

Beyond Hypertension: Clinical Heterogeneity and Cardiovascular Outcomes in Midaortic Syndrome

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ABSTRACT

Midaortic syndrome (MAS) is a rare vascular disorder characterized by segmental or diffuse narrowing of the distal thoracic and/or abdominal aorta, frequently involving the renal and visceral arteries. We aimed to describe the clinical characteristics, cardiovascular findings, management strategies, and follow-up outcomes of patients with MAS treated at a tertiary referral center.

This retrospective observational case series included all patients with radiologically confirmed MAS managed between 2010 and 2026. Demographic characteristics, clinical presentation, associated syndromic and cardiovascular abnormalities, electrocardiographic and echocardiographic findings, imaging studies, treatment strategies, and follow-up outcomes were reviewed.

Five patients were included. Hypertension was present in four patients at diagnosis, and the remaining patient developed hypertension during follow-up; all patients were managed medically. Three patients had associated genetic syndromes (Turner, Williams and Noonan syndromes), and four had concomitant congenital cardiovascular abnormalities. Three patients had undergone cardiac catheterization before the diagnosis, one of them with balloon angioplasty of coarctation of the aorta, whereas none required surgical reconstruction or bypass surgery for MAS. No deaths, heart transplantation, ventricular assist device implantation, or sustained arrhythmias occurred during follow-up. Notably, this cohort includes the long-term follow-up of a previously published patient with Turner syndrome and MAS from our institution, extending the available clinical data beyond the initial case report.

MAS demonstrates substantial clinical heterogeneity and is frequently associated with congenital cardiovascular abnormalities and genetic syndromes. Early diagnosis, comprehensive vascular imaging, individualized management, and long-term follow-up may contribute to favorable cardiovascular outcomes.

Keywords: Midaortic syndrome, Hypertension, Pediatrics

Introduction

Midaortic syndrome (MAS) is a very rare vascular disorder characterized by segmental or diffuse narrowing of the distal thoracic and/or abdominal aorta, often accompanied by stenosis of the renal and visceral arteries. It accounts for fewer than 1% of cases of aortic coarctation and has an estimated incidence of about 1 per million people.¹ Despite its rarity, MAS is one of the most important causes of severe renovascular hypertension in children and adolescents, because the disease is progressive and can lead to irreversible end-organ damage if left untreated.²⁻⁵ The etiology of MAS is heterogeneous.² Congenital disease predominates in children, whereas acquired causes such as Takayasu arteritis are less common.⁶ MAS is also associated with several genetic syndromes, including Williams

syndrome, Noonan syndrome, neurofibromatosis type 1 and Alagille syndrome, and pathogenic variants in ELN, JAG1, GATA6, RNF213 and NF1 have recently been identified, broadening the genetic spectrum of the disease.⁷⁻⁹

The clinical presentation is variable. Hypertension, which is often resistant to medical therapy, is the most common finding,⁵ but patients may also present with heart failure, lower-extremity claudication, abdominal pain, growth retardation or renal dysfunction, or may remain asymptomatic until hypertension is detected incidentally.⁵ This variability frequently delays the diagnosis, particularly in young children.^{5,10} Long-standing hypertension may lead to left ventricular hypertrophy, ventricular dysfunction, vascular remodeling and chronic kidney injury, and renal artery involvement, which is present in most pediatric patients, is a major determinant of

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disease severity that usually requires multidisciplinary management.^{3,5,10,11} Computed tomography and magnetic resonance angiography are now the principal diagnostic modalities, as they define the extent of aortic narrowing, branch-vessel involvement and collateral circulation, while catheter angiography is largely reserved for intervention or hemodynamic assessment.^{3,5,11}

Management remains challenging because the disease is rare and prospective data are lacking. Medical therapy controls blood pressure but is rarely definitive; endovascular procedures may provide temporary benefit in selected patients, although restenosis is common in growing children; and surgical reconstruction is preferred for extensive disease once patient size permits a durable repair, with reintervention still required in some patients.^{2-5,10,12,13} Consequently, most available evidence comes from small single-center series and case reports, and the natural history, associated cardiovascular abnormalities, optimal timing of intervention and long-term outcomes of pediatric and young adults MAS remain incompletely defined.^{2-5,10,12,13}

In the present study, we report the clinical characteristics, cardiovascular findings, imaging features, medical management, and follow-up outcomes of five patients with radiologically confirmed midaortic syndrome managed at a tertiary referral center.

