LETTER TO THE EDITOR

Renal Infarct from Traumatic Thrombosis of Aberrant Renal Artery: An Under-recognized Vascular Injury in the Emergency Setting

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Dear Editor,

Traumatic renal artery thrombosis (TRAT) is an extremely rare complication of blunt trauma, seen in only 0.05% of over 945,000 trauma cases.[1] Aberrant renal artery thrombosis after trauma is extremely rare, with an in-depth literature review of the English language showing a single case report published previously.[2]

Left-sided injuries predominate (left-to-right ratio of 1.4:1 to 2:1) due to the left kidney's greater mobility and the sharper angle of the renal artery off the aorta.

TRAT may result from either sudden deceleration causing arterial stretch and intimal disruption or direct compression against the spine.[3]

Case Description

We report two cases of aberrant renal artery thrombosis, diagnosed prospectively on computed tomography (CT) angiography, done as part of the patients' trauma workup.

The first patient was a 40-year-old man with no relevant past medical history, who presented to the trauma center of our institute after sustaining a road traffic injury. He was hemodynamically stable (BP: 132/80 mm Hg; pulse: 88 bpm), alert, and had abdominal bruising with left-sided tenderness. No hematuria was observed. An extended focused assessment with sonography for trauma (eFAST) scan showed minimal free fluid in the right pararenal space. A biphasic contrast-enhanced CT revealed a nonenhancing, wedge-shaped infarct in the lower pole of the left kidney, with normal opacification of the main renal arteries. Further evaluation identified a thrombosed left aberrant renal artery (ARA) supplying the infarcted segment (Fig. 1). This was classified as an American Association for the Surgery of Trauma (AAST) grade IV renal trauma. Associated injuries included right-sided L1-L4 transverse process fractures, bilateral adrenal hematoma, rib fractures, and minimal hemoperitoneum. Urea and creatinine levels were normal on admission and at discharge (38 mg/dL and 0.36 mg/dL, respectively). After 1 year of follow-up, the patient had no fresh complaints, with normal renal function tests (urea = 28 mg/dL and creatinine = 0.5 mg/dL). Follow-up ultrasound showed normal kidney size (8.2 cm), with focal reduced echogenicity noted in the lower pole, likely secondary to previous vascular injury.

The second patient was a young woman with no relevant past medical history and an alleged history of fall from a height (~15 ft), following which she developed neck pain, and on examination, there was cervical tenderness. There was no hematuria. Glasgow Coma Scale (GCS) score was 15/15. The patient was unable to move the lower limbs, with 3/5 power in the upper limbs and 0/5 in the lower limbs. The abdomen was nontender, and no guarding was present. The patient was started on IV noradrenaline 8 mg in 500 mL normal saline at 40 mL/hour in view of nonfluid-responsive shock (BP = 80/55 mm Hg). eFAST was positive for free fluid in the abdomen. Subsequently, CT angiography of the abdomen was done (Fig. 2), which showed a devascularized lower pole with thrombosed aberrant right renal artery (AAST grade IV right renal injury), with an AAST grade III liver injury and minimal ascites. Baseline urea (17 mg/dL) and creatinine (0.58 mg/dL) were normal. The blood pressure rose to 110/76 mm Hg. Urology was consulted, and the patient was kept under conservative management, with monitoring of vitals, urine output, and close observation for development of hematuria. After 3 months of follow-up, the patient had mild residual bilateral leg spasticity and weakness, with difficulty in walking. The power in the upper limbs recovered to 4/5 and that in the lower limbs to 3/5. Repeat renal function tests (urea = 20 mg/dL and creatinine = 0.62 mg/dL) were within normal limits. The patient underwent follow-up ultrasound, which revealed normal kidney size (right kidney: 9 cm; left kidney: 9.2 cm), shape, and outline, with mild heterogeneity noted in the lower pole at the site of previous devascularization. No hydronephrosis or perinephric collection was seen.

