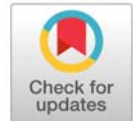


Case of Isolated Absence of the Right Renal Vein with Drainage via an Enlarged Cortical Vein into the Ipsilateral Gonadal Vein

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ABSTRACT

Background: Right-renal-vein agenesis is an exceptionally rare congenital anomaly. Absent the orthotopic vein, renal blood diverts through collaterals and, in rare cases, a single hypertrophied cortical vein draining into the ipsilateral gonadal vein. Unrecognised, it can mimic thrombosis or tumour and, if the solitary channel is injured, cause catastrophic haemorrhage.

Objective: To detail the presentation and multimodality imaging cues of right-renal-vein agenesis with exclusive cortical-to-gonadal drainage.

Case Presentation: A 43-year-old expatriate man in Dubai reported vague right-flank discomfort and occasional tea-coloured urine. Examination and renal biochemistry were normal; urinalysis showed microscopic haematuria. Multiphase contrast-enhanced CT revealed no right renal vein entering the inferior vena cava; perirenal channels merged into a hypertrophied cortical vein descending to a dilated right gonadal vein. Colour and spectral Doppler demonstrated low-velocity monophasic venous flow, excluding shunt or arteriovenous malformation. The contralateral renal vein and both renal arteries were unremarkable. With preserved renal function and mild symptoms, conservative management was adopted; at one-year review the patient remained asymptomatic and imaging findings were unchanged.

Conclusion: Identifying an absent orthotopic vein, dominant cortical collateral, preserved enhancement and venous Doppler waveform prevents misdiagnosis and averts inadvertent ligation during surgery. When blood pressure and renal function are stable, periodic imaging alone ensures a favourable prognosis.

Keywords: Renal, Agenesis, Gonadal, Collateral, Doppler, Venography.



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INTRODUCTION

The renal venous system emerges from an intricate sequence of embryological events that unfold between the sixth and tenth weeks of gestation. During this interval, three paired channels—the posterior cardinal, subcardinal and supracardinal veins—undergo selective anastomosis, regression and fusion to create the definitive inferior vena cava (IVC) and its tributaries [1]. The right renal vein ordinarily develops when the right supracardinal segment joins the subcardinal system and, moments later in developmental time, connects with the nascent IVC. Because this junction lacks the extensive anastomotic collar that encircles the aorta on the left, the right renal

vein is uniquely susceptible to perturbations in embryogenesis. Arrested maturation, premature involution or aberrant persistence of neighbouring channels can culminate in complete agenesis or extreme hypoplasia of the vessel. The true prevalence of isolated renal-vein agenesis is unknown, but large autopsy series place it below 0.05 %, with a clear right-sided preponderance [2].

In the absence of a direct renal-to-IVC conduit, the developing kidney experiences a transient rise in venous pressure that promotes enlargement of embryonic collateral pathways. Three decompressive routes are consistently described. The first traverses the periureteric venous plexus to the common iliac veins; the second

ascends via the lumbar and azygos–hemiazzygos system to the superior vena cava; and the third, least common, route recruits capsular or cortical veins that coalesce and drain inferiorly into the gonadal vein [3]. The calibre and efficiency of these collaterals dictate clinical presentation. Robust networks preserve near-normal renal haemodynamics, rendering the anomaly clinically silent and typically discovered incidentally on imaging. Inadequate collateralisation, however, may produce flank pain, microscopic or gross haematuria, varicocele or—rarely—renin-mediated hypertension due to sustained venous congestion [4].

Modern cross-sectional imaging has revolutionised the recognition of these variants. Multiphase contrast-enhanced CT and MR venography provide high-resolution maps of venous anatomy, whereas colour and spectral Doppler ultrasound can non-invasively confirm flow direction and waveform characteristics. These modalities are indispensable when evaluating potential living kidney donors, planning retroperitoneoscopic surgery or deploying IVC filters [5]. Misinterpretation as renal-vein thrombosis, tumoural invasion or postoperative ligation can prompt unnecessary anticoagulation, invasive biopsies or altered surgical strategy. More importantly, inadvertent ligation of a solitary collateral during nephrectomy or gonadal-vein surgery may precipitate catastrophic haemorrhage or renal venous infarction [6].

