

## Renovascular hypertension secondary to renal artery compression by diaphragmatic crura

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### ABSTRACT

Median arcuate ligament syndrome is the result of celiac axis compression by the diaphragmatic crura. Although the celiac artery is the most common vessel to have compression, the renal arteries may also rarely be compressed by the crural fibers of the diaphragm, which may cause secondary hypertension. We present two cases of renovascular hypertension secondary to renal artery compression by the diaphragmatic crura. The first patient was treated with open decompression and wide resection of the crural fibers, and the second patient was decompressed laparoscopically. Neither case required renal artery reconstruction. Antihypertensives were discontinued in both patients postoperatively. (*J Vasc Surg Cases and Innovative Techniques* 2020;6:239-42.)

**Keywords:** Renal artery; Median arcuate ligament; Renovascular hypertension; Laparoscopic; Crura

Median arcuate ligament (MAL) compression of the celiac axis is a well-documented phenomenon, with up to 24% of the population having this anatomic finding.<sup>1</sup> However, <1% of patients are symptomatic with MAL syndrome.<sup>1</sup> Rarely, the renal arteries are compressed by the crural fibers of the diaphragm, resulting in secondary hypertension.<sup>2-4</sup> Given the rarity with no standard treatment, we present two cases of renovascular hypertension secondary to diaphragmatic crura compression of the renal arteries with a review of the literature. Both patients consented to publication.

### CASE REPORTS

#### Case 1

A 20-year-old man presented with shortness of breath in the setting of a recent upper respiratory infection. A computed tomography (CT) pulmonary embolism protocol was negative for pulmonary embolism but demonstrated a left renal artery (LRA) kink with associated stenosis. He was hypertensive with systolic pressures of 190 mm Hg. Medical management was suboptimal with three agents. Laboratory evaluation was remarkable for renal insufficiency (creatinine concentration progression from 1.4 to 1.7 mg/dL). A mercaptoacetyltriglycine



**Fig 1.** Angiographic image of the left renal artery (LRA) demonstrating proximal stenosis with caudal displacement of the artery, consistent with extrinsic compression (performed at the referring institution).

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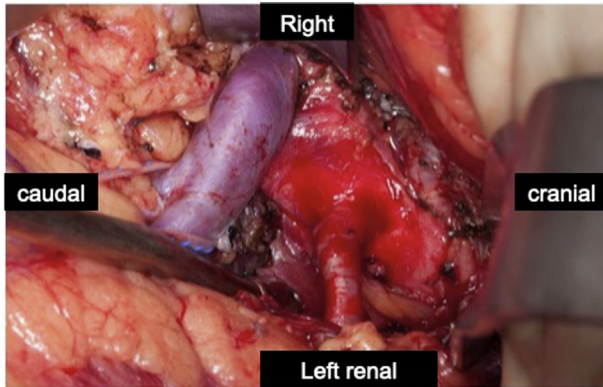
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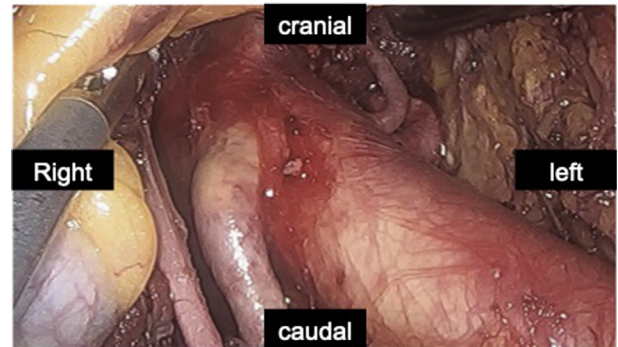
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scan revealed 82% right kidney function and 18% left kidney function. He underwent unsuccessful transfemoral renal stenting locally (Fig 1) and was referred to our institution for evaluation approximately 3 months after initial presentation.

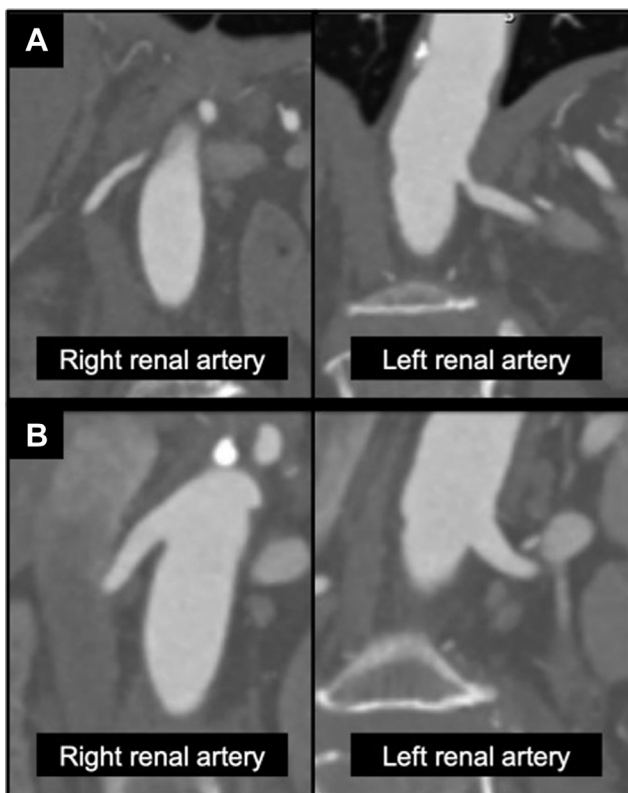
On examination, there were no abdominal bruits, and pulses were palpable in all extremities. Repeated angioplasty and stenting through a transbrachial approach was attempted because of the downgoing orientation of the LRA origin. Despite successful prolonged dilation, there was significant arterial recoil and continued kinking. Extrinsic compression was therefore suspected. The LRA had a high origin at T12 (above the superior mesenteric artery) and adjacent MAL compression of the artery



