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Refractory hyperglycemia in a geriatric patient with diabetic ketoacidosis: A critical care nursing case report

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Abstract

Background: Refractory hyperglycemia in diabetic ketoacidosis (DKA) presents a critical challenge in geriatric patients due to multifactorial physiological and clinical complexities. Early recognition and integrated management are essential, with critical care nursing as a pivotal component.

Purpose: This case report aims to describe and analyze the multifactorial causes of refractory hyperglycemia in a geriatric DKA patient, highlighting the importance of early detection, comprehensive correction, and collaborative nursing roles.

Method: A descriptive analytic approach was employed to examine the clinical course of a 75-year-old woman with DKA and persistent hyperglycemia. Data were collected from direct patient observation, systematic physical examinations, laboratory assessments, imaging, and multidisciplinary clinical records. Daily therapeutic interventions and patient responses were meticulously documented.

Results: Persistent hyperglycemia resulted from several interacting factors: the impact of glucocorticoids on glycemic control, effects of severe pulmonary infection on insulin resistance, the role of electrolyte disturbances in exacerbating hyperglycemia, the relationship between heart failure and diuretic use, the impact of malnutrition and sarcopenia on insulin resistance, and the need for an integrated therapeutic approach. Each factor contributed to increased insulin resistance and complicated glycemic control.

Conclusion: Optimal management of refractory hyperglycemia in elderly DKA patients requires early identification and simultaneous correction of underlying factors, supported by intensive critical care nursing and multidisciplinary collaboration.

Keywords: Critical Care Nursing; Diabetic Ketoacidosis; Geriatric Patients; Refractory Hyperglycemia.

INTRODUCTION

Diabetes mellitus (DM) comprises a group of metabolic disorders characterized by hyperglycemia resulting from impaired insulin secretion, impaired insulin action, or both (Carreira, Gonçalves, Wahnon, Dâmaso, & Martins, 2021). Globally, the number of individuals with diabetes mellitus (DM) increased from 108 million in 1980 to 422 million in 2014, with an adult prevalence of 8.5%, particularly affecting the elderly population (Elendu, David, Udoyen, Egbunu, Ogbuiyi-Chima, Unakalamba, & Ramon-Yusuf, 2023).

The risk of DM-related complications, such as diabetic ketoacidosis (DKA), rises with advancing age due to physiological changes (e.g., immunosenescence), multiple comorbidities (e.g., infections and cardiovascular diseases), and polypharmacy (Illés, Terzin, Holzinger, Kosár, Róka, Zsóri, & Czakó, 2016). DKA, characterized by hyperglycemia, ketonemia, and metabolic acidosis, is often overlooked in older adults due to atypical presentations, such as mental disorientation or shortness of breath, rather than gastrointestinal

symptoms (Carreira et al., 2021). Evidence suggests that infections, particularly pneumonia and urinary tract infections, are the primary triggers of DKA in elderly patients (those over 70 %). In contrast, therapeutic non-adherence is more commonly observed in non-geriatric patients (Singh, Saroch, Pannu, Sachin, Sharma, & Dutta, 2019).

Mortality rates in elderly patients with DKA reach up to 48.1%, significantly higher than in younger populations (20.8%), driven by advanced age, lower Glasgow Coma Scale (GCS) scores, and infection as primary triggers (Singh et al., 2019; Piro & Purrello, 2021). Elderly individuals are also susceptible to complications such as atrial fibrillation, acute kidney injury, and euglycemic DKA associated with the use of sodium-glucose cotransporter-2 (SGLT2) inhibitors, exacerbating hyperglycemia and elevating mortality risks (Sehgal & Ulmer, 2019). Polypharmacy further complicates clinical management by increasing the risk of drug interactions and insulin non-adherence, especially in cognitively impaired patients (Sehgal & Ulmer, 2019).

Early detection of undiagnosed diabetes mellitus (DM) in intensive care units (ICUs) is critical, given that many patients are first diagnosed during episodes of diabetic ketoacidosis (DKA) (Shazniza Shaaya, Halim, Leong, Ku, Lim, Tan, & Wong, 2021). The implementation of evidence-based management in ICUs, including monitoring of glucose levels, ketones, electrolytes, and acid-base status, has been shown to reduce complications and hospital length of stay (Melki, Laham, Karim, Komal, Kumar, Barham, & Baddoura, 2019). The critical care nurse plays a crucial role in monitoring hemodynamics, managing fluid and ventilation, and collaborating with multidisciplinary teams to ensure optimal intervention (Carreira et al., 2021).