Materials and Methods

Study Design and Patient Population: This retrospective observational case series was conducted between 2010 and 2026 in Marmara Faculty of Medicine, Department of Pediatric Cardiology. The ethics committee at Marmara University approved this retrospective analysis (date/no:09.2026/26-0746). Written informed consent was obtained from the patients and/or their parents. Medical records of patients diagnosed with midaortic syndrome (MAS) were retrospectively reviewed. All patients with radiologically confirmed MAS who were followed at our institution between 2010 and 2026 were eligible for inclusion. The diagnosis of MAS was established by computed tomography angiography (CTA) or, in one patient, magnetic resonance angiography, demonstrating segmental or diffuse narrowing of the distal thoracic and/or abdominal aorta with or without involvement of the renal or visceral arteries. Patients without definitive radiological confirmation or with incomplete clinical records were excluded. A total of five

patients met the inclusion criteria and constituted the study cohort.

Data Collection: Clinical data were retrieved from the institutional electronic medical record system. Demographic characteristics, age at diagnosis, sex, presenting symptoms, associated syndromic conditions, congenital cardiovascular abnormalities, physical examination findings, laboratory investigations, electrocardiographic findings, echocardiographic measurements, cross-sectional imaging findings, medical treatment, previous cardiovascular interventions, and follow-up outcomes were collected. Hypertension was diagnosed and treated according to current guidelines.¹⁴

Follow-up data included antihypertensive therapy, serial echocardiographic findings, left ventricular ejection fraction, development of cardiac complications, additional cardiovascular interventions, and survival status at the last outpatient visit.

Cardiac Evaluation: Transthoracic echocardiography was performed as a part of routine clinical evaluation according to institutional practice. Left ventricular systolic function, structural cardiac abnormalities, ventricular hypertrophy, ventricular morphology, valvular abnormalities, and pericardial effusion were evaluated using standard pediatric echocardiographic measurements.

Routine echocardiography was performed on admission and every 3, 6 or 12 months thereafter. Additional echocardiography was performed in some cases if needed and the frequency was decided according to the severity of the disease. Echocardiography was performed by experienced pediatric cardiologists using Philips iE33 and EPIQ 7C cardiovascular ultrasound machines (Philips Healthcare®). Standard echocardiographic measurements were obtained according to the American Society of Echocardiography guidelines.¹⁵ All echocardiographic parameters were calculated using the published reference data.¹⁶⁻¹⁸

Standard 12-lead electrocardiograms obtained at the initial evaluation were reviewed. Heart rhythm, heart rate, corrected QT interval (QTc), conduction abnormalities, and ST-T wave changes were recorded. Cross-sectional angiographic imaging (CTA in four patients and magnetic resonance angiography in one) was reviewed to confirm the diagnosis of MAS and to evaluate the anatomical location and extent of aortic narrowing as well as associated vascular abnormalities.

Cardiac magnetic resonance imaging was not routinely performed in this cohort.

Statistical Analysis: Because of the small sample size, only descriptive statistical analyses were performed. Continuous variables are presented as median (minimum–maximum) and categorical variables as frequencies and percentages. Statistical analyses were performed with the SPSS 20.0 statistical package (IBM Corp., Armonk, NY, USA). No comparative statistical analyses or hypothesis testing were performed because of the limited number of patients.

Results

A total of five patients with radiologically confirmed diagnosis of midaortic syndrome (MAS) were included in this study. The cohort consisted of four females (80%) and one male (20%). The median age at diagnosis was 108 months (range:37-288). The median follow-up duration was 96 months (range:14-120). Three patients had an associated genetic syndrome (Williams, Noonan and Turner syndromes), and four had concomitant congenital cardiovascular abnormalities. Hypertension was present at diagnosis in four patients (80%), and the remaining patient developed hypertension during follow-up. Each patient is described below; their clinical characteristics are summarized in Table 1 and their antihypertensive treatment in Table 2.

