WebLog Journal of Nephrology and Hypertension

Case Report
Published: 13 May, 2026

Abdominal Aortic Dissection Associated with Primary Aldosteronism: A Case Report and Review of Emerging Evidence

Giulia Percudani#, Maria Giulia Canè#, Davide Cunzi, Debora Severi, Giorgia Prampolini, Jorge Luis Zoino and Aurelio Negro*

Internal Medicine and Hypertension Center, Sant'Anna Hospital Castelnovo ne' Monti, Azienda USL-IRCCS di Reggio Emilia, Reggio Emilia, Italy

#*Both authors contributed equally*

Abstract

Primary aldosteronism (PA) is a common and potentially curable cause of secondary hypertension associated with increased cardiovascular morbidity and mortality beyond blood pressure levels. Aldosterone excess contributes to vascular damage through pro-inflammatory, pro-fibrotic and remodelling effects on the arterial wall. An association between PA and aortic diseases has been increasingly reported but remains underrecognized in clinical practice.

We report a case of a 52-year-old woman referred to the emergency room due to acute abdominal and lower limb pain with detection of subrenal aortic dissection on contrast-enhanced computed tomography (CECT). Given her young age and newly diagnosed hypertension, a secondary cause was investigated. An elevated aldosterone-to-renin ratio was detected and autonomous aldosterone secretion was confirmed by saline infusion test. Review of imaging revealed a left adrenal nodule. Adrenal vein sampling (AVS) showed significant left-sided lateralization. The patient was deemed eligible for left adrenalectomy. Following surgery, she progressively reduced antihypertensive therapy. Genetic analysis showed a germline mutation in CLCN2, indicative of familial hyperaldosteronism type II.

This case supports the emerging link between PA and aortic dissection suggesting that aldosterone excess may play a direct pathogenic role in aortic wall vulnerability, independently of hypertension severity. Screening for PA should be considered in patients with aortic dissection and hypertension as early diagnosis and targeted treatment may reduce the risk of severe cardiovascular (CV) complications.

Keywords: Primary Aldosteronism; Aortic Dissection; Mineralocorticoid Receptor; Endocrine Hypertension; Vascular Remodeling; Familial Hyperaldosteronism

OPEN ACCESS

Correspondence:

Giulia Percudani, Internal Medicine and Hypertension Center, Sant'Anna Hospital Castelnovo ne' Monti, Azienda USL-IRCCS di Reggio Emilia, Reggio Emilia, Italy,

E-mail: giulia.percudani@ausl.re.it

Received Date: *28 Apr 2026*

Accepted Date: *11 May 2026*

Published Date: *13 May 2026*

Citation:

Percudani G, Canè MG, Cunzi D, Severi D, Prampolini G, Zoino JL, et al. Abdominal Aortic Dissection Associated with Primary Aldosteronism: A Case Report and Review of Emerging Evidence. wjnh.2026.e1304. https://doi.org/10.5281/zenodo.20210925

Introduction

Acute aortic dissection (AD) is a rare life-threatening vascular emergency which could develops in aortic rupture. It is associated to high early mortality approaching 50% before hospital admission and up to 90% within the first 48 hours if left untreated [1]. The pathogenesis is related to medial degeneration resulting from elastic fibers fragmentation, smooth muscle cell loss and extracellular matrix remodelling leading to structural weakening of the aortic wall [2]. While traditional CV risk factors and inherited connective tissue disorders are well established, an increasing number of cases occurs in patients without clear predisposing conditions suggesting additional biological mechanisms beyond hemodynamic stress alone.

PA is a common cause of secondary hypertension affecting approximately 5–10% of hypertensive individuals and 20–30% of patients with resistant hypertension [1]. Beyond its pressor effects, aldosterone exerts direct deleterious actions on the cardiovascular system promoting endothelial dysfunction, vascular inflammation, oxidative stress, fibrosis, extracellular matrix remodelling and increased arterial stiffness [2]. These non-hemodynamic effects contribute to an additive cardiovascular risk to the patients. Growing experimental and clinical evidence suggest a potential link between aldosterone excess and aortic disease [2]. We report a case of spontaneous abdominal aortic dissection as the initial manifestation of genetically confirmed familial primary aldosteronism

type II. We suggest the importance of including endocrine etiologies in the differential diagnosis of aortic dissection.

Case Report

A 52-year-old woman was admitted to the emergency department due to sudden-onset severe abdominal pain radiating to the lower back. She had no past medical history and no chronic medications assumption. On arrival, she presented hypertensive with blood pressure (BP) 180/115 mmHg, heart rate 60 beats/min, oxygen saturation 100% on room air and normal body temperature.

