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Severe Hyperglycemia in a Febrile Patient: Sepsis as an Underlying Cause

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Introduction

Hyperglycemia is a common finding in hospitalized patients and is often attributed to uncontrolled diabetes. However, in acutely ill patients, particularly those with fever, hyperglycemia may signal an underlying systemic infection such as sepsis. The stress response to infection involves the release of counter-regulatory hormones and cytokines that promote gluconeogenesis and insulin resistance, leading to elevated blood glucose levels even in non-diabetics. Failure to identify sepsis as a precipitating cause of hyperglycemia may delay appropriate treatment and increase morbidity. Early recognition and prompt intervention are essential, as both hyperglycemia and sepsis independently contribute to poor outcomes. This case emphasizes the importance of considering infection as a trigger for acute metabolic derangement. Moreover, distinguishing stress-induced hyperglycemia from chronic poorly controlled diabetes is critical, as their management and prognostic implications differ. Stress hyperglycemia typically resolves with treatment of the underlying illness, whereas persistent elevation may warrant further evaluation for undiagnosed diabetes. Clinicians should be alert to abrupt changes in glucose levels, especially in non-diabetic patients, as these may be early indicators of systemic infection or organ dysfunction. Incorporating routine glucose monitoring into the assessment of febrile or critically ill patients can aid in the early detection of sepsis and guide timely initiation of antimicrobial and supportive therapy [1].

Description

Severe hyperglycemia in the context of acute infection can result from stress-induced insulin resistance and increased hepatic glucose output. In patients with previously well-controlled diabetes, a sudden and marked elevation in blood glucose levels should prompt evaluation for an acute underlying illness, particularly sepsis. This metabolic response is driven by inflammatory cytokines such as TNF-α and IL-6, which impair insulin signaling, alongside stress hormones like cortisol and catecholamines that promote gluconeogenesis and lipolysis. As a result, hyperglycemia may occur even in non-diabetic individuals, and its severity often correlates with the intensity of the inflammatory response. The use of HbA1c levels is invaluable in distinguishing chronic hyperglycemia from an acute metabolic derangement. A relatively normal HbA1c with extreme hyperglycemia points toward stress hyperglycemia rather than poor glycemic control. This distinction carries significant implications for both treatment and prognosis. Even in the absence of diabetic ketoacidosis or hyperosmolar hyperglycemic state, stress hyperglycemia can contribute to endothelial dysfunction, impaired immune response, and increased morbidity if not managed appropriately. Rapid identification and treatment of the infectious trigger, coupled with glucose-lowering interventions, are essential to reverse the metabolic disturbance and mitigate complications [2].

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Clinicians should maintain a high index of suspicion for underlying infections such as pneumonia, urinary tract infections, or intra-abdominal sepsis in diabetic patients who present with sudden, unexplained hyperglycemia and systemic symptoms. Empirical antibiotic therapy, guided by local epidemiology and clinical presentation, should be initiated promptly after obtaining relevant cultures. Intensive insulin therapy using intravenous infusions is effective in achieving rapid glycemic control during the acute phase, with transition to subcutaneous insulin as the patient stabilizes. Close glucose monitoring and careful fluid management further support recovery and prevent iatrogenic complications. As the interplay between infection and glucose metabolism becomes better understood, future strategies may focus on early identification of stress hyperglycemia as a prognostic marker rather than a secondary finding. Biomarkers of inflammation and stress, such as procalcitonin, IL-6, or cortisol levels, could be integrated into clinical protocols to help differentiate between stress-induced and chronic hyperglycemia. This could facilitate more tailored interventions and earlier sepsis recognition, improving outcomes in both diabetic and non-diabetic populations [3].

Precision medicine approaches, including Continuous Glucose Monitoring (CGM) in hospitalized patients, may offer real-time insights into glucose dynamics during infection and recovery. Combined with predictive analytics, CGM data could help clinicians anticipate glycemic excursions, personalize insulin regimens, and reduce the risk of both hyperglycemia and hypoglycemia in acutely ill patients. Incorporating artificial intelligence into these systems could further optimize treatment algorithms and resource utilization in high-dependency settings. On a broader scale, post-discharge follow-up protocols should be enhanced for patients who experience stress hyperglycemia during hospitalization. Identifying those at risk of developing new-onset diabetes or persistent glucose intolerance following an acute illness is crucial. Longitudinal studies are needed to understand the long-term metabolic implications of stress hyperglycemia and to develop preventive strategies, including lifestyle counseling and metabolic screening. As healthcare systems move toward holistic and preventive care, bridging acute and chronic disease management will be essential for improving outcomes in patients affected by infection-related hyperglycemia. Expanding clinical awareness and education about stress hyperglycemia among healthcare providers is also critical [4].

Emergency and primary care physicians are often the first to evaluate patients presenting with fever and hyperglycemia. Training initiatives that emphasize the importance of evaluating for occult infections especially in patients without a known history of diabetes can lead to faster diagnoses and improved care. Including stress hyperglycemia in standard medical curricula and continuing education programs will help reduce diagnostic delays and mismanagement, particularly in resource-limited settings where the overlap of infectious diseases and metabolic disorders is common.Additionally, there is growing interest in exploring pharmacological agents that can modulate the inflammatory and hormonal cascades driving stress hyperglycemia. Targeted therapies that address cytokine-mediated insulin resistance or stress hormone dysregulation may offer adjunctive treatment options, particularly in critically ill patients. From a public health standpoint, integrating infection screening and glucose monitoring into community-level care for individuals with known or suspected diabetes may help reduce hospital admissions due to acute metabolic complications. Community health workers can play a pivotal role in identifying early signs of infection or hyperglycemia, providing patient education, and ensuring timely referrals. This approach is particularly relevant in low- and middle-income countries, where the dual burden of infectious diseases and diabetes is increasing. Strengthening the link between primary care and inpatient services through standardized care pathways can enhance continuity of care and ultimately improve outcomes for at-risk populations [5].

Conclusion

Hyperglycemia in febrile patients should prompt evaluation for underlying sepsis, especially when glycemic levels are significantly elevated without corresponding chronic indicators. This case demonstrates that acute infections can disrupt glucose homeostasis even in previously controlled diabetics. Early diagnosis, aggressive infection control, and insulin therapy can reverse the metabolic imbalance and improve prognosis. Clinicians must recognize hyperglycemia as a potential red flag rather than solely a metabolic concern in acutely ill patients.

Acknowledgment

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Conflict of Interest

None.

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