

Addison Disease: Early Detection and Treatment Principles

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Primary adrenal insufficiency, or Addison disease, has many causes, the most common of which is autoimmune adrenalitis. Autoimmune adrenalitis results from destruction of the adrenal cortex, which leads to deficiencies in glucocorticoids, mineralocorticoids, and adrenal androgens. In the United States and Western Europe, the estimated prevalence of Addison disease is one in 20,000 persons; therefore, a high clinical suspicion is needed to avoid misdiagnosing a life-threatening adrenal crisis (i.e., shock, hypotension, and volume depletion). The clinical manifestations before an adrenal crisis are subtle and can include hyperpigmentation, fatigue, anorexia, orthostasis, nausea, muscle and joint pain, and salt craving. Cortisol levels decrease and adrenocorticotropic hormone levels increase. When clinically suspected, patients should undergo a cosyntropin stimulation test to confirm the diagnosis. Treatment of primary adrenal insufficiency requires replacement of mineralocorticoids and glucocorticoids. During times of stress (e.g., illness, invasive surgical procedures), stress-dose glucocorticoids are required because destruction of the adrenal glands prevents an adequate physiologic response. Management of primary adrenal insufficiency or autoimmune adrenalitis requires vigilance for concomitant autoimmune diseases; up to 50% of patients develop another autoimmune disorder during their lifetime. (*Am Fam Physician*. 2014;89(7):563-568. Copyright © 2014 American Academy of Family Physicians.)

CCME This clinical content conforms to AAFP criteria for continuing medical education (CME). See CME Quiz Questions on page 515.

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► Patient information: A handout on this topic is available at <http://familydoctor.org/familydoctor/en/diseases-conditions/addison-disease.html>.

More than 150 years ago, Thomas Addison described a group of patients with anemia and diseased adrenal glands at autopsy, a condition now known as primary adrenal insufficiency. Autoimmune adrenalitis is the most common cause of primary adrenal insufficiency, or Addison disease, in the United States. Less common causes include infection, hemorrhage, metastatic cancer, medication use, and adrenoleukodystrophy.

Autoimmune adrenalitis is a disorder in which the adrenal cortex is destroyed, resulting in the loss of mineralocorticoid, glucocorticoid, and adrenal androgen hormone production. Addison disease can be part of the autoimmune polyglandular syndromes (type 1 and 2), or it may present as an isolated disorder.¹ This article focuses on the diagnosis and treatment of Addison disease as an isolated disorder, with a focus on the pathophysiology and treatment considerations of autoimmune adrenalitis.

Pathogenesis

Autoimmune adrenalitis can be divided into stages of progression^{2,3} (Table 1). As the

disease develops, individuals lose adrenocortical function over a period of years. In the first three stages, the human leukocyte antigen genes confer genetic risk; an unknown precipitating event initiates antiadrenal autoimmunity; and 21-hydroxylase antibodies are produced, which predict future disease. The production of these antibodies can precede symptom onset by years to decades, and they are present in more than 90% of recent-onset cases.^{2,4-7} In the fourth stage, overt adrenal insufficiency develops. One of the first metabolic abnormalities to occur is an increase in plasma renin level, followed by the sequential development of other abnormalities, including a decreased response to adrenocorticotropic hormone (ACTH) stimulation in the fifth stage. If symptoms of adrenal insufficiency are present but go undiagnosed, an Addisonian crisis can occur.

Clinical Diagnosis

Because the estimated prevalence of Addison disease is one in 20,000 persons in the United States and Western Europe, a high clinical suspicion is needed to avoid misdiagnosing a life-threatening adrenal crisis.⁸ Signs and symptoms can be subtle and nonspecific.

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