



Addison's Disease: An Overview and Nursing Intervention Plans

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Abstract:

Background: Addison's disease, also known as primary adrenal insufficiency, is a rare endocrine disorder characterized by the destruction of the adrenal cortex, leading to a deficiency in glucocorticoids, mineralocorticoids, and androgens. Autoimmune adrenalitis is the predominant cause in developed countries, with non-specific symptoms often delaying diagnosis. Severe cases can progress to adrenal crises, requiring immediate intervention.

Aim: This article explores the etiology, pathophysiology, and clinical management of Addison's disease, with a focus on nursing interventions to improve patient outcomes.

Methods: A literature review was conducted to examine the causes, risk factors, and clinical manifestations of Addison's disease. Emphasis was placed on nursing diagnoses, management strategies, and their integration into multidisciplinary care plans.

Results: The findings highlight the multifaceted nature of Addison's disease, encompassing autoimmune, infectious, genetic, and neoplastic etiologies. Early signs include fatigue, weight loss, and hyperpigmentation, with laboratory markers like hyponatremia and hyperkalemia aiding diagnosis. Effective nursing interventions, such as infection prevention, fluid management, and patient education, are critical to mitigating complications like adrenal crises. Collaborative care strategies enhance long-term outcomes.

Conclusion: Addison's disease requires a comprehensive approach to care due to its complex presentation and potential for life-threatening complications. Nursing interventions play a pivotal role in early detection, ongoing management, and education, ensuring patient safety and improved quality of life. Multidisciplinary collaboration is essential to optimize care strategies.

Keywords: Addison's disease, primary adrenal insufficiency, autoimmune adrenalitis, adrenal crisis, nursing interventions, glucocorticoid deficiency, multidisciplinary care

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Introduction:

Addison's disease is an acquired primary adrenal insufficiency, a rare yet potentially life-threatening endocrine condition resulting from bilateral adrenal cortex destruction. This destruction leads to decreased production of essential adrenocortical hormones, including cortisol, aldosterone, and androgens. The disease often follows an insidious course, beginning with glucocorticoid deficiency and subsequently progressing to mineralocorticoid deficiency. However, it may also present acutely, particularly during periods of intercurrent illness, emphasizing the need for prompt recognition and management. The term "Addison's disease" is specifically employed to describe primary adrenal insufficiency, distinguishing it from secondary and tertiary causes of adrenal dysfunction. Autoimmune adrenalitis is the leading cause of primary adrenal insufficiency, particularly in developed nations, and is associated with elevated levels of 21-hydroxylase antibodies, which serve as a hallmark of the condition [1][2]. Despite its rarity, Addison's disease represents a significant clinical challenge due to its nonspecific presentation and the potential for delayed diagnosis. The pathophysiology of the disorder involves immune-mediated destruction of the adrenal cortex, often linked to broader autoimmune processes. Patients may initially present vague symptoms, including fatigue, weakness, and hyperpigmentation, which may obscure the diagnosis. Severe cases can lead to adrenal crisis, characterized by profound hypotension, shock, and electrolyte imbalances, necessitating immediate medical intervention. Understanding the underlying mechanisms and clinical manifestations of Addison's disease is crucial for early diagnosis and effective management. This requires a multidisciplinary approach to improve patient outcomes and mitigate the risks of potentially fatal complications. Continued research into its etiology and treatment remains vital to advancing care strategies.

