Collaborative Nursing Interventions in Geriatric Patients with Adrenal Crisis and Blood Glucose Fluctuations: A Case Report

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Abstract

Adrenal crisis is an endocrine emergency condition, with one of its clinical manifestations being hypoglycemia. To date, its management focuses on the administration of corticosteroid therapy primarily hydrocortisone as the first-line treatment alongside glucose administration. This study aims to observe and report blood glucose levels, hemodynamic status, and multidisciplinary collaboration in the administration of corticosteroids, as a collaborative intervention in stabilizing blood glucose. This case study was conducted on a 70 years old geriatric patient who experienced an adrenal crisis due to the sudden discontinuation of long-term steroid therapy without a doctor's prescription. Data were collected over five days through patient interviews, family reports, and medical records. The patient experienced severe hypoglycemia despite receiving parenteral glucose. Following the administration of dexamethasone, the patient's blood glucose levels improved and began to stabilize. Dexamethasone proved effective as an alternative initial therapy for adrenal crisis when hydrocortisone was unavailable or the diagnosis is not yet confirmed. Nurses played a vital role in the early recognition of adrenal crisis signs, monitoring of blood glucose levels and hemodynamic parameters. Education for the family is essential, considering the dependency of geriatric patients on caregivers in managing daily health needs. Multidisciplinary collaborative management and educational support are key to successful care in geriatric patients with adrenal crisis.

Keywords: Adrenal Crisis, Corticosteroid, Geriatric, Hypoglycemia

Introduction

Adrenal crisis or acute adrenal insufficiency is a life-threatening medical condition caused by an acute deficiency of cortisol, a hormone produced by the adrenal glands (Arlt, 2016). Adrenal crisis is a further complication of adrenal insufficiency (AI). Adrenal insufficiency is an inadequate secretion of corticosteroids due to damage to the adrenal glands, which leads to an adrenal crisis (Adhiarta & Soetedjo, 2017). Cortisol deficiency can cause hypotension, hypoglycemia, and electrolyte imbalance, which can be fatal if not treated promptly (Bancos et al., 2015). The adrenal glands secrete two essential hormones for the body: mineralocorticoids and glucocorticoids. One of the glucocorticoid hormones is cortisol. Cortisol is essential for regulating metabolism, immune response, blood pressure, and electrolyte balance. When cortisol production is disrupted or insufficient, the body can enter a critical condition known as adrenal crisis. This condition commonly occurs in patients with primary adrenal insufficiency (Addison's disease) as well as secondary adrenal insufficiency (Arlt, 2016).

The prevalence of adrenal crisis varies across countries. A study involving over 400 AI patients estimated the incidence of adrenal crisis to be 8 out of 100 patients per year, with a mortality rate of 0.5 out of 100 patients per year (Dineen et al., 2019). Data from Norway indicate that 1 in 7 patients with Addison's disease died from adrenal crisis (Amrein et al., 2018). However, in Indonesia, epidemiological data on adrenal crisis remains limited. Individual variability may increase the risk of recurrent adrenal crises (Bizzarri et al., 2023). In addition, the sudden discontinuation of synthetic steroid therapy in patients who have been on long-term use is also a significant risk factor (Bancos et al., 2015).

Corticosteroids are currently used as therapy for various medical conditions and are widely accessible, including for unsupervised use that disregards indications, proper dosage, and treatment duration. One of the side effects of corticosteroids is adrenal suppression due to their impact on the endocrine system (Wardani, 2023). Toxicity can occur when steroid therapy is abruptly stopped, leading to disease relapse and acute adrenal insufficiency due to suppression of the HPA (Hypothalamus-Pituitary-Adrenal) axis, which can no longer function properly (Azis, 2011). Every individual has different responses in terms of dosage or duration of corticosteroid therapy, making it difficult to determine the relative risk of adrenal crisis in each case.

