INVITED ARTICLE

Parturient with Endocrine Disorders in the Intensive Care Unit

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Abstract

Almost every endocrine axis is influenced by pregnancy. The diagnosis in acute cases is challenging as the classical symptoms are often masked. Thyroid storm is found in only 1–2% of hyperthyroid parturients (0.1–0.4% of all pregnancies). Burch and Wartofsky scoring system is useful for the identification of thyroid storms. Myxedema coma is an extremely rare complication of overt hypothyroidism with a 20% mortality rate. Diabetic ketoacidosis usually reported in the second and third trimesters carries a risk of fetal loss in 10–25% of cases. The size of the tumor rises in 2.7% of microprolactinomas and 22.9% of macroprolactinomas during pregnancy. Adrenal insufficiency in pregnancy is usually caused by primary adrenal failure, which is mostly autoimmune in origin. Pheochromocytoma may present as preeclampsia during pregnancy. Unrecognized pheochromocytoma is associated with a maternal mortality rate of 50%. Shared decision-making and close coordination between critical care, anesthesiology, obstetrics, and endocrinology can help in assuring good maternal and fetal outcomes.

Keywords: Acute adrenal insufficiency, Diabetic ketoacidosis, Myxedema coma, Pheochromocytoma, Pituitary emergencies, Pregnancy, Thyroid storm.

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Introduction

Despite a declining trend of maternal mortality rates with improvements in healthcare infrastructure and awareness, the elevated rate (113 deaths per 100,000 live births in 2016–18, from 130 per 100,000 live births in 2014–16) in comparison with the western countries is still a major concern for healthcare delivery system in India.[1] As the accessibility of institutionalized maternal healthcare is improving, our critical care units are going to have a greater number of parturient.

Almost every endocrine axis is revamped in pregnancy due to the interactions between the matriarchal endocrine system and the fetoplacental unit. The majority of the endocrinopathies can be emergencies and detrimental for both mother and fetus if overlooked. The diagnosis and therapeutic management in acute cases are challenging as the classical symptoms are often muted or masked, as well as the laboratory chemical reference ranges for endocrinological parameters are influenced by pregnancy. Pharmacological options are limited with stricter therapeutic goals than nonpregnant patients.

This review article summarizes the endocrinological situations a critical care physician might realistically face in an intensive care unit (ICU) and their management. However, it is not an exhaustive review of all endocrinological disorders of pregnancy and should be supplemented by further reading.

Acute Thyroid Disorders

Alteration in thyroid function is among the frequently encountered endocrine conditions in females of the reproductive age-group. In pregnancy, the following changes are reflected in the thyroid function to meet the increase in metabolic needs:

- Serum thyroid-binding globulin (TBG) rises almost two-fold
- Total T4 and T3 rise by 50% and reach a steady state by 20 weeks of gestation.
- Decrease in serum thyroid-stimulating hormone (TSH) during the first trimester due to the stimulation of the TSH receptors by elevated levels of serum human chorionic gonadotropin (hCG).

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Since pregnancy itself affects thyroid function, it is common for previously well-managed thyroid disease to become uncontrolled, requiring intensive monitoring and care.

Hyperthyroid Emergencies

Thyrotoxicosis

Thyrotoxicosis, which refers to overexposure to thyroid hormones secondary to overproduction (Table 1), is present in 0.1–0.4% of all pregnancies.[2] Hyperthyroidism can cause hypertension, preeclampsia, and congestive heart failure and can increase the chances of preterm delivery and placental abruption. The fetus can be affected by the transplacental transfer of TSH receptor antibodies (TRAb), which can lead to fetal and neonatal hyperthyroidism. Risks include growth retardation, prematurity cardiac conduction abnormalities, and death.

Hyperthyroidism, which is subclinical, mild, and asymptomatic, does not usually warrant treatment during pregnancy. With overt hyperthyroidism, the principles of treatment remain the same, that is, to control thyroid dysfunction. Patients with life-threatening hyperthyroidism should be managed in the ICU.

