

# Altered Mental Status and Endocrine Diseases

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## KEYWORDS

- Altered mental status • Diabetic ketoacidosis • Thyroid storm • Myxedema coma
- Pheochromocytoma • Addison disease

## KEY POINTS

- Although altered mental status is a common presentation in the emergency department, altered mental status caused by endocrine emergencies is rare.
- The differential diagnosis for altered mental status is always varied and it is sometimes difficult to uncover the ultimate cause in the emergency department.
- When considering the differential diagnosis of an altered patient, clinicians must consider the age and sex of the patient, prior medical history, medications, and risk factors for developing endocrinopathies.

## INTRODUCTION

The chief complaint of altered mental status represents up to 10% of all emergency department (ED) visits, and 5% of these are ultimately diagnosed with endocrine causes.<sup>1</sup> Being altered is a term that includes a spectrum of presentations including being comatose, combative, confused, having personality changes, or being difficult to arouse. It could pose a challenge to diagnose a patient with altered mental status secondary to an endocrine disorder, especially if a prior history of an endocrine disease is unknown. The diagnosis of these diseases requires a high clinical suspicion, and information gleaned from the history, physical examination findings, and laboratory studies. When considering the differential diagnosis of an altered patient, clinicians must consider the age and sex of the patient, prior medical history, medications, and risk factors for developing endocrinopathies. The cause of the endocrine disease and the precipitating factor for the diseases commonly have similar presentations, which causes increasing complexity in the diagnosis of these diseases.

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The authors have nothing to disclose.

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## EVALUATION AND TREATMENT OF THE PATIENT

### *History*

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All patients who present to the ED should be approached with a stepwise algorithm. As with any patient who presents to the ED, a thorough assessment of the ABCs (airway, breathing, and circulation) is required. After the patient's airway and circulatory status are assessed and stabilized, the cause of the presentation can be sought. The act of taking the history may prove difficult depending on the patient's mental status. The emergency physician may develop a plausible differential diagnosis primarily from speaking with the patient, or this may necessitate involvement of family members or caretakers. Because of the wide variety of presentations, from the subtle alterations of sensorium to a floridly psychotic state, eliciting a history that suggests an endocrinopathy as the cause of the mental status change can be difficult. Any information could be vital to diagnosing and treating the patient appropriately.

The history should begin with an adequate understanding of baseline mental status, and any reports of bizarre behavior need to be supported with specific examples. The history should focus on the onset of the symptoms and variability of the symptoms through time. Emergency physicians should not hesitate to call those who can provide the most accurate information, such as the nursing home staff, family and friends, or other health care providers who may have information about the patient. To the extent that it is possible, the history should also include a thorough medical history, and this may require a review of the patient's medical records, if available. The pertinent aspects of the medical history include any new or changed medications, substance abuse, and any antecedent illnesses.

Part of the difficulty in diagnosing endocrinopathies is that they can be exacerbated, or masked, by other processes. A thorough review of the history is necessary to evaluate the possibility of other processes, which include infection, polysubstance abuse, cerebrovascular accidents, psychiatric illness, dementia, or head trauma.

### *Physical Examination*

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The physical examination should always begin with an overview of the vital signs, which may provide an initial clue to the underlying cause. Examples of vital sign abnormalities associated with endocrinopathies are listed in [Table 1](#).

After an assessment of the vital signs, a thorough head-to-toe physical examination should be performed. The examination should start with a general overview of the patient's appearance because this may give clues to the cause of their presentation. As with any patient who presents with a change in mental status, a complete neurologic examination is warranted. The neurologic examination should begin with an assessment of mental status; a commonly used tool is the mini-mental screening examination (MMSE). A quick MMSE allows the practitioner to easily and reliably determine the patient's cognitive ability. Next, the assessment should include a neurologic examination with focus on the cranial nerves to evaluate for a possible cerebrovascular accident. Other crucial components of the neurologic examination include examination of muscle tone, strength, and reflexes. Although an extensive examination of the integumentary system is rarely performed, this can be beneficial in the obtunded patient. For example, abnormalities of the skin and hair can give clues to previous hypothyroidism. [Table 2](#) shows other endocrinopathies and examples of pertinent physical examination findings that can be associated with them.