Adrenal crisis can cause serious complications and even death. Therefore, rapid diagnosis and treatment are key to preventing further complications. In clinical practice, the management of adrenal crisis refers to guidelines such as those recommended by Bornstein et al., 2016, which include intravenous hydrocortisone as first line therapy administered as a 100 mg bolus injection followed by 200 mg over 24 hours or 50 mg every 6 hours along with intravenous fluid resuscitation and hypoglycemia correction (Arlt et al., 2020). Short-acting glucocorticoids should always be used as the main treatment option (Nowotny et al., 2021). If the patient is hypoglycemic, parenteral glucose therapy is necessary (Dineen et al., 2019). A lack of glucose in systemic circulation, especially during hypoglycemia, can have serious effects on brain function, as the brain relies heavily on glucose as its primary energy source for maintaining metabolic and neurological functions. Adrenal crisis cases in geriatric patients often present with atypical symptoms and are at risk of being missed in early recognition. Therefore, it is important to closely monitor blood glucose levels to detect early signs of hypoglycemia, which indicate the need for further interventions, including steroid administration.

Management of adrenal crisis, especially in geriatric patients, requires a collaborative multidisciplinary approach. Geriatric patients usually have comorbidities that may worsen their condition during an adrenal crisis. In this context, multidisciplinary collaboration in acute care management is essential. Nurses, as frontline caregivers, play a critical role in monitoring blood glucose and hemodynamics, recognizing signs of adrenal crisis, and collaborating with the medical team especially doctors for the

administration of corticosteroid therapy. The accuracy of nurses in identifying fluctuations in blood glucose levels and changes in patient hemodynamics is crucial in reducing mortality rates due to delayed diagnosis or treatment. If nurses do not understand the importance of blood glucose monitoring and its relationship to adrenal crisis, the patient's condition may deteriorate without timely intervention. Therefore, this study is essential to observe and report assessments of blood glucose levels, hemodynamics, and multidisciplinary collaboration, especially with doctors in the administration of corticosteroids in adrenal crisis patients, as an effort to strengthen evidence-based practice in healthcare services. In addition, educating patients and families with adrenal insufficiency on adjusting steroid dosages during stress conditions is also a key aspect in preventing adrenal crisis.

Method

The research method used in this study is descriptive quantitative with a case report approach, aimed at providing a detailed description of nursing care for a patient with adrenal crisis due to the sudden discontinuation of long-term steroid therapy (more than 10 years) without a doctor's prescription to manage joint and knee pain due to rheumatoid arthritis, resulting in severe hypoglycemia as one of the clinical manifestations. The case study in this report involves Mrs. A, a 70-year-old, was brought to the emergency department (ED) with the chief complaint of generalized weakness since being at home, accompanied by decreased consciousness. Upon arrival in the inpatient unit, her random blood glucose level (RBG) was recorded at 20 mg/dL. From the interview, it was revealed that the patient had a history of hypertension for over 10 years. Laboratory results on November 20, 2024, showed hemoglobin at 7.1 g/dL, leukocytes at 11,040/mm³, urea at 136 mg/dL, and normal electrolyte levels except for elevated potassium (K: 6.4 mmol/L). She received treatment for five days. Data sources were obtained primarily through direct assessment of the patient and secondarily from family information and medical records. In conducting this study, the researcher adhered to research ethics by maintaining patient confidentiality. In addition, informed consent was obtained, and the researcher ensured that the patient or family had given approval.

Results

Table 1. Random Blood Glucose (RBG) Values on Day 1

Vital Signs	Time	RBG Value	Intervention
BP: 190/100 mmHg HR: 82 bpm RR: 22/min Temp: 36.5 C GCS: E3V4M5 (12)	19.00	35	Bolus of 3 vials D40%
	20.00	45	
	21.00	30	
	22.00	20	
	23.00	44	
	24.00	24	

On the first day (November 19, 2024), the patient experienced significant fluctuations in random blood glucose (RBG) levels, with a trend toward severe hypoglycemia (20–45 mg/dL). Hourly monitoring was performed, and the patient received D40% bolus (3 vials) every hour. Despite temporary increases, glucose levels remained unstable, necessitating tight monitoring and evaluation of collaborative interventions.