Although scattered case reports describe renal outflow through periureteric or azygos collaterals, exclusive drainage via a single enlarged cortical vein emptying into the ipsilateral gonadal vein remains extraordinarily rare. Fewer than ten such cases have been published—most in the radiological literature, with limited clinicopathological correlation [7]. This gap underscores the need for detailed descriptions that integrate embryological insight, multimodality imaging and clinical decision-making. Against this backdrop, we report the case of a 43-year-old expatriate man living in Dubai with right-renal-vein agenesis whose entire renal parenchyma drains through a dominant cortical vein terminating in the right gonadal vein. We emphasise the embryological basis, characteristic imaging findings and practical implications for surgeons and interventional radiologists [8].

CASE PRESENTATION: A 43-year-old expatriate man living in Dubai presented to the Dubai Health Authority (DHA) outpatient clinic with a six-month history of an intermittent, dull ache in the right flank. The discomfort was never severe enough to limit activity and was occasionally accompanied by transient tea-coloured urine, but he denied fever, dysuria, weight loss, nausea or lower-limb swelling. He reported no trauma, urolithiasis or family history of renal or vascular anomalies and had no cardiovascular risk factors, nor did he use tobacco or alcohol.

On examination, blood pressure was 118/74 mm Hg, pulse 78 beats min^{-1} and body-mass index 23.6 kg m^{-2} .

There was no flank tenderness, abdominal bruit or varicocele. Laboratory tests showed: serum creatinine 0.9 mg dL^{-1} (estimated glomerular filtration rate 112 $\text{ml min}^{-1} 1.73 \text{ m}^2$); urea 29 mg dL^{-1} ; normal electrolytes; haemoglobin 14.6 g dL^{-1} ; platelet count $263 \times 10^9 \text{ L}^{-1}$; C-reactive protein $< 1 \text{ mg L}^{-1}$. Urinalysis revealed 5–10 red blood cells per high-power field without proteinuria or casts, and urine culture was sterile.

Screening abdominal ultrasonography showed kidneys of normal size and echogenicity without hydronephrosis, but colour Doppler incidentally demonstrated a striking flow focus at the right renal hilum suggestive of an aberrant venous channel. Multiphase contrast-enhanced CT confirmed preserved cortical enhancement yet complete absence of a right renal vein entering the inferior vena cava (IVC) (Figure 1). Closer axial sections revealed numerous perirenal channels coalescing into a single enlarged cortical vein at the mid-posterior hilum (Figure 2). Coronal reformats traced this vein caudally along the ureteric border, where it emptied into a markedly ectatic right gonadal vein that drained normally into the IVC below the renal hilum (Figures 3 and 4). The left renal vein and both renal arteries were orthotopic and patent; no retroperitoneal mass, lymphadenopathy or renal-vein thrombosis was detected, and the pampiniform plexus was not dilated.

Targeted duplex sonography refined the haemodynamic profile: colour Doppler depicted a homogeneous vascular blush within the cortical collateral and proximal gonadal vein, while spectral Doppler recorded a low-velocity (mean 12 cm s^{-1}), continuous monophasic waveform—findings consistent with low-pressure venous outflow and excluding an arteriovenous malformation (Figures 5 and 6). Key observations from each modality and their clinical implications are summarised in Table 1, underscoring how complementary techniques established the diagnosis of isolated right-renal-vein agenesis with cortical-to-gonadal collateral drainage.

