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Mini Review

Associations between Supra-Ventricular Tachycardias and Adrenal Insufficiency

Nadhem Abdallah[1*], Abdilahi Mohamoud[1], Meriam Abdallah[2], Mohammed Abdullah[3], Hisham Daher[3]

[1]Department of Internal Medicine, Hennepin Healthcare, Minneapolis, USA; [2]Department of Internal Medicine, University of Saskatchewan, Saskatchewan, Canada; [3]Department of Internal Medicine, University of Debrecen Medical School, Debrecen, Hungary

ABSTRACT

Background: Adrenal Insufficiency (AI) has significant implications for cardiovascular health, influencing electrolyte balance, hemodynamics and atherosclerosis. Understanding its impact on arrhythmia disorders is crucial for improving patient outcomes.

Objectives: This mini review aims to explore the association between AI and cardiovascular complications, particularly in patients with Supra Ventricular Tachycardias (SVTs), to underscore the importance of recognizing AI as a high-risk factor for adverse cardiovascular events.

Methods: PubMed and Google Scholar were searched using terms including "hypoadrenalism," "adrenal insufficiency," and various arrhythmia disorders. Criteria included English-language, peer-reviewed studies focusing on their association. Data extraction and quality assessment were conducted to synthesize findings for analysis and dissemination.

Discussion: The review found that AI is associated with increased in-hospital mortality, a higher need for intensive care, longer hospital stays and higher hospitalization costs in patients with SVTs. These outcomes are attributed to electrolyte imbalances, altered hemodynamics and accelerated atherosclerosis in AI patients.

Conclusions: AI significantly impacts cardiovascular health and is a predictor of adverse outcomes in patients with SVTs. Clinicians should be aware of this association to improve management strategies. Further research is needed to unravel the underlying mechanisms and enhance clinical outcomes for this high-risk population.

Keywords: Tachycardias; Adrenal insufficiency; SVTs; Hemodynamics

INTRODUCTION

Numerous studies and case reports in the literature have indicated a connection between adrenal insufficiency and cardiovascular disease. This link is believed to arise from the essential role adrenal hormones play in regulating the cardiovascular system. Patients with Adrenal Insufficiency (AI), who often undergo long-term treatment with glucocorticoids, either alone or combined with mineralocorticoids, are susceptible to inadequate adrenal hormone levels (either before diagnosis or due to insufficient treatment during periods of physical stress) or excessive exposure to these hormones. This imbalance in adrenal hormones leads to disruptions in electrolyte levels, changes in blood flow dynamics and accelerated development of arterial hardening, potentially increasing the risk of adverse cardiac events [1]. Despite these findings, there is limited data on the outcomes of arrhythmia disorders such as atrial fibrillation, atrial flutter and Paroxysmal Supraventricular Tachycardias (PSVT) in individuals with AI. Arrhythmias are abnormalities in the heart's rhythm and they can have serious consequences, including stroke and heart failure. Understanding how adrenal insufficiency may influence the development and progression of these arrhythmias is crucial for optimizing patient care. This mini review aims to examine existing literature on this topic to highlight the association and advocate for future research that could enhance patient outcomes. By synthesizing current evidence and identifying gaps in knowledge, this review seeks to stimulate further investigation into the relationship between adrenal insufficiency and arrhythmia disorders. A deeper understanding of this association

Correspondence to: Nadhem Abdallah, Department of Internal Medicine, Hennepin Healthcare, Minneapolis, USA; E-mail: nadhem.abdallah@hcmed.org

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could lead to improved risk stratification, prevention strategies and therapeutic interventions for individuals with adrenal insufficiency and comorbid cardiovascular conditions.

LITERATURE REVIEW

Adrenal insufficiency occurs when the adrenal glands can no longer produce adequate amounts of mineralocorticoid and glucocorticoid hormones to meet the body's physiological needs. Treatment for adrenal insufficiency varies depending on its classification and severity, but primary management includes optimizing electrolytes, fluid resuscitation and supplementing deficient glucocorticoid and/or mineralocorticoid hormones. Additionally, patients with AI in acute conditions often require higher doses of steroid supplementation due to their increased hormone demands, a practice known as stress dosing. This need for stress dosing can further complicate hospitalization, as patients often require more complex steroid dosage adjustments. This situation can lead to prolonged hospital stays, increased demand for healthcare resources, and higher Total Healthcare Costs (THC) [2].