**Fig 2.** Intraoperative photograph of the left renal artery (LRA) after wide resection of the diaphragmatic crura.



**Fig 4.** Intraoperative photograph of laparoscopic median arcuate ligament (MAL) release.



**Fig 3.** Computed tomography angiography (CTA) demonstrating compression of the bilateral renal arteries by diaphragmatic crura during inspiration (A) and expiration (B).

on CT. We therefore planned for open decompression and possible reconstruction.

**Operative technique.** Through a left subcostal incision, a left retroperitoneal exposure was performed to isolate the left kidney, artery, and vein. The distal LRA was diminutive. The left crural fibers traversed both anterior and posterior to the LRA, causing a scissor-like compression. The muscle was widely resected, freeing the LRA in its entirety (Fig 2). The resection was

carried cephalad to the base of the superior mesenteric artery and celiac axis. Although the distal LRA appeared diminutive compared with the proximal vessel, this normalized with topical papaverine. Intraoperative duplex ultrasound confirmed patency of the LRA, without stenosis.

His postoperative course was unremarkable, and he remained off antihypertensive medication. He was discharged on postoperative day 6. At 17-month follow-up, he continued to be normotensive without medication. Duplex ultrasound confirmed a widely patent LRA, and the left kidney measured 11.5 cm compared with 9 cm preoperatively.

#### Case 2

A 65-year-old woman presented with poorly controlled hypertension with three medications. She had a recent hypertensive emergency, with systolic blood pressures of 230 mm Hg. Diagnostic evaluation included an ultrasound scan demonstrating renal artery stenoses that prompted subsequent CT angiography (CTA), which demonstrated noncalcified stenoses of the renal origins with overlying diaphragmatic crura. We therefore performed CTA with respirophasic maneuvers that demonstrated severe compression by crural fibers of bilateral renal arteries during inspiration, with complete resolution during expiration (Fig 3).

Examination was unremarkable, with no abdominal bruits and palpable pulses in all extremities. Laboratory evaluation findings were normal, with a creatinine concentration of 0.5 mg/dL. To correct her poorly controlled renovascular hypertension, we planned for operative intervention. After discussion of both open and laparoscopic options, she elected for a minimally invasive approach.

**Operative technique.** Under general endotracheal anesthesia, pneumoperitoneum was established and a laparoscopic Kocher maneuver was performed. The anterior aspect of the inferior vena cava and the infrarenal aorta were exposed, and the right renal artery (RRA) was identified. The proximal 5 cm of the RRA was cleared of surrounding crural fibers, some of which were fibrotic. The artery was nonfibrotic and normal caliber. Resection of the crura was then extended several centimeters cephalad and caudal to the RRA, freeing it circumferentially. The LRA and accessory LRA were identified and freed, with a

**Table.** Reports of renovascular hypertension secondary to renal artery compression by median arcuate ligament (MAL): Patient demographics, treatment modality, and outcomes, including the current report

Author	Year	Age, years	Sex	Presentation	Laterality	Treatment	Reconstruction	Resolution
d'Abreu <sup>6</sup>	1962	–	–	–	–	–	–	–
Villanueva <sup>7</sup>	1972	–	–	–	–	–	–	–
Silver <sup>4</sup>	1976	24	Male	Hypertension	Left	Open	No	Yes
Silver <sup>4</sup>	1976	4	Male	Hypertension	Left	Open	Vein patch <sup>a</sup>	Yes
Silver <sup>4</sup>	1976	33	Female	Hypertension	Right	Open	No	Yes
Spies <sup>8</sup>	1987	20	Male	Hypertension	Left	Open	Vein patch <sup>a</sup>	Yes
Clement <sup>9</sup>	1990	26	Female	Hypertension	Left	Open	No	Yes
Vahdat <sup>10</sup>	1991	23	–	Hypertension	Right	Open	Vein graft	Yes
Bacourt <sup>11</sup>	1992	21	Male	Hypertension	Left	Open	No	Yes
Baguet <sup>12</sup>	2003	74	Male	Hypertension	Left	Stent/open	Aortorenal bypass	Yes
Kopecky <sup>13</sup>	1997	–	Male	Hypertension	Bilateral	–	–	Yes
Thony <sup>5</sup>	2005	–	–	Hypertension	–	Stent	N/A	No
Thony <sup>5</sup>	2005	–	–	Hypertension	–	Stent	N/A	No
Thony <sup>b,5</sup>	2005	77	Female	Hypertension	–	N/A	N/A	N/A
Thony <sup>c,5</sup>	2005	–	Male	Hypertension	Right	N/A	N/A	N/A
Gaebel <sup>14</sup>	2009	19	Female	Hypertension	Left	Open	No	Yes
Singham <sup>d,15</sup>	2010	20	Female	Hypertension	Left	N/A	N/A	N/A
Sari <sup>3</sup>	2013	44	Female	Hypertension	Right	N/A	N/A	N/A
Mirza		20	Male	Hypertension	Left	Open	No	Yes
Arazińska <sup>16</sup>	2016	30	Female	Hypertension	Left	Medical <sup>e</sup>	N/A	N/A
Mirza	2018	65	Female	Hypertension	Bilateral	Laparoscopic	No	Yes

