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Steroid-Induced Diabetic Ketoacidosis Following High-Dose Intravenous Methylprednisolone Pulse Therapy in a Patient with Dermatomyositis: A Case Report

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Abstract

Background: Diabetic ketoacidosis (DKA) is a life-threatening acute metabolic complication of diabetes mellitus characterized by hyperglycemia, metabolic acidosis, and ketosis. Although glucocorticoid therapy is a well-recognized cause of steroid-induced hyperglycemia, the development of DKA in patients without previously diagnosed diabetes is uncommon. High-dose intravenous methylprednisolone pulse therapy may significantly increase insulin resistance and precipitate severe metabolic decompensation, particularly in patients with underlying inflammatory disorders.

Case Presentation: We report the case of a 62-year-old patient with active dermatomyositis who developed severe diabetic ketoacidosis shortly after receiving high-dose intravenous methylprednisolone pulse therapy. Before hospitalization, the patient had no history of diabetes mellitus, and self-monitored blood glucose levels had remained within the normal range. Following pulse therapy, the patient presented with profound hyperglycemia (545 mg/dL), positive ketonuria, metabolic acidosis (pH 7.20, bicarbonate 14.8 mmol/L), and an elevated anion gap, fulfilling the diagnostic criteria for DKA. Intensive management, including aggressive intravenous fluid resuscitation, continuous intravenous insulin infusion, electrolyte replacement, and close metabolic monitoring, resulted in gradual correction of hyperglycemia and metabolic acidosis. Despite metabolic stabilization, the patient experienced progressive muscle weakness, dysphagia, and respiratory deterioration related to active dermatomyositis.

Conclusion: This case highlights high-dose glucocorticoid pulse therapy as a potential precipitating factor for diabetic ketoacidosis, even in individuals without previously diagnosed diabetes mellitus. Clinicians should closely monitor blood glucose levels before, during, and after corticosteroid pulse therapy, particularly in patients with autoimmune inflammatory diseases receiving prolonged glucocorticoid treatment. Early recognition and prompt management of steroid-induced DKA are essential to reduce morbidity and improve clinical outcomes.

Keywords: diabetic ketoacidosis; steroid-induced diabetes; methylprednisolone pulse therapy; dermatomyositis; glucocorticoids; hyperglycemia; case report.

Introduction

Diabetic ketoacidosis (DKA) is an acute, life-threatening metabolic emergency characterized by hyperglycemia, ketonemia, and high anion gap metabolic acidosis resulting from absolute or relative insulin deficiency. Although DKA is most frequently associated with type 1 diabetes mellitus, it may also occur in individuals with type 2 diabetes or, less commonly, in patients without a previous diagnosis of diabetes when exposed to significant metabolic stress or diabetogenic medications (1,2,3). Prompt recognition and treatment are essential, as delayed diagnosis may result in severe electrolyte disturbances, cerebral edema, multiple organ dysfunction, and death (4,5,6).

The incidence of DKA has increased worldwide over the past two decades despite advances in diabetes care. Recent epidemiological studies estimate an annual incidence of approximately 8–46 episodes per 1,000 patients with type 1 diabetes, whereas DKA occurs considerably less frequently in patients with type 2 diabetes. Mortality rates have markedly declined in developed countries and are generally below 1–5% when evidence-based management protocols are applied; however, mortality remains substantially higher among older adults and patients with significant comorbidities (7,8).

Several precipitating factors have been identified, including infections, omission or inadequate administration of insulin, myocardial infarction, stroke, acute pancreatitis, trauma, surgery, and medications that impair glucose metabolism. Among pharmacological triggers, systemic glucocorticoids are well recognized for inducing hyperglycemia through increased hepatic gluconeogenesis, enhanced peripheral insulin resistance, and impaired pancreatic β-cell function. Nevertheless, progression from steroid-induced hyperglycemia to overt diabetic ketoacidosis remains uncommon, particularly in individuals without previously diagnosed diabetes mellitus.