Nursing Diagnosis

Nursing care for patients with Addison's disease focuses on addressing critical risks associated with the condition's underlying pathophysiology and complications. One significant concern is the risk of infection, which arises due to immunocompromised states associated with chronic glucocorticoid deficiency. Fever and susceptibility to opportunistic infections often signify this increased risk. Nurses must monitor patients for early signs of infection and implement preventive measures to mitigate these risks. Another prominent nursing diagnosis is the risk of fluid volume depletion. This condition is primarily attributed to salt-wasting mechanisms inherent in mineralocorticoid deficiency, evidenced by hyponatremia, fatigue, and dizziness. Addressing this issue necessitates meticulous monitoring of fluid balance and serum electrolytes, alongside tailored interventions to ensure hydration and electrolyte restoration. Additionally, alteration in perfusion is a critical concern linked to hypotension, which frequently manifests in Addison's disease due to reduced aldosterone production. This is evidenced by low blood pressure and reduced tissue perfusion, potentially leading to life-threatening outcomes if left unaddressed. Effective management involves close hemodynamic monitoring, administration of prescribed corticosteroids, and patient education to prevent complications. Nurses play a pivotal role in patient education, particularly regarding adherence to pharmacological therapies and recognizing early warning signs of adrenal crisis. By adopting a comprehensive approach that integrates prevention, monitoring, and patient engagement, nursing interventions can significantly improve the quality of life for individuals living with Addison's disease. Collaborative efforts between nursing staff and multidisciplinary teams are essential to optimizing patient care and preventing adverse outcomes.

Causes

The etiology of Addison's disease, a primary adrenal insufficiency, encompasses various pathological processes that damage the adrenal cortex. In developed nations, autoimmune adrenalitis represents the predominant cause. This condition, characterized by immune-mediated destruction of the adrenal glands,

may occur in isolation or as part of autoimmune polyglandular endocrinopathies, including types 1 and 2. Patients with autoimmune adrenalitis are frequently predisposed to developing additional autoimmune disorders, further complicating their clinical course [3][4]. Infectious causes, though less prevalent in modern healthcare settings, remain significant contributors, particularly in regions with limited resources. Tuberculosis, HIV, and sepsis are notable infections linked to adrenal insufficiency, often leading to bilateral adrenal damage. Adrenal hemorrhages also account for a subset of cases, frequently associated with coagulopathies, trauma, or severe infections such as meningococemia. Neoplastic processes involving the adrenal glands, including metastases and primary malignancies, can also impair adrenal function. Rarer etiological factors include infiltrative disorders, such as sarcoidosis and amyloidosis, as well as fungal infections. Genetic conditions, such as adrenoleukodystrophy and Wolman disease, are additional causes, often presenting in pediatric populations or familial clusters. Each of these causes contributes uniquely to the disease's progression, requiring precise diagnostic evaluation to tailor appropriate management strategies. Understanding the diverse etiologies of Addison's disease is crucial for its diagnosis, as each underlying cause may necessitate specific interventions. Improved diagnostic tools and awareness of these causes can facilitate early identification and reduce the burden of this life-threatening condition.

Risk Factors

The prevalence of Addison's disease is estimated at 4 to 11 per 100,000 individuals, with an annual incidence of approximately 0.6 per 100,000. Women are more frequently affected than men, and the condition most commonly manifests in individuals aged 30 to 50 years. Autoimmune adrenalitis, the most common cause of Addison's disease in developed countries, is strongly associated with other autoimmune disorders, highlighting the importance of screening for comorbidities in affected patients. Individuals with a history of autoimmune diseases, such as type I diabetes, hypoparathyroidism, or chronic thyroiditis, exhibit a significantly higher risk of developing Addison's disease. Other autoimmune conditions linked to increased susceptibility include Graves' disease, pernicious anemia, hypopituitarism, and vitiligo. Additionally, rare autoimmune dermatoses, such as dermatitis herpetiformis, and neuromuscular conditions, such as myasthenia gravis, may coexist in individuals with Addison's disease. Genetic predisposition is another notable risk factor, as familial clustering of autoimmune disorders often occurs. Environmental triggers, such as infections or stressors, may exacerbate the risk in genetically predisposed individuals. Understanding these risk factors is critical for early identification and prevention of complications. Healthcare providers must remain vigilant in recognizing the clinical and familial contexts that predispose patients to this condition. This proactive approach can facilitate timely diagnosis, appropriate intervention, and improved patient outcomes. Ongoing research into the interplay of genetic, environmental, and immunological factors is essential to refining prevention and management strategies for Addison's disease.

