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Is It Really Diabetic Ketoacidosis? Double Trouble

Gerçekten Diyabetik Ketoasidoz mu? Çifte Bela

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ABSTRACT

Introduction: In a patient admitted to the emergency service with complaints of nausea, vomiting, polydipsia, and polyuria and with findings of hyperglycemia, ketonemia, and acidosis, the first diagnosis to be considered is diabetic ketoacidosis (DKA). DKA is more common among young patients, of whom 50% to 85% are adults. Uremia, lactic acidosis, and intoxication with drugs and substances, such as salicylates, methanol, paraldehyde, and ethylene glycol, may present clinically as diabetic ketoacidosis.

Case Report: In this paper, we presented an 18-year-old patient who was first treated with the diagnosis of DKA and was diagnosed with salicylate intoxication afterwards during the emergency service follow-up.

Conclusion: The final diagnosis and necessary treatment might be impeded in such patients if the treatment is initiated considering DKA. Thus, when the clinical findings cannot be explained with the history, physical exam, and laboratory tests, intoxication with drugs, particularly with easily accessible salicylates, should be undoubtedly considered.

ÖZET

Giriş: Bulantı, kusma, çok su içme, çok idrara çıkma nedeni ile acil servislere başvuran ve ek olarak hiperglisemi, ketonemi ve asidoz saptanan hastalarda öncelikli tanı diyabetik ketoasidozdur (DKA). Daha çok gençlerde görülmekle birlikte olguların %50-85' i erişkin yaşlardadır. Üremi ve laktik asidoz ile metanol, paraldehit, etilen glikol ve salisilat gibi maddelerle zehirlenmeler DKA benzeri klinik tablo oluşturabilir.

Olgu Sunumu: Yazımızda DKA tanısı ile tedavisine başlanan ve acil servis takibi sırasında salisilat zehirlenmesi tanısı alan 18 yaşında bir olguyu sunduk.

Sonuç: DKA düşünülerek tedavi başlanan ancak bu gruplara giren olgularda tanı ve uygun tedavi gecikebilir. Bu nedenle hikaye, fizik muayene ve laboratuar tetkikleri ile açıklanamayan klinik bulguları olan olgularda özellikle salisilatlar gibi kolaylıkla ulaşılabilen ilaçlarla zehirlenmeler mutlaka düşünülmelidir.

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Anahtar Kelimeler: Diyabetik ketosidoz, salisilat, zehirlenme
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Introduction

Diabetic ketoacidosis (DKA) is the first diagnosis to be considered when patients with hyperglycemia, ketonemia, and acidosis have complaints of polydipsia, polyuria, nausea, and vomiting. Yet, consumption of salicylates in high doses also results in similar clinical and laboratory findings. When patients or their companions report that there is a possibility of intoxication, it is easier for the physician to diagnose. However, this possibility may be overlooked when there is insufficient information about the patient's history.

In the literature, there are limited publications reporting on cases diagnosed with salicylate poisoning following the pre-diagnosis of DKA. Most of these cases are in the pediatric age group (1, 2). Yet, adult patients may have the same problem. Thus, in this paper, we will discuss this misdiagnosis using a case that first received treatment for DKA but was then diagnosed with salicylate poisoning.

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Case Report

An 18-year-old female patient was referred to the emergency department (ED) due to her altered state of consciousness. Her companions said that the patient started complaining of shortness of breath and distress 6 hours ago and started suffering from nausea and vomiting 2-3 hours ago. The patient had no additional sickness or disorder other than a congenital deformation in her lower extremities. From the initial health care center that the patient reported to, the following laboratory findings were obtained: blood glucose 214 mg/dl, pH: 7.27, HCO_3: 6.1 mmol/L, PO_2: 95 mm Hg, PCO_2: 13 mm Hg arterial blood gas, and negative urine ketones. In the initial health care center, 18 IU crystallized insulin was administered subcutaneously to the patient with an intravenous injection of 10 mg metoclopramide, 50 mg ranitidine, and 1500 ml of normal saline. The patient was transferred to our ED for further investigation and management. We were informed that the patient had had a cardiac arrest; the ambulance team applied cardiopulmonary life support to the patient for about 3-4 minutes and thus enabled spontaneous cardiac activity during the transfer.

In the ED, the patient's Glasgow Coma Scale was 3, her pupils were isochoric, and light reflex was bilaterally weak. Her blood pressure was 80/50 mm Hg, heart pulse rate was 79 beats/min, body temperature was 36.4 ˚C, oxygen saturation was 80% (when supported with an oxygen mask, 8-10 liters/min), respiratory rate was 16 breaths/min, and her respiration was in the form of sighing. Bag-valve mask ventilation was applied to improve respiration. The patient was given insulin at the initial health care center; thus we measured her blood sugar level to preclude hypoglycemia. Blood glucose measurement from the fingertip provided a value of 408 mg/dl. Rapid sequence intubation was applied to assure airway security and oxygen support. Electrocardiography revealed only non-specific ST-T wave changes. The first blood gas parameters in the ED were as follows: pH: 6.69, pCO_2: 32.9 mm Hg, pO_2: 74 mm Hg, HCO_3: 3.9 mmol/L, BE: -32.2 mmol/L, and lactate: 18.11 mmol/L. Then, fluid replacement therapy and 0.1 U/kg/h insulin infusion were administered to the patient; 100 mmol bicarbonate infusion was initiated to be infused in 2 hours. Complete blood count and biochemical tests were ordered. However, in the course of her treatment, blood pressure fell below measurable levels, and dopamine infusion was applied at 10 mcg/kg/min. Her blood sample was sent to the laboratory for toxicological studies, because the patient's history could not be ascertained due to her unconscious state, and the acidosis continued despite treatment. The patient developed cardiac arrest in the 25th minute of her arrival to the ED. Return of spontaneous circulation was received in the third minute of advanced cardiac life support (ACLS). After the return of spontaneous circulation, the blood gas parameters in the control were as follows; pH: 6.72, HCO_3: 9.2, and lactate: 16.05. Her anion gap was 33 mEq/L. Follow-up in the intensive care unit was required for the patient; thus, the internal medicine and anesthesia-reanimation departments, which provided intensive care at our hospital, were called for consultation. Meanwhile, ACLS was resumed due to the onset of ventricular fibrillation. At the end of 1-hour ACLS and control of cardiac contraction with bedside ultrasonography, the patient was declared dead, as there was no spontaneous circulation. After the ACLS, the toxicology results showed that the salicylate level of the patient was 316 mg/ml.