Table 2. Random Blood Glucose (RBG) Values on Day 2

Vital Signs	Time	RBG Value	Intervention
BP: 190/100 mmHg HR: 82 bpm RR: 22/min Temp: 36.5°C GCS: E3V4M5 (12)	01.00	20	Bolus 3 vials D40% + D10% maintenance + D40 drip 2 vials/12 hr (1 line)
	02.00	22	
	03.00	23	
	04.00	22	
	05.00	20	
	06.00	23	
	07.00	32	
BP: 170/80 mmHg HR: 77 bpm RR: 21/min Temp: 36.2°C GCS: 15	08.00	46	
	09.00	57	
	10.00	82	
	13.00	44	
BP: 140/90 mmHg HR: 87 bpm RR: 21/min Temp: 36.6°C GCS: 15	17.00	18	
	19.00	18	
BP: 160/90 mmHg HR: 88 bpm RR: 20/min Temp: 36.7°C GCS: 15	21.00	22	
	23.00	36	

On day 2 (November 20, 2024), the patient continued to experience severe hypoglycemia with RBG levels ranging from 18–82 mg/dL. Despite continuous administration of D40% bolus, D10% maintenance, and D40 drip, fluctuations persisted. Glucose levels peaked at 82 mg/dL at 10:00 but dropped to as low as 18 mg/dL by evening. This indicates impaired glucose regulation likely due to adrenal insufficiency, reinforcing the need for corticosteroid therapy and close glucose monitoring.

Table 3. Random Blood Glucose (RBG) Values on Day 3

Vital Signs	Time	RBG Value	Intervention
BP: 160/90 mmHg HR: 88 bpm RR: 20/min Temp: 36.7°C GCS: 15	03.00	38	Bolus 3 vials D40%, D10% maintenance, D40 drip 2 vials/12 hr (2 lines)
	06.00	31	
BP: 130/70 mmHg HR: 80 bpm RR: 20/min Temp: 37.2°C GCS: 15	07.00	43	Bolus 3 vials D40%, D10% maintenance, D40 drip 2 vials/12 hr (2 lines) Dexamethasone 10 mg IV, Flamicort 40 mg IM
	08.00	67	Bolus 3 vials D40%, D10% maintenance, D40 drip 2 vials/12 hr (2 lines)

BP: 140/90 mmHg HR: 96 bpm RR: 20/min Temp: 36.5°C GCS: 15	**14.00**	**97**	**Bolus 3 vials D40%, D10% maintenance, D40 drip 2 vials/12 hr (2 lines), dexamethasone 10 mg iv**
	20.00	**30**	**Bolus 3 vials D40%, D10% maintenance, D40 drip 2 vials/12 hr (2 lines)**
BP: 140/90 mmHg HR: 79 bpm RR: 20/min Temp: 36.6°C GCS: 15	**21.00**	**90**	

On the third day of treatment, November 21, 2024, the patient's blood glucose levels (GDS) continued to fluctuate, with a tendency toward recurrent hypoglycemia. At 07:00, the patient began receiving corticosteroid therapy in the form of dexamethasone 3 × 10 mg intravenously and flamicort 40 mg intramuscularly as additional hormone therapy to address possible adrenal crisis. Following corticosteroid administration, the GDS increased to 67 mg/dL at 08:00, indicating an initial positive response to the combination of glucose and corticosteroid therapy. However, between 10:00 and 13:00, one of the intravenous lines was used for packed red cell (PRC) transfusion, so glucose infusion was only administered through a single line. After the dual-line infusion was resumed at 13:00, the GDS reached to 97 mg/dL at 14:00, the highest value recorded that day. However, the GDS dropped sharply again to 30 mg/dL at 20:00, despite the continued administration of glucose therapy through two lines.

Corticosteroid therapy was initiated on the third day due to the patient's persistently fluctuating GDS levels despite bolus glucose therapy. Dexamethasone provided a temporary positive effect on blood glucose stability. However, hypoglycemia still occurred, especially when interventions were disrupted or suboptimal, indicating that the patient's metabolic condition remained unstable and required strict monitoring and therapeutic adjustments. This also shows that the initial administration of dexamethasone still resulted in fluctuating glucose levels.