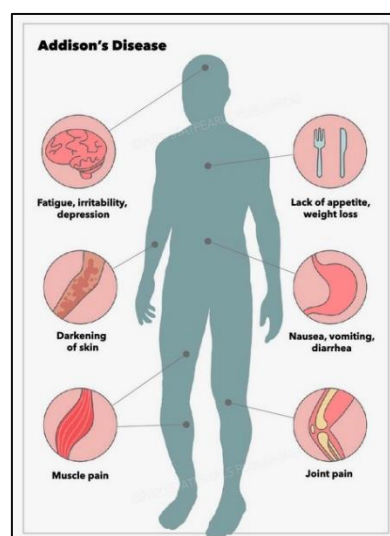


Figure 1: Addison's Disease Symptoms.

Assessment:

Addison's disease typically manifests with an insidious onset of vague and non-specific symptoms, often leading to a delayed diagnosis. Frequently, the condition is recognized only when a patient experiences an acute adrenal crisis, characterized by hypotension, hyponatremia, hyperkalemia, and hypoglycemia, often precipitated by stressors such as infections, trauma, surgery, vomiting, or diarrhea. Although Addison's disease can emerge at any age, it most commonly affects individuals in their second or third decade of life. Symptoms are often generalized and include fatigue, weakness, unintentional weight loss, nausea, vomiting, abdominal discomfort, dizziness, tachycardia, and postural hypotension. Hyperpigmentation of the skin and mucosal surfaces, particularly in sun-exposed regions, emerges later in the disease course, resulting from elevated adrenocorticotrophic hormone (ACTH) levels. This phenomenon occurs because ACTH and melanocyte-stimulating hormone (MSH) share the same precursor, pro-opiomelanocortin (POMC). Given its nonspecific presentation, clinicians should maintain a high index of suspicion for Addison's disease when encountering unexplained symptoms like persistent fatigue, poor appetite, chronic abdominal discomfort, or weight loss. Laboratory findings may include hyponatremia, hyperkalemia, and hypotension. An Addisonian crisis, which may present severe dehydration, refractory hypotension, and shock, necessitates immediate medical attention. This critical condition is particularly suspected in patients undergoing corticosteroid therapy, those who are hemodynamically unstable despite aggressive fluid resuscitation, and individuals experiencing septic shock. Prompt recognition and intervention are essential to prevent fatal outcomes [5].

Evaluation:

Hyponatremia is the most common initial laboratory abnormality in Addison's disease, primarily caused by low cortisol and aldosterone levels. Cortisol deficiency stimulates the excessive release of antidiuretic hormone (ADH) due to hypovolemia and increased hypothalamic secretion of corticotropin-releasing hormone (CRH), thereby compounding ADH hypersecretion. Furthermore, the absence of cortisol removes the negative feedback mechanism on CRH and ADH production. Aldosterone deficiency exacerbates the electrolyte imbalance through natriuresis and potassium retention, potentially resulting in life-threatening hyperkalemia. Hypoglycemia, a frequent finding, is multifactorial, stemming from inadequate glucocorticoid levels required for gluconeogenesis and reduced dietary intake. Diagnostic criteria often reveal low random or stimulated cortisol and aldosterone levels, with cortisol levels below 18–20 micrograms/dL being diagnostic. Elevated ACTH levels confirm primary adrenal insufficiency, while central adrenal insufficiency is indicated by normal or low ACTH levels. Increased plasma renin activity (PRA) is a hallmark of advanced mineralocorticoid deficiency. Autoimmune etiology can be established by detecting anti-adrenal antibodies, particularly 21-hydroxylase antibodies. In cases of suspected adrenal hemorrhage, an abdominal CT scan can provide valuable diagnostic information. Additional evaluations, such as a chest radiograph to identify a small heart or a purified protein derivative (PPD) test for tuberculosis, may be necessary. For patients with familial predispositions or uncertain etiology, a plasma very-long-chain fatty acid profile should be conducted to investigate adrenal leukodystrophy. Comprehensive assessment is pivotal for accurate diagnosis and effective treatment planning [5].

