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Case Report

Recurrent Septic Illness in Pregnancy Complicated by Primary Adrenal Insufficiency: Successful Prevention of Adrenal Crisis

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Abstract

Background: Primary adrenal insufficiency is a rare endocrine disorder characterized by deficient cortisol production, with or without aldosterone deficiency, and carries a significant risk of adrenal crisis during pregnancy due to increased physiological stress demands. This case aimed to highlight the diagnostic and therapeutic challenges of managing adrenal insufficiency complicated by recurrent infection during pregnancy.

Case Report: A 39-year-old gravida 4 para 3+0 African woman with known primary adrenal insufficiency presented twice during pregnancy with recurrent multidrug-resistant septic illness. At 29+3 and 32 weeks of gestation, she developed fever, dysuria, respiratory symptoms, and systemic inflammatory features consistent with sepsis. Investigations revealed elevated inflammatory markers, electrolyte imbalance, and multidrug-resistant urinary infection. She was managed with intravenous antibiotics, intensive monitoring, and escalation from oral to stress-dose intravenous hydrocortisone (50 mg every 6 hours), with multidisciplinary input from obstetrics, endocrinology, and infectious disease teams. An elective cesarean section with bilateral tubal ligation was performed at 37 weeks under perioperative steroid coverage. Maternal recovery was uneventful, and a healthy 2.8 kg neonate was delivered.

Conclusion: This case highlights that early recognition and prompt stress-dose steroid therapy are critical in preventing adrenal crisis during pregnancy. Recurrent infection significantly increases physiological stress and risk of endocrine decompensation. Multidisciplinary management is essential to optimize maternal and foetal outcomes.

Keywords: *Primary Adrenal Insufficiency; Addison Disease; Pregnancy; Adrenal Crisis; Sepsis; Hydrocortisone; Multidisciplinary Care*

Introduction

Pregnancy is a state of profound endocrine adaptation. It requires coordinated changes in adrenal function to maintain maternal cardiovascular stability and foetal growth. Cortisol is one of the main hormones involved in this adaptation [1]. It promotes vascular tone, glucose control, and immune modulation. When adrenal function is impaired, this adaptive system becomes inadequate. Primary adrenal insufficiency (PAI), commonly referred to as Addison's disease, is a chronic endocrine disorder caused by destruction or dysfunction of the adrenal cortex [1,2]. It results in deficient cortisol production, with or without aldosterone deficiency [3,4]. The most common cause in developed countries is autoimmune adrenalitis [3,5]. This damage via the immune process results in a permanent loss of function. As gestation progresses, this mismatch becomes more clinically relevant [6,7].

Primary adrenal insufficiency occurs infrequently in clinical settings. The estimation of prevalence is about 100 - 140 per million in developed regions [4]. However, women are affected by this condition more often than men, especially in the reproductive age [8,9]. This distribution makes it more relevant to obstetric populations. Recent data show a rising frequency of detections, presumably because of better survival and diagnostic recognition [10]. Overall, the prevalence of these cases is rare, but increasing in high-risk obstetric environments [3].

Adrenal physiology is profoundly changed by pregnancy. The production of corticosteroid-binding globulin increases gradually, which in turn increases cortisol levels [6]. Pregnancy increases total cortisol because of increased corticosteroid-binding globulin, but free cortisol also rises**,** particularly later in pregnancy. Aldosterone also rises to maintain the plasma volume expansion [8]. Concurrently, progesterone inhibits the mineralocorticoid receptors. This impacts sodium and fluid balance. These changes may create physiological instability even in healthy pregnancies.

These adaptations also create diagnostic challenges. Adrenal insufficiency symptoms are similar to those of normal pregnancy. Both conditions result in fatigue, nausea, vomiting, dizziness and hypotension [6,9]. Consequently, adrenal insufficiency can be overlooked until it reaches an advanced stage. Diagnostic delay is a critical risk factor in the clinical setting [4].

Adrenal crisis is a life-threatening condition resulting from acute adrenal insufficiency. It is characterized by a critical deficiency of cortisol, typically triggered by physiological stress [11,12]. Common causes are infection, surgery, labour, hyperemesis gravidarum and sepsis [13,14]. These conditions increase cortisol demand rapidly. In adrenal insufficiency, this response is absent or insufficient. This leads to circulatory collapse.