Table 4. Random Blood Glucose (RBG) Values on Day 4

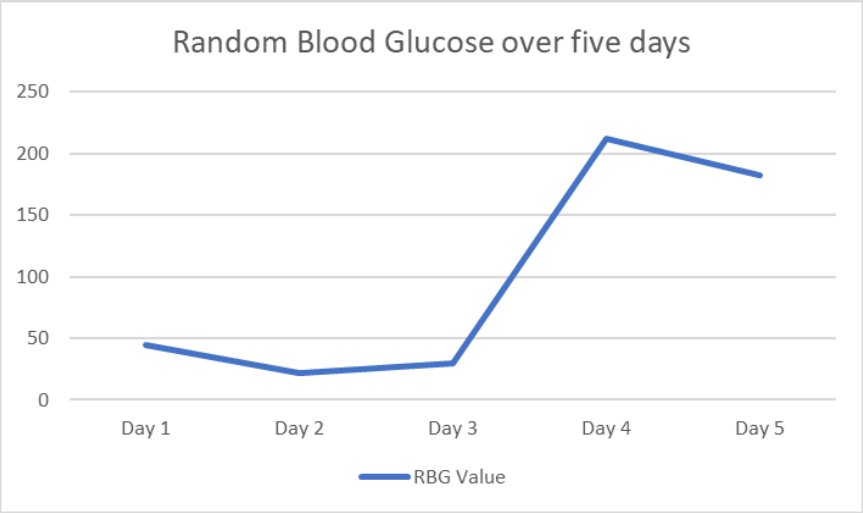
Vital Signs	Time	RBG Value	Intervention
BP: 160/80 mmHg HR: 78 bpm RR: 20/min Temp: 37.1°C GCS: 15	**04.00**	**96**	**D10% maintenance + D40 drip 2 vials/12 hr (2 lines)**
	10.00	**120**	
BP: 120/80 mmHg HR: 74 bpm RR: 20/min Temp: 36.5°C GCS: 15	**16.00**	**243**	**D10% maintenance + D40 drip (1 line)**
	20.00	**212**	

On the fourth day of treatment, November 22, 2024, the patient's blood glucose levels showed significant improvement and tended to stabilize. All of this improvement occurred while the patient continued to receive maintenance D10% and D40% drip (2 flacons every 12 hours) through two intravenous lines, along with corticosteroid therapy—dexamethasone 3x5 mg IV. This condition indicates that the patient's body had started to respond to the glucose and corticosteroid therapy. The increase in blood glucose levels suggests improvement in metabolic function and a possible achievement of adrenal hormone stability. However, the spike to over 200 mg/dL should be closely monitored, and a gradual adjustment of the glucose infusion rate is necessary. Starting at 21:00, the patient was given D10% only, without drip, through a single line.

Table 5. Random Blood Glucose (RBG) Values on Day 5

Vital Signs	Time	RBG Value	Intervention
BP: 150/90 mmHg HR: 69 bpm RR: 21/min Temp: 36.5°C GCS: 15	04.00 10.00	96 182	D10% maintenance 1500 cc/24 hr (1 line)

On the fifth day of treatment, November 23, 2024, during this period, the patient only received maintenance D10% at a volume of 1500 cc over 24 hours through a single intravenous line, without additional bolus or D40 drip. The significant increase in GDS despite only receiving maintenance therapy indicates that the patient's metabolic condition was beginning to stabilize and the body was able to maintain blood glucose levels without intensive therapy. This also suggests an improvement in hormonal function, particularly following the administration of corticosteroids.



Graph 1. Random Blood Glucose (RBG) over five days

Monitoring of random blood glucose levels (RBG) over the five-day period showed fluctuations. On the first day at 20:00, the RBG was recorded at 45 mg/dL, indicating hypoglycemia. A further drop occurred on the second day, with RBG reaching 22 mg/dL, and on the third day it fell again to 30 mg/dL, indicating recurrent severe hypoglycemia. On the fourth day, there was a drastic increase in RBG to 212 mg/dL at 20:00, indicating hyperglycemia. This was likely a response to glucose and corticosteroid therapy beginning to show systemic effects. On the fifth day, the RBG level remained elevated at 182 mg/dL at 10:00, but appeared to be stabilizing compared to the spike on the previous day. These fluctuations suggest that the patient's physiological response to therapy had not yet fully stabilized, and strict monitoring of RBG remains necessary.