**Table 1**  
**Abnormal vital signs indicating possible endocrine emergencies**

Hyperthermia	Thyroid storm
Hypothermia	Myxedema coma
Hypertension	Thyroid storm, pheochromocytoma
Hypotension	Adrenal crisis, DKA/HHNK (if dehydrated)
Tachycardia	Thyroid storm, pheochromocytoma, adrenal crisis (if patient is dehydrated), DKA/HHNK
Bradycardia	Myxedema coma, adrenal crisis
Tachypnea	DKA
Bradypnea	Myxedema coma

*Abbreviations:* DKA, diabetic ketoacidosis; HHNK, hyperglycemic hyperosmolar nonketotic.

## GLUCOSE-RELATED CAUSES OF ALTERED MENTAL STATUS

### *Hypoglycemia*

Because the endocrine system is a highly regulated series of interconnected glands that control most aspects of metabolism and homeostasis, disorders can negatively affect glucose levels. Because glucose is the primary and preferred energy source for the central nervous system, this causes alterations in mental status. The most obvious organ that may contribute to this is the insulin-producing pancreas, but almost every endocrine gland and its associated maladies can have an effect on glucose metabolism. The hypothalamic-pituitary-adrenal axis is responsible for the control of cortisol and epinephrine, among other hormones, which are crucial in the production and use of glucose by the body. Hypoglycemia is most likely seen in diabetics, but can also be seen in thyrotoxicosis, myxedema coma, pheochromocytoma, and adrenal crisis.

The main clinical differentiation that needs to be made is to determine whether the hypoglycemia is causing the patients' clinical presentation or whether is a result of another process that is causing the hypoglycemia. Simple hypoglycemia as a cause of altered mental status should show resolution of symptoms once normoglycemia has been achieved.<sup>2</sup> However, if the neurologic impairment remains, other causes should be considered. Just as a patient with severe sepsis can have concomitant

**Table 2**  
**Physical examination findings suggesting endocrine emergencies**

Disorder	Physical Examination Findings
DKA	Kussmaul respirations, signs of dehydration, decline in mental status, fruity odor of ketones
Myxedema coma	Patient has severe decline in mental status, hypothermia, delayed deep tendon reflexes, dry skin and hair, pretibial myxedema, macroglossia, signs of heart failure
Thyroid storm	Proptosis, goiter, diaphoresis, tremors, thin hair, hyperreflexia, fever, wide pulse pressures, signs of CHF, atrial fibrillation
Adrenal crisis	Orthostatic hypotension, nausea, vomiting, darkened skin
Pheochromocytoma	Vacillating blood pressure/heart rate/temperature, diaphoresis, tremor, pallor secondary to vasoconstriction

*Abbreviation:* CHF, congestive heart failure.

hypoglycemia from several causes, so can the patient with an endocrinopathy. For example, the patient in myxedema coma may have hypoglycemia, and this could be caused by the infection that exacerbated the endocrine disorder, the dysfunction of the thyroid gland causing abnormal metabolism, or a combination of both. In addition to a dysfunctional endocrine gland causing problems, the use of medications can also cause hypoglycemia. Insulin is the most frequent cause in hypoglycemia, but oral antidiabetic medications, especially sulfonylureas, can also cause prolonged and severe hypoglycemia.<sup>3</sup> **Box 1** lists possible causes, including medication, for hypoglycemia.

**Box 1****Causes of hypoglycemia**

## Diabetic medications

- Insulin
- Sulfonylureas
- Meglitinides

## Antidiabetic medications

- $\beta$ -Blockers
- Angiotensin-converting enzyme inhibitors
- Pentamidine
- Fluoroquinolones

## Organ dysfunction

- Liver failure (decreased gluconeogenesis and glycogenolysis)
- Kidney failure (decreased insulin clearance)

## Stress states

- Infection and sepsis
- Burns

## Malnutrition

- Alcohol abuse
- Eating disorder
- Malabsorption states

## Endocrinopathies

- Adrenal insufficiency
- Hypothyroidism

## Neoplastic disease (rare)

- Insulinoma
- Liver metastases

## Psychiatric

- Surreptitious insulin use
- Overdose

*Modified from* Alsahli M, Gerich JE. Acute and chronic complications of diabetes hypoglycemia. *Endocrinol Metabol Clin* 2013;42(4):657–76.