Maternal consequences are severe, including hypotension, shock, electrolyte disturbances, and admission to intensive care. These complications are hypotension, shock, electrolyte disturbances and intensive care admission [11]. The foetal effects are also important. Increased placental under-perfusion may result in foetal distress, growth restriction or premature delivery [10]. In delayed or untreated cases, maternal mortality is still a concern. Inappropriate steroid use in pregnancy and postpartum has been demonstrated as a preventable cause of death in confidential enquiry reports [15]. Cohort studies also indicate that having a chronic disease before pregnancy does not eliminate the risk of developing other intercurrent illnesses during pregnancy, such as an acute illness, during pregnancy [9].

Management depends on timely glucocorticoid replacement. During an acute illness, the use of stress dose steroids is essential [11]. This method is designed to simulate the physiological behavior of cortisol in the body. Requirements rise even more during pregnancy. In the 3rd trimester, the dosing is increased by 20 - 40% as the physiological requirement increases during the last trimester [10,16]. During the perinatal period, steroid coverage is recommended at the time of delivery to prevent hemodynamic collapse [17]. Clinical monitoring consists of checking the stability of electrolytes, glucose, and blood pressure [14]. Collaborative management is beneficial. Strong teamwork and communication between the obstetrics, endocrinology, intensive care, and infectious disease groups are required [9,17].

Despite extensive literature, important gaps remain. There is still limited pregnancy-specific information available on adrenal insufficiency [6]. Recurrent infection-induced adrenal stress is especially poorly described. This is significant because one of the most common triggers of decompensation is an infection [10]. Systemic inflammation and delayed treatment further complicate cases involving multidrug-resistant infections [17]. There is also a lack of research on metabolic complications during repeated steroid step-up. Complications observed include glucose instability and electrolyte disturbance [18].

The case presented repeated physiological stress during pregnancy, including multiple septic illnesses with multidrug-resistant organisms. Despite high risk factors, the patient did not develop adrenal crisis, emphasizing the need for timely escalation of stress dose steroids and the coordinated management by multidisciplinary teams. This case report aimed to describe a complex pregnancy in

primary adrenal insufficiency complicated by recurrent multidrug-resistant sepsis. It focused on repeated stress-dose glucocorticoid use and multidisciplinary management. It also emphasized early recognition and prevention of adrenal crisis.

Case Presentation

Patient background

The patient was a 39-year-old African woman who had come to the Middle East for geopolitical and economic reasons. She was gravida 4, para 3+0, with three previous cesarean sections. She had a known diagnosis of primary adrenal insufficiency for 8 years and was maintained on oral hydrocortisone replacement therapy. She had been pregnant with steroid-associated hyperglycemia, which had necessitated intermittent insulin therapy. No early pregnancy endocrine optimization records were available.

Physical examination

The patient at 29+3 weeks of gestation presented with fever, dysuria, lower abdominal pain, fatigue and generalized weakness. On examination, she was febrile, tachycardic, and clinically dehydrated. Blood pressure was borderline low. Cardiovascular and respiratory examinations were otherwise unremarkable. Abdominal examination revealed suprapubic tenderness. Transvaginal ultrasound examination of the uterus indicated no irritability and that it was at the correct size for gestational age. Foetal heart rate and growth parameters were reassuring.

She returned to the hospital at 32-weeks' gestation, with fever, tachycardia, tachypnea, and deterioration of her respiratory status indicative of sepsis. Her systemic inflammatory response pattern was evident with the need for high dependency monitoring. Obstetric and foetal evaluations were unchanged and no distress was observed.

Initial clinical investigations

Clinical examination showed that the infection was also severe and had multidrug resistance with extensive metabolic derangements as shown in table 1.

Investigation	Result	Reference Range	Interpretation
Hemoglobin	Low	Normal	Anemia of illness
CRP	Elevated	Normal	Active systemic inflammation
Procalcitonin	Elevated	Normal	Severe bacterial infection
Potassium	Low	Normal	Electrolyte imbalance
ABG	Respiratory alkalosis	Normal	Sepsis-related metabolic change
Urine culture	MDR *E. coli*	Negative	Multidrug-resistant UTI
High vaginal swab	MRSA positive	Negative	Infective colonization
Chest X-ray	No acute pathology	Normal	No pulmonary source
Renal ultrasound	Nephrocalcinosis, renal stones	Normal	Chronic urinary tract disease
Pelvic ultrasound	Normal pregnancy	Normal	No obstetric complication

Table 1: *Laboratory and imaging findings during hospital admissions.*

Diagnosis

The patient was diagnosed with recurrent multidrug-resistant urinary tract infection with sepsis in pregnancy. She had known primary adrenal insufficiency with a high risk of adrenal crisis during acute illness. Additional diagnoses included steroid-induced hyperglycemia and persistent electrolyte imbalance. The clinical picture was complicated by overlapping symptoms between sepsis and adrenal insufficiency.