Discussion

This case illustrates that ARA thrombosis can occur in isolation, without main renal artery involvement or hematuria, underscoring the value of multiphasic contrast-enhanced CT. Aberrant renal arteries are typically smaller and more mobile, making them more susceptible to intimal tears during deceleration injuries due

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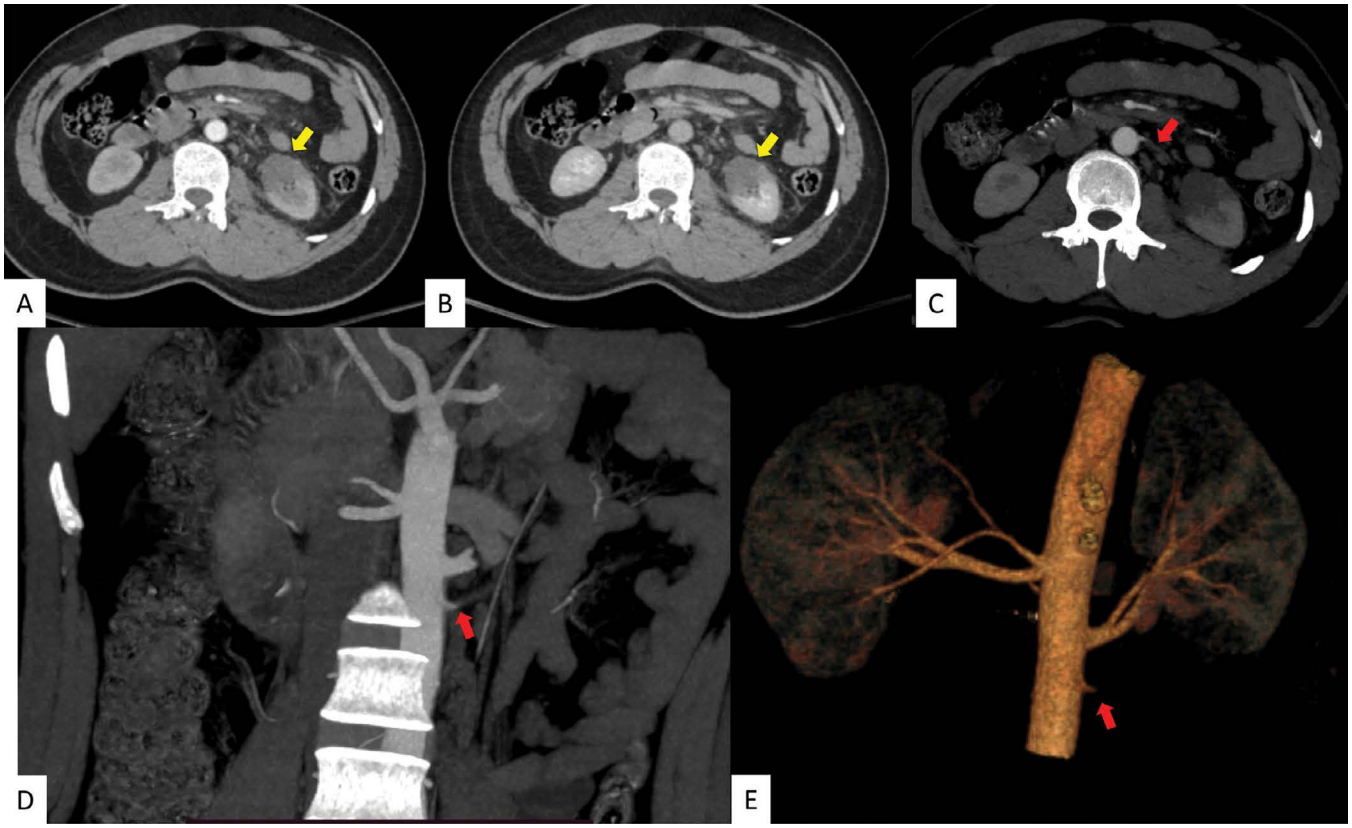
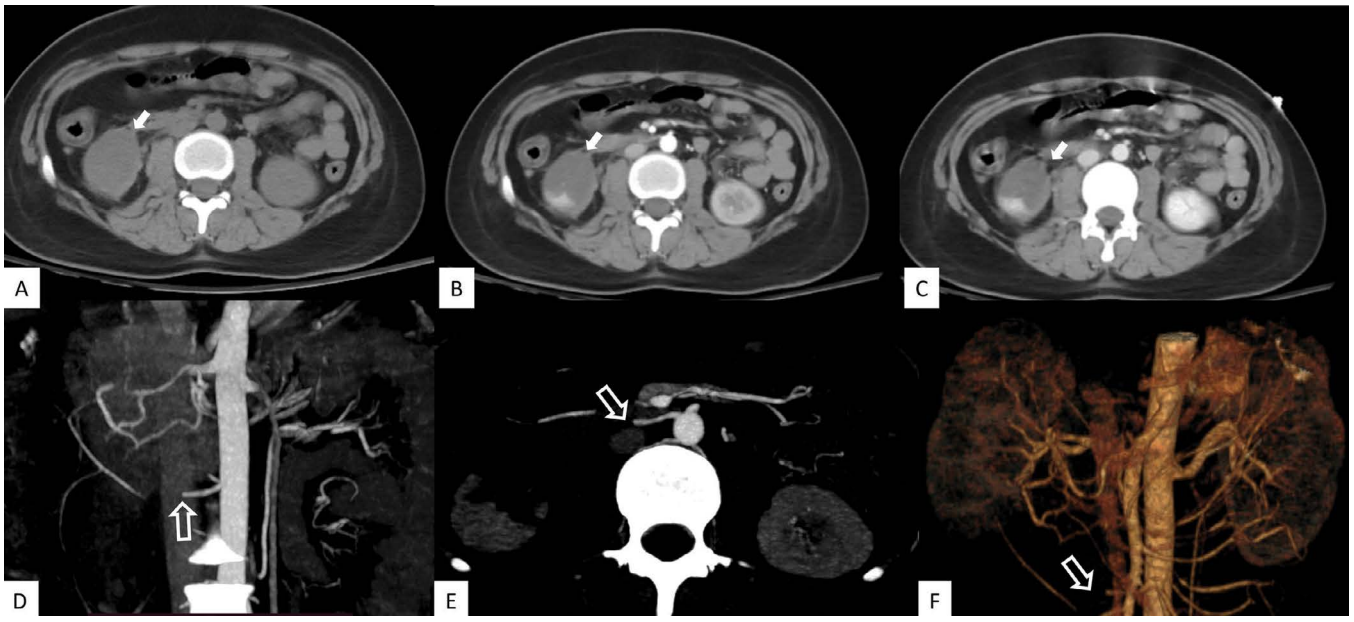