Although Adrenal Insufficiency (AI) is reported to be relatively rare in the general population, with an incidence of approximately 10-15 per 100,000 individuals [1], a recent retrospective study using nationwide inpatient data revealed that 0.2% (or 200 per 100,000) of patients in the United States admitted with Supraventricular Tachycardias (SVTs) also had AI, indicating that AI may be more common in patients with arrhythmia disorders [3]. This finding aligns with existing literature suggesting a significant association between AI and cardiovascular disease. Patients with adrenal insufficiency often require prolonged glucocorticoid therapy, with or without mineralocorticoids, which can lead to steroid insufficiency or excess. Excess glucocorticoids are linked to a higher risk of obesity, hypertension, metabolic syndrome and various cardiomyopathies [4,5]. Conversely, glucocorticoid deficiency can lead to severe hypotension due to the impaired regulation of the glucocorticoid receptor on endothelial vasculature, thus contributing to poor cardiovascular health [6].

Moreover, acute adrenal crisis has been extensively documented to precipitate severe cardiovascular complications, as highlighted in several compelling case reports [7]. Esposito et al., have postulated a plausible mechanism wherein adrenal insufficiency predisposes individuals to lower blood pressure levels. This, when coupled with underlying coronary artery disease, can precipitate a significant myocardial perfusion imbalance [8]. Similarly, Mastorakos et al., have proposed a compelling hypothesis regarding the heightened risk of cardiovascular disease in individuals with adrenal insufficiency. They suggest a potential link between lower levels of mineralocorticoids and elevated levels of inflammatory cytokines, including IL-6, IL-1 and TNF-alpha [9]. This inflammatory milieu may contribute to endothelial dysfunction and vascular remodeling, further exacerbating the risk of adverse cardiovascular events.

The pathophysiological cascade underlying cardiovascular events in adrenal insufficiency likely involves accelerated atherosclerosis, a process intricately linked to hormonal dysregulation. Elevated levels of Adrenocorticotropic Hormone (ACTH) have been implicated in the promotion of atheroma plaque calcification through interactions with osteochondrogenic mesenchymal cells, thereby fostering their enhanced differentiation [10]. This phenomenon underscores the multifactorial nature of cardiovascular complications in adrenal insufficiency, implicating hormonal, inflammatory and vascular factors in their pathogenesis.

These insights underscore the pressing need for further research endeavors aimed at elucidating the intricate mechanisms underpinning the heightened cardiovascular risks associated with adrenal insufficiency. By unraveling the complex interplay between hormonal dysregulation, inflammation, and atherosclerosis, researchers can pave the way for targeted therapeutic interventions aimed at mitigating these risks and improving patient outcomes. Additionally, a deeper understanding of the pathophysiology of adrenal crisis-induced cardiovascular complications can inform clinical practice guidelines, empowering healthcare providers to promptly recognize and manage these potentially life-threatening events.

Moreover, recent research indicates that serum electrolyte imbalance and acidosis due to adrenal hormone deficiency can result in myocardial reperfusion injuries and significantly affect the ionic balance within the myocardial intracellular and extracellular environment, predisposing patients to rhythm disturbances. Hypopituitarism is frequently associated with electrocardiographic abnormalities. Iga et al., suggested that hypoglycemia-induced catecholamine release might lead to arrhythmias and/or abnormal wall motion of the left ventricle in patients with adrenal insufficiency. Similarly, Nunoba et al., discuss the impact of hypomagnesemia in patients with AI, noting that it can cause an intracellular-extracellular electrolytic imbalance in myocytes, resulting in the shortening of the effective refractory period and prolongation of the relative refractory period of cardiac conductive tissue. Several case reports have documented an increased propensity for ventricular arrhythmias in patients with adrenal insufficiency and other studies have reported a higher incidence of ventricular arrhythmias in patients with postpartum Sheehan syndrome.