Common presenting symptoms for hypoglycemia include diaphoresis, irritability, and tremors. As the serum glucose gets lower, neurologic and cognitive signs begin to show, such as cognitive impairment, paralysis, and eventually seizures and coma.<sup>2</sup> As with any patient presenting with an alteration in mental status, whether obvious or subtle, the first test that should be obtained is a bedside glucose. An aspect of hypoglycemia that is clinically relevant is that the level of hypoglycemia may not relate to the clinical presentation. For example, a diabetic patient who regularly has difficulty controlling blood glucose may be tolerant to the effects of hypoglycemia. In addition, hypoglycemia can unmask previous clinical manifestations of cerebrovascular accident that had been clinically silent. In short, hypoglycemia can be caused by, or associated with, many endocrine disorders, and a quick bedside determination of glucose status is crucial to the approach to a patient with altered mental status.

Hypoglycemia in patients with altered mental status should be diagnosed and treated promptly. Prolonged severe hypoglycemia has been shown to be associated with brain injury and can lead to permanent neurocognitive impairment.<sup>4</sup> Initial stabilization is often done in the prehospital setting. Alert hypoglycemic patients should be given oral glucose, which can be in the form of glucose tablets or a high-carbohydrate juice such as orange juice. In a hypoglycemic patient who is obtunded or comatose, parenteral agents should be used. Intravenous (IV) 50% dextrose solutions should be first-line treatment. If IV access is not established, intramuscular glucagon can be administered, but this has a slower onset of action. Octreotide is another medication that has been used for refractory hypoglycemia caused by sulfonylurea overdose with good results.<sup>5</sup>

Frequent monitoring of glucose levels is necessary to avoid recurrent hypoglycemia. Patients may require subsequent dextrose boluses, and some patients may even require a constant infusion of dextrose to maintain normoglycemia. Once a patient is able to safely tolerate food, the patient should eat a meal that includes both easily digested carbohydrates and protein, for example a turkey sandwich on white bread. After the blood sugar is stabilized, the provider should then explore what precipitated the hypoglycemic episode.

### ***Diabetic Ketoacidosis/Hyperglycemic Hyperosmolar Syndrome***

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Although thyroid and adrenal emergencies are rarer phenomena encountered in the ED, diabetes-related complications are commonplace in comparison. In hyperglycemic patients, diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS) should be considered as possible causes of a patient having altered mental status. Even with minor derangements in laboratory tests, patients can be considered in DKA if their blood glucose is more than 250 mg/dL, they have an anion gap greater than 10, and pH less than 7.3. The severity of the altered sensorium correlates more with the serum osmolality (>320 mOsm/L) than other laboratory findings such as the degree of hyperglycemia or the acidosis.<sup>6</sup> The presence of acidosis is the main differentiation between DKA and HHS. However, the altered sensorium associated with their clinical presentations can be similar, mainly because of the hyperosmolality.<sup>7</sup>

Patients may have classic complaints of hyperglycemia, such as excessive thirst and frequent urination, or they may complain of nausea and vomiting. Findings on physical examination for a patient in DKA include signs of dehydration such as dry mucous membranes, tachycardia, hypotension, Kussmaul respirations, and abdominal tenderness. Mental status changes do not have to occur in every patient in DKA, but, if they do, patients can have a varying presentation from lethargy to coma.

Patients with HHS have some element of neurologic dysfunction ranging from hemiparesis and partial seizures to stupor and frank coma.<sup>7,8</sup>

One of the most serious complications of DKA, especially for pediatric patients, is cerebral edema. Cerebral edema occurs in about 1% of pediatric patients in DKA and should be a consideration when the patient undergoing treatment of DKA experiences a sudden change in mental status or shows signs of increased ICP.<sup>9,10</sup> In general, this complication is only seen in pediatric patients and although the pathophysiology is not clear, the cause centers on the inadequate increase in serum sodium levels in conjunction with the high osmolarity of the serum, high blood urea nitrogen levels, and hypocapnia.<sup>9,10</sup> The initial glucose or sodium levels on presentation do not affect the patient's chances of developing cerebral edema.<sup>9</sup> **Box 2** lists clinical as well as diagnostic results that are risk factors for patients to develop cerebral edema.