This case report aims to describe and analyze the factors contributing to refractory hyperglycemia in a geriatric patient with diabetic ketoacidosis (DKA), highlighting the importance of early identification, multifaceted correction, and the collaborative roles of critical care nursing in managing complex metabolic conditions in elderly populations. A noteworthy aspect of this case is the persistent elevation of blood glucose despite frequent insulin titration.

RESEARCH METHOD

This case report utilized a descriptive clinical approach to detail the course of a geriatric patient with diabetic ketoacidosis and persistent refractory hyperglycemia. Data were obtained from direct observation, electronic health records, and daily multidisciplinary team documentation. Serial assessments included a physical examination, laboratory tests (glucose, electrolytes, and ABG), and a chest X-ray. The analysis focused on identifying multifactorial contributors to refractory hyperglycemia and the critical care nursing interventions applied. Clinical responses to insulin titration and corrective measures were monitored daily. All patient information was anonymized in accordance with ethical standards.

RESEARCH RESULTS

A 75-year-old woman presented to the emergency department (ED) of Dr. H. A. Rotinsulu Pulmonary Hospital on 20 Feb 2025, at 18:39, with the main complaint of severe shortness of breath. The patient reported experiencing dyspnea for two days before admission. Accompanying symptoms included productive cough and reduced appetite, with a reported weight loss of 7 kg (from 67 kg), indicating a deterioration in nutritional status. A medical history of heart disease was confirmed by current medications (Atorvastatin, Candesartan, Clopidogrel, ISDN, Nitrokafe, Spironolactone). Upon ED examination, the patient was fully alert (compos mentis) and exhibited accessory muscle use, nasal flaring, and diffuse wheezing in both lungs. The extremities were warm, with a capillary refill time (CRT) of under 3 seconds; however, the patient appeared pale and weak. Vital signs were as follows: respiratory rate, 41 breaths/min; pulse, 119 beats/min; blood pressure, 160/74 mmHg; temperature, 36°C; and oxygen saturation, 99%. Initial laboratory results revealed a haemoglobin level of 9.7 g/dL, a leukocyte count of 5,670/mm³, a random blood glucose (RBG) level of 255 mg/dL, a urea level of 40.7 mg/dL, a creatinine level of 1.1 mg/dL, and an albumin level of 2.8 g/dL. Arterial blood gas (ABG) analysis showed pH 7.23, pCO₂ 40 mmHg, pO₂ 68 mmHg, HCO₃ 17.1 mmol/L, base excess (BE) -9 mmol/L, sodium 122 mmol/L

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(hyponatremia), potassium 5.6 mmol/L (hyperkalemia), and calcium 1.26 mmol/L. Chest radiography confirmed increased broncho vascular markings and perihilar and pericardiac infiltrates, suggestive of bilateral pneumonia accompanied by cardiomegaly.

Initial management in the ED included oxygen therapy via nasal cannula at 5 L/min, intravenous Asering infusion (500 mL every 8 hours), and inhalation therapy using a combination of Combivent and Pulmicort repeated after 15 minutes. The patient also received intravenous methylprednisolone 125 mg and omeprazole 40 mg. Shortness of breath slightly improved after one hour, although weakness persisted. The patient was subsequently transferred to a second-class inpatient ward, where they received pulmonology-directed treatment consisting of a 500 mL normal saline (NaCl 0.9%) infusion, aminophylline drip, moxifloxacin antibiotic, methylprednisolone 125 mg administered twice daily, and cardiology-directed medications including furosemide, spironolactone, aspirin, atorvastatin, and candesartan.

The next morning (21 Feb 2025), ABG revealed pH 7.35, HCO₃ 19.2 mmol/L, and BE -5 mmol/L, while fasting blood glucose was 260 mg/dL and two-hour postprandial glucose reached 301 mg/dL (HbA1C 7.1%). After consultation with an internist, the patient was prescribed Novorapid insulin 10-10-10 units subcutaneously (SC) and Lantus 15 units SC (at night), following an hourly insulin titration protocol based on glucose monitoring results. At 21:00, blood glucose remained poorly controlled (RBG 451 mg/dL), prompting escalation to continuous intravenous insulin infusion at 15 units/hour. The patient's condition deteriorated at 22:15, necessitating transfer to the ICU at 23:00; detailed clinical progress is available in the table below.

This sequence of events underscores the multifactorial nature of refractory hyperglycemia in elderly patients with diabetic ketoacidosis, pulmonary dysfunction, and cardiovascular comorbidities. The complexity of critical care management necessitates a collaborative approach, emphasising rigorous insulin regulation, cautious electrolyte correction, monitoring nutritional status, and infection control in older adults who are prone to metabolic instability.