The laboratory showed normal inflammatory markers and organ function: hemoglobin 15.6 g/dl (n.v. 12.0-16.0), serum creatinine 0.98 mg/dl (n.v. 0.50-1.10), sodium 139 mmol/l (n.v. 135-145), potassium 3.3 mmol/l (n.v. 3.5-4.5), cardiac troponin was negative. Electrocardiography revealed no acute ischemic changes. Ultrasonography raised suspicion for infrarenal abdominal aortic dissection. A CECT scan of the chest, abdomen and supra-aortic vessels confirmed an infrarenal abdominal aortic dissection extending to the aortic bifurcation with an intimal flap, an anterior false lumen and a mural hematoma extended cranially to the thoracoabdominal junction (figure 1). The dissection involved the left aspect of the aortic bifurcation with extension to the origin of the left common iliac artery. A small 6-mm aneurysm of a post-divisional branch of the inferior mesenteric artery was also noted. The thoracic aorta was normal in caliber.

After vascular surgery consultation, conservative management was recommended. The patient was admitted to the intensive care unit and intravenous clevidipine and labetalol were started. Blood pressure control was progressively reached and de-escalation to oral antihypertensive therapy with carvedilol, amlodipine, ramipril and indapamide was performed. Serial CT examinations demonstrated stability of dissection.

Given the young age and the absence of known cardiovascular risk factors, an etiologic evaluation was undertaken. 18F-FDG positron emission tomography demonstrated grade 2 aortic wall uptake according to Meller criteria without evidence of active vasculitis. Immunological testing including erythrocyte sedimentation rate, complement levels, antinuclear antibodies, extractable nuclear antigens antibodies, anti-neutrophil cytoplasmic antibodies and rheumatoid factor were negative. Transthoracic echocardiography excluded structural heart disease and significant aortic dilation.

During follow-up, severe hypertension requiring multiple agents and associated hypokalemia prompted evaluation for PA. The laboratory showed suppressed plasma renin (<0.1 ng/dl), aldosterone 232 pg/ml (n.v. 30-150) and altered aldosterone-to-renin ratio. A saline infusion test and a captopril challenge test demonstrated inadequate aldosterone suppression (96 pg/ml post saline infusion and 127 pg/ml post captopril respectively), confirming autonomous aldosterone secretion.

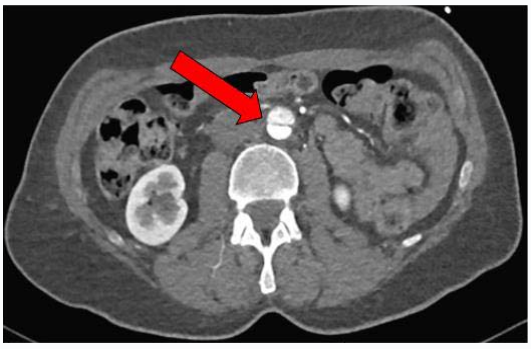

Figure 1: CT angiography showing infrarenal abdominal aortic dissection with intimal flap and false lumen.

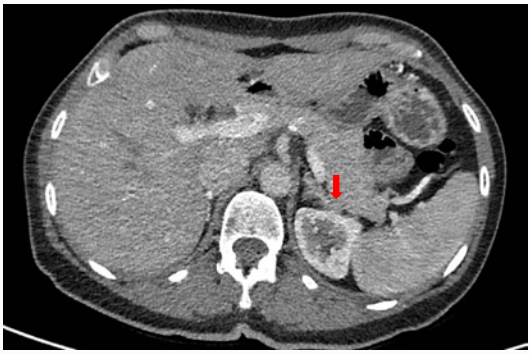

Figure 2: Abdominal CT scan demonstrating left adrenal adenoma.

Retrospective review of abdominal imaging identified a left adrenal nodule with Hounsfield unit (HU) 9 typical for adenoma (Figure 2).

AVS without cosyntropin stimulation was performed. An optimal selectivity and a significant left lateralization were observed (laterality index 7.06). The patient underwent laparoscopic left adrenalectomy. BP improved substantially and the patient reached an optimal BP control with carvedilol 6.25 mg twice daily.

Histological examination identified diffuse aldosterone-producing adrenal hyperplasia characterized by a broad and uninterrupted band of zona glomerulosa cells with more than 50% of cells showing CYB11B2 positivity (APDH), according to the HISTALDO Consensus Classification for Unilateral Primary Aldosteronism.

Genetic analysis identified a heterozygous gain-of-function mutation in the *CLCN2* gene (position 3q27.1 exon 15, protein variation p.Val521Met) consistent with familial hyperaldosteronism type 2.

At the most recent evaluation, the patient is normotensive without drug therapy. The most recent blood tests showed renin 1 ng/dl (n.v. 0.1-2.4), aldosterone 128 pg/ml, creatinine 0.91 mg/dl, potassium 4.4 mmol/l. Ongoing follow-up includes periodic CT imaging, 24-hour ambulatory blood pressure monitoring and reassessment of renin and aldosterone levels.

Discussion

We report a case of a patient presenting with acute aortic dissection in whom subsequent evaluation revealed PA due to a familial type II form associated with a *CLCN2* mutation.