Pathophysiology:

Addison's disease (AD) is primarily caused by autoimmune factors, but its etiology has evolved over time. In the 1930s, tuberculosis (TBC) was the leading cause of AD in Europe, responsible for up to 70% of cases. However, as TBC prevention improved, its contribution to AD declined, with the prevalence of TBC-related AD dropping to just 3% in Italy by recent years. In contrast, autoimmune causes have increasingly dominated, now accounting for 75–96% of AD cases in Europe. As a result, while non-autoimmune causes like TBC remain present, the overall increase in AD cases reflects a rise in autoimmune-related AD. The prevalence of autoimmune AD (AAD) varies based on geographic location and patient demographics. For example, 96% of Norwegian AD cases are autoimmune, while other regions report lower percentages. In adult cases in Italy, 77.7% of AD cases are autoimmune, with 9% related to TBC and smaller percentages linked to genetic or other rare causes. In contrast, most cases of pediatric AD in Canada and Turkey are

attributed to genetic disorders, particularly in those under 15 years of age. Additionally, recent studies suggest a significantly higher prevalence of AD among patients with type 1 diabetes, with a tenfold increase in incidence compared to the general population.

AAD typically occurs in adults between the ages of 30 and 50, and women are more frequently affected than men. Many AAD patients also develop other autoimmune diseases, forming part of autoimmune polyendocrine syndromes (APS). APS-2 is the most common form, characterized by a combination of AAD, autoimmune thyroid disease, and type 1 diabetes. Other associated autoimmune conditions like vitiligo, celiac disease, and premature ovarian failure can also occur. APS-1, a rarer form, involves multiple autoimmune disorders and is caused by mutations in the AIRE gene. This gene's mutations lead to defective immune tolerance, allowing autoreactive T cells to escape deletion, causing autoimmune responses. APS-4, a less defined syndrome, includes AAD with other autoimmune diseases not seen in APS-1 or APS-2. Isolated AAD, without any other associated autoimmune diseases, occurs in a smaller percentage of cases, and it typically presents in adults with a mean onset age of around 28 years. Monitoring for associated autoimmune diseases is essential, and regular screening for thyroid and gastric autoimmunity, type 1 diabetes, and other conditions is recommended for AAD patients.

Histologically, the adrenal glands in AAD patients may be normal or reduced in size, showing lymphocytic infiltration in the adrenal cortex, along with plasma cells, macrophages, and fibrosis. The adrenal medulla is usually unaffected. In terms of family history, AAD can occur in multiple members of the same family, particularly in APS-1, where 25% of patients have a family history of the disease. Autoimmunity in AAD is primarily driven by the presence of adrenal cortex autoantibodies (ACA) and 21-hydroxylase antibodies (21-OHABs). These markers were first identified in the 1960s and remain crucial in diagnosing and monitoring the disease. The identification of 21-OH as the primary autoantigen has been instrumental in developing diagnostic assays, making it easier to detect autoimmune adrenal insufficiency in clinical settings. The presence of 21-OHABs is highly specific to AAD and is commonly used as a biomarker for the disease.