Table Patient Condition Progression

Date and Time	Clinical Status & Objective Findings	Interventions	Evaluation / Progress
21 Feb 2025 22.15 (Worsening of condition in regular inpatient care)	Respiratory rate: 45 breaths/min. ABG: pH 7.398; PCO ₂ 39 mmHg; PO ₂ 202 mmHg; HCO ₃ 17.0 mmol/L; BE -8 mmol/L. Electrolytes: Na 119 mmol/L, K 5.3 mmol/L, Cl 101.9 mmol/L, Ca 1.18 mmol/L. Lactate 1.3 mmol/L. A random blood glucose level of 598 mg/dL indicates refractory hyperglycemia.	ABG and laboratory re-evaluation. ICU transfer team preparation.	ABG confirmed mild metabolic acidosis and severe hyperglycemia. The decision was made to escalate to the ICU.
21 Feb 2025 23:00 (ICU)	Somnolence; blood pressure 75/30 mmHg; heart rate 122/min; respiratory rate 36/min; temperature 36.8°C; SpO ₂ 97% (O ₂ nasal cannula 5 lpm); ronki -/-, wheezing +/-; Urine output 0 cc.	Insulin drip 20 IU/hour. Aggressive fluid resuscitation with NaCl 0.9%. Sodium bicarbonate 50 mEq every 6 hours. Following DKA resuscitation, new rhonchi were detected; a furosemide drip of 2 mg/hour was initiated.	Blood pressure started to improve, but urine output remained low (hourly monitoring recommended). Rhonki appeared after fluid overload; diuretics were titrated accordingly.

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Date and Time	Clinical Status & Objective Findings	Interventions	Evaluation / Progress
22 Feb 2025 03.00	Improved consciousness; blood glucose increased again to 400 mg/dL; vital signs relatively stable.	Hyponatremia correction: NaCl 3% (gradual infusion). Potassium correction based on serial results. Adjust insulin drip to 20–25 IU/hour. Intensive monitoring of pulmonary, hemodynamic, and metabolic status (ABG and laboratory every 4–6 hours post correction).	Fluctuating blood glucose levels persisted (target blood glucose 140–180 mg/dL).
23 Feb 2025 07:30	Post-correction laboratory: Na 126 mmol/L; Cl 103.2 mmol/L; K 4.5 mmol/L; blood glucose 348 mg/dL.	The insulin drip was continued at 22 IU/hour. Maintenance fluids have been switched to isotonic solutions; diuretic titration is performed based on fluid balance. Family education and clinical progress documentation were conducted.	Hyponatremia improved; potassium normalized; blood glucose decreased but remained above the target range.
24 Feb 2025 08.10	The insulin drip was increased to 25 IU/hour. Intubation and PSIMV ventilation: respiratory rate 14/min, pressure support 16, pressure control 16, PEEP 5, I:E ratio 1:2, FiO ₂ 100% (gradual weaning). Tidal volume 300–360 mL. Norepinephrine 0.5 µg/kg/min. Fentanyl drip 20 mg/hour IV.	FiO ₂ successfully reduced to 60% within 3 hours (SpO ₂ > 95%). Blood pressure stabilized at 90–100/50–60 mmHg. Blood glucose trended down (250–280 mg/dL) over 6 hours.	The insulin drip was increased to 25 IU/hour. Intubation and PSIMV ventilation: respiratory rate 14/min, pressure support 16, pressure control 16, PEEP 5, I:E ratio 1:2, FiO ₂ 100% (gradual weaning). TV 300–360 mL. Norepinephrine 0.5 µg/kg/min. Fentanyl drip 20 mg/hour IV.

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DISCUSSION

A 75-year-old female patient experienced diabetic ketoacidosis (DKA) with refractory hyperglycemia precipitated by several interacting clinical factors. Glycemic control in DKA is significantly more challenging among elderly populations due to various physiological changes, especially reduced insulin sensitivity caused by ageing, as well as insulin resistance resulting from acute inflammation, malnutrition, and specific pharmacological agents (Sehgal & Ulmer, 2019). Five major factors contributed to refractory hyperglycemia in this case: high-dose glucocorticoid therapy, severe pulmonary infection, significant electrolyte disturbances, heart failure with diuretic therapy, and profound malnutrition exacerbated by sarcopenia.

Administration of glucocorticoids, specifically methylprednisolone at a dose of 250 mg/day, constituted the principal factor complicating glycemic control. Glucocorticoids increase insulin resistance by stimulating hepatic gluconeogenesis and inhibiting peripheral glucose uptake, leading to persistent hyperglycemia (Hwang & Weiss, 2014; Shah, Kalra, Yadav, Deka, Lathia, Jacob, & Das, 2022). This was evident in the present case, where blood glucose remained uncontrolled despite high-dose subcutaneous insulin therapy. Studies demonstrate that elderly patients receiving high-dose steroids often require a more aggressive intravenous insulin protocol due to markedly increased insulin resistance (Alnuaimi, Mach, Reynier, Filion, Lipes, & Yu, 2024).