## THYROID-RELATED CAUSES OF ALTERED MENTAL STATUS

### *Myxedema Coma*

Although the prevalence of hypothyroidism is not rare, the severe form of the disease, myxedema coma, is infrequently seen in the ED. The usual description of a patient's sensorium includes a precipitous decline from the patient's baseline and presentation may range from lethargy to coma. The patient's baseline mental status is likely to encompass some findings of hypothyroidism such as amnesia, depression, or slow cognitive processing. The textbook clinical scenario of a patient in myxedema coma is an elderly woman with a history of hypothyroidism who presents in stupor after exposure to cold weather.<sup>11,12</sup> The severity of the hypothermia correlates with the prognosis, with the more severe the hypothermia, the worse the prognosis for the patient.<sup>11</sup> The overall mortality is approximately 25% for patients in myxedema coma despite appropriate medical intervention.<sup>11-14</sup>

If available, a history of hypothyroidism may be obtained from the family, previous medical records, or medication bottles brought in by emergency medical service providers. A thyroidectomy scar may be present on the neck. Other possible pertinent physical examination findings include hypothermia; depressed deep tendon

#### **Box 2**

#### **Risk factors for pediatric DKA-induced cerebral edema**

New-onset diabetes

Younger age

Longer duration of DKA symptoms

Degree of acidosis at presentation (pH<7.1)

Greater hypocapnia (Pco<sub>2</sub><20 mm Hg)

Greater initial rate of fluid resuscitation for treatment of severe DKA (>50 mL/kg in the first 4 hours)

Administration of insulin during the first hour of fluid resuscitation

Slower increase in measured serum sodium concentration during treatment

Bicarbonate treatment

*From* Olivieri L, Chasm R. Diabetic ketoacidosis in the pediatric emergency department. *Emerg Med Clin North Am* 2013;31(3):755-73.

reflexes; dry, doughlike skin and hair; pretibial myxedema; macroglossia; and signs of heart failure. As with any disease, the physical examination findings are on a spectrum, and the more severe the myxedema the higher the severity of the presenting signs and symptoms. Hypoglycemia is frequently present in these patients, because patients in myxedema coma have impaired glucose metabolism and retain free water. The decline in mental status can occur abruptly and usually has a trigger that causes a metabolic stress and thus the rapid decline.<sup>15</sup> Most commonly the onset of an infection, trauma, or a myocardial infarction starts the deterioration into myxedema coma.<sup>14</sup> Exogenous or iatrogenic factors, such as cold exposure, and medications that decrease central processes, such as antiepileptics,  $\beta$ -blockers, or sedatives, can also exacerbate the symptoms.<sup>14</sup> In the process of working up a patient in myxedema coma, the practitioner should entertain a broad differential diagnosis including cardiac and infectious causes while simultaneously investigating medication changes or other recent physiologic stresses. Patients with myxedema coma may present with seizures, further complicating the clinical presentation. One-quarter of the patients with myxedema coma develop seizures secondary to hyponatremia, hypoglycemia, or decreased oxygenation of the cerebral vasculature.<sup>11</sup> In such cases, it is important to first stabilize the patient with first-line seizure therapy and then, once the patient is stabilized, the cause can be investigated.

There should be a low threshold to intubate the patient with myxedema coma because mental status can continue to decline in the department while work-up and treatment are ongoing. It is prudent to prepare for a difficult airway in these patients secondary to an edematous pharynx and possibly even angioedema.<sup>16</sup>

If myxedema coma is suspected based on the initial inspection of the patient, a full thyroid panel and cortisol levels should be sent. Treatment should not wait until the laboratory values return.<sup>12,14</sup> Few clinicians find fault in studies associated with a general altered mental status work-up such as computed tomography head, lumbar puncture, urine drug screen, levels of medications that the patient was taking, alcohol level, ammonia level, and possibly an arterial blood gas. In small number of studies, most patients in myxedema had increased cerebrospinal fluid pressure and total protein content, likely secondary to the increased vascular permeability of the meninges.<sup>15</sup> A bedside echocardiogram might reveal a pericardial effusion as well. Treatment consists of airway management, thyroid repletion, and stress dose steroids. Patients in myxedema coma often have concomitant adrenal insufficiency. Once thyroid hormone is administered, there is a subsequent increase in metabolism, which requires more cortisol.<sup>14</sup>

### ***Thyroid Storm***

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Although patients with hyperthyroidism may present with mild agitation, patients with thyroid storm can have varied presentations. Early in the progression of the disease the patient usually presents with signs and symptoms of a hypermetabolic state.<sup>17</sup> The clues to diagnosis can be found in the physical examination findings such as fever, tachycardia, hypertension, tremors, and agitation. Some other neurologic findings include hyperreflexia, muscle weakness, ataxia, and even myopathy.<sup>12,18,19</sup> The patient may also have gastrointestinal (GI) symptoms including vomiting and diarrhea. As the disease progresses the patient can present with psychosis and then enter a comatose state as the disease becomes fulminant. The overall prevalence of thyroid storm is 1% to 2% of those diagnosed with hyperthyroidism, with mortality between 20% and 50%.<sup>18,20</sup>