Dermatomyositis is a rare autoimmune inflammatory myopathy characterized by progressive proximal muscle weakness, characteristic cutaneous manifestations, and potential involvement

of the lungs, gastrointestinal tract, and other organs. High-dose intravenous methylprednisolone pulse therapy remains an important treatment option for severe or rapidly progressive disease because of its potent anti-inflammatory and immunosuppressive effects. However, pulse-dose glucocorticoids may precipitate profound metabolic disturbances, including severe hyperglycemia, especially in susceptible individuals (9).

We present the case of a 62-year-old patient with active dermatomyositis who developed severe steroid-induced diabetic ketoacidosis shortly after receiving high-dose intravenous methylprednisolone pulse therapy despite having no previous history of diabetes mellitus. This case highlights the importance of careful glucose monitoring before, during, and after glucocorticoid pulse therapy and emphasizes that diabetic ketoacidosis should be considered in the differential diagnosis of patients presenting with acute metabolic deterioration following high-dose corticosteroid administration.

Etiology and Pathophysiology

Diabetic ketoacidosis (DKA) develops as a consequence of absolute or relative insulin deficiency combined with increased secretion of counter-regulatory hormones, including glucagon, catecholamines, cortisol, and growth hormone. This hormonal imbalance results in impaired glucose utilization by peripheral tissues, excessive hepatic gluconeogenesis and glycogenolysis, accelerated lipolysis, and increased hepatic ketogenesis. The accumulation of ketone bodies, primarily β-hydroxybutyrate and acetoacetate, leads to high anion gap metabolic acidosis, while persistent hyperglycemia causes osmotic diuresis, dehydration, electrolyte depletion, and intravascular volume contraction (10,11).

The most common precipitating factors for DKA include infections, inadequate insulin therapy or treatment discontinuation, newly diagnosed diabetes mellitus, acute myocardial infarction, stroke, pancreatitis, surgery, trauma, and certain medications. In recent years, several pharmacological agents, including sodium-glucose cotransporter-2 (SGLT2) inhibitors, atypical antipsychotics, immune checkpoint inhibitors, and systemic glucocorticoids, have been increasingly recognized as potential triggers of DKA (12).

Glucocorticoids induce hyperglycemia through multiple mechanisms. They stimulate hepatic gluconeogenesis, reduce peripheral glucose uptake by skeletal muscle and adipose tissue, suppress insulin-mediated glucose utilization, and impair pancreatic β-cell function, resulting in increased insulin resistance and relative insulin deficiency. High-dose corticosteroid therapy, particularly intravenous methylprednisolone pulse therapy, produces rapid and profound metabolic alterations that may overwhelm pancreatic compensatory mechanisms, especially in patients

with predisposing factors such as obesity, prediabetes, advanced age, chronic inflammatory diseases, or prolonged glucocorticoid exposure (13).

Dermatomyositis itself may further contribute to the development of severe metabolic disturbances. Active systemic inflammation is associated with increased production of pro-inflammatory cytokines, including tumor necrosis factor-α (TNF-α), interleukin-1 (IL-1), and interleukin-6 (IL-6), which promote insulin resistance and impair insulin signaling. The combined effects of systemic inflammation and high-dose glucocorticoid therapy markedly increase the risk of severe hyperglycemia and, in susceptible individuals, may precipitate diabetic ketoacidosis (14,15).

In the present case, the development of DKA was most likely multifactorial. Long-term glucocorticoid treatment, recent administration of high-dose intravenous methylprednisolone pulse therapy, active dermatomyositis-associated systemic inflammation, and previously unrecognized chronic hyperglycemia, as suggested by the elevated glycated hemoglobin (HbA1c 9.9%), probably acted synergistically to precipitate acute metabolic decompensation. This case illustrates that diabetic ketoacidosis should be considered in any patient who develops severe hyperglycemia and metabolic acidosis following glucocorticoid pulse therapy, even in the absence of a previously established diagnosis of diabetes mellitus (16,17,18).

Case Presentation

A 62-year-old patient was admitted to the emergency department with severe generalized weakness, nausea, repeated vomiting, diffuse abdominal discomfort, polyuria, and polydipsia. On admission, marked hyperglycemia was detected, which persisted despite initial insulin administration.

The patient had been diagnosed with dermatomyositis in December 2025 and was under regular rheumatologic follow-up. The disease was characterized by progressive proximal muscle weakness, mild cutaneous rash, impaired ambulation, difficulty rising from a seated position, and dysphagia.