Laboratory diagnosis of hyperthyroidism is confirmed with a suppressed serum TSH in the setting of elevated free T4 levels without the presence of nodular goiter or thyroid mass. A downward shift of the TSH reference range occurs during pregnancy, with a

Table 1: Common causes of hyperthyroidism in pregnancy[2]

- Graves disease
- Toxic adenoma
- Hyperemesis gravidarum
- Trophoblastic disease
- Thyroiditis (chronic, subacute, viral)
- Exogenous thyroid hormone
- Toxic multinodular goiter

reduction in both the lower (decreased by about 0.1–0.2 mU/L) and the upper limits of maternal TSH (decreased by about 0.5–1.0 mU/L), relative to the typical nonpregnant TSH reference range. In this reduction too, there are ethnic and racial differences. The American Thyroid Association recommends the use of trimester-specific ranges and cutoffs.[3]

Thyroid Storm

In acute exacerbation of hyperthyroidism, although found only 1–2% of hyperthyroid parturient, the consequences can be disastrous for both mother and fetus. Usually seen in patients with poorly controlled hyperthyroid states, with one or more of the following manifestations: high-grade fever (104–106°F), tachycardia, cardiac dysrhythmias, agitation, anxiety, delirium, psychosis, stupor, or coma, features of congestive heart failure, nausea, vomiting, and abdominal pain. Infections, trauma, surgery, or exposure to anesthesia, labor, and hypertensive emergencies may be the triggering events. A physical examination can reveal goiter, ophthalmopathy (in the presence of Graves' disease), lid lag, hand tremor, and warm and moist skin.

Burch and Wartofsky introduced a scoring system using precise clinical criteria for the identification of thyroid storms (Table 2).[4] While the strongest association of thyroid storm is seen in a score of ≥45, a score between 25 and 44 is the marker of an impending risk, the score <25 has almost no risk. Thyroid function should be assessed in all suspected cases of thyroid storm, but treatment should not be delayed in anticipation of the results.

Management

The principles of management of parturients with hyperthyroid emergencies are as follows:

- Treatment needs to be immediately instituted with beta-blockers for alleviating the responses due to elevated adrenergic tone. Typically, propanolol 60–80 mg orally or via NG, every 4–6 hours is given, or 0.5–1 mg intravenously over 10 minutes followed by 1–2 mg intravenously over 10 minutes every few hours. An advantage of propanolol is that it inhibits the type 1 deiodinase, which may help reduce serum T3 levels. The Japan Thyroid Association recommends esmolol (loading dose of 250–500 µg/kg IV, followed by an infusion at 50–100 µg/kg/minute) over propranolol because of concerns over increased mortality in patients with congestive heart failure treated with propranolol.[5] In patients with reactive airway disease, cardioselective beta-blockers such as metoprolol can be used. Atenolol is avoided in pregnancy as it causes fetal growth restriction. It is advisable to minimize the duration of β-adrenergic blocker therapy during gestation and to wean the patient off these agents once the thionamides are controlling the hyperthyroid symptoms. Calcium channel blockers can be used to achieve rate control in situations where beta-blockers are contraindicated.

Table 2: Burch and Wartofsky scoring system for thyroid storm[4]

Thermoregulatory dysfunction	
Temperature (°F|°C)	
99–99.9|37.2–37.7	5
100–100.9|37.8–38.2	10
101–101.9|38.3–38.8	15
102–102.9|38.9–39.4	20
103–103.9|39.4–39.9	25
≥104.0|>40.0	30
Central nervous system effects	
Mild	10
Agitation	
Moderate	20
Delirium	
Psychosis	
Extreme lethargy	
Severe	30
Seizure	
Coma	
Gastrointestinal-hepatic dysfunction	
Moderate	10
Diarrhea	
Nausea/vomiting	
Abdominal pain	
Severe	20
Unexplained jaundice	
Cardiovascular dysfunction	
Tachycardia	
99–109	5
110–119	10
120–129	15
130–139	20
≥140	25
Atrial fibrillation	10
Heart failure	
Mild	5
Pedal edema	
Moderate	10
Bibasilar rales	
Severe	15
Pulmonary edema	
Precipitant history	
Negative	0
Positive	10

*A score of 45 or more is highly suggestive of thyroid storm, a score of 25–44 supports the diagnosis, and a score below 25 makes thyroid storm unlikely (Burch and Wartofsky[4])