Because renal function, systemic blood pressure and parenchymal enhancement were all normal and symptoms were mild, the multidisciplinary team (nephrology, urology, vascular surgery and radiology) deemed intervention unnecessary. The patient was counselled on the benign nature of the anomaly and the surgical risk posed by inadvertent ligation of the solitary drainage vein during any future abdominal or pelvic procedure. A conservative surveillance plan was instituted—annual clinical review, serum creatinine measurement and duplex ultrasound. At 12-month follow-up, he remained asymptomatic; blood pressure and renal function were unchanged, and repeat imaging showed stable calibre and flow in the cortical–gonadal collateral with no new varices or hydronephrosis, confirming satisfactory long-term adaptation of the renal venous outflow pathway.



Figure-1: Axial portal-venous phase CT confirming absence of a right renal vein entering the IVC.

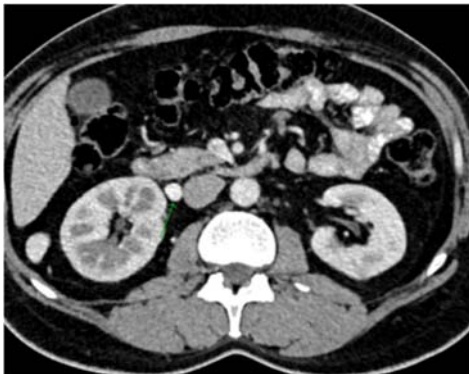


Figure-2: Magnified axial CT (green arrow) demonstrating a prominent cortical draining vein



Figure-3: Coronal CT tracing the cortical vein (arrow) descending to join the dilated gonadal vein

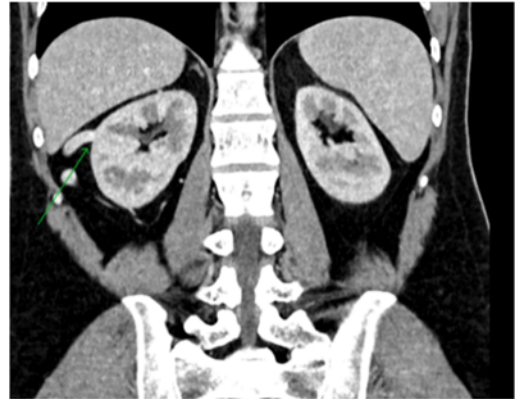


Figure-4: Coronal CT depicting the cranial capsular segment of the cortical collateral (arrow)

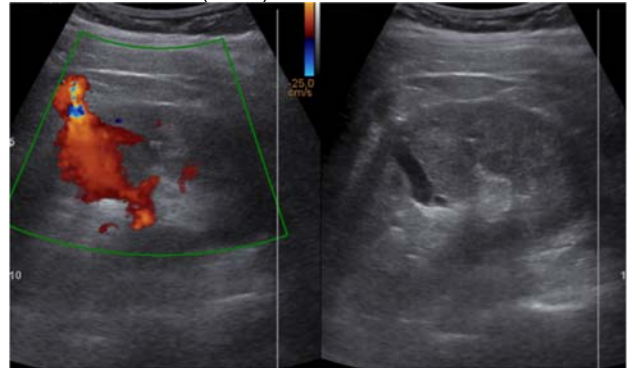


Figure-5: Color Doppler ultrasound showing vascular blush within the cortical collateral and draining gonadal vein

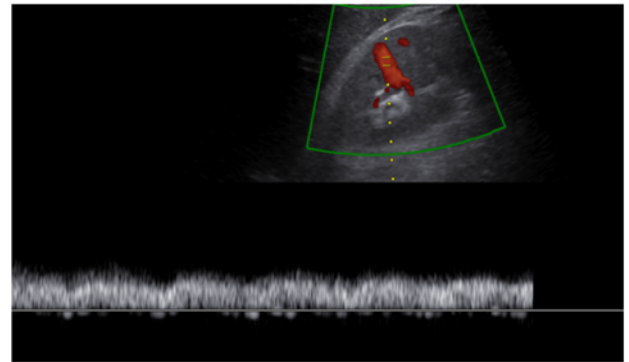


Figure-6: Spectral Doppler displaying low-velocity, continuous venous flow within the cortical vein