Medical Management

Management of Addisonian crisis, a potentially fatal condition, necessitates immediate intervention upon clinical suspicion. Prior to initiating therapy, blood samples should be obtained for subsequent ACTH and cortisol measurements. A single random cortisol level cannot confirm or exclude Addison's disease unless significantly elevated. Elevated ACTH levels combined with low cortisol levels confirm primary adrenal insufficiency, while an ACTH stimulation test may clarify equivocal cases. Elevated PRA levels, indicative of mineralocorticoid deficiency, often accompany low aldosterone levels. Treatment protocols include fluid resuscitation with intravenous (IV) normal saline to address hypovolemia and dextrose administration for hypoglycemia correction. Hormonal therapy with hydrocortisone is the cornerstone of management. Stress-dose hydrocortisone, with significant mineralocorticoid activity, typically negates the need for fludrocortisone during acute phases. The recommended stress dose of hydrocortisone ranges from 50–100 mg/m², delivered via continuous infusion. Subsequent maintenance therapy involves oral hydrocortisone (10–15 mg/m²/day in divided doses) or alternative glucocorticoids such as prednisone or dexamethasone for patients requiring less frequent dosing. Fludrocortisone supplementation, at doses of 50–200 micrograms/day, is necessary for mineralocorticoid replacement. Hydrocortisone dosage must be adjusted during febrile illnesses or infections to mimic physiological stress responses. Close monitoring of electrolytes, cortisol, ACTH, and plasma renin activity is essential to ensure adequate replacement therapy. Identification and treatment of precipitating factors, such as sepsis, are critical for successful outcomes [6–8].

Nursing Management

Effective nursing management plays a vital role in stabilizing patients with Addisonian crisis. Comprehensive assessment and vigilant monitoring are essential components of care. Initial steps involve evaluating the patient's condition, recording vital signs, and establishing intravenous access to initiate normal saline infusion. Regular laboratory monitoring, including complete blood counts, lactate levels,

metabolic panels, and arterial blood gases, is critical for tracking the patient's clinical progress and identifying complications. Blood cultures should be obtained to investigate potential infections as underlying triggers for adrenal insufficiency. Intake and output must be meticulously monitored to ensure adequate hydration and fluid balance. Administering prescribed medications and observing for therapeutic responses are fundamental nursing responsibilities. Particular attention should be given to skin pigmentation changes, as they provide important diagnostic clues and reflect disease progression. Additionally, nurses should promptly report any clinical deterioration, such as unstable vital signs or refractory hypotension, to the healthcare provider for immediate intervention. Collaboration with the multidisciplinary team and patient education on medication adherence and stress management strategies further contribute to positive outcomes. By maintaining a proactive and patient-centered approach, nurses ensure comprehensive care and support for individuals with Addisonian crisis.

When to Seek Help

Patients and healthcare providers should remain vigilant for signs warranting immediate medical attention. Clinical indicators include persistent hypotension, unstable vital signs, and a body temperature exceeding 101°F. Early recognition and intervention are crucial to mitigate complications and optimize patient outcomes.

Outcome Identification

Successful management of Addison's disease is marked by the resolution of precipitating factors and stabilization of the patient's condition. Indicators of positive outcomes include the absence of infections, correction of volume depletion and dehydration, and effective medication administration without adverse reactions. Restoration of the patient to their baseline health status signifies the achievement of therapeutic goals.

Monitoring

Patients with Addison disease require meticulous monitoring to detect any changes in their condition and mitigate complications. Healthcare providers must frequently evaluate laboratory parameters, including electrolytes, complete blood counts, and markers of infection, to ensure appropriate management. Regular assessments of blood pressure, respiration, and body temperature are critical, as they provide insight into the patient's cardiovascular and systemic stability. Monitoring body weight is also essential to identify fluid imbalances or weight loss due to insufficient glucocorticoid or mineralocorticoid replacement. Observation for signs of infection, such as fever or elevated inflammatory markers, is crucial given the patient's vulnerability to infections. Skin turgor and other indications of dehydration, such as dry mucous membranes, should be assessed to identify hypovolemia. Electrolyte imbalances, including hyponatremia and hyperkalemia, must be promptly addressed to avoid complications. The presence of an irregular heartbeat or dysrhythmia, often indicative of severe electrolyte disturbances, necessitates immediate intervention. Continuous vigilance and timely reporting of abnormal findings to the healthcare team ensure optimal outcomes for patients with Addison disease [6].