- Application of thionamides blocks de novo thyroid hormone synthesis. However, they do not have any effect on the release of the stored hormone. Propylthiouracil (PTU) 600–800 mg orally followed by 150–200 mg per oral/4–6 hourly (alternatively by NG tube or as a rectal suppository) or methimazole 20 mg orally every 4–6 hours should be given. For life-threatening

thyroid storms, PTU is preferred over methimazole as it blocks the peripheral conversion of T4 to T3 and causes a more rapid fall in serum T3 levels. Methimazole has a longer half-life and is less hepatotoxic as compared to PTU and can be used for severe thyrotoxicosis, which is not life-threatening. On the flip side, it is recommended to avoid methimazole in the first trimester as there are concerns about increased teratogenic risk. There are no intravenous preparations available for the drugs although there are some methods described to dissolve tablets in saline to make a stop-gap IV [6,7] or rectal formulations.[8–10]

- An iodine preparation should be administered to stop the release of stored hormone by inhibiting the proteolysis of thyroglobulin. Iodine initially stimulates the production of thyroid hormone. Thus, it should be given only after PTU has been administered, typically after 1–2 hours. The following formulations can be given:
  - Saturated solution of potassium iodide 2–5 drops/8 hourly, or
  - Sodium iodide 0.5–1 g IV/8 hourly, or
  - Lugol's solution 8 drops/6 hourly, or
  - Lithium carbonate 300 mg orally/6 hourly.
- Glucocorticoids to reduce T4-to-T3 conversion promote vasomotor stability, possibly reduce the autoimmune process in Graves' disease, and possibly treat an associated relative adrenal insufficiency. This can be either dexamethasone 2 mg intravenously or intramuscularly every 6 hours for four doses or hydrocortisone 300 mg/d IV or prednisone 60 mg/d orally.
- Cholestyramine, a bile acid sequestrant that reduces the enterohepatic circulation of T4 and T3 and enhances elimination is given orally in the dose of 4 g every 6 hours.
- Supportive therapy in terms of alleviation of hyperpyrexia, maintenance of oxygenation, fluid therapy, nutritional support, and search for, and treatment of infection focus is essential for a favorable outcome.

Patients with multiple organ failure often require intensive therapy for congestive heart failure, respiratory failure, acute kidney injury, hepatic insufficiency, and coma. Plasmapheresis and plasma exchange have been reported to be successful in patients who do not tolerate thionamide therapy. Continuous fetal monitoring should be performed.

Hypothyroid Emergencies

Hypothyroidism is relatively common with the following etiologies: iodine deficiency, autoimmune disorders, and hypothalamic or pituitary gland pathologies, following thyroidectomy, or radio ablative therapy. Most of the women usually possess preexisting hypothyroidism before pregnancy. A concurrent load on the thyroid gland during pregnancy can manifest as new-onset hypothyroidism in previously euthyroid patients.

An acute onset central hypothyroidism during parturiency can occur due to Sheehan syndrome, or pituitary macroadenomas, head injuries, or lymphocytic hypophysitis, or sarcoidosis.

Myxedema Coma

Myxedema coma is an extremely rare complication of overt hypothyroidism in pregnancy. Lack of healthcare penetration and access to proper prenatal care increases the incidence and is relatively more common in remote and underserved areas.

The presentation can be varied and misleading. It may mimic preeclampsia with hypertension, proteinuria, and encephalopathy. Changes in mentation, extreme cold intolerance, hypothermia, bradycardia, hypoventilation, generalized edema, and effusions should raise the suspicion of myxedema coma in patients with preexisting and uncontrolled hypothyroidism.

The etiology is poorly understood although a precipitating event is common. Sepsis, preeclampsia, exposure to cold temperature, use of codeine, sedatives, anesthetics, lithium, ferrous sulfate and calcium supplements, labor, and surgery can trigger myxedema coma.

Thyroid function tests are abnormal but TSH, T4, and T3 levels do not always correlate with the severity of the condition. Cortisol levels should also be checked because patients with central hypothyroidism may have associated hypopituitarism and secondary adrenal insufficiency. Metabolic derangements such as hyponatremia, hypoglycemia, acidosis, and anemia are common. If left untreated, progression to severe hypoventilation with carbon dioxide retention, hypothermia, cardiac arrhythmias, hypotension with multiorgan failure, and coagulopathy and coma will ensue.

A patient with myxedema coma must be managed in the ICU and must include the following:

- Mechanical ventilation for hypoventilation and airway protection in severely altered or comatose patients
- Coagulopathy correction
- Hypothermia correction—active warming may result in rapid vasodilation and thereby precipitating hypotension and shock. A normalizing trend should be expected within 36–48 hours.
- Severe hyponatremia with seizures should be treated with 3% sodium chloride until serum sodium is >120. An increase in serum sodium should be achieved per guidelines for the treatment of severe symptomatic hyponatremia.
- Close fetal monitoring and careful timing of delivery after maternal stabilization are important.
- Search and treatment of precipitating cause.
- Replacement therapy.

