CASE REPORT

Varicella Zoster-Induced Severe Diabetic Ketoacidosis

Asri Mukti Nanta[1], Agustia Sukri Ekadamayanti[2], Krishna Wardhana Sucipto[2], Hendra Zufry[2,3*]

1 Residency Program in Internal Medicine, Department of Internal Medicine, Faculty of Medicine, Universitas Syiah Kuala
2 Division of Endocrinology, Metabolism, and Diabetes, Department of Internal Medicine, Faculty of Medicine/Dr. Zainoel Abidin Hospital, Universitas Syiah Kuala
3 Innovation and Research Center of Endocrinology, Faculty of Medicine, Universitas Syiah Kuala

*Corresponding author:
Hendra Zufry, MD.
Division of Endocrinology, Metabolism, and Diabetes-Thyroid Centre, Department of Internal Medicine, Faculty of Medicine, Universitas Syiah Kuala, Banda Aceh, Indonesia.
Email: Hendra_zufry@unsyiah.ac.id

ABSTRACT

Diabetic ketoacidosis (DKA) is a life-threatening complication of diabetes. There are not many cases reported relate to this case. This case report aims to present a case of DKA which is a common and potentially life-threatening complication in T1DM and can be the first sign of undiagnosed diabetes. A 36-year-old female presented with altered mental status and breathing difficulty. The patient's three children had recently contracted varicella (chickenpox). The patient was diagnosed with type 1 diabetes mellitus 8 months ago, with irregular adherence to treatment. Vital signs included a blood pressure of 84/60 mmHg on norepinephrine at 12 mcg/min, heart rate of 135 bpm, respiratory rate of 33 breaths/min, and temperature of 38.9°C. Physical examination revealed multiple lenticular, circumscribed vesicles with an erythematous base across the entire body. Laboratory results showed leukocytosis (leukocytes: 16,000 x 10^3/µL), hyperglycemia (random blood glucose: 273 mg/dL), severe metabolic acidosis (pH: 7.002, HCO3: 5.7 mEq/L, BE: -23.6 mEq/L), hypoxemia (pO2: 38 mmHg, SaO2: 52.8%), hypoalbuminemia (albumin: 3.48 g/dL), and stage I acute kidney injury (creatinine: 1.36 mg/dL). Urinalysis revealed the presence of ketone bodies. The patient was subsequently diagnosed with severe diabetic ketoacidosis, varicella zoster infection, septic shock with multiorgan failure, and type 1 diabetes mellitus. Despite initial treatment efforts, the patient's condition continued to deteriorate, with no signs of clinical improvement. After 3 days, the patient deceased. In conclusion, although varicella zoster infection is an uncommon precipitant of DKA, the present case report highlights the critical role of varicella zoster vaccination and optimal glycemic control in DMT1 to prevent infection-related DKA progression.

Keywords: Varicella, diabetic ketoacidosis, vaccine, complication

INTRODUCTION

Diabetes mellitus (DM) is a global health concern with significant prevalence, contributing to high morbidity and mortality rates worldwide.[1-3] Diabetic ketoacidosis (DKA) is the most frequent and potentially life-threatening acute complication of diabetes mellitus.[4] The primary precipitating factors for DKA include infections and inadequate insulin therapy.[5] Infections account for more than 50% of DKA cases, with specific infections such as pneumonia and urinary tract infections frequently identified as triggers.[6,7] In patients with uncontrolled DM, the likelihood of developing DKA is markedly increased, and these patients are also more susceptible to infections, which serve as major triggers for DKA.[8,9] The physiological stress induced by infections elevates the body's insulin requirements, and failure to meet these demands can precipitate DKA.[10]