Before hospitalization, the patient's maintenance treatment included hydroxychloroquine 200 mg twice daily, prednisolone 10 mg/day, mycophenolate mofetil twice daily, tacrolimus (oral capsules and topical ointment), esomeprazole 40 mg/day, and rivaroxaban 10 mg/day. During the two weeks preceding admission, oral methylprednisolone 16 mg twice daily had been added because of worsening disease activity.

The patient had no previous diagnosis of diabetes mellitus. Self-monitoring of blood glucose during chronic corticosteroid therapy had shown fasting glucose levels ranging from 108 to 115 mg/dL and postprandial glucose levels of approximately 128 mg/dL.

On the day of admission, according to the rheumatologist's recommendation, the patient received high-dose intravenous methylprednisolone pulse therapy consisting of 1 g methylprednisolone administered in 250 mL of 0.9% sodium chloride solution. Shortly after completion of the infusion, the patient developed profound hyperglycemia (36.36 mmol/L), accompanied by generalized weakness, dizziness, nausea, dry mouth, and marked polyuria, prompting emergency hospital admission.

The patient reported that mild xerostomia and increased urinary frequency had been present for approximately one month before admission; however, severe hyperglycemia had never previously been documented. The medical history was also notable for partial right nephrectomy performed in 2025 for a renal cyst confirmed by histopathological examination.

On physical examination, the patient appeared acutely ill but remained conscious and cooperative, with delayed verbal responses. Hemodynamic parameters were stable. Cardiac auscultation revealed regular rhythm with muffled heart sounds and no audible murmurs. Pulmonary examination demonstrated bilateral vesicular breath sounds without adventitious sounds. The tongue and oral mucosa were markedly dry. The abdomen was soft with diffuse tenderness on palpation but without signs of peritoneal irritation. No costovertebral angle tenderness was present, and urine output was preserved. Neurological examination demonstrated no focal deficits or meningeal signs. Musculoskeletal examination revealed no skeletal deformities.

Electrocardiography showed normal sinus rhythm without evidence of acute ischemic changes.

Initial laboratory investigations demonstrated a white blood cell count of $7.84 \times 10^3/\mu L$ with neutrophil predominance (94.6%), hemoglobin of 116 g/L, platelet count of $124 \times 10^3/\mu L$, serum glucose of 545 mg/dL, and serum creatinine of 146.6 µmol/L. Urinalysis revealed marked glucosuria, positive ketonuria, urinary pH of 5.0, and a specific gravity of 1.015.

Arterial blood gas analysis confirmed metabolic acidosis with a pH of 7.20, bicarbonate concentration of 14.8 mmol/L, partial pressure of carbon dioxide (pCO_2) of 38.1 mmHg, an anion gap of 18.6 mmol/L, and serum potassium of 4.99 mmol/L. Glycated hemoglobin (HbA1c) was 9.9%.

Based on the clinical presentation and laboratory findings, the diagnosis of diabetic ketoacidosis was established.

Immediate treatment included aggressive intravenous fluid resuscitation, continuous intravenous insulin infusion using insulin glulisine (Apidra), electrolyte replacement therapy, oxygen supplementation, and close monitoring of blood glucose, electrolyte concentrations, and acid-base status. Because blood glucose levels initially exceeded the upper measurable limit of the glucometer (HI), 10 units of subcutaneous insulin glulisine were administered without significant improvement. Continuous intravenous insulin infusion was subsequently initiated (50 units of insulin glulisine diluted in 50 mL of 0.9% sodium chloride, infused at 4 units/hour), together with intensive fluid replacement and serial biochemical monitoring.

The patient's metabolic parameters gradually improved during treatment. Arterial pH increased from 7.20 to 7.39, serum bicarbonate increased from 14.8 to 20.9 mmol/L, and blood glucose decreased from 545 mg/dL to 227 mg/dL.

Despite successful correction of diabetic ketoacidosis, the patient's overall clinical condition deteriorated because of progressive dermatomyositis. Marked proximal muscle weakness, worsening dysphagia, progressive respiratory insufficiency, and increasing oxygen requirements developed during hospitalization, reflecting progression of the underlying autoimmune disease.