Case 1. A girl with epilepsy who had been followed for coarctation of the aorta, and who had undergone balloon angioplasty of the coarctation on two occasions, was diagnosed with MAS at the age of 9 years. She was asymptomatic and physical examination was unremarkable, but she was hypertensive at presentation. Electrocardiography was normal. Echocardiography showed preserved left ventricular systolic function (left ventricular ejection fraction [LVEF] 70%) without hypertrophy, valvular regurgitation or pericardial effusion. Computed tomography angiography (CTA), performed during follow-up of the coarctation, incidentally revealed diffuse narrowing of the abdominal aorta consistent with MAS, with only minimal residual narrowing (8 mm) of the distal arch adjacent to the left subclavian artery. Hypertension was controlled with ramipril and hydrochlorothiazide, spironolactone and amlodipine. After 4 years of follow-up, she remains asymptomatic with controlled blood pressure, an LVEF of 78% and no left ventricular hypertrophy.

Case 2. A 3-year-old girl with Williams syndrome was referred for cardiac evaluation during the work-up of delayed walking and was found to be hypertensive; physical examination was otherwise unremarkable. Electrocardiography was normal. Echocardiography showed hyperdynamic left ventricular function (LVEF 80%) with mild left ventricular hypertrophy and mild mitral regurgitation. CTA demonstrated diffuse narrowing of the infrarenal abdominal aorta and both iliac arteries (Figure 1). Because of severe hypertension she was hospitalized, including a short period in the intensive care unit, and blood pressure was controlled with carvedilol, amlodipine and spironolactone. At the last visit, 14 months after diagnosis, blood pressure was controlled, LVEF was 71%, mitral regurgitation had resolved and mild left ventricular hypertrophy persisted.

Case 3. A 24-year-old man with Noonan syndrome, known coarctation of the aorta and bronchiectasis presented with chest pain radiating to the left shoulder. He was normotensive, and physical examination showed only the syndromic features. Electrocardiography was normal. Echocardiography showed an LVEF of 67%, mild-to-moderate mitral regurgitation and increased flow velocity in the descending aorta, which prompted CTA. CTA showed a 40% narrowing of the proximal descending thoracic aorta consistent with coarctation, together with narrowing of the abdominal aorta consistent with MAS. A diagnostic cardiac catheterization had been performed before the diagnosis of MAS. No intervention was performed. He developed hypertension 82 months (6.8 years) after diagnosis, which has been controlled with atenolol monotherapy. After 8 years of follow-up, LVEF is 65%, mitral regurgitation is trivial, and no arrhythmia or cardiac event has occurred.

Case 4. A 12-year-old girl with Turner syndrome was referred from the pediatric endocrinology clinic because of a newly noted 3/6 systolic murmur. She was hypertensive and asymptomatic. Electrocardiography was normal. Echocardiography showed an LVEF of 73% without hypertrophy or valvular regurgitation, and findings consistent with coarctation of the aorta. Diagnostic catheter angiography confirmed the coarctation and demonstrated progressive tapering of the aorta from the midaortic level downwards, consistent with MAS: the aortic diameter was 17.7 mm at the midaortic level and decreased distally, and the femoral arteries were also narrowed, more markedly on the right (minimal diameter 3.5 mm

Table 1: Demographic Characteristics, Associated Cardiac and Genetic Diagnoses, Mode of Diagnosis, Cardiac Procedures Performed Before the Diagnosis of Midaortic Syndrome, Echocardiographic Findings and Follow-Up Outcomes of The Five Patients with Midaortic Syndrome

Patient	Sex	Age at diagnosis (months)	Associated cardiac diseases	Syndrome	LVEF (%)	LV hypertrophy on echocardiography	Follow-up (months)	Mode of diagnosis	of Cardiac procedures before MAS diagnosis
1	Female	108	Coarctation of aorta	None	70	No	48	Incidental, CTA during follow-up	on Balloon angioplasty of CoA (×2)
2	Female	37	None	Williams syndrome	80	Yes	14	Work-up of hypertension	None
3	Male	288	Coarctation of aorta (mild)	Noonan	67	No	96	Increased descending aortic flow on echocardiography performed for chest pain	Diagnostic catheterization
4	Female	144	Coarctation of aorta (mild), bicuspid aortic valve, aortic stenosis (mild)	Turner syndrome	73	No	120	Work-up of hypertension and murmur on physical examination	Diagnostic catheterization
5	Female	84	Bicuspid aortic valve, mild aortic stenosis (valvular), moderate supravalvular pulmonary stenosis	None	67	No	102	Work-up of hypertension	None