Diabetic patients are more susceptible to infections due to an immunocompromised state, which is exacerbated by hyperglycemia that impairs immune function and may lead to more severe infections.[11,12] Varicella zoster virus infections commonly affect immuno-compromised patients, with diabetes mellitus associated to increased risk, particularly in those with poorly controlled blood glucose levels.[13] Although skin complications associated with DKA are uncommon, those specifically triggered by varicella zoster infections are exceedingly rare.[14] To the best of our knowledge, only one case of DKA triggered by varicella zoster have been reported in the literature.[14] This case report aimed to present a case of varicella zoster-induced DKA in a patient with uncontrolled type 1 diabetes mellitus. The present case report was prepared in accordance to case report (CARE) guideline.[15]

CASE ILLUSTRATION

A 36-year-old female patient presented to the Emergency Unit of Dr. Zainoel Abidin Hospital in Banda Aceh, Indonesia, with gradually altered mental status and breathing difficulty, unaccompanied by seizures, projectile vomiting, or severe headache. The patient reported experiencing a fever for 5 days prior to admission, followed by the development of painful blistering skin lesions and vesicular rashes that began on the neck and subsequently spread across the entire body. The patient did not self-administer any medications for symptomatic relief prior to hospital admission. The patient's family reported that the patient's three children had recently contracted varicella (chickenpox). The patient denied any prior history of varicella infection or varicella vaccination. The patient has a history of recently diagnosed type 1 diabetes mellitus, identified approximately 8 months ago, with irregular adherence to treatment and HbA1c levels of 10%.

The patient's vital signs upon examination were as follows: blood pressure of 84/60 mmHg while on norepinephrine at 12 mcg per minute, heart rate of 135 beats per minute, respiratory rate of 33 breaths per minute, and a temperature of 38.9°C. This patient weighs 70 kg and has a body mass index (BMI) that falls within the overweight category. Physical examination revealed multiple lenticular, circumscribed vesicles with an erythematous base across the entire body. Laboratory results showed leukocytosis (leukocytes: 16,000 x 10^3/µL), anemia (hemoglobin: 16.4 g/dL, hematocrit: 22%, erythrocyte: 2.6 x 10^3/µL), hyperglycemia (random blood glucose: 273 mg/dL), severe metabolic acidosis (pH: 7.002, HCO3: 5.7 mEq/L, BE: -23.6 mEq/L), hypoxemia (pO2: 38 mmHg, SaO2: 52.8%), hypocalcemia (calcium: 7.6 mg/dL) and corrected with Ca gluconate 1g/12 hours, hypoalbuminemia (albumin: 3.48 g/dL), stage I acute kidney injury (creatinine: 1.36 mg/dL), electrolyte imbalances (sodium: 134 mEq/L, potassium: 4.6 mEq/L, chloride: 105 mEq/L). Urinalysis revealed the presence of ketone bodies.

The patient was subsequently diagnosed with severe diabetic ketoacidosis, varicella zoster infection, septic shock with multi-organ failure, stage I acute kidney injury, and type 1 diabetes mellitus. In the emergency department, the patient was administered 4,000 mL of 0.9% NaCl over the first 6 hours and was started on

an insulin drip with rapid-acting insulin aspart 2 IU/hour. Throughout the treatment, the patient was continuously monitored for vital signs, urine output, blood glucose, and electrolyte levels. Blood glucose was checked hourly, and ketone levels were monitored to assess the effectiveness of the treatment. The insulin dose was titrated based on blood glucose levels. The random blood glucose levels during the infusion were 235 mg/dL in the first hour, decreasing to 213 mg/dL in the second hour, then to 210 mg/dL, 186 mg/dL, and eventually stabilizing at 160 mg/dL. This patient's urine output is 1500/24 hours. Additionally, acyclovir was given at an oral dose of 800 mg five times daily (4.000 mg daily). Despite initial treatment efforts, the patient's condition continued to deteriorate, with no signs of clinical improvement. The patient was urgently referred to the Respiratory Intensive Care Unit at Dr. Zainoel Abidin Hospital in Banda Aceh, Indonesia. Intensive supportive care was provided, including mechanical ventilation to manage respiratory failure and intravenous meropenem 1000 mg every 8 hours to combat potential bacterial infections. Despite these aggressive interventions, the patient's condition remained critical. After 3 days in the respiratory intensive care unit, the patient deceased.