Initial and preoperative management

She was admitted under high-dependency care for continuous monitoring. Intravenous fluid resuscitation was initiated along with culture-guided broad-spectrum antibiotics. Oral hydrocortisone was converted to intravenous hydrocortisone at a stress-dose regimen (50 mg every 6 hours). Early endocrinology and infectious disease consultations were obtained. Continuous maternal and foetal monitoring was maintained.

Treatment plan and management

Management was focused on infection control, endocrine stabilization and metabolic correction. Gradual tapering of stress-dose glucocorticoids was continued during both septic episodes, after stabilization. The electrolyte imbalance was resolved by potassium supplementation. Sliding scale insulin therapy was used for blood glucose control. A multi-disciplinary team, including specialists from obstetrics, endocrinology, infectious disease and intensive care, provided care.

Postoperative care

An elective cesarean section was performed at 37 weeks following multidisciplinary planning. Bilateral tubal ligation was completed after counseling. Perioperative steroid coverage was escalated with intravenous hydrocortisone and gradually tapered postoperatively based on clinical response.

Hospital course

The patient was admitted to the hospital twice during pregnancy for septic illness, where steroid therapy had to be escalated and intensive monitoring was necessary. Antibiotic and endocrine optimization resulted in clinical stabilization. Clinical stabilization was achieved following antibiotic therapy and endocrine optimization. The postpartum course was complicated by transient hypertension, hyperglycemia, and electrolyte imbalance, which gradually improved with supportive management.

Discharge plan

She was discharged on double-dose oral hydrocortisone with a structured tapering regimen under endocrine supervision. She was educated on sick-day rules, emergency steroid administration, and early recognition of adrenal crisis symptoms. Follow-up was arranged with endocrinology and obstetrics services.

Follow-up and outcomes

At the 6-week postpartum review, the patient remained clinically stable with no evidence of adrenal decompensation. The neonate weighed 2.8 kg at birth, required 24-hour observation, and was discharged in good health without complications. Maternal endocrine control remained stable on maintenance therapy.

Discussion

Primary adrenal insufficiency in pregnancy is widely recognized as a high-risk endocrine condition, particularly due to the inability of the adrenal cortex to meet increasing gestational cortisol demands. As demonstrated by Hahner., *et al*. (2021) and Husebye., *et al*.

(2021), this condition is extremely rare in obstetrics, but it is becoming more common because of better survival and awareness [3,4]. In this case, no single stressor precipitated an adrenal crisis, unlike some previously reported cases. Recurrent infections appeared to cause physiological stress without leading to adrenal decompensation, representing a clinical course somewhat different from typical reports [9].

Consistent with the findings highlighted by Lee and Torpy (2023), pregnancy is associated with a gradual increase in cortisol production that is due to the increase in corticosteroid-binding globulin [6]. Another study by Alexandraki and Kaltsas (2022) also described that progesterone has a functional antagonistic effect on mineralocorticoid receptors and that this effect results in fluid balance changes in late gestation [8]. However, while these physiological adaptations are well described in the literature, pregnancy itself increases demands, but vulnerability depends on adequacy of replacement therapy. In contrast, the present case did not follow the commonly demonstrated pattern of rapid decompensation, despite repeated physiological stress from infection.

Diagnostic difficulty in adrenal insufficiency during pregnancy has been widely discussed. Similar to observations by Husebye., *et al*. (2021) and Bothou., *et al*. (2020), symptoms such as fatigue, nausea, hypotension, and vomiting are frequently misinterpreted as physiological pregnancy changes or sepsis-related manifestations [4,9]. Delayed recognition has been described in cohort studies, often contributing to poor outcomes. However, in contrast to cases reported in the literature where diagnostic delay led to adrenal crisis or maternal deterioration, the current case was recognized early during both admissions, allowing prompt steroid escalation and prevention of endocrine collapse [1,12].

