

SPECIAL CASE

# Renal Artery Stenosis in a Patient With Heart Disease

*How low-flow hemodynamics and irregular rhythm alter renal Doppler interpretation*

**CORE CHALLENGE** A globally damped, beat-to-beat variable Doppler examination can hide a focal renal artery stenosis. The sonographer must separate a cardiac inflow effect from a true renal vascular lesion.

| Case type         | Advanced vascular ultrasound teaching case                                     |
|-------------------|--------------------------------------------------------------------------------|
| Primary diagnosis | Hemodynamically significant right renal artery stenosis                        |
| Cardiac modifier  | Ischemic cardiomyopathy with reduced ejection fraction and atrial fibrillation |
| Audience          | Sonography students, educators, and practicing vascular sonographers           |

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This is a fictional, de-identified composite case created for education. It is not a substitute for local laboratory criteria, physician interpretation, or patient-specific medical care.

## 1. Learning objectives

- Apply a complete native renal arterial duplex protocol in a technically difficult cardiac patient.
- Recognize how low cardiac output and atrial fibrillation change peak systolic velocity, acceleration, and waveform reproducibility.
- Distinguish global cardiac waveform alteration from a focal stenotic jet and unilateral downstream tardus-parvus change.
- Use direct and indirect criteria as an integrated pattern rather than relying on a single threshold.
- Write a cautious, clinically useful preliminary impression and recommend correlation when data are discordant.

## 2. Clinical presentation

A 74-year-old man is referred for renal artery duplex because of resistant hypertension and progressive renal dysfunction. Blood pressure remains 176/92 mm Hg despite four antihypertensive agents. Serum creatinine increased from 1.3 to 2.0 mg/dL over six months. The patient reports two admissions for sudden pulmonary edema.

| Clinical factor        | Finding                                                                           |
|------------------------|-----------------------------------------------------------------------------------|
| Cardiovascular history | Prior myocardial infarction; ischemic cardiomyopathy; chronic atrial fibrillation |
| Recent echocardiogram  | LVEF 30%; low stroke volume; moderate calcific aortic stenosis                    |
| Renal risk profile     | Hypertension, diabetes, hyperlipidemia, former smoking                            |
| Medication context     | Beta blocker, loop diuretic, ACE inhibitor held after creatinine rise             |
| Indication             | Evaluate for renovascular hypertension / ischemic nephropathy                     |

**CLINICAL SUSPICION** The combination of resistant hypertension, worsening kidney function, diffuse atherosclerotic disease, and recurrent flash pulmonary edema increases concern for clinically important renovascular disease.

### 3. Examination protocol and technical approach

The patient fasted for eight hours. A low-frequency curvilinear transducer was used. The examination included grayscale renal survey, color Doppler, spectral Doppler of the aorta and bilateral main renal arteries, renal veins, and upper/lower pole intrarenal arteries. Angle correction was maintained at 60 degrees or less and aligned with the direction of flow.

- Record the cardiac rhythm and blood pressure at the time of the examination.
- Sample the aorta at or just above the renal artery origins before calculating a renal-aortic ratio (RAR).
- Interrogate each main renal artery at the origin/proximal, mid, and distal segments; add flank or coronal windows when needed.
- Measure intrarenal resistive index (RI) and acceleration time (AT) in upper and lower pole segmental or interlobar arteries.
- With atrial fibrillation, record multiple beats and avoid isolated short or long R-R intervals. Use several representative cycles and document variability.

### 4. Duplex findings

| Site                           | PSV (cm/s) | EDV (cm/s) | Key waveform observation                                        |
|--------------------------------|------------|------------|-----------------------------------------------------------------|
| Aorta at renal level           | 42         | 8          | Low-amplitude, irregular beat-to-beat output                    |
| Right renal artery - origin    | 92         | 18         | Suboptimal angle; mild color disturbance                        |
| Right renal artery - proximal  | 214-286    | 48-67      | Focal aliasing, spectral broadening; highest repeatable PSV 272 |
| Right renal artery - mid       | 118        | 27         | Post-stenotic turbulence                                        |
| Right renal artery - distal    | 71         | 19         | Damped downstream waveform                                      |
| Left renal artery - proximal   | 86         | 21         | No focal velocity step-up                                       |
| Left renal artery - mid/distal | 74 / 69    | 18 / 17    | Globally low velocities, preserved contour                      |

Calculated RAR using the highest repeatable right proximal PSV:  $272/42 = 6.5$ . This ratio is striking, but the denominator is abnormally low because of reduced systemic flow. Therefore, RAR may be exaggerated and must not be interpreted alone.

| Intrarenal sample | RI   | AT (ms) | Morphology                                |
|-------------------|------|---------|-------------------------------------------|
| Right upper pole  | 0.64 | 112     | Delayed systolic upstroke; rounded peak   |
| Right lower pole  | 0.62 | 106     | Tardus-parvus pattern                     |
| Left upper pole   | 0.70 | 58      | Low amplitude but brisk relative upstroke |
| Left lower pole   | 0.69 | 61      | Similar to left upper pole                |

Kidneys measure 9.2 cm on the right and 10.8 cm on the left. The right renal cortex is mildly thinned. Renal veins are patent.