The American Thyroid Association recommends empiric glucocorticoid coverage (hydrocortisone 50–100 mg IV every 6–8 hours) followed by a bolus of 200–400 µg levothyroxine. A judicious dosage is advised for patients with a smaller stature, or with a history of coronary disease or arrhythmia. Thereafter, a daily oral dose of 1.6 µg/kg body weight should be given once the patient becomes ambulatory.[3,11] In India, the injectable form is not widely available and typically levothyroxine tablets are given via NG tube, although absorption might be unpredictable due to ileus and poor gastric motility. Some authors suggest supplementing T3 (Cytomel) as well in young patients with low cardiovascular risk in the dose of 10 µg IV every 8 hours.

Diabetic Ketoacidosis

Globally, diabetic ketoacidosis or DKA is one of the dreaded complications of diabetes mellitus. The hallmark triad is hyperglycemia, anion gap metabolic acidosis, and ketosis. Although it is not common in pregnancy, usually reported in the second and third trimesters, it carries a significant risk of poor outcomes with a risk of fetal loss in 10–25% of cases.

A detailed explanation of the pathogenesis of DKA is outside the scope of this article. It is the result of an exaggerated counterregulatory response to a paucity of glucose at the cellular level in terms of ketogenesis, facilitated by glucagon, catecholamines, and cortisol. These hormones further promote lipolysis and hepatic free fatty acid release leading to resultant acidosis.

Pregnancy is a state of heightened insulin resistance, and by 36 weeks of gestation, insulin sensitivity is estimated to be 50% of that of a nonpregnant female.[12] Several hormones unique to pregnancy either increase insulin resistance or promote hyperglycemia (Table 3).

Diabetic, pregnant females are more sensitive to starvation due to poor nutritional intake and vomiting. Infections, poor compliance with treatment, use of beta-sympathomimetic agents for tocolysis, and use of steroids for fetal lung maturation can induce hyperglycemia and lipolysis, resulting in maternal and fetal acidosis. Thus, these patients warrant close monitoring.

Acute renal failure, myocardial infarction, acute lung injury, cerebral edema, preterm delivery, fetal hypoxia, adverse neurobehavioral outcomes of the newborn, and maternal demise have all been attributed to DKA.

The signs and symptoms of DKA are similar to nonpregnant patients (Table 4). A high level of suspicion is warranted as an episode of DKA may be the first presentation of diabetes, especially in our country with poor healthcare penetration.

The principles of management remain the same although the patient should be treated in an intensive care setting as close maternal and fetal observation is warranted. Maternal left lateral decubitus positioning and oxygen supplementation are essential to optimize uteroplacental perfusion. Aggressive fluid resuscitation, intravenous insulin therapy, metabolic and electrolyte correction, and correction of the underlying pathology are the basis of treatment. Typically, the fluid deficit is 100 mL/kg body weight, which amounts to 6–10 L of deficit varying with maternal body weight. The aim is to replace approximately 75% of this deficit within the first 24 hours and the remaining in the next 24 hours. Initially, 1–2 L of 0.9% normal saline is administered over the first 2 hours following which infusion rate is slowed down to 250–500 mL/hour and continued till glucose levels are below 250 mg/dL. Once this level is reached, 5% dextrose infusion is started. At this moment, the choice of IV fluid depends on hemodynamic stability and electrolyte status and it should be continued till the deficit is taken care of. For example, in the presence of hyponatremia, 0.45% saline should be used to replace the free water loss. Insulin should be administered intravenously, 0.1 U/kg boluses followed by 0.1 U/kg/hour infusion until the serum bicarbonate and acidosis normalize and serum ketones become absent.

Serum potassium should be kept between 4 and 5 mEq/L. This is achieved by intravenous administration of potassium chloride. Table 5 shows the suggested protocol for potassium replacement.[13]

DKA alone is not an indication for delivery as it may increase maternal morbidity. Also, delivery of a premature, acidotic, and hypoxic neonate may not be the best choice. The decision to terminate the pregnancy must be individualized based on maternal response to treatment and fetal monitoring.