Coordination of Care

Managing Addison disease effectively requires a collaborative, interprofessional approach due to its multi-organ impact and potential life-threatening complications. A team of healthcare specialists, including endocrinologists, intensivists, infectious disease experts, gastroenterologists, and pharmacists, plays a pivotal role in comprehensive patient care. Early diagnosis is critical, as delayed treatment significantly increases morbidity and mortality. Nurses are integral to this process, administering prescribed treatments, monitoring the patient's response, and providing updates to the healthcare team. Pharmacists contribute by ensuring proper medication dosing and educating patients about the importance of adherence to corticosteroid therapy. Physicians, alongside nurses and pharmacists, must educate patients about recognizing symptoms of Addisonian crises and the need for immediate medical attention when experiencing changes in vital signs. Patients should be encouraged to wear medical alert identification to facilitate timely intervention during emergencies. They must also understand the necessity of adjusting

corticosteroid doses during stress, such as infections or minor illnesses. Proper coordination and patient education are essential to improving outcomes and reducing mortality [6, 9].

Health Teaching and Health Promotion

Educating patients with Addison disease is a cornerstone of effective management and long-term health maintenance. Patients must be thoroughly counseled on the importance of medication adherence, as consistent glucocorticoid and mineralocorticoid therapy prevents life-threatening complications. Self-care strategies, including maintaining adequate dietary sodium, monitoring blood pressure, and tracking weight changes, are crucial for early identification of imbalances. Regular clinical checkups enable ongoing evaluation and timely adjustments to therapy. Patients should wear medical alert identification to ensure rapid recognition and treatment in emergencies. It is equally important to keep an emergency dose of cortisol readily available and understand the correct technique and timing for administration. Educating patients to recognize the early signs of Addisonian crises, such as severe fatigue, dizziness, or sudden hypotension, empowers them to seek prompt medical care. Comprehensive health promotion, supported by a collaborative healthcare team, enhances patient safety and quality of life [10].

Risk Management

Effective risk management strategies are vital in mitigating potential complications in patients with Addison disease. Ensuring the correct dosage and timely administration of medications minimizes adverse effects and prevents under-treatment or over-treatment. Fall prevention measures are essential, as patients may experience dizziness or postural hypotension due to adrenal insufficiency. Infection prevention is critical, given the immunosuppressive effects of glucocorticoid therapy. Patients must also avoid dehydration by maintaining adequate fluid intake, especially during illnesses or high-stress situations. By implementing these risk management strategies, healthcare providers can significantly enhance patient safety and reduce the likelihood of adverse events.

Discharge Planning

Comprehensive discharge planning is essential for patients with Addison disease to ensure continued care and optimal recovery. Patients must receive clear instructions regarding follow-up visits with their healthcare provider to monitor their condition and adjust treatment as necessary. Detailed guidance on medication regimens, including timing and dosing, should be provided to promote adherence. Patients should also be educated about potential medication side effects of medication and advised to seek medical attention if adverse reactions occur. Laboratory parameters, such as serum electrolytes and cortisol levels, should be monitored periodically as directed by the clinician. Patients should be vigilant for signs of infection and dehydration, reporting any concerning symptoms promptly. Thorough discharge planning helps facilitate a smooth transition to self-management and prevents readmission.

Other Issues

Recognizing Addison disease can be challenging due to its nonspecific symptoms, which require a high index of suspicion for timely diagnosis. Acute presentations, such as cardiovascular collapse and hemodynamic instability, necessitate immediate medical attention. In cases of Addisonian crisis, treatment with glucocorticoids and fluid resuscitation should not be delayed for diagnostic confirmation, as delays can be fatal. During periods of stress, such as fever or infections, glucocorticoid doses must be doubled to meet increased physiological demands. Prompt recognition, aggressive treatment, and preventive strategies are essential to reduce morbidity and mortality associated with Addison disease.