CoA: coarctation of the aorta; CTA: computed tomography angiography; LV: left ventricular; LVEF: left ventricular ejection fraction at diagnosis; MAS: midaortic syndrome. Age at diagnosis and follow-up duration are given in months. All patients were alive at the last follow-up visit

on the right and 4.3 mm on the left). This patient was previously reported by our institution as a case of MAS in Turner syndrome.¹⁹ No intervention was performed. Hypertension was controlled with propranolol. Over 10 years of follow-up, left ventricular systolic function has remained preserved (LVEF 61% at the last visit) without hypertrophy, arrhythmia or cardiac events.

Case 5. A 7-year-old girl with ichthyosis presented with hypertension, growth retardation (height and weight below the 3rd percentile) and easy

fatigability. Physical examination revealed a 2–3/6 systolic murmur. Electrocardiography was normal. Echocardiography showed an LVEF of 67%, a bicuspid aortic valve with mild aortic stenosis and mild supravalvular pulmonary stenosis, without hypertrophy or regurgitation. Magnetic resonance angiography demonstrated a diffusely hypoplastic abdominal aorta with a maximal diameter of 6.5 mm, establishing the diagnosis (Figure 2). Hypertension was resistant and required four agents (ramipril, atenolol, nifedipine and doxazosin), with which blood pressure was

Table 2: Hypertension at Admission, Resistant Hypertension and Antihypertensive Drugs in the Five Patients with Midaortic Syndrome

Patient	Hypertension at admission	Resistant Hypertension	Hypertension treatment
1	Yes	Yes	ACEI and thiazide diuretic (ramipril and hydrochlorothiazide), MRA (spironolactone), CCB (amlodipine)
2	Yes	Yes	Beta-blocker (carvedilol), CCB (amlodipine), MRA (spironolactone)
3	No	No	Beta-blocker (atenolol)
4	Yes	No	Beta-blocker (propranolol)
5	Yes	Yes	ACEI (ramipril), beta-blocker (atenolol), alpha-blocker (doxazosin), CCB (nifedipine)

ACEI: angiotensin-converting enzyme inhibitor; CCB: calcium channel blocker; MRA: mineralocorticoid receptor antagonist. Resistant hypertension was defined as blood pressure above target despite three antihypertensive agents of different classes including a diuretic, or blood pressure controlled with four or more agents