DISCUSSION

Recent data suggests that among patients admitted with atrial fibrillation, atrial flutter and PSVT, co-existing AI is linked to higher odds of in-hospital mortality, an increased need for intensive care, longer hospital stays and higher total charges compared to patients without AI. Patients with AI experiencing acute events, such as decompensation of chronic atrial fibrillation or new-onset atrial flutter with rapid ventricular response, often exhibit a heightened demand for hormones. This necessitates increased doses of steroid supplementation. The disparity between hormone demand and supply can lead to acute relative steroid deficiency in AI-arrhythmia patients, exacerbating hypotension and requiring vasopressor or mechanical ventilation support. Conversely, excessive inpatient steroid supplementation in AI patients can also result in unfavorable outcomes, as excess glucocorticoid intake is associated with an increased risk of adverse cardiovascular

events. Regarding the extended mean length of stay and elevated total hospitalization charges, adverse outcomes such as the increased need for vasopressors and mechanical ventilation likely contribute to these results. Additionally, literature suggests that adjustments in steroid dosages can prolong hospital stays and escalate the demand for healthcare resources among hospitalized patients with AI. Therefore, precise and cautious glucocorticoid dosing is imperative during hospitalizations for atrial fibrillation, atrial flutter and PSVT to mitigate the risks associated with both under- and over-supplementation. A multidisciplinary approach involving both cardiovascular and endocrinology specialists is essential to optimize patient outcomes in this high-risk population.

CONCLUSION

This burgeoning body of evidence unveils a significant clinical challenge, necessitating a comprehensive understanding of the underlying mechanisms driving this association. It serves as a clarion call for concerted efforts to delve deeper into the intricacies of AI-related cardiovascular complications, thereby paving the way for targeted interventions that can ameliorate patient outcomes. Given the paucity of data elucidating the specific mechanisms linking AI to adverse cardiovascular events, further research endeavors are imperative. Robust investigations are warranted to unravel the intricate pathways by which AI predisposes individuals to heightened cardiovascular risks, including arrhythmia disorders such as SVTs. Moreover, a nuanced exploration of potential therapeutic strategies tailored to mitigate these risks is essential. The imperative for rigorous scientific inquiry extends beyond academic curiosity; it directly impacts clinical decision-making and patient care. By unraveling the enigma surrounding the intersection of AI and cardiovascular health, clinicians can refine risk stratification strategies, optimize treatment modalities and ultimately enhance patient outcomes. Therefore, investment in translational research efforts is paramount, with the collective goal of transforming these insights into tangible clinical benefits for individuals grappling with AI-associated cardiovascular complications.

REFERENCES

1. Esposito D, Bobbio E, Di Fraia R, Mone P, Accardo G, de Bellis A, et al. Patients with adrenal insufficiency have cardiovascular features associated with hypovolemia. Endocrine. 2020;70:412-420.
2. Bancos I, Hahner S, Tomlinson J, Arlt W. Diagnosis and management of adrenal insufficiency. Lancet Diabetes Endocrinol. 2015;3(3):216-226.
3. Abdallah N, Mohamoud A, Kearns A, Abdallah M, Linzer M. Relationships between adrenal insufficiency and cardiovascular outcomes in patients with atrial fibrillation, atrial flutter and paroxysmal supraventricular tachycardia. Curr Probl Cardiol. 2024:102641.
4. Stewart PM, Biller BM, Marelli C, Gunnarsson C, Ryan MP, Johannsson G. Exploring inpatient hospitalizations and morbidity in patients with adrenal insufficiency. J Clin Endocrinol Metab. 2016;101(12):4843-4850.
5. Filipsson H, Monson JP, Koltowska-Haggstrom M, Mattsson A, Johannsson G. The impact of glucocorticoid replacement regimens on metabolic outcome and comorbidity in hypopituitary patients. J Clin Endocrinol Metab. 2006;91(10):3954-3961.
6. Krug JJ. Cardiac arrest secondary to Addison's disease. Ann Emerg Med. 1986;15(6):735-737.
7. Zhao R, Luo S, Wang S, Wen Y, Xiong F. Coronary artery disease in a patient with Addison's disease: A case report and literature review. BMC Cardiovasc Disord. 2023;23(1):54.
8. Mastorakos G, Paltoglou G, Greene M, Ilias I, Papamichalopoulos A, Dimopoulos S, et al. Inappropriately normal plasma ACTH and cortisol concentrations in the face of increased circulating interleukin-6 concentration in exercise in patients with sarcoidosis. Stress. 2013;16(2):202-210.
9. Evans JF, Ragolia L. Systemic and local ACTH produced during inflammatory states promotes osteochondrogenic mesenchymal cell differentiation contributing to the pathologic progression of calcified atherosclerosis. Med Hypotheses. 2012;79(6):823-826.
10. Iga K, Hori K, Gen H. Deep negative T waves associated with reversible left ventricular dysfunction in acute adrenal crisis. Heart Vessels. 1992;7:107-117.