Figs 1A to E: Axial arterial (A) and venous phase (B) images showing a focal area of hypodensity in the lower pole of the left kidney without any change across phases, suggestive of renal infarct (yellow arrow); axial (C) and coronal (D) arterial-phase maximum-intensity-projection (MIP) images showing a proximally opacified left-sided aberrant renal artery with distal nonopacification seen in the form of hypodensity (red arrow); (E) three-dimensional volume-rendering technique showing the cutoff of the left-sided accessory renal artery (red arrow), with normal opacification of the bilateral main renal arteries



Figs 2A to F: Axial noncontrast (A), arterial (B), and venous-phase (C) images showing a focal area of hypodensity in the lower pole of the right kidney without any change across phases, suggestive of renal infarct (white arrow); coronal (D), axial (E), and arterial-phase maximum-intensity-projection (MIP) images showing a proximally opacified right-sided aberrant renal artery with distal nonopacification (white hollow arrow in D and E); (F) three-dimensional volume-rendering technique showing the cutoff of the right-sided accessory renal artery (white hollow arrow), with normal opacification of the bilateral main renal arteries

to shear stress. In ARA thrombosis, intervention (e.g., stenting) poses higher risk than benefit, as the infarcted segment is small, and anticoagulation could lead to further complications such as bleeding and hematoma formation.

In a pooled analysis of 54 cases of blunt renal vascular injury treated with percutaneous revascularization, technical success reached 88.9%, and clinical success (no new hypertension, stable creatinine, or preserved split renal function) was 75%, although failures still occurred.[4] By contrast, isolated accessory/segmental artery thrombosis usually supplies a small volume of renal parenchyma and is most often observed conservatively because procedural risks outweigh the expected benefit.