Conclusion:

Addison's disease, though rare, poses significant clinical challenges due to its nonspecific symptoms and potential for severe complications like adrenal crises. Early recognition and diagnosis are critical for improving outcomes and preventing life-threatening situations. The complex interplay of autoimmune, infectious, genetic, and other etiologies underscores the need for comprehensive diagnostic evaluations, including laboratory tests and imaging studies. The role of nursing in managing Addison's disease cannot

be overstated. Nurses serve as the first line of defense in monitoring patients for early signs of adrenal insufficiency, such as fatigue, hypotension, and hyperpigmentation. Vigilant assessment of fluid balance, electrolyte levels, and hemodynamic stability is essential for mitigating risks like hypovolemia and hyperkalemia, common in mineralocorticoid deficiency. Nurses also play a key role in managing adrenal crises, initiating life-saving interventions such as intravenous hydration, electrolyte correction, and corticosteroid administration. Education is another cornerstone of nursing care in Addison's disease. Patients must be informed about the importance of adhering to prescribed glucocorticoid and mineralocorticoid therapies, recognizing early signs of adrenal crises, and adjusting medications during stress or illness. This education fosters self-management and reduces the risk of complications. The multidisciplinary approach to managing Addison's disease further enhances care quality. Collaboration between nurses, endocrinologists, dietitians, and other healthcare professionals ensures that all aspects of the disease are addressed, from hormonal replacement therapy to nutritional and psychosocial support. In conclusion, the management of Addison's disease demands a multifaceted approach that integrates medical, nursing, and patient-centered strategies. Early diagnosis, effective nursing interventions, and collaborative care can significantly improve patient outcomes and quality of life. Ongoing research into the disease's pathophysiology and treatment options will further enhance care strategies, enabling better prevention and management of this challenging condition.

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الملخص:

الخلفية: يُعرف مرض أديسون، أو القصور الكظري الأولي، بأنه اضطراب غدي نادر يتميز بتدمير قشرة الغدة الكظرية، مما يؤدي إلى نقص في الجلوكوكورتيكويدات والمينيرالوكورتيكويدات والأندروجينات. ويُعتبر التهاب الغدة الكظرية المناعي الذاتي السبب الرئيسي في الدول المتقدمة، حيث تؤدي الأعراض غير المحددة غالباً إلى تأخير التشخيص. في الحالات الشديدة، يمكن أن يتطور المرض إلى أزمة كظرية تتطلب تدخلاً فورياً.

الهدف: يستعرض هذا المقال أسباب مرض أديسون، وآلياته المرضية، وإدارته السريرية، مع التركيز على التدخلات التمريضية لتحسين نتائج المرضى.

الطرق: تم إجراء مراجعة للأدبيات لفحص أسباب المرض، وعوامل الخطر، والمظاهر السريرية لمرض أديسون. وتم التركيز على تشخيصات التمريض واستراتيجيات الإدارة ودمجها في خطط الرعاية متعددة التخصصات.

النتائج: تُبرز النتائج الطبيعة المتعددة الجوانب لمرض أديسون، بما في ذلك الأسباب المناعية الذاتية، والعدوى، والوراثية، والورمية. تشمل العلامات المبكرة التعب، وفقدان الوزن، وفطر التصبغ، مع وجود مؤشرات مخبرية مثل نقص الصوديوم وفطر البوتاسيوم التي تساعد في التشخيص. تعد التدخلات التمريضية الفعالة، مثل الوقاية من العدوى، وإدارة السوائل، وتنقيف المرضى، ضرورية للتخفيف من مضاعفات مثل الأزمات الكظرية. كما أن استراتيجيات الرعاية التعاونية تعزز النتائج طويلة الأجل.

الاستنتاج: يتطلب مرض أديسون نهجاً شاملاً للرعاية بسبب تعقيداته وإمكانية حدوث مضاعفات تهدد الحياة. يلعب التمريض دوراً محورياً في الاكتشاف المبكر والإدارة المستمرة والتنقيف، مما يضمن سلامة المرضى وتحسين جودة حياتهم. كما أن التعاون متعدد التخصصات ضروري لتحسين استراتيجيات الرعاية.

الكلمات المفتاحية: مرض أديسون، القصور الكظري الأولي، التهاب الغدة الكظرية المناعي الذاتي، الأزمة الكظرية، التدخلات التمريضية، نقص الجلوكوكورتيكويد، الرعاية متعددة التخصصات.