Table 3: Triggering factors for diabetic ketoacidosis in pregnancy[12]

- Human chorionic gonadotropin–induced nausea and vomiting may precipitate starvation ketosis.
- Progesterone affects GI motility and increases carbohydrate absorption.
- Human placental lactogen and prolactin decrease insulin sensitivity.
- Increased maternal metabolic rate.
- Pregnancy is a state of respiratory alkalosis due to increased minute ventilation. Often it is compensated with elevated renal bicarbonate excretion.

Table 4: Clinical features of diabetic ketoacidosis in pregnancy[13]

Signs and symptoms	*Laboratory findings*
• Tachypnea	• Plasma glucose >250 mg/dL
• Sinus tachycardia	• Arterial pH <7.30
• Altered sensorium	• Anion gap >12 mEq/L
• Kussmaul respirations	• ↑Base deficit
• Nausea or vomiting	• Serum bicarbonate ≤15 mEq/L
• Blurred vision	• ↑Serum blood urea nitrogen and creatinine
• Muscle weakness	• Positive serum/urine ketones, especially 3b-hydroxybutyrate
• Hypotension or dehydration	• Falsely normal potassium level might be present.
• Nonreassuring fetal tracing	
• Abdominal pain or contractions	

Table 5: Potassium replacement in diabetic ketoacidosis during pregnancy[13]

Potassium level	*Intervention*
>5 mEq/L	No treatment
4–5 mEq/L	20 mEq/L replacement
3–4 mEq/L	30–40 mEq/L replacement
3 mEq/L or less	40–60 mEq/L replacement

Pituitary Emergencies in Pregnancy

Prolactinomas

Prolactinomas are the most common pituitary tumors seen in women of childbearing age.[14] In nonpregnant patients, prolactinomas are usually managed with a dopamine agonist (bromocriptine or cabergoline), which shrinks the tumor and normalizes the serum prolactin levels. But, the management is more complicated in pregnancy.

An increase in tumor size is often manifested as headaches, changes in the visual field, or more ominously as loss of vision in the case of apoplexy. If there is documented increase in the size of a prolactinoma, treatment with a dopamine agonist is initiated. In the absence of response to treatment, transsphenoidal debulking of the adenoma can be considered keeping in mind the fact that surgery in the second and third trimesters can increase the risk of fetal loss.

Pituitary size is known to increase with advancing pregnancy due to the increasing lactotrophs, in response to the elevated estrogen levels. Interruption of blood supply (due to hypotension secondary to peri- or postpartum hemorrhage) to the anterior pituitary can lead to infarction giving rise to Sheehan's syndrome.

Sheehan's Syndrome

Sheehan's syndrome is a rare condition characterized by postpartum hypopituitarism due to ischemic pituitary necrosis. The most common presentation is the failure of lactation and the absence of resumption of menses in the postpartum period. Diabetes insipidus may also be seen due to failure of the posterior pituitary. Subclinical vasopressin deficiency is common. Infections and surgery might precipitate an acute adrenal crisis. Rarely, the first presentation might be an acute adrenal crisis many years after

delivery. The acute management is fluid resuscitation and timely hormonal replacement. Appropriate fluid resuscitation aids in limiting pituitary necrosis by improving perfusion. Long-term hormone replacement is often necessary.

Sudden bleeding into the pituitary, is known as pituitary apoplexy, is an endocrine emergency with a potential for maternal and fetal morbidity. It is associated with radiation therapy, hypertension, diabetes mellitus, anticoagulant therapy, the use of bromocriptine, and disseminated intravascular coagulopathy. Acute clinical findings like sudden onset of headache, nausea and vomiting, diplopia, loss of vision, and panhypopituitarism are observed. Surgical decompression can reverse visual disturbances and, in some cases, hypopituitarism as well. Hormone replacement is imperative, is the mainstay of treatment.

Adrenal Insufficiency

Adrenal insufficiency in pregnancy is usually caused by primary adrenal failure, which is mostly autoimmune in origin. In the case of known AI, close monitoring needs to be maintained to optimize steroid substitution as levels of cortisol-binding globulin (CBG) and cortisol keep rising throughout pregnancy. Abrupt withdrawal of exogenous glucocorticoids, infection, trauma, surgery, pain, and labor can trigger an acute adrenal crisis, which can manifest as volume depletion and hypotension. The biochemical hallmarks are hyperkalemia and hyponatremia. Fluid resuscitation should be initiated as soon as possible in an intensive care setting to optimize maternal and fetal outcomes without any delay for diagnostic confirmation. Empiric glucocorticoid replacement with hydrocortisone is advisable[15,16] (Table 6).