N/A, Not applicable.  
<sup>a</sup>Exploration without abnormality and vein patch closure.  
<sup>b</sup>Considered for open surgery, but it was not performed because of comorbidities.  
<sup>c</sup>Aborted because of intraoperative difficulty.  
<sup>d</sup>Patient not treated and lost to follow-up.  
<sup>e</sup>Because of patient's preference.

similar resection of the left-sided crura (Fig 4). Again, the LRAs were nonfibrotic and normal caliber.

The postoperative course was significant for orthostatic hypotension refractory to conservative management. Orthostasis resolved with midodrine therapy, and she was discharged on day 7. CTA demonstrated patent bilateral renal arteries with respiratory maneuvers. Her course was complicated by viral gastroenteritis with dehydration, resulting in readmission for recurrent orthostasis. Midodrine was discontinued on day 9, and after supportive care, she was discharged on postoperative day 15 off all antihypertensives.

## DISCUSSION

The literature describing renovascular hypertension secondary to compression by the diaphragm is sparse.<sup>3-5</sup> There is a wide range of age at presentation, and patients are invariably hypertensive (Table). Of the cases with information on laterality, there was compression of the LRA in 10, of the RRA in 4, and of bilateral renal arteries in 2 patients. On review of all CT and magnetic resonance scans during a 7-year period, Thony et al<sup>5</sup> reported the radiographic finding in 15 patients, with RRA accounting for

73% of cases. The renal artery origin was cephalad to the middle of the L1 vertebra in 40% and occurred in the setting of hypertrophic crura in 53% of patients.<sup>5</sup> Our first patient had a high origin of the LRA above the superior mesenteric artery at T12, and the second patient had both renal arteries originate between T12 and L1. Multiple reports, including ours, also describe hypertrophic and fibrotic bands of crura.<sup>14</sup> Arazińska et al<sup>16</sup> diagnosed secondary renovascular hypertension due to MAL compression of an LRA at T12 with hypertrophic muscle also kinking the distal descending thoracic aorta. Extrinsic compression should therefore be considered in unusually high renal origins with adjacent prominent MAL in the setting of renovascular hypertension.

As with other compressive conditions, treatment has focused on surgical decompression through a transperitoneal or retroperitoneal approach.<sup>4,5,14</sup> We chose a retroperitoneal approach in the first case to allow ample exposure of the entire LRA in the event that reconstruction was necessary. Currently, we offer both open and laparoscopic decompression for MAL syndrome in general. With laparoscopic release, recovery is usually faster,

and reconstruction can still be performed in a separate setting if indicated. This approach is advantageous in the morbidly obese, those with hostile abdomens, and those at high risk for wound complications. In our second case, the patient elected for a laparoscopic approach to avoid laparotomy, understanding that reconstruction may have been indicated after postoperative imaging evaluation. There are three reports of stenting of renal artery MAL compression. One was performed with a self-expanding stent that fractured at 2 years, requiring aortorenal bypass.<sup>11</sup> In two cases, balloon-expandable stents were used and were patent at 6 months, but long-term patency was not provided.<sup>4</sup> Our practice has been to avoid stenting for extrinsic compression syndromes in the absence of decompression as stent fracture is highly likely.

Reconstruction appears to be infrequently required. On literature review, two cases of vein patch were performed after inspection of the intima showed no irregularity.<sup>4,8</sup> There was one aortorenal saphenous vein bypass without MAL decompression. This was complicated by graft thrombosis, resulting in postoperative recognition of the extrinsic compression. Revision was performed with MAL release.<sup>10</sup> Most reports, including the two presented herein, describe normal appearance and caliber of renal arteries after decompression and therefore no reconstruction.<sup>4,9</sup>

## CONCLUSIONS

Renovascular hypertension secondary to renal artery compression by the diaphragm crura is rare. It should be considered in patients with high renal artery origins, with adjacent prominent crura. Workup requires astute judgment to ensure that respirophasic imaging is performed to assess dynamic compression. Laparoscopic decompression is a viable approach as reconstruction is not always mandated.

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