DISCUSSION

The present case report identified a severe case of varicella zoster-induced DKA with septic shock and multi-organ failure in a patient with uncontrolled type 1 diabetes mellitus. Despite these aggressive interventions, the patient's condition continued to deteriorate. After 3 days in the respiratory intensive care unit, the patient deceased. The association between infections and the onset of DKA is well-established, with infections often serving as a significant precipitating factor for DKA.[8,10,11] The mechanism underlying infection-induced DKA involves the body's stress response, which triggers the release of counter-regulatory hormones such as glucagon, cortisol, catecholamines, and growth hormone.[16] These hormones oppose insulin action and dysglycemia, leading to insulin resistance.[17] Glucagon, in particular, promotes hepatic gluconeogenesis and glycogenolysis, increasing blood glucose levels, and it additionally induces ketone production.[18] Simultaneously, catecholamines and cortisol reduce glucose uptake in peripheral tissues, exacerbating hyperglycemia and precipitating DKA due to insufficient insulin activity.[19] In addition to promoting hyperglycemia, these counter-regulatory hormones also stimulate lipolysis, the breakdown of triglycerides into free fatty acids, in adipose tissue.[20] Free fatty acids are subsequently transported to the liver, where glucagon stimulation promotes the conversion into ketone bodies, primarily acetoacetate and β-hydroxybutyrate, serving as an alternative energy source in the absence of sufficient insulin.[21] The accumulation of ketone bodies leads to metabolic acidosis, one of the hallmarks of DKA.[22] Furthermore, the infection-induced inflammatory response exacerbates insulin resistance and promotes hyperglycemia.[23] Proinflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), are released in response to infection and contribute to the impairment of insulin signaling pathways, further reducing the effectiveness of insulin and worsening hyperglycemia.[24-26] This cascade of events ultimately overwhelms the body's compensatory mechanisms, leading to the clinical manifestation of DKA.[27]

Varicella zoster infection, though rare, is a notable trigger of DKA.[14] To the best of our knowledge, only one case of DKA triggered by varicella zoster have been reported in the literature.[14] A 17-year-old male patient presented with DKA exacerbated by a varicella zoster infection.[14] Despite a decade-long history of type 1 diabetes mellitus, the patient developed severe DKA, characterized by vomiting, dehydration, and a generalized vesicular rash indicative of chickenpox.[14] Upon admission, the patient required intensive management, including aggressive fluid replacement, intravenous insulin therapy, and electrolyte monitoring.[14] The chickenpox was

treated with intravenous acyclovir and supportive care.[14] Despite initial complications, including hypokalemia and renal impairment, the patient's metabolic parameters gradually stabilized, and his condition improved.[14] The patient was subsequently discharged with outpatient follow-up and continued antiviral therapy.[14] Unlike previous case report, the present case report highlights the severe and potentially fatal consequences of varicella zoster-induced DKA, highlighting how this condition, particularly when combined with other factors septic shock and multi-organ failure, can rapidly deteriorate a patient's health. Despite aggressive medical interventions, the disease's progression can be relentless, ultimately leading to death.

