

Primary Hyperaldosteronism: A Rare Potential Risk Factor for Acute Aortic Dissection, an Interesting Case Report

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ABSTRACT

Primary hyperaldosteronism is overproduction of aldosterone from the adrenal cortex with wide electrolyte imbalance and cardiovascular complications like aortic dissection. While the association between primary hyperaldosteronism and aortic dissection has been a subject of interest, the exact relationship and potential causative mechanisms remain unclear. We present here a 51-year-old male with no significant past medical history who came to the emergency department with severe chest pain, sudden-onset radiating to the back, shortness of breath (SOB), and diaphoresis. A chest-to-abdominal Computed Tomography (CT) angiography showed a small tear in the inner lining of the aortic arch, just below the left subclavian artery. The descending aorta had a large mural thrombosis up to the T7 level. The pressure was controlled with intravenous drugs, and then oral antihypertensive drugs and electrolytes were corrected. The patient was recommended to go outside where cardiovascular surgery is available and is now doing well in cardiology outpatient follow-up with well-controlled blood pressure.

Keywords: Primary Hyperaldosteronism, Aortic Dissection, Renin Aldactone Ratio

Introduction

Primary hyperaldosteronism, Conn's syndrome, is a disorder characterized by the overproduction of aldosterone from the adrenal cortex, leading to various clinical manifestations such as hypertension, hypokalemia, and associated cardiovascular complications [1]. Aortic dissection, on the other hand, is a life-threatening condition involving the separation of the layers within the aortic wall, often associated with severe chest or back pain and high mortality rates if not promptly diagnosed and managed [2]. While the association between primary hyperaldosteronism and aortic dissection has been a subject of interest, the exact relationship and potential causative mechanisms remain to be fully elucidated [3]. Diagnosing primary hyperaldosteronism involves assessing aldosterone levels, renin activity, and the aldosterone-renin ratio with confirmatory tests such as the saline infusion test or the captopril challenge test [4]. Imaging studies such as Computed Tomography (CT) and magnetic resonance imaging (MRIs) are also used to identify adrenal tumors or structural abnormalities. In the case of aortic dissection, imaging modalities such as computed tomography angiography (CTA) and transesophageal echocardiography (TEE) play a crucial role

in the accurate diagnosis and classification of the dissection [5]. Additionally, a thorough clinical evaluation and patient history are essential for identifying risk factors and presenting symptoms associated with aortic dissection [6]. The potential association between primary hyperaldosteronism and aortic dissection is complex and multifaceted. Primary hyperaldosteronism can lead to hypertension as well as vascular remodeling, potentially contributing to the weakening of the aortic wall and increasing the risk of dissection [3]. This paper aims to explore the potential link between primary hyperaldosteronism and aortic dissection, drawing on relevant case reports and literature to provide a comprehensive understanding of this association.

Case Presentation

A 51-year-old male presented to the emergency department with sudden-onset, severe chest pain radiating to his back. The pain began abruptly while he was at rest and was associated with shortness of breath and diaphoresis. The patient reported no significant medical history, but he had experienced intermittent episodes of hypertension in recent months. On examination, he appeared distressed and anxious. His blood pressure was significantly elevated at 190/110 mmHg, with a heart rate of 110 beats per minute. Physical examination revealed diminished bilateral pulses in the lower extremities and a difference in blood

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pressure between the right and left arms. Chest auscultation revealed a diastolic murmur, and he exhibited signs of mild respiratory distress. Immediate investigations were initiated, including a 12-lead electrocardiogram (ECG), which showed nonspecific changes. Right away, a contrast-enhanced computed tomography (CT) angiogram of the chest and abdomen was done, which showed a Stanford type B aortic dissection involving the descending aorta. Small intimal dissection in the aortic arch distal to the left subclavian artery with large mural thrombosis of the descending aorta up to the T7 level was seen (Figure 1 A&B). In addition to the CT angiogram, transthoracic echocardiography (TTE) was performed to assess for any aortic valve involvement or other cardiac abnormalities. TTE revealed normal left ventricular function and the absence of significant valvular pathology. On a lab test, the patient had severe hypokalemia, mildly elevated hypernatremia, and a markedly elevated aldosterone-to-renin ratio, which was consistent with primary hyperaldosteronism (Figure 2). The patient was diagnosed with an acute Stanford type B aortic dissection secondary to primary hyperaldosteronism. As soon as possible, medical stabilization began, with aggressive blood pressure control using intravenous beta-blockers and a glycerate trinitrate infusion with potassium replacement. The patient was also started on oral antihypertensive medications, including the mineralocorticoid receptor antagonist Spiro lactone 50mg twice a day. Given the extent of the dissection, the patient was closely monitored for signs of end-organ malperfusion, and surgical intervention was considered.



Figure 1: CT angiogram showing Small intimal dissection in the aortic arch distal to the left subclavian artery (panel A&B) with large mural thrombosis of the descending aorta up to the T7 level.

Sodium	153	mEq/L	135-150
Potassium	2.48	mEq/L	3.5-5.5
Renin Aldactone ratio	52		0-12

Figure 2: Lab test showing elevated renin to Aldactone ratio and severe hypokalemia

Discussion

The association between primary hyperaldosteronism and aortic dissection has been a topic of interest in the medical literature [3]. Ahmed et al. presented a case report that strengthened the hypothesis that primary hyperaldosteronism is a potential independent risk factor for aortic dissection [3]. This finding suggests a potential direct link between the pathophysiological effects of aldosterone excess and the structural integrity of the aortic wall, leading to the development of dissection.

Furthermore, Safi et al. described a patient with long-lasting secondary hypertension caused by primary aldosteronism, along with coronary artery aneurysms and aortic dissection, highlighting the potential cardiovascular implications of primary hyperaldosteronism [7]. These findings add to the clinical evidence that there may be a link between primary hyperaldosteronism and aortic dissection and highlight the need for more research into the underlying mechanisms and risk factors that cause this to happen. In addition to the cardiovascular implications, Ito et al. showed in a case report that primary hyperaldosteronism has been associated with severe hypokalemia, which can lead to complications such as rhabdomyolysis. The electrolyte disturbances associated with hyperaldosteronism may contribute to the pathogenesis of aortic dissection through mechanisms involving vascular smooth muscle function and integrity [8]. While the exact pathophysiological mechanisms linking primary hyperaldosteronism to aortic dissection remain to be fully elucidated, the existing literature provides compelling evidence of a potential association between these two conditions. The role of aldosterone in modulating vascular function, electrolyte balance, and blood pressure regulation may contribute to the development of structural changes in the aortic wall, predisposing individuals with primary hyperaldosteronism to aortic dissection. The association between primary hyperaldosteronism and aortic dissection represents a complex and clinically significant relationship that warrants further investigation. The synthesis of case reports and literature reviews provides valuable insights into the potential pathophysiological links and clinical implications of this association, highlighting the need for continued research to better understand the underlying mechanisms and optimize the management of patients with primary hyperaldosteronism who are at risk of aortic dissection.

Conclusion

This case highlights the importance of considering the secondary causes of hypertension, such as primary hyperaldosteronism, in patients presenting with acute aortic dissection. Timely diagnosis and management are crucial for optimizing outcomes in such complex cases, involving a multidisciplinary approach that includes cardiology, endocrinology, and vascular surgery.

Consent for Publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal upon request.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising, or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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