The increased CV risk associated with PA has historically been attributed primarily to chronic hypertension. However, accumulating clinical and experimental evidence shows aldosterone direct pathogenic effects on the vascular wall independent of blood pressure levels [2]. Animal models have demonstrated that mineralocorticoid receptor (MR) activation associated with high salt intake can induce aneurysm formation and aortic dissection [3]. MR activation

promotes oxidative stress, inflammatory signalling, extracellular matrix degradation, collagen remodelling and vascular fibrosis [3]. These processes may contribute to medial degeneration and loss of structural integrity facilitating dissection independently of systemic BP. The literature has described aortic dissection and aneurysm formation in patients with PA, often occurring at a relatively young age and occasionally representing the initial clinical sign of PA leading to endocrine diagnosis [1]. Although such observations cannot establish causality, the recurrence of similar clinical patterns across diverse geographic and clinical contexts suggests that the association may not be coincidental. In some cases, stabilization of vascular disease following adrenalectomy or targeted medical therapy has been reported supporting the hypothesis of a pathophysiological link between aldosterone excess and aortic wall vulnerability.

The literature has further characterized the clinical spectrum of aortic involvement in PA highlighting the relatively young age at presentation, the predominance of unilateral adrenal disease and suggesting that surgical treatment may be associated with lower recurrence of vascular events compared to medical therapy alone [1]. Case–control studies have demonstrated higher plasma aldosterone concentrations in hypertensive patients with aortic aneurysm or dissection compared with matched hypertensive controls without major vascular pathologies [4]. This association is particularly evident in patients with suppressed renin activity supporting the hypothesis that autonomous aldosterone secretion represents a biologically distinct vascular risk condition. Although limited by retrospective design and potential residual confounding, the independent replication of findings across cohorts strengthens their internal consistency.

Recent genetic epidemiology has provided additional support for a potential causal relationship. A Mendelian randomization study using genetic variants associated with aldosterone excess as instrumental variables has demonstrated a significant increase in the risk of aortic pathology, particularly AD [5]. Interestingly, these analyses suggest a differential association across aortic segments with stronger effects observed for thoracic aortic disease and dissection compared with abdominal aneurysm formation. These findings raise the possibility that aldosterone-mediated mechanisms may preferentially promote acute structural instability rather than progressive aneurysmal dilation, although the biological basis of this pattern remains incompletely understood.

The identification of a germline *CLCN2* mutation in this patient highlights the potential relevance of genetic forms of PA in vascular risk stratification. Familial hyperaldosteronism type II remains incompletely characterized with respect to long-term vascular outcomes and current evidence is insufficient to define genotype-specific risk profiles. Nevertheless, this finding underscores the importance of considering inherited endocrine disorders in patients presenting with severe vascular events at a relatively young age.

Several limitations must be acknowledged as the current evidence remains largely observational. The absolute risk of aortic complications among patients with PA is unknown and prospective longitudinal data are lacking. Despite these limitations, this case has relevant clinical implications. Endocrine causes of secondary hypertension should be considered in patients presenting with AD, reduced renin levels or difficult BP control. Future prospective studies are needed to clarify the prevalence of PA among patients with aortic disease and to determine whether targeted treatment can influence long-term aortic outcomes.

In summary, this case strengthens the hypothesis that aldosterone excess may contribute to aortic wall vulnerability and supports the evolving concept of PA as a systemic vascular disorder with potential implications for screening and multidisciplinary management.

On behalf of all authors, the corresponding author states that there are no conflict of interest and no external funding was received. Patient informed consent was obtained.

References

1. Gorji MG, Keshtkar A, Rafiei A, Alidousti A, Zarepour F, Keshtkar A. A case of simultaneous adrenalectomy and dissection repair with direct sheath placement into the aorta and systematic review of cases with hyperaldosteronism and vascular dissection: a case report. *J Med Case Reports.* 2025; 19: 237.

2. Hung CS, Sung SH, Liao CW, Pan CT, Chang CC, Chen ZW, et al. Aldosterone induces vascular damage: a wave reflection analysis study. *Hypertension.* 2019; 74(3): 623-629.

3. Liu S, Xie Z, Daugherty A, Cassis LA, Pearson KJ, Gong MC, et al. Mineralocorticoid receptor agonists induce mouse aortic aneurysm formation and rupture in the presence of high salt. *Arterioscler Thromb Vasc Biol.* 2013; 33(7): 1568-1579.

4. Pu Y, Yang G, Pan X, Wang F, Zhang Y, Zheng A, et al. Higher plasma aldosterone concentrations in patients with aortic diseases and hypertension: a retrospective observational study. *Eur J Med Res.* 2023; 28(1): 541.

5. Liu S, Wan J, Yang Y, Wang D, Hou J, Wang P. Causal association between genetically predicted primary aldosteronism and aortic aneurysm and aortic dissection: a Mendelian randomization study. *Medicine (Baltimore).* 2025; 104(12): e41951.