Discussion

Diabetic ketoacidosis (DKA) is a serious acute metabolic complication that usually develops in patients with type 1 diabetes mellitus but may also occur in individuals with type 2 diabetes or previously undiagnosed diabetes under conditions of severe physiological stress. The development of DKA following glucocorticoid therapy is uncommon, and only a limited number of cases have been described in the literature. This case highlights the potential for high-dose intravenous methylprednisolone pulse therapy to precipitate severe DKA in a patient with active dermatomyositis and no previously established diagnosis of diabetes mellitus.

Glucocorticoids are widely used in the treatment of autoimmune and inflammatory diseases because of their potent anti-inflammatory and immunosuppressive effects. However, they profoundly affect glucose metabolism by increasing hepatic gluconeogenesis, decreasing peripheral glucose uptake, promoting insulin resistance, and impairing pancreatic β-cell function. These mechanisms result in steroid-induced hyperglycemia, which is frequently observed during corticosteroid therapy. Nevertheless, progression to diabetic ketoacidosis is rare and generally occurs in susceptible individuals with impaired β-cell reserve or previously unrecognized abnormalities of glucose metabolism.

Several factors likely contributed to the development of DKA in the present patient. First, prolonged exposure to oral glucocorticoids had already increased insulin resistance. Second, administration of high-dose intravenous methylprednisolone pulse therapy resulted in an abrupt increase in circulating glucocorticoid concentrations, further aggravating hyperglycemia. Third, active dermatomyositis itself represents a state of systemic inflammation characterized by increased production of pro-inflammatory cytokines, including tumor necrosis factor-α, interleukin-1, and interleukin-6, all of which are known to interfere with insulin signaling and promote insulin resistance. The combined effects of these mechanisms likely exceeded the patient's endogenous insulin secretory capacity, leading to uncontrolled hyperglycemia, ketogenesis, and metabolic acidosis.

Although the patient had no documented history of diabetes mellitus, the glycated hemoglobin level of 9.9% suggests that chronic hyperglycemia had been present before hospitalization. Previous self-monitored glucose values remained near the normal range, indicating that significant deterioration occurred only after methylprednisolone pulse therapy. It is therefore likely that glucocorticoid administration precipitated acute metabolic decompensation in a patient with previously unrecognized diabetes or significant impairment of glucose tolerance.

Current international guidelines recommend prompt recognition and aggressive treatment of DKA with intravenous isotonic fluids, continuous intravenous insulin infusion, careful potassium replacement, correction of electrolyte abnormalities, and frequent monitoring of acid-base status and blood glucose levels. Early initiation of this standardized treatment protocol in our patient resulted in rapid correction of metabolic acidosis, normalization of serum glucose concentrations, and closure of the anion gap, demonstrating the effectiveness of evidence-based management.

Despite successful metabolic stabilization, the patient's overall clinical condition deteriorated because of progressive dermatomyositis, manifested by worsening proximal muscle weakness, dysphagia, respiratory insufficiency, and increasing oxygen requirements. This observation emphasizes that successful treatment of DKA does not necessarily improve the underlying autoimmune disease and that multidisciplinary management involving endocrinologists, rheumatologists, and intensive care specialists is essential in such complex clinical situations.

The present case has several important clinical implications. Patients receiving high-dose glucocorticoid pulse therapy, particularly those with autoimmune inflammatory diseases, advanced age, prolonged corticosteroid exposure, or other risk factors for impaired glucose metabolism, should undergo careful glucose assessment before treatment and close glycemic monitoring throughout therapy. Early identification of steroid-induced hyperglycemia may allow timely intervention and prevent progression to life-threatening diabetic ketoacidosis.

In conclusion, this case demonstrates that high-dose intravenous methylprednisolone pulse therapy can precipitate severe diabetic ketoacidosis even in patients without a previously established diagnosis of diabetes mellitus. Awareness of this rare but potentially fatal complication is essential for physicians managing autoimmune diseases, as early recognition and prompt treatment may significantly improve patient outcomes.