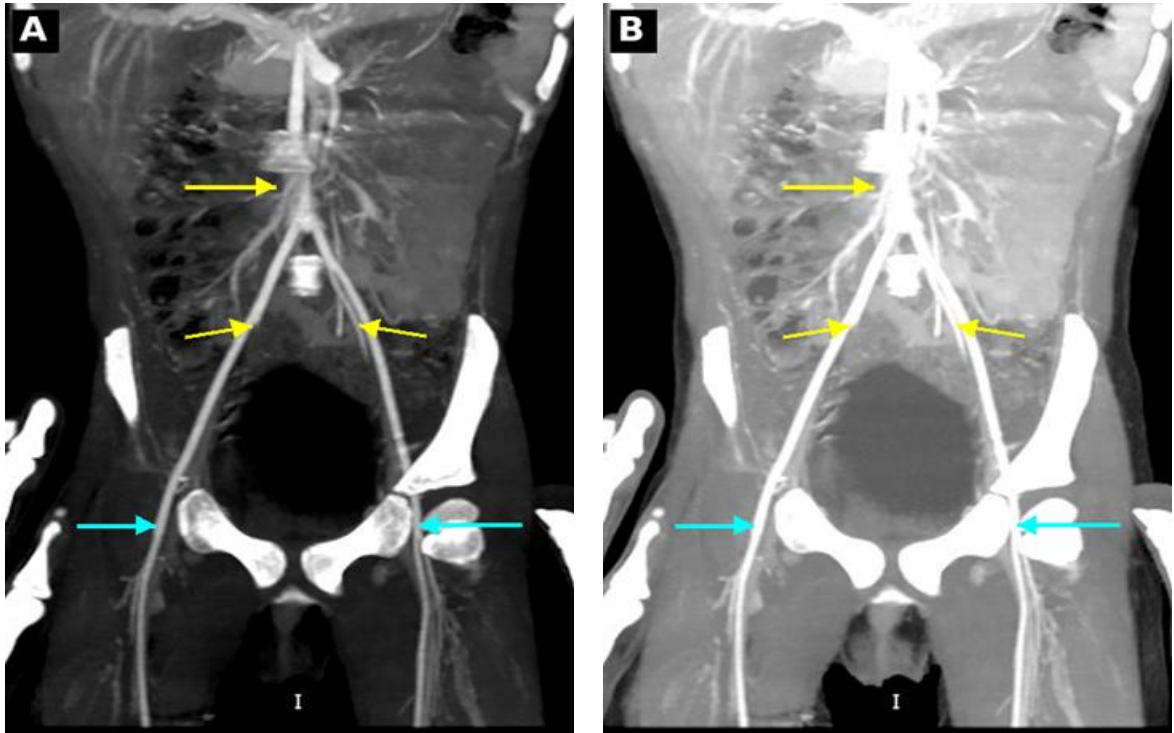


Fig. 1. Contrast-enhanced computed tomography angiography of a 3-year-old girl. The infrarenal abdominal aorta (single yellow arrow), common iliac arteries (paired yellow arrows) and the external iliac and common femoral arteries (blue arrows) are diffusely narrowed

eventually controlled. After 8 years of follow-up, she is clinically stable; the last echocardiogram showed an LVEF of 77%, minimal mitral regurgitation, mild aortic stenosis and moderate supra-valvular pulmonary stenosis, and no left ventricular hypertrophy. Renal denervation is planned but has not yet been performed.

No renal artery involvement was detected on abdominal CTA in any patient. No patient had heart failure at diagnosis, and baseline electrocardiography was normal in all. Left ventricular systolic function was preserved in every patient both at diagnosis and at the last follow-up; mild left ventricular hypertrophy was