Discussion

Diabetes patients that characteristically suffer from polyuria, polydipsia, polyphagia, and weight loss may have non-specific complaints, such as lassitude and fatigue, when they develop DKA. Nausea, vomiting, and stomach ache are also among common complaints. Patients may be taken to the ED due to an altered state of consciousness and coma (5). DKA is more common among young patients, of whom 50% to 80% are adults. Moreover, 20% to 25% of attacks are seen among newly diagnosed diabetic patients (6).

The underlying metabolic situation is absolute or relative lack of insulin and an increase in anti-insulin hormones. The lack of balance among hormones leads to disorders in carbohydrate, protein, and fat metabolism. The result may be in the form of acidosis, hyperglycemia, and ketonemia (4).

However, particularly in patients without a complete history, other causes of acidosis should be taken into consideration in the differential diagnosis, particularly if the patient suffers from a change in the state of consciousness. It is essential to seek the causes of metabolic acidosis with increased anion gap, which may be caused by lactic acidosis, chronic renal failure, and drugs, such as salicylate, methanol, ethylene glycol, or paraldehyde (4). In our case, because the patient did not respond to DKA treatment and acidosis continued at a very critical level, we sought other possible causes. The toxicological tests showed that the level of salicylate was high.

Salicylates that are frequently used as analgesics, antipyretics, and anti-inflammatories may cause mortality when they are taken in high doses acutely or used chronically (1). Salicylates, stimulating the respiratory center and hence damaging mitochondrial oxidative phosphorylation, cause an increase in lactate and metabolic acidosis. In this process, the metabolism of fat acids and ketone bodies increases. In order to compensate for acidosis, the respiration rate and the renal excretion of bicarbonates increase (5). When compensation is insufficient, pH reduces, all bicarbonate is used up, and lactic acid builds up.

End-organ damage occurs in patients that suffer from clinical instability. The onset of clinical findings is parallel to the metabolism. Due to the stimulation of medullary chemoreceptors, vomiting starts in 3 to 8 hours following the consumption of salicylate. The increase in respiratory and metabolic activity causes increased fluid loss. It also increases capillary permeability in pulmonary and cerebral tissues, leading to the development of edema. In further stages, hypertension elevation due to acidosis and fluid loss results in myocardial depression (6). The medical condition of the patient may finally lead to cardiopulmonary arrest.

Salicylates may also cause hyperglycemia, reducing aerobic metabolism and increasing glucosis-6 phosphatase activity, or

hypoglycemia, increasing the use of glucose in peripheral tissues or inhibiting gluconeogenesis. In salicylate toxicity causing hyperglycemia, the patient may suffer from glycosuria and ketonemia, and the clinical picture may be confused with diabetic ketoacidosis (6).

In salicylate toxicity, symptoms and findings may vary according to the age of the patient, as well as the amount of salicylate taken (5). The serum salicylate level, which is expected to reach the ultimate level in 2 to 4 hours following acute consumption, is usually not parallel to the clinical findings (3). Nevertheless, slight symptoms (gastrointestinal irritation, tinnitus, or tachypnea) are seen if the level of serum salicylate is between 30 and 50 mg/dl; medium symptoms (fever, sweating, or agitation) are observed if the level is between 50 and 100 mg/dl; and severe clinical symptoms (dysarthria, coma, crisis, or pulmonary edema) appear if the level is over 100 mg/dl (4). It is recommended that the patient be screened for subsequent salicylate and blood gas levels (6).

For patients diagnosed with salicylate poisoning, the primary methods of treatment are the replacement of intravenous fluid and serum-urine alkalization. In case of end-organ damage, early and aggressive resuscitation is essential. It should be kept in mind that a patient requiring intubation may also need hemodialysis.

In our case, the increase in the level of blood glucose and acidosis despite DKA treatment led us to consider that the patient might be suffering from a problem other than DKA. The patient might have taken another drug or material that could not be detected by the toxicology laboratory. However, clinical findings and a high level of salicylate supported the diagnosis of salicylate toxicity.

Conclusion

In patients with hyperglycemia and coma, salicylate toxicity should be considered as a possibility when the history is unreliable and there is clinical suspicion. It should also be kept in mind that acidosis and/or ketosis may be seen in the case of acute salicylate toxicity.

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