Infection is reported as the most significant precipitating factor for adrenal crisis. Lentz., *et al*. (2022) documented that systemic infection markedly increases cortisol demand, often exceeding adrenal reserve in affected patients [14,19]. Similarly, Afzal., *et al*. (2025) discussed that multidrug-resistant infections significantly prolong inflammatory stress and complicate metabolic stability [17]. Felker., *et al*. (2025), in maternal mortality reviews, also highlighted that delayed steroid administration during infection-associated stress contributed to preventable deaths [15]. In comparison, although the present case involved recurrent multidrug-resistant urinary tract infections with sepsis-like presentations, adrenal crisis did not develop, which contrasts with the most severe outcomes demonstrated in the literature under similar infectious burden.

Stress-dose glucocorticoid therapy is consistently emphasized across endocrine and obstetric guidelines. Nowotny., *et al*. (2021) described that early hydrocortisone escalation during acute illness and pregnancy-related stress is essential to prevent adrenal crisis [11]. Dineen., *et al*. (2019) further explained that glucocorticoid replacement restores vascular responsiveness and prevents circulatory collapse [13]. Similar to these recommendations, the present case required repeated escalation to intravenous hydrocortisone during septic episodes. However, whereas delayed or inadequate steroid adjustments have been associated with shock in some reports, the timely administration in this case may have helped prevent progression to adrenal crisis.

Electrolyte disturbances in adrenal insufficiency are classically described as hyponatremia and hyperkalemia due to aldosterone deficiency. However, Adrogué., *et al*. (2022) highlighted that biochemical patterns may be altered in acute illness and with concurrent therapies [18]. Similarly, Green., *et al*. (2024) noted that insulin therapy and pregnancy-related metabolic shifts may mask classical adrenal biochemical findings [10]. In this case, hypokalemia was observed rather than the expected hyperkalemia, which differs from the typical pattern highlighted in Addison's disease [20]. This atypical presentation aligns with emerging literature suggesting that biochemical profiles in pregnancy may not reliably reflect underlying adrenal status during acute infection.

Multidisciplinary management has been consistently demonstrated as a key determinant of favorable outcomes. Afzal., *et al*. (2025) and Botho., *et al*. (2020) described that coordinated care between obstetrics, endocrinology, and intensive care significantly improves maternal outcomes in endocrine emergencies [9,17]. Similar multidisciplinary collaboration was employed in this case, involving

infectious disease and critical care teams. In this case, hypokalemia was observed instead of the more commonly expected hyperkalemia, which differs somewhat from the typical pattern described in Addison's disease.

From a broader healthcare perspective, Hahner., *et al*. (2021) highlighted that fragmented medical records and patient migration can contribute to inconsistent endocrine management and delayed optimization [3]. Similar challenges were observed in this case, where prior endocrine documentation was limited due to relocation. This represents a recognized, though less frequently highlighted, systemic factor that can contribute to the complexity of managing chronic endocrine diseases during pregnancy.

Similar to previously demonstrated literature, this case emphasise that infection is the primary precipitant of adrenal stress during pregnancy and that early steroid escalation is essential. However, in contrast to most described cases, repeated multidrug-resistant septic episodes did not progress to adrenal crisis, likely due to timely multidisciplinary intervention and prompt glucocorticoid adjustment. This divergence from expected outcomes highlights the critical role of early recognition and strict adherence to stress-dose steroid protocols in preventing maternal deterioration.

Conclusion

This case highlights primary adrenal insufficiency in pregnancy complicated by recurrent multidrug-resistant septic illness, creating repeated episodes of physiological stress. Early recognition of clinical deterioration and timely escalation of glucocorticoid therapy were critical in preventing adrenal crisis. Recurrent infection significantly increased maternal risk, requiring continuous metabolic and hemodynamic monitoring. Coordinated multidisciplinary care ensured maternal stabilization and a favorable foetal outcome. This case reinforces the importance of prompt steroid adjustment, infection control, and structured endocrine–obstetric collaboration to prevent life-threatening complications in pregnancy.

Ethics Approval

Not required as per institutional case report policy.

Consent for Publication

Written informed consent was obtained from the patient for publication.

Conflict of Interest

The authors declare no conflict of interest.

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Author Contributions

All authors contributed to case management, data collection, and manuscript preparation.

Data Availability

Available from the corresponding author upon reasonable request.

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