Pheochromocytoma

Catecholamine-secreting tumors that arise from chromaffin cells of the adrenal medulla and sympathetic ganglia are collectively referred to as pheochromocytomas. The classic triad of symptoms involves episodic headaches, sweating, and tachycardia. Around 50% of patients have hypertension at presentation. Thus, during pregnancy, pheochromocytoma may masquerade as preeclampsia and may be treated as such. A classic description of a heavily pregnant patient with pheochromocytoma is a rise in blood pressure instead of the expected fall while lying supine (paradoxical supine hypertension). This is because of the gravid uterus pressing down on the adrenal tumor and causing a catecholamine release.

Maternal and fetal mortalities are high in cases that are not diagnosed till delivery.[17] Thus, timely diagnosis is essential. Diagnosis is typically made by the measurement of 24-hour urinary and plasma metanephrines and catecholamines. Imaging modalities like magnetic resonance imaging (MRI) and computed tomography (CT) aid in establishing the diagnosis. Fludeoxyglucose–positron emission tomography (FDG-PET) is more sensitive than CT or MRI although this too has been superseded by gallium-68 (Ga-68) DOTA-0-Phe1-Tyr-3 octreotate (Ga-68 DOTATATE)–positron emission tomography (Ga-68 DOTATATE PET), which is more sensitive than all three. In pregnant patients, MRI without Gadolinium contrast is the imaging modality of choice as the other techniques mentioned previously are not considered safe in pregnancy.

Typically, one might encounter a pheochromocytoma patient in the ICU after a spell has been the trigger. In such a scenario, immediate control of the blood pressure is the primary concern as severe systolic hypertension is an important factor in the incidence of adverse cerebral events.[18] Common triggers for a pheochromocytoma crisis are labor, drugs such as tricyclic antidepressants, anesthesia induction, surgery, opiates, and contrast media. There is consensus that women with severe hypertension (defined as systolic blood pressure ≥160 mm Hg and/or diastolic blood pressure ≥110 mm Hg) persisting for ≥15 minutes should be treated to reduce the risk of maternal stroke and heart failure and other serious maternal complications (Table 7).

For acute control of blood pressure in pheochromocytoma, treatment options are similar to those for nonpregnant patients. Spontaneous labor and delivery should be avoided, and a caesarean section is the preferred mode of delivery.

Table 7: Antihypertensive agents in pregnancy[18]

Safe	*Unknown*	*Avoid*
Beta-blockers Calcium channel blockers Methyldopa Nitroglycerine Hydralazine Clonidine	Thiazide diuretics Amiloride Eplerenone	Angiotensin converting enzyme (ACE) inhibitors—fetal renal abnormalities Angiotensin receptor blockers—fetal renal abnormalities Spironolactone—antiandrogenic activity Nitroprusside—risk of fetal and maternal cyanide poisoning

Table 6: Management of adrenal insufficiency in pregnancy[16]

Emergency measures	*Subacute measures after stabilization of the patient*
• Establishment of large bore intravenous access	• Continuation of intravenous isotonic saline at a slower rate for the next 24–48 hours.
• Emergency evaluation of electrolytes and glucose and routine measurement of plasma cortisol and adrenocorticotropic hormone (ACTH). Do not wait for laboratory results.	• Search for the precipitating causes
• Infusion of 2–3 L of isotonic saline or 5% dextrose in isotonic saline as quickly as possible.	• A short ACTH stimulation test to confirm the diagnosis of adrenal insufficiency
• Hydrocortisone (100 mg intravenous bolus), followed by 50 mg intravenously every 6 hours (or 200 mg/24 hours as a continuous intravenous infusion for the first 24 hours). Other alternatives are prednisolone, prednisone, and dexamethasone.	• Tapering of parenteral glucocorticoid over 1–3 days to oral glucocorticoid maintenance dose.
• Use supportive measures	• For patients with primary adrenal insufficiency, the mineralocorticoid replacement should be started with oral fludrocortisone, 0.1 mg.

Conclusion

Finally, the complex interplay of maternal and fetoplacental physiology and the proverbial Damocles sword of "two lives in one body" make the decision-making complicated, thus, shared decision-making and close coordination between intensivists, anesthesiologists, obstetricians, and endocrinologists are required for assuring good maternal and fetal outcomes. This review hopes to give a brief overview of the nuances of management of such a patient. We hope that the readers find it useful.

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