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მაღალი დოზით ინტრავენური მეთილპრედნიზოლონის პულს-თერაპიით ინდუცირებული დიაბეტური კეტოაციდოზი დერმატომიოზიტის მქონე პაციენტში: კლინიკური შემთხვევა

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ანოტაცია

შესავალი: დიაბეტური კეტოაციდოზი (DKA) შაქრიანი დიაბეტის ერთ-ერთი ყველაზე მძიმე და სიცოცხლისათვის საშიში მწვავე გართულებაა, რომელიც ხასიათდება ჰიპერგლიკემიით, მეტაბოლური აციდოზითა და კეტოზით. მიუხედავად იმისა, რომ გლუკოკორტიკოიდებით გამოწვეული ჰიპერგლიკემია ფართოდ არის ცნობილი, დიაბეტური კეტოაციდოზის განვითარება პაციენტებში, რომლებსაც მანამდე შაქრიანი დიაბეტი არ ჰქონდათ, იშვიათ მოვლენას წარმოადგენს. მაღალი დოზით ინტრავენურმა მეთილპრედნიზოლონის პულს-თერაპიამ შესაძლოა მკვეთრად გაზარდოს ინსულინრეზისტენტობა და გამოიწვიოს მძიმე მეტაბოლური დეკომპენსაცია.

კლინიკური შემთხვევა: წარმოდგენილია 62 წლის პაციენტის კლინიკური შემთხვევა აქტიური დერმატომიოზიტით, რომელსაც მაღალი დოზით ინტრავენური მეთილპრედნიზოლონის პულს-თერაპიის ჩატარების შემდეგ განუვითარდა მძიმე დიაბეტური კეტოაციდოზი. პაციენტს ანამნეზში შაქრიანი დიაბეტი არ აღენიშნებოდა, ხოლო თვითკონტროლისას გლიკემიის მაჩვენებლები ნორმის ფარგლებში იყო. პულს-თერაპიის დასრულების შემდეგ განვითარდა გამოხატული ჰიპერგლიკემია (545 მგ/დლ), კეტონურია, მეტაბოლური აციდოზი (pH 7.20; HCO_3^- 14.8 mmol/L) და ანიონური ნაპრალის მომატება, რაც შეესაბამებოდა დიაბეტური კეტოაციდოზის დიაგნოსტიკურ კრიტერიუმებს. დაუყოვნებლივ დაიწყო ინტენსიური ინფუზიური თერაპია, ინტრავენური ინსულინის უწყვეტი ინფუზია, ელექტროლიტური დარღვევების კორექცია და მუდმივი მონიტორინგი, რის შედეგადაც მიღწეულ იქნა ჰიპერგლიკემიისა

და მეტაბოლური აციდოზის ეტაპობრივი კორექცია. მიუხედავად მეტაბოლური პარამეტრების გაუმჯობესებისა, პაციენტს აღენიშნებოდა დერმატომიოზიტის პროგრესირებასთან დაკავშირებული კუნთოვანი სისუსტე, დისფაგია და სუნთქვის უკმარისობის გაღრმავება.

დასკვნა: წარმოდგენილი შემთხვევა ხაზს უსვამს, რომ მაღალი დოზით გლუკოკორტიკოიდული პულს-თერაპია შეიძლება გახდეს დიაბეტური კეტოაციდოზის მაპროვოცირებელი ფაქტორი იმ პაციენტებშიც კი, რომლებსაც მანამდე შაქრიანი დიაბეტი არ ჰქონდათ. გლუკოკორტიკოიდებით მკურნალობის დროს, განსაკუთრებით აუტოიმუნური დაავადებების მქონე პაციენტებში, აუცილებელია სისხლის გლუკოზის დონის რეგულარული მონიტორინგი, რათა თავიდან იქნას აცილებული მძიმე მეტაბოლური გართულებები და უზრუნველყოფილი იყოს დროული დიაგნოსტიკა და მკურნალობა.

საკვანძო სიტყვები: დიაბეტური კეტოაციდოზი; გლუკოკორტიკოიდები; სტეროიდებით ინდუცირებული ჰიპერგლიკემია; მეთილპრედნიზოლონის პულს-თერაპია; დერმატომიოზიტი; კლინიკური შემთხვევა.