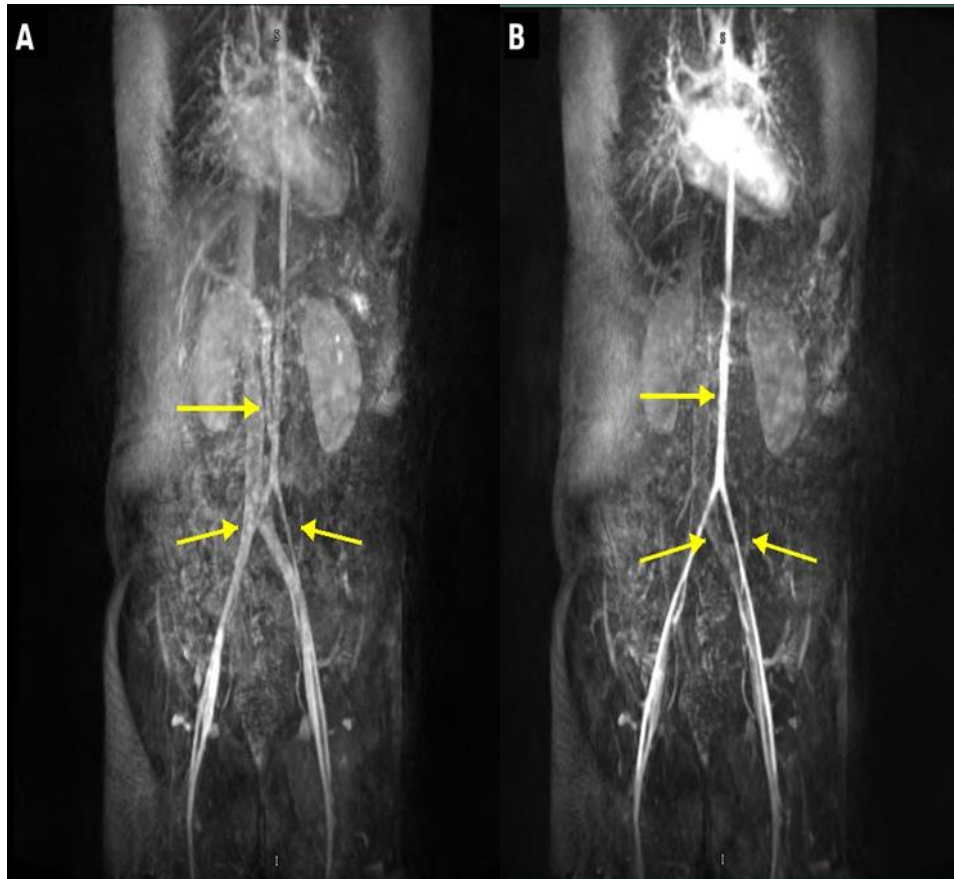


Fig. 2. Contrast-enhanced magnetic resonance angiography of a 7-year-old girl. The infrarenal abdominal aorta shows diffuse, long-segment narrowing (single arrows) extending to the aortic bifurcation, with narrowing of both common iliac arteries (paired arrows)

present in one patient, and no patient had hemodynamically significant valvular regurgitation or pericardial effusion. Four patients were hypertensive at diagnosis, and one patient (Case 3) developed hypertension 82 months after diagnosis; thus, all five patients eventually required antihypertensive therapy, and three fulfilled the criteria for resistant hypertension. Treatment was tailored to the individual needs of each patient, and blood pressure was controlled with medical therapy in all. No drug-related adverse effects occurred, and no antihypertensive agent had to be discontinued or replaced during follow-up. Before the diagnosis of MAS, three patients had undergone cardiac catheterization: the procedure was purely diagnostic in two (Cases 3 and 4), whereas one patient (Case 1) had undergone balloon angioplasty of coarctation of the aorta twice. No patient underwent stent implantation, surgical reconstruction or bypass surgery for MAS during the study period. Screening for intracranial aneurysms was not performed in any patient. During follow-up no patient experienced sustained arrhythmia or required a permanent pacemaker, implantable

cardioverter-defibrillator, ventricular assist device or heart transplantation, and no deaths occurred.

Discussion

Midaortic syndrome (MAS) is a rare but clinically important cause of pediatric renovascular hypertension. Because it is so uncommon, current knowledge comes largely from retrospective cohorts, systematic reviews and single-center experience, and standardized diagnostic and therapeutic strategies are lacking.^{3,4} In this series of five patients with radiologically confirmed MAS, the disease was markedly heterogeneous in its presentation, associated cardiovascular abnormalities, treatment and outcome. Although small, our cohort reflects the clinical complexity described in earlier pediatric series and adds tertiary-center data to the literature on this condition.^{3,8,13}

A principal observation was the variability of the initial presentation. Although hypertension is the hallmark of MAS, not all our patients were diagnosed because of severe or refractory

hypertension; in several, the diagnosis was made during evaluation of congenital heart disease or an associated syndrome. Brunet-Garcia et al.⁸ similarly showed that pediatric MAS may remain clinically silent until hypertension is detected incidentally or vascular involvement causes target-organ damage, and Musajee et al.⁴ emphasized that symptom onset depends on the severity and distribution of the vascular lesions, which often delays diagnosis in childhood.

Associated congenital cardiovascular abnormalities and genetic syndromes were also common: three patients had a recognized genetic disorder, and several others had additional congenital cardiac anomalies. This supports the concept that MAS is often part of a broader developmental arteriopathy rather than an isolated vascular lesion. Recent reviews have highlighted its association with Williams syndrome, neurofibromatosis type 1, Alagille syndrome, Noonan syndrome and other disorders of vascular development, and suggest that careful genetic and systemic evaluation be considered in affected children.^{3,4,20}

A further strength of this study is the long-term follow-up of a patient with Turner syndrome whom our institution previously reported as a single case of MAS presenting with systemic hypertension.¹⁹ Her inclusion extends that isolated case report into longitudinal follow-up and provides additional information on the natural history, cardiovascular evolution and long-term outcome of MAS in Turner syndrome.¹⁹

Despite the anatomical severity of the vascular involvement and the presence of hypertension in all patients at some point during the disease course, left ventricular systolic function remained preserved throughout follow-up, and no patient required mechanical circulatory support or transplantation or died. Similar favorable cardiac outcomes have been reported in contemporary pediatric cohorts when blood pressure was controlled before advanced end-organ damage developed.^{3,8}