As with any endocrine disease that causes altered mental status, clinicians must first consider the diagnosis before it can be made. It is fortunate if a history of

hyperthyroidism is uncovered on a review of the patient's past medical history. The diagnosis of thyroid storm is usually made on clinical grounds alone because laboratory studies can have prolonged turnaround times causing delays in management. Left untreated, the patient rapidly decompensates, shows signs of heart failure and hypotension, and succumbs to respiratory failure. With such a wide array of presentations, clinicians may decide to rule out other conditions such as meningitis, cerebrovascular accident, heat stroke, alcohol withdrawal, drug overdose, neuroleptic malignant syndrome, and diabetic ketoacidosis. Laboratory studies can be markedly abnormal but do not help distinguish between many diseases. The patient often has a concomitant process that has precipitated the thyroid storm. Treatment involves blocking thyroid hormone production with antithyroid medication and then iodine, blocking systemic effects with beta-blockade and corticosteroids, and treating the precipitating event.<sup>12,19</sup>

## ADRENAL-RELATED CAUSES OF ALTERED MENTAL STATUS

### *Adrenal Insufficiency*

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Adrenal emergencies, similar to thyroid emergencies, can manifest either as minor mental status changes like depression or severe diseases like frank psychosis or coma. Adrenal insufficiency may have many underlying causes, including autoimmune, iatrogenic, and infectious processes. Like most endocrine diseases, adrenal insufficiency can have primary or secondary causes. Primary adrenal insufficiency is most commonly caused by autoimmune adrenalitis in Western countries such as the United States.<sup>21</sup> Tuberculosis should be considered in areas that are endemic or in patients who have traveled to or lived in those endemic areas because tuberculosis remains the most common infectious cause.<sup>21,22</sup> Human Immunodeficiency virus (HIV) is also a common infectious cause of adrenal destruction, and the clinician must be cognizant with patients known to be HIV seropositive.<sup>23</sup> Secondary adrenal insufficiency is caused by disorders of the hypothalamic pituitary axis, like pituitary apoplexy or tumors. Whether the patient has primary or secondary adrenal insufficiency, the patient presents similarly when in adrenal crisis.

There is a history of chronic adrenal insufficiency, especially in primary adrenal insufficiency, which can include symptoms of salt craving; hyperpigmentation of skin; GI distress such as nausea, vomiting, and diarrhea; and generalized muscle wasting and fatigue. In the acute setting, it manifests as a sudden worsening of these symptoms along with hemodynamic instability as shown by the body's inability to support the additional stress of a medical crisis such as an infection, trauma, or surgical procedure. When a patient requires a sudden increase of stress hormones or exogenous hormones are removed, the patient manifests a profound inability to maintain adequate amounts of glucocorticoids and mineralocorticoids, resulting in mental status changes, tenuous vital signs, and multiple laboratory derangements. With circulatory collapse, the patient can present as being less responsive, delirious, or in coma as the brain is hypoperfused.<sup>22</sup>

Primary adrenal insufficiency can produce marked hyponatremia, hypoglycemia, hypercalcemia, hyperkalemia, and possibly metabolic acidosis from inadequate organ perfusion manifested by an increased lactic acid level. Beginning the inpatient work-up by sending random cortisol and adrenocorticotropic hormone levels is warranted if there are standard findings of hypovolemia or hemodynamic instability that cannot be explained otherwise, especially if the hemodynamic instability is refractory to standard therapies.<sup>21,24</sup> A cosyntropin stimulation test can be performed for confirmation of the diagnosis of adrenal insufficiency, but should not delay treatment.

**Box 3****Causes of adrenal insufficiency**

## Primary

## Vascular

Adrenal infarction, hemorrhage (lupus anticoagulant or HIT)

## Infiltrative

Lymphoproliferative disorder, metastatic disease, granulomatous disease (eg, sarcoidosis)

## Autoimmune

Autoimmune adrenalitis, polyglandular autoimmune syndrome I and II

## Traumatic

Abdominal blunt trauma or back trauma

## Drugs

Ketoconazole (especially in patients with acquired immunodeficiency syndrome),  
etomidate

## Congenital

Adrenoleukodystrophy, adrenal enzyme deficiencies

## Secondary

## Vascular

Pituitary infarction (Sheehan syndrome)