The primary goals of management become monitoring for stability while accepting the loss of the small infarcted renal segment, keeping the patient on follow-up with renal function tests and blood pressure monitoring, typically up to 1 year.[5] According to American Urological Association (AUA) guidelines, a repeat CT scan at 48 hours is advised for grade IV-V injuries managed nonoperatively to assess for evolving complications.[6]

The take-home message is that multiphasic CT is essential to detect isolated accessory renal artery thrombosis, and the emergency physician, in conjunction with the urologist, must act as the balancing force to weigh the risks versus benefits and guide the need for endovascular intervention (or the lack thereof) in such cases.

Recognition of TRAT is critical in emergency settings due to its nonspecific presentation and potential for misdiagnosis, particularly when involving accessory renal arteries, which are present in up to 30% of the population. Thrombosis of such aberrant arteries can result in segmental infarction, presenting with flank pain, hematuria, or no symptoms if the infarcted segment is small.[2] Routine laboratory or urinalysis findings may underestimate the injury, so high clinical suspicion is warranted in patients with acute flank pain, hematuria, or new-onset hypertension after trauma.[5]

A missed or delayed diagnosis almost guarantees the permanent loss of the kidney, converting a potentially salvageable organ into a nonfunctional, atrophic remnant that may later cause refractory hypertension.[5] Management is typically conservative in hemodynamically stable patients, with close monitoring and specialist referral for extensive infarction, uncontrolled hypertension, or complications. Although management is usually conservative, awareness of aberrant renal vascular anatomy and communication with urology and vascular surgery teams remain critical to prevent missed segmental devascularization, avoid inadvertent vascular injury during subsequent interventions, and ensure appropriate follow-up rather than unnecessary anticoagulation or invasive procedures. Reporting isolated aberrant renal artery thrombosis is clinically significant, as differentiating it from main renal artery occlusion directly influences management decisions, prevents misdiagnosis of segmental renal infarction, and justifies structured long-term surveillance for delayed hypertension or renal dysfunction.

This report is limited by its small sample size (two cases from one institution), restricting generalizability and precluding statistical analysis. The literature review was limited to English-language sources, potentially missing non-English data. Follow-up is incomplete for the second case, adding uncertainty to long-term outcomes.

The identification of aberrant renal artery thrombosis, even when managed conservatively, has important diagnostic, prognostic, and educational implications for emergency physicians evaluating blunt abdominal trauma.

Data Availability Statement

Anonymized case details are accessible upon reasonable request from the corresponding author.

Ethics Approval and Patient Consent to Participate

Institutional ethical approval was not taken, as no identifying information was revealed or collected during the preparation of the case. Patient consent was obtained for publication.

Acknowledgment

Nil.

Author Contributions

All authors contributed to the study conception and design. Material preparation, data collection, and analysis were performed by AaS and MS. Clinical input, management and follow up: AdS. The first draft of the manuscript was written by AaS. Manuscript editing and revision by MS and AdS. All authors read and approved the final manuscript.

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References

1. Sangthong B, Demetriades D, Martin M, et al. Management and hospital outcomes of blunt renal artery injuries: analysis of 517 patients from the National Trauma Data Bank. J Am Coll Surg 2006;203(5):612–617. DOI: 10.1016/j.jamcollsurg.2006.07.004
2. Basavanagowda PA, Prabhakar N, Prabhakar A, et al. Blunt trauma abdomen leading to bilateral renal segmental infarction: resolving the enigma. Indian J Nephrol 2023;33(6):476–477. DOI: 10.4103/ijn.ijn_274_23
3. Rossi UG, Cariati M. Blunt renal artery trauma: a therapeutic dilemma. Turk J Urol 2019;45(3):233–236. DOI: 10.5152/tud.2018.91145
4. Jahangiri Y, Ashwell Z, Farsad K. Percutaneous renal artery revascularization after prolonged ischemia secondary to blunt trauma: pooled cohort analysis. Diagn Interv Radiol 2017;23(5):371–378. DOI: 10.5152/dir.2017.16415
5. Xia X, Zhao G, Feng Q, et al. Traumatic renal artery thrombosis: a case report and literature review. Front Med (Lausanne) 2024;11:1480275. DOI: 10.3389/fmed.2024.1480275
6. Morey AF, Brandes S, Dugi DD 3rd, et al. Urotrauma: AUA guideline. J Urol 2014;192(2):327–335. DOI: 10.1016/j.juro.2014.05.004