Management of MAS requires an individualized, multidisciplinary approach in which the priority is control of hypertension and prevention of hypertensive end-organ injury.⁴ No patient in our study underwent invasive treatment specifically for MAS. Antihypertensive therapy is therefore central to initial management and may achieve satisfactory control in selected patients, although several agents are often needed, particularly when renal artery involvement is significant.^{4,21} Invasive treatment should be considered when

hypertension remains uncontrolled despite optimized medical therapy, when drug-related adverse effects become limiting, or when significant aortic or renovascular obstruction is causing progressive end-organ damage.⁴ Treatment decisions should therefore rest not on the anatomical severity of the narrowing alone, but on blood pressure control, renal and other end-organ function, vascular anatomy, etiology, and the age and growth potential of the child.^{4,21}

When medical therapy is insufficient, endovascular intervention can relieve hemodynamically significant aortic or renal arterial obstruction early, particularly in young children in whom definitive reconstruction is best deferred until further somatic growth.^{4,21} Balloon angioplasty is the principal catheter-based option, whereas stenting is reserved for selected aortic lesions or as a salvage procedure after complications such as flow-limiting dissection or thrombosis; restenosis, vascular injury, aneurysm formation and the need for repeated procedures remain important limitations in growing children.^{4,21} In the 30-year series reported by Porras et al.²¹, freedom from reintervention after percutaneous treatment was 58% at 1 year and 33% at 5 years, emphasizing that catheter-based therapy frequently represents part of a staged treatment strategy rather than a definitive solution.²¹ Definitive surgery, most often aorto-aortic bypass or patch aortoplasty with renal or visceral artery reconstruction when needed, provides more durable correction in appropriately selected patients.^{4,5,21} Consistent with this, a systematic review and meta-analysis found persistent hypertension to be more frequent after endovascular treatment than after surgery among juvenile patients, although the available evidence remains predominantly observational and treatment selection is strongly influenced by anatomy, age, and disease severity.²²

Although three of our patients needed three or more antihypertensive agents, none underwent endovascular or surgical treatment of the mid-aortic segment. This decision was made by a multidisciplinary team (pediatric cardiology, pediatric nephrology and cardiovascular surgery) for two reasons: the narrowing was diffuse and long-segment in all patients, without a discrete stenosis suitable for angioplasty or stenting; and blood pressure was ultimately controlled with medical therapy, renal function remained normal, and there was no evidence of progressive target-organ damage or of visceral or lower-limb ischemia. In one patient renal denervation has been planned, but she is currently managed

medically. This approach is consistent with current evidence, which reserves endovascular treatment for focal lesions or as a bridge to surgery and favors definitive surgical reconstruction once patient size permits a durable repair.^{4,5,10} All patients remain under close surveillance, and intervention will be reconsidered if hypertension becomes uncontrollable, renal function deteriorates or ischemic symptoms develop.

Long-standing hypertension and aortic arteriopathies such as coarctation are recognized risk factors for intracranial aneurysm, and an increased prevalence has been reported in patients with coarctation of the aorta.²³ Screening for intracranial aneurysms was not performed in our cohort, which we acknowledge as a limitation. Although neither pediatric hypertension guidelines nor the MAS literature gives specific recommendations, we believe that non-invasive screening with magnetic resonance angiography should be considered in patients with MAS, particularly those with long-standing or resistant hypertension, coexisting coarctation or a syndromic vasculopathy, and at transition to adult care.

Although our study included only five patients, its findings reinforce several important concepts emerging from contemporary MAS literature in children and young adults. MAS should be suspected in children presenting with hypertension, unexplained cardiovascular abnormalities, or syndromic disorders associated with vascular disease. Comprehensive imaging is essential for defining disease extent and planning individualized management. Long-term multidisciplinary follow-up remains mandatory because recurrent hypertension, progressive vascular disease, and the need for reintervention may occur even after apparently successful initial treatment.

Limitation: This study is limited by its retrospective, single-center design and small sample size, which precluded comparative statistical analyses and limits the generalizability of the findings. In addition, heterogeneous clinical characteristics and variable follow-up durations may restrict the assessment of long-term outcomes.

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