Over five days, the nursing interventions evolved from emergency hypoglycemia management to metabolic stabilization and recovery. The novelty of this case lies in the nurse-led early recognition of hypoglycemia, continuous evaluation of glucose delivery strategies, and proactive interdisciplinary collaboration that facilitated timely initiation of corticosteroid therapy. These nursing-driven actions were pivotal in stabilizing blood glucose levels in a patient with adrenal crisis. Furthermore, systematic nursing documentation provided critical clinical evidence to support ongoing evaluation and collaborative therapeutic decision-making.

Discussion

Adrenal crisis is an endocrine emergency that occurs due to an acute deficiency of

cortisol, the primary hormone secreted by the adrenal glands, which plays a crucial role in maintaining the body's homeostasis. Cortisol deficiency triggers a cascade of metabolic disturbances, including hypoglycemia, hypotension, hyponatremia, and sometimes hyperkalemia, all of which can pose serious clinical risks to the patient (Barthel et al., 2019). A lack of cortisol in the body can lead to decreased gluconeogenesis and increased insulin sensitivity, ultimately resulting in hypoglycemia. This is particularly dangerous because the brain relies entirely on glucose for energy, and a glucose deficiency can cause neurological disturbances such as confusion, loss of consciousness, seizures, and even coma. Additionally, aldosterone deficiency leads to sodium loss and potassium retention via the kidneys, causing hyponatremia, hyperkalemia, and severe dehydration.

In the geriatric population, aging is marked by a gradual decline in organ function, including hormonal changes. Cortisol levels tend to increase in older adults due to alterations in the hypothalamic-pituitary-adrenal (HPA) axis, which leads to reduced negative feedback sensitivity to regulate cortisol levels (Stamou et al., 2023). Circadian rhythm also affects cortisol levels, with higher levels typically occurring in the afternoon and evening, impacting sleep patterns (Yiallouris et al., 2019). However, in certain conditions, cortisol levels may drop, especially due to physical or biological stress. In this case, the patient had chronic joint inflammation requiring long-term use of anti-inflammatory medications like Rhemafar and piroxicam. Prolonged exposure to exogenous corticosteroids causes the body to reduce its endogenous cortisol production. When steroid therapy is abruptly stopped, the endocrine system cannot compensate quickly enough, leading to adrenal crisis, especially in elderly patients.

In this case, the patient presented with decreased consciousness, weakness, and severe hypoglycemia, with the lowest recorded random blood glucose level (RBG) at 20 mg/dL. This is consistent with clinical manifestations of adrenal crisis, in which cortisol deficiency impairs glucose metabolism and the physiological stress response (Bancos et al., 2015). Cortisol plays an essential role in maintaining blood glucose levels by stimulating gluconeogenesis, mobilizing free fatty acids, and enhancing the response to catecholamines. A lack of cortisol reduces the body's ability to maintain stable blood glucose, particularly during stress, infection (e.g., tuberculosis), or the sudden discontinuation of steroid therapy (Beuschlein et al., 2024).

Initial treatment involved intravenous glucose administration (D40% boluses) and a D10% drip, which aimed to elevate blood glucose levels. However, the patient's RBG levels remained low and fluctuating during the first two days of hospitalization, indicating that supportive therapy alone was insufficient. This suggests that the primary issue was not merely glucose deficiency, but hormonal dysfunction due to cortisol deficiency. Normally, cortisol supports hepatic gluconeogenesis and inhibits insulin action, thereby helping maintain blood glucose levels, especially during periods of poor food intake. In the absence of sufficient cortisol, the body fails to compensate, leading to recurrent hypoglycemia even with parenteral glucose administration.

In managing adrenal crisis, the choice of corticosteroid is crucial due to its role in replacing deficient cortisol and aldosterone. Hydrocortisone is the first-line recommended therapy because of its balanced glucocorticoid and mineralocorticoid activity (Beuschlein et al., 2024). Mineralocorticoid activity is essential for stabilizing blood pressure and normalizing sodium and potassium levels—common disturbances in adrenal crisis. First-line therapy includes administering 100 mg hydrocortisone IV bolus, followed by 50 mg IM or IV every 6 hours for 24–48 hours, or 200 mg/day continuously (Lewis et al., 2023). In acute settings, mineralocorticoid replacement is typically unnecessary because hydrocortisone provides sufficient mineralocorticoid activity (40 mg hydrocortisone = 100 mcg fludrocortisone). According to Lewis et al. (2023), dexamethasone is not an ideal glucocorticoid substitute because it lacks mineralocorticoid effects, making it ineffective in correcting electrolyte imbalance and blood pressure. However, dexamethasone still has a role in certain situations, particularly during

ACTH stimulation testing to confirm adrenal insufficiency, as it does not interfere with serum cortisol levels (Barthel et al., 2019).