## 5. The cardiac Doppler variation: what it changes

Ischemic cardiomyopathy lowers stroke volume and may reduce velocities throughout the aorta and renal arteries. Aortic stenosis may produce a delayed, rounded systemic systolic contour. Atrial fibrillation adds variable cycle length and variable ventricular filling, so successive waveforms may have different peak velocities and acceleration slopes.

| Cardiac effect                          | Possible renal duplex consequence                                                        | Interpretive response                                                                                                            |
|-----------------------------------------|------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------|
| Low cardiac output                      | Globally reduced PSV; severe focal stenosis may not reach a usual absolute PSV threshold | Look for a focal velocity ratio/step-up, color aliasing, broadening, post-stenotic disturbance, and unilateral downstream change |
| Atrial fibrillation                     | Beat-to-beat PSV, EDV, and acceleration variability                                      | Acquire multiple cycles; avoid post-pause beats; report a representative range and reproducible maximum                          |
| Aortic stenosis / prolonged LV ejection | Bilaterally delayed systolic upstroke that can mimic tardus-parvus                       | Compare both kidneys and the aortic waveform; true unilateral downstream delay supports ipsilateral RAS                          |

| Cardiac effect      | Possible renal duplex consequence | Interpretive response                           |
|---------------------|-----------------------------------|-------------------------------------------------|
| Very low aortic PSV | Artificially high RAR             | Do not use RAR as the sole diagnostic criterion |

**PATTERN THAT RESOLVES THE CASE** The patient has global cardiac damping, but only the right kidney shows a focal proximal jet, post-stenotic turbulence, smaller renal size, and clearly longer intrarenal acceleration time. The asymmetry supports superimposed right renal artery stenosis rather than cardiac disease alone.

## 6. Interpretation and diagnostic reasoning

### Direct evidence

- Focal, repeatable right proximal renal artery PSV elevation to approximately 272 cm/s.
- A marked local velocity step-up compared with adjacent segments and the contralateral artery.
- Color aliasing, spectral broadening, and post-stenotic turbulence at/after the lesion.

### Indirect evidence

- Unilateral right intrarenal tardus-parvus morphology with AT greater than 100 ms.
- Right-left kidney length asymmetry and mild right cortical thinning.
- Clinical phenotype compatible with atherosclerotic renovascular disease.

### Important limitation

The RAR is unreliable as a stand-alone measure because the aortic PSV is depressed. Absolute velocity thresholds may also be unstable across irregular beats. The diagnosis therefore rests on concordant focal and unilateral findings, not one number.

## 7. Final impression

**SUGGESTED REPORT** Focal hemodynamically significant stenosis of the proximal right renal artery is suspected, supported by repeatable focal velocity elevation, post-stenotic turbulence, and ipsilateral intrarenal tardus-parvus waveforms. No focal left renal artery stenosis is identified. Globally low and irregular arterial velocities reflect the patient's reduced cardiac output and atrial fibrillation; the renal-aortic ratio may be artificially elevated and should be interpreted cautiously. Correlation with clinical findings and confirmatory CTA or MRA may be considered if the result would change management.

The interpreting physician should integrate kidney function, contrast risk, treatment goals, local validated duplex criteria, and the full cardiovascular picture before recommending additional imaging or intervention.

## 8. Differential diagnosis

| Alternative                                    | Why considered                             | Why less favored here                                                                   |
|------------------------------------------------|--------------------------------------------|-----------------------------------------------------------------------------------------|
| Cardiac low-flow state alone                   | Explains globally low, variable velocities | Does not explain the focal right-sided jet, turbulence, or unilateral AT prolongation   |
| Aortic stenosis causing systemic tardus-parvus | Can delay both renal waveforms             | Delay is substantially greater on the right; left contour is relatively preserved       |
| Technical/angle artifact                       | Can falsely elevate PSV                    | Elevation was repeatable from two windows with concordant color and downstream findings |
| Fibromuscular dysplasia                        | Can cause RAS, often mid/distal            | Age, risk factors, and proximal ostial pattern favor atherosclerosis                    |
| Renal parenchymal disease                      | May elevate RI and reduce perfusion        | Does not produce a focal main renal artery jet                                          |

## 9. Sonographer pitfalls and quality checklist

- Do not call bilateral renal artery stenosis solely because both kidneys show a delayed upstroke in a patient with aortic stenosis or cardiomyopathy.
- Do not dismiss stenosis solely because PSV fails to exceed a conventional threshold in a low-output patient.
- Do not calculate RAR from an aortic sample affected by poor angle, ectasia, severe plaque, or an unusually low/high velocity without stating the limitation.
- Do not measure only the strongest post-pause beat in atrial fibrillation; document representative variability.
- Confirm a suspected lesion with multiple concordant findings and, when possible, more than one acoustic window.

## 10. ARDMS-style review questions

**1. Which finding most strongly separates right RAS from a global low-output cardiac effect?**

- A. Bilaterally low renal artery PSV
- B. Irregular spectral cycles
- C. Focal right proximal velocity step-up with ipsilateral prolonged AT
- D. Aortic PSV of 42 cm/s

**Answer: C**

**2. Why may the RAR be misleading in this case?**

- A. Renal EDV is too high
- B. The low aortic PSV makes the ratio artificially high
- C. Kidney length cannot be measured
- D. Atrial fibrillation always lowers RAR

**Answer: B**

**3. What is the best acquisition strategy in atrial fibrillation?**

- A. Measure the single highest beat
- B. Measure only the shortest R-R interval
- C. Record several representative cycles and document variability
- D. Avoid spectral Doppler

**Answer: C**

**4. Which bilateral finding would favor aortic valve/cardiac influence over unilateral RAS?**

- A. Similar delayed upstrokes in both kidneys without focal main-artery jets
- B. Right focal aliasing and spectral broadening
- C. Right renal atrophy
- D. Right post-stenotic turbulence

**Answer: A**

## 11. Teaching summary

**TAKE-HOME MESSAGE** Cardiac disease changes the baseline waveform; renal artery stenosis creates a focal and asymmetric disturbance. In complex patients, compare sides, compare segments, sample multiple beats, and require concordance among direct, indirect, morphologic, and clinical findings.

## References and professional guidance

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