnosis and classification in the figure below (Table 1). In the case presented, the patient did not present a picture of EHH.

Table 1. Diagnosis/Classification of CAD and EHH. Matipó, MG, Brazil, 2022

	DIABETIC KETOACIDOSIS			HYPERGLYCEMIC STATUS HYPEROSMOLAR
	LIGHT	MODERATE	SEVERE	
Blood glucose (mg/dL)	> 250	> 250	> 250	> 600
pH	7,5 - 7,3	7,0 - 7,24	< 7,0	> 7,3
HCO3 (mEq/L)	15 - 18	10 - 14,99	< 10	> 18
Urinary and/or serum ketone bodies	+	++	+++	Rare
Osmolarity*	Variable	Variable	Variable	>320 mOsm/kg
Anion gap*	> 10	> 12	> 12	< 12
Sensory	Alert	Obtunded	Torporous	Torpor/coma

Note: *Osmolarity - 2. [As measured in mEq/L] + (glucose in mg/dL)/18 ---- normal 290 + or -. ** Anion gap = Na - Cl - HCO3 (in mEq/L) ---- normal 9 – 12.
Source: Zoppi[9].

As we observed in the patient's decompensated DM condition, with regard to thrombocytopenia, there is no concrete evidence of how this occurred, since diabetic patients usually have platelet levels considered normal, as in patients without the disease. However, it is known that hyperglycemia, dyslipidemia and hypertension can independently cause vascular lesions, which can directly affect the intact state of the vessels, which in turn prevent

platelet aggregation, thus preventing increased adhesion and platelet aggregation. In view of this, decompensated diabetic patients are more prone to thrombotic events.

Considering the case presented, we have that the Basic Health Units (UBS) have protocols for the specific care of diabetics. In these units, professionals receive guidance and are prepared to accompany diabetic patients, when the patient does not seek the UBS, as in the case reported, the Community Health Agents (CHA) together with the doctor make home visits in order to monitor and explain the patient's need to perform the treatment correctly so that there are no future complications. As the patient was already in a serious condition, if her monitoring was monitored at home, there could be a referral to Hiperdia Minas, These centers are secondary health care units, aimed at assisting diabetics and hypertensive patients at high and very high risk levels[10].

While Diabetes Mellitus can promote serious complications that lead to complications and death, educational measures can be taken to build self-care dependency. In addition, health education based on guidelines during care and professional training/updating translate into one of the greatest and most important indicators of the quality of health care and patient compliance to the treatment[11].

Final Considerations

From the analysis of the literature findings, we can conclude that DM is characterized as a metabolic disorder caused by low levels of insulin in the body and/or by the inability of this hormone to function properly, resulting in hyperglycemia. It is worth mentioning that CAD, like HHE, is an acute complication of DM and carries risks, as it is one of the main clinical emergencies of this comorbidity.

As explained in the case report, the patient was diagnosed with DKA, an acute metabolic complication of diabetes characterized by hyperglycemia, hyperketonemia and metabolic acidosis. To reach this diagnosis, laboratory tests and evaluation of the patient's signs and symptoms were performed. At that moment, she reported not having been previously diagnosed as having diabetes and not having performed follow-up or treatment to improve the condition at another time. As a result, there was a significant decompensation of DM and it had damages such as visual acuity due to vascular lesions as a result of hyperglycemia, damage that could progress to partial or total blindness. In view of this, hyperketonemia compromises the function of the beta cells of the pancreatic islets with hyperglycemia. Thus, she developed metabolic acidosis due to the accumulation of ketones in the body that acidify the blood.

Therefore, due to the need for significant intravenous insulin administration to try to compensate the patient, she presented hypokalemia as a result of insulin treatment, as it takes potassium (K) into the cells. In addition, if serum K is not monitored and replaced as needed, potentially fatal hypokalemia can result. Although she received treatment indicated for CAD and was transferred to a referral hospital, the patient was very decompensated and died.

The case study presented confirms the importance of early diagnosis of diabetes mellitus, as well as the need for knowledge about the signs and symptoms, in order to avoid a potentially fatal condition of decompensated ketoacidosis, as in the patient in question. Once DM is diagnosed, it is also important not to neglect the patient's demand for adherence to self-treatment, especially in the setting of chronic diseases, such as latent autoimmune diabetes in adults. It is understood that in addition to therapy, it is also necessary to ensure healthy eating habits. In short, it is emphasized that the family doctor, as the patient's manager, must encompass all of the patient's problems, promoting good eating habits and supporting adaptation to this chronic disease. In addition, it is known that the correct analysis of a patient's laboratory parameters allows the accurate diagnosis of diabetic ketoacidosis, which can help in the treatment and recovery of the patient.

In specific cases with complications such as diabetic ketoacidosis, it is necessary to assess the degree of complication, as depending on the parameters it is possible to stabilize the patient and treat him. In more isolated cases, like the one mentioned, it can be more serious and irreversible, being fatal. This discussion is increasingly necessary, since there is an increase in the incidence and prevalence of Diabetes Mellitus because it causes a greater need for discussions on the subject. It is hoped that new studies can bring real clinical reports to academic and professional discussion.

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