



One Gram of Prevention Is Worth a Kilogram of Cure: Preventing Kidney Problems in Patients with Coarctation of the Aorta

Reza Abdollahi ^{1,*}

¹ Student Research Committee, Urmia University of Medical Sciences, Urmia, Iran

*Corresponding Author: Student Research Committee, Urmia University of Medical Sciences, Urmia, Iran. Email: rezaabdollahi97@yahoo.com

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Dear Editor,

Coarctation of the aorta (CoA) is a congenital condition characterized by narrowing of the thoracic aorta, leading to proximal hypertension and distal hypoperfusion. Although the cardiovascular consequences of CoA are well documented, its direct and sustained impact on kidney health is often underemphasized during clinical follow-up. This letter argues that, beyond monitoring for recoarctation, lifelong care for patients with CoA should include a targeted renal protection strategy (1). The primary objective is to synthesize current evidence linking CoA to kidney injury and to propose a more focused framework for prevention, shifting from general surveillance to specific nephroprotective interventions.

The hemodynamic disturbance in CoA directly compromises renal function. Aortic narrowing reduces blood flow and perfusion pressure distal to the stenosis, including to the renal arteries. This hypoperfusion is a potent stimulus for activation of the renin-angiotensin-aldosterone system (RAAS), leading to systemic vasoconstriction and fluid retention that further exacerbate hypertension and perpetuate a cycle of renal stress (1). The clinical consequences of this mechanism can be severe and acute. For instance, Saliccioli and Zachariah described cases in which the hemodynamic compromise associated with CoA precipitated critical events, such as recurrent acute kidney injury in a 38-year-old man, directly linking the anatomic defect to renal parenchymal damage (2). In an even more dramatic pediatric case, Zhang et al. reported acute renal infarction in a 10-year-old boy secondary to thrombosis in a coarcted aortic segment, underscoring the potential for irreversible kidney damage when flow

is critically compromised (3). These examples illustrate that the threat to the kidneys is not merely theoretical but a tangible clinical reality requiring proactive management.

Effective prevention of these outcomes hinges on a dual approach: timely relief of the obstruction and rigorous, lifelong blood pressure management tailored to the unique physiology of CoA. The primary intervention, whether surgical or catheter-based, aims to restore normal aortic flow, thereby improving renal perfusion pressure and mitigating the RAAS-driven hypertensive stimulus (4). As Ye et al. noted, successful intervention often normalizes blood pressure and improves renal hemodynamics, as evidenced by enhanced perfusion after angioplasty and stenting (5). However, relief of the obstruction does not equate to a cure for all patients. Hypertension can persist or recur years later because of altered vascular compliance, residual gradients, or aortic arch abnormalities. Therefore, the cornerstone of renal protection is meticulous blood pressure control. Standard office measurements are insufficient; ambulatory blood pressure monitoring (ABPM) is essential for detecting masked or nocturnal hypertension, which exerts continuous, subclinical strain on the renal microvasculature (6). A lower threshold for initiating and intensifying antihypertensive therapy may be justified in this population to prevent the long-term progression to chronic kidney disease.

In conclusion, CoA should be reconceptualized not only as a congenital heart defect but also as a state of chronic kidney threat. The message for the clinical community is clear: to prevent kidney problems in patients with CoA, clinicians must look beyond the

coarctation site itself and adopt a targeted, dual-pronged approach. First, timely correction of the anatomic obstruction is needed to restore renal perfusion. Second, aggressive long-term hypertension management guided by ABPM is equally critical to protect the kidneys from persistent pressure overload. This focused care paradigm, which prioritizes renal protection from the outset, embodies the principle that one gram of prevention is worth a kilogram of cure.

Footnotes

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