Severe pulmonary infection in the form of bilateral pneumonia triggered a systemic inflammatory response, directly aggravating insulin resistance. This infection induces the release of inflammatory cytokines, including TNF- α , IL-6, and IL-1 β , which disrupt insulin signalling, increase lipolysis, stimulate hepatic gluconeogenesis, and promote ketone production, thereby exacerbating hyperglycemia (Othman, Nashwan, Alfayoumi, Khatib, Abujaber, Othman, & AbuJaber, 2023). The inflammatory response substantially increased insulin requirements, necessitating an insulin drip of up to 20 units/hour, far exceeding the standard recommended dose of 0.1 units/kg/hour (Mustafa, Haq, Dashora, Castro, Dhatariya, & Joint British

Diabetes Societies (JBDS) for Inpatient Care Group 2023).

Significant electrolyte disturbances, namely severe hyponatremia (Na 119 mmol/L) and mild hyperkalemia (K 5.3 mmol/L) contributed substantially to the worsening of insulin resistance. Hyperkalemia in DKA typically results from potassium redistribution due to insulin deficiency rather than total body excess (Yamada, Funazaki, Kakei, Hara, & Ishikawa, 2017). Aggressive insulin therapy can rapidly shift potassium intracellularly, increasing the risk of acute hypokalemia and fatal arrhythmias, necessitating close monitoring before insulin initiation (Coregliano-Ring, Goia-Nishide, & Rangel, 2022). Severe hyponatremia also delays the insulin response through impaired glucose transport and must be corrected cautiously to avoid the risk of osmotic demyelination (Aditioningsih, Djaja, & George, 2017).

Chronic heart failure and prolonged administration of diuretics, including furosemide and spironolactone, further aggravated insulin resistance. Furosemide impairs glucose transport into adipose tissue and reduces skeletal muscle glycolysis, thereby disrupting effective glucose utilization (Widiarti, Saputra, Savitri, Putranto, & Alkaff, 2025). Spironolactone, although generally considered safe, has been associated with increased blood glucose and HbA1c levels in some observational studies, although this evidence requires cautious interpretation (Zhao, Xu, Lin, & Schooling, 2016). Heart failure itself slows insulin distribution due to poor tissue perfusion, making optimal glycemic control more difficult (Sezer, Karacan, Tuygun, & Şenel, 2024).

Severe malnutrition, as evidenced by a significant weight loss of 7 kg over a short period and low albumin levels (2.8 g/dL), directly exacerbated insulin resistance in this patient. This condition is closely associated with reduced muscle mass (sarcopenia), resulting in markedly decreased peripheral glucose uptake and an increased severity of metabolic acidosis. Routine nutritional assessment using the Malnutrition Universal Screening Tool (MUST) or Nutrition Risk Screening (NRS-2002) is essential to improve the metabolic response (Tamzil, Yaacob, Noor, & Baharuddin, 2023).

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This case represents a highly complex clinical scenario where therapeutic interventions could not rely solely on aggressive escalation of insulin dosage. Comprehensive correction of the underlying factors contributing to refractory hyperglycemia, such as severe pulmonary infection, adverse effects of high-dose steroids, significant electrolyte disturbances, cardiac dysfunction with long-term diuretic use, and severe malnutrition, constituted the cornerstone of patient management. Successful therapy depended on intensive monitoring by ICU nurses and close multidisciplinary coordination to adjust insulin therapy, correct electrolyte imbalances, manage fluids precisely, control infections, and provide timely nutritional support (Carreira et al., 2021).

This case demonstrates that refractory hyperglycemia in geriatric patients with DKA cannot be resolved by standard insulin therapy alone. Clinical management must specifically address the underlying causes of hyperglycemia to achieve optimal clinical outcomes, including reductions in morbidity and mortality among elderly patients with similar conditions (Melki et al., 2019; Sehgal & Ulmer, 2019).

CONCLUSION

This case confirms that refractory hyperglycemia in geriatric patients with diabetic ketoacidosis (DKA) results from multifactorial interactions, including high-dose glucocorticoid use, severe pulmonary infection, significant electrolyte disturbances, chronic heart failure with prolonged diuretic therapy, and malnutrition that exacerbates insulin resistance. Management requires not only intensification of insulin therapy but also early identification and simultaneous correction of these contributing factors. The role of the critical care nursing team in strict clinical monitoring and multidisciplinary collaboration proves essential for accelerating recovery and preventing fatal complications in geriatric populations with complex metabolic disorders.

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