Table-1: Imaging modalities and key findings

Modality	Key observation	Clinical implication
CT – axial (Figure 1)	No orthotopic right renal vein; peri-renal venous plexus.	Suggests congenital agenesis.
CT – axial zoom (Figure 2)	Dominant cortical vein drains posteriorly.	Identifies collateral origin.
CT – coronal (Figures 3–4)	Cortical vein empties into a dilated gonadal vein.	Maps the entire collateral pathway.
Colour Doppler (Figure 5)	Vascular blush within the cortical collateral.	Confirms patency and direction.
Spectral Doppler (Figure 6)	Low-velocity monophasic waveform.	Verifies venous nature; excludes AV malformation.

DISCUSSION

Isolated agenesis of the right renal vein occurs when the embryonic junction between the right subcardinal and supracardinal veins fails to mature and fuse with the developing inferior vena cava (IVC). In the absence of a direct reno-caval conduit, transient intrarenal venous hypertension stimulates enlargement of collateral channels that decompress the kidney [9]. Three principal escape routes are recognised: (1) peri-ureteric veins that drain into the iliac system, (2) ascending lumbar veins that join the azygos-hemiazygos network, and (3) corticocapsular veins that coalesce and empty inferiorly into the gonadal vein. In the present case, the entire renal parenchyma relied on this third pathway [10].

Differentiating congenital renal-vein absence from acquired conditions such as acute renal-vein thrombosis or tumoural invasion is essential. Congenital agenesis is characterised by smooth caval walls, a normally enhancing kidney and well-formed collaterals without intraluminal filling defects [11]. Thrombosis, by contrast, produces a venous filling defect, delayed cortical enhancement, perinephric oedema and renal enlargement, while tumoural invasion appears as eccentric soft tissue within the vein or cava. In our patient, colour and spectral Doppler proved decisive: the slow, monophasic waveform throughout the cortical collateral and gonadal vein confirmed low-pressure venous drainage rather than a high-velocity shunt or arteriovenous malformation [12].

Although many patients remain asymptomatic, episodic flank pain and microscopic haematuria can result from transient elevations in renal venous pressure or rupture of thin-walled capsular veins. When the gonadal vein is the sole outflow channel, men may develop a varicocele, and women may develop pelvic congestion syndrome. Intervention is seldom required unless symptoms are severe or renal function deteriorates [13]. The principal clinical danger lies in inadvertent disruption of the solitary collateral during nephrectomy, IVC interventions or procedures on the gonadal vein, which can precipitate catastrophic haemorrhage or renal venous infarction. Meticulous pre-operative venous mapping with contrast-enhanced CT or MR venography, supplemented by Doppler ultrasound, is therefore mandatory whenever anomalous venous channels are encountered [14, 15].

CONCLUSION

Isolated absence of the right renal vein with exclusive cortical-to-gonadal drainage is rare yet clinically significant. Recognition depends on four features: an absent orthotopic renal vein, a dominant cortical collateral coursing to the gonadal axis, preserved renal enhancement and a venous Doppler waveform. When renal function and blood pressure are normal and symptoms are mild, conservative follow-up with periodic imaging is

appropriate. The paramount clinical priority is clear documentation, alerting future surgeons and interventional radiologists that a single collateral vein sustains renal outflow and must not be ligated or injured.

Patient Consent: Written informed consent was obtained from the patient for publication of anonymized clinical data and images.

Conflict of Interests: The author declares no competing interests.

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Authors Contribution: SA conceived the study, interpreted the images, and drafted the manuscript. The author read and approved the final version.

Data Availability Statement: The data used in this study are available upon reasonable request from the corresponding author, subject to ethical and institutional guidelines.

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