## Neoplasm

Pituitary adenoma, metastasis

## Infective

Tuberculosis, HIV, and fungal infection

## Infiltrative

Granulomatous disease (sarcoidosis, histiocytosis), hemochromatosis

## Inflammatory

Lymphocytic hypophysitis

## Traumatic

Brain injury

## Radiation

After pituitary radiation

## Drugs

Patient cessation of prolonged glucocorticoid therapy (including inhaled glucocorticoid)

## Critical illness

Relative glucocorticoid deficiency

## Others

ACTH deficiency

*Data from Adler S, Wartofsky L. Myxedema coma. In: Berghe GV, editor. Acute endocrinology: from case to consequence. New York: Humana Press; 2008. p. 29–44.*

Treatment involves fluid resuscitation, repletion of electrolyte deficiencies (especially glucose), and steroid replacement therapy. Stress dose steroids given as hydrocortisone 100 mg IV as a bolus every 8 hours is sufficient for patients with adrenal insufficiency.<sup>21</sup> Hydrocortisone contains glucocorticoid and mineralocorticoid and sufficiently replaces both. As an alternative, dexamethasone 4 mg is also an acceptable treatment. Dexamethasone treatment has the advantage of not affecting the cosyntropin stimulation test should the provider desire to perform it, whereas hydrocortisone does affect the result. The disadvantage of dexamethasone is that it does not contain any mineralocorticoid and may need to be given in combination with fludrocortisone, a mineralocorticoid.<sup>21,25</sup> Although uncovering the cause of adrenal insufficiency may become a secondary consideration for the emergency physician when the patient is in an acutely decompensated state, **Box 3** lists the common causes to consider in the work-up and treatment when a patient is found to have adrenal insufficiency.

### ***Pheochromocytoma***

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Although rare, and even more rarely diagnosed in the ED, pheochromocytomas need to remain on the differential diagnosis of any altered patient. The general findings may be similar to a patient having a panic attack, which can dangerously and easily be dismissed if not considering a broader differential. The 5 Ps represent the symptoms associated with pheochromocytomas: pressure (hypertension), pain (headache and chest pain), palpitations, perspiration, and pallor (caused by vasoconstriction).<sup>26</sup> Again, the difficulty in diagnosis lies in recognition because pheochromocytoma can encompass a wide spectrum of presentations.

The underlying mechanism of the symptoms is caused by the sudden release of epinephrine and norepinephrine into the blood stream by the adrenal tumor. The wave of catecholamine excess can thus cause symptoms similar to a fight-or-flight response. Mental status changes can also range from subtle to frank psychosis.<sup>27</sup> One case report details the diagnosis of a pheochromocytoma in a patient who continued to have what was labeled as a relapsing paranoid psychosis.<sup>28</sup> Other findings that suggest a pheochromocytoma include a paroxysmal increase in blood pressure, heart rate, temperature, a sudden onset of headache, dysrhythmias, acute GI distress, and refractory response to antihypertensive medications.<sup>27</sup> These paroxysms can be brought on by physical exertion, bowel movements, trauma, or even certain medications that may stimulate the sympathetic nervous system.<sup>27</sup> The differential diagnosis usually includes drug overdose, hyperthyroidism, and psychiatric disturbances, which may be the initial impression of the evaluating physician. It is important to thoroughly evaluate medical conditions that could need prompt treatment even if they have a pertinent history of drug abuse or psychiatric issues.

If the diagnosis is made and confirmed in the ED, the provider should focus on catecholamine blockade to control symptoms. Alpha-blockade with phentolamine or phenoxybenzamine is the preferred method to control symptoms. The addition of a  $\beta$ -blocker for selective symptoms can also be used. The pharmacologic treatment of a pheochromocytoma is solely a temporizing measure to bridge the patient until surgical removal of the gland.<sup>28</sup>

### **SUMMARY**

The differential diagnosis for altered mental status is always varied and it is sometimes difficult to uncover the ultimate cause in the ED. Part of what distinguishes astute clinicians is considering the atypical diagnoses and pursuing them under reasonable

suspicion. Endocrinopathies, although not common, are still fascinating and their diagnosis is laudable. Sorting through the history, physical examination, laboratory findings, and imaging can become a satisfying endeavor when arriving at an answer that will save the patient and restore their mental status to baseline. The problematic but remarkable aspect of the endocrine system is that it affects many other systems of the human body and the presentation can be perplexing. Knowledge of the endocrine emergencies and how they can present in the ED will inform the differential diagnosis of altered mental status and will possibly be called on at some point in the clinician's career for the treatment of such patients.

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