Dexamethasone is more potent than hydrocortisone and has a longer duration of action (>36 hours). It exerts a stronger suppressive effect on the HPA axis compared to hydrocortisone (Nachawi et al., 2024). In this case, dexamethasone administration proved effective as part of the adrenal crisis management. It was chosen due to limited access or unconfirmed diagnosis at the time. Following corticosteroid therapy with dexamethasone and Flamicort, the patient's blood glucose levels became more stable and began to rise between the third and fifth day of hospitalization. This improvement supports the notion that cortisol deficiency was the primary cause of hypoglycemia, and that exogenous glucocorticoid replacement with dexamethasone effectively corrected the metabolic imbalance.

The clinical decision to use dexamethasone was based on technical and efficiency considerations. In acute conditions where the patient's blood pressure remained stable, the priority was to address glucocorticoid deficiency. Thus, dexamethasone administration was appropriate and yielded a positive clinical response. However, due to its long half-life, glucose levels remained variable despite dexamethasone treatment. Other risk factors that worsened the patient's condition included advanced age, a history of chronic disease (hypertension), and thyroid enlargement. This combination increased vulnerability to endocrine dysfunction and the risk of recurrent adrenal crises.

Managing adrenal crisis in geriatric patients follows adult guidelines but requires stricter monitoring, especially in those with comorbidities. In this case, management was not limited to collaborative therapy but included comprehensive nursing care. A key intervention was hourly RBG monitoring to prevent hypoglycemia and assess response to glucose and steroid therapy. Regular assessments of consciousness, vital signs, and general condition were also performed to detect clinical deterioration. Glucose and corticosteroid therapies were administered collaboratively with physicians, and patient responses were carefully documented. In line with the study findings, nursing interventions played a crucial role in the early identification, management, and stabilization of adrenal crisis. Continuous, nurse-led assessment, particularly hourly random blood glucose monitoring and vital signs evaluation, and hemodynamic, as well as in interdisciplinary collaboration when glucose therapy is ineffective.

Understanding the close relationship between corticosteroids, glucose metabolism regulation , and adrenal crisis is crucial appropriate hormone replacement is a key component in managing similar cases and determining the success of clinical interventions. Overall, this case implies that nursing practice should emphasize early vigilance for hypoglycemia as a potential indicator of adrenal crisis, particularly in geriatric patients with a history of long-term corticosteroid use. Timely and appropriately dosed corticosteroid therapy has been shown to significantly improve metabolic conditions and prevent complications. To prevent recurrence, comprehensive patient and family education to improve early detection about the disease, treatment regimen, risk situations, and when to seek medical care is also essential (Çamtosun & Sangün, 2025).

Conclusion

Adrenal crisis is an endocrine emergency caused by cortisol deficiency that can lead to hypoglycemia and death. In this case, a 70-year-old patient experienced severe hypoglycemia despite receiving parenteral glucose therapy. After administering dexamethasone, the patient's blood glucose levels gradually improved and stabilized. Dexamethasone proved effective in stabilizing blood glucose. Future clinical evidence should further evaluate the use of dexamethasone and flamicort in adrenal crisis, particulary in cases of hypoglycemia. Although not the first-line therapy for adrenal crisis, dexamethasone can be a suitable alternative in certain situations, particularly when the diagnosis is unconfirmed or hydrocortisone is unavailable, as it provides rapid glucocorticoid effects without interfering with cortisol assays. Additional prospective studies and structured nursing documentation

are needed to clarify optimal dosing, timing, and clinical outcomes associated with dexamethasone, as well as the supportive role of flamicort in metabolic and hemodynamic stabilization, to strengthen evidence-based, multidisciplinary management of adrenal crisis. Multidisciplinary management combining medical monitoring and educational support is key to preventing recurrence and improving the quality of life for patients with adrenal crisis.

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