Although varicella zoster-induced DKA is rare, caution is also needed with herpes zoster-induced DKA, as both can cause severe metabolic disturbances, potentially leading to fatal outcomes. To the best of our knowledge, the existing literature documents only two case reports of herpes zoster-induced DKA, each originating from different years and countries.[28,29] These cases offer unique insights into this uncommon complication. The present case report is similar to a 2018 case involving a 75-year-old female patient from Indonesia who presented with declining consciousness and skin lesions consistent with herpes zoster.[28] Despite a history of uncontrolled type 2 diabetes mellitus, the patient had not been compliant with insulin therapy.[28] Upon admission, the patient was diagnosed with DKA triggered by herpes zoster infection.[28] The treatment included intravenous fluids, insulin, ceftriaxone for pneumonia, and acyclovir for the herpes zoster.[28] Unfortunately, despite initial improvement, the patient succumbed to complications from sepsis during hospitalization.[28] Another case, a 17-year-old female patient from the United Kingdom, presented in 1991 with diabetic ketoacidosis precipitated by a genital herpes infection.[29] The patient's symptoms included vulval soreness, drowsiness, and hypothermia.[29] The patient was treated with intravenous insulin and acyclovir, which led to a slow recovery over 72 hours.[29]

The risk of varicella virus infection is elevated in elderly or immunocompromised patients.[30,31] The 2022 American Association of Clinical Endocrinology Clinical Practice Guideline recommends varicella vaccination for patients with type 2 diabetes mellitus.[32] Hata et al found that demonstrated that varicella zoster vaccine safely enhanced varicella zoster virus specific immunity in elderly people with or without diabetes.[33] However, the detailed procedures, efficacy, and safety of the varicella zoster vaccine in type 1 diabetes mellitus patients have not yet been fully established.

The current recommendation specifically pertains to herpes zoster vaccination for immunocompromised patients.[34-36] Immuno-compromised individuals experience a higher incidence of herpes zoster and related complications.[37-39] Herpes zoster has a 25-30% lifetime risk and can lead to severe complications, including death.[40] Advisory Committee on Immunization Practices (ACIP) recommended the Zoster Vaccine Recombinant, Adjuvanted (Shingrix, GlaxoSmithKline [GSK], GSK Research Triangle Park, North Carolina, USA), a 2-dose subunit vaccine initially approved by Food and Drug Administration (FDA) for preventing herpes zoster in immunocompetent adults aged ≥ 50 years, with moderate to high vaccine efficacy and an acceptable safety profile.[36] Additionally, National Advisory Committee on Immunization (NACI) in Canada recommended the recombinant zoster vaccine for adults over 50 without contraindications and noted it may be considered for immunocompromised adults.[34] However, in 2022, the FDA broadened the vaccine's use to include adults aged 18 years and older at increased risk due to immunodeficiency or immunosuppression, which is also relevant for patients with type 1 diabetes mellitus.[35] Furthermore, a meta-analysis by Racine et al confirmed that the recombinant subunit herpes zoster vaccine has a favorable safety profile and effectively induces

immunity in a significant proportion of immunocompromised patients aged 18-49.[41]

However, recent literature advises caution when using the live attenuated zoster vaccine. A case report by Alexander et al highlighted that live zoster vaccination (Zostavax, Merck & Co., Inc, New Jersey, USA) in an immunocompromised patient led to disseminated varicella-zoster virus infection and death, underscoring the need for a non-live vaccine for immunocompromised patients.[42] Li-Kim-Moy et al demonstrated that disseminated varicella-zoster virus can be life threatening and primarily affects individuals with severe immunosuppression following the live attenuated herpes zoster vaccine.[43] ACIP deferred recommendations of recombinant zoster vaccine for immunocompromised patients until more data were available.[35]

A limitation of the present case report is the absence of polymerase chain reaction (PCR) testing to confirm the varicella zoster infection, primarily due to financial constraints, which could have provided more definitive diagnostic clarity. Future research is recommended to conduct large scale studies evaluating the efficacy and safety of varicella zoster vaccines in diverse immunocompromised populations, including those with specific conditions such as type 1 and type 2 diabetes mellitus.

CONCLUSION

Varicella zoster infection rarely precipitates DKA, yet it may occur due to an ineffective immune response to the varicella-zoster virus in patients with diabetes mellitus. The present case report highlights the critical role of varicella zoster vaccination and optimal glycemic control in DMT1 to prevent infection-related DKA progression.

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