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Case Report

Hypoplasia of right renal vein with aberrant drainage into ipsilateral spermatic vein: Case report

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ABSTRACT

The objective of this study was to describe a case of marked hypoplasia of the right renal vein with drainage into ipsilateral gonadal vein. A 66-year-old man, known for hypertension and previous smoking, underwent an abdominal ultrasound exam, which detected a juxtarenal aortic aneurysm. Computed tomography scan confirmed the presence of the aortic aneurysm; furthermore it showed an abnormal right kidney venous drainage consistent in a dilated and tortuous vein, which originated at the hilar region, heading caudally and joining the right spermatic vein at level of aortic carrefour. A thin vein—located more cranially with mild and late contrast enhancement—was also demonstrated from right kidney hilum to inferior vena cava, probably representing a remnant of the right main renal vein. To our knowledge, this anatomic variant was never reported in the peer-reviewed literature.

Introduction

The development of renal veins is a complex process that involves multiple steps. The fusion of the supra- and subcardinal veins generates 2 renal veins (ventral and dorsal), nevertheless the dorsal veins ordinarily regress and the ventral vein forms the renal vein. Eventually, caudal stretch of the subcardinal veins produces the gonadal vessels [1]. Consequently, complete or partial duplication of the right renal vein is the most common anatomic variation. Usually, it is clinically silent and can be incidentally detected by imaging performed for other reasons [2]. On the other hand, the absence of the right renal vein (with normal kidney) is a very rare condition; only 3 cases were previously reported [3–5].

We describe a case of marked hypoplasia of the right renal vein with drainage into ipsilateral gonadal vein: this anatomic variant was never reported in the peer-reviewed literature.

Competing Interests: The authors have declared that no competing interests exist.

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Case report

A 66-year-old man, known for hypertension and previous smoking, was admitted to our emergency department for a fall with skull-facial trauma, reporting an intraparenchymal hemorrhage of the right lenticular nucleus. The patient was admitted to the neurosurgery department and was treated conservatively. Throughout hospitalization, an abdominal ultrasound exam showed an uncomplicated abdominal aortic aneurysm. Therefore, after some days, a computed tomography (CT) study (Aquilion 64, Toshiba Medical Systems, Ōtawara, Tochigi, Japan) was performed. Two acquisitions—in angiographic and late urographic phase— were extended, respectively, from C7 vertebra to pubic bone (100 kVp, modulated mAs, section thickness 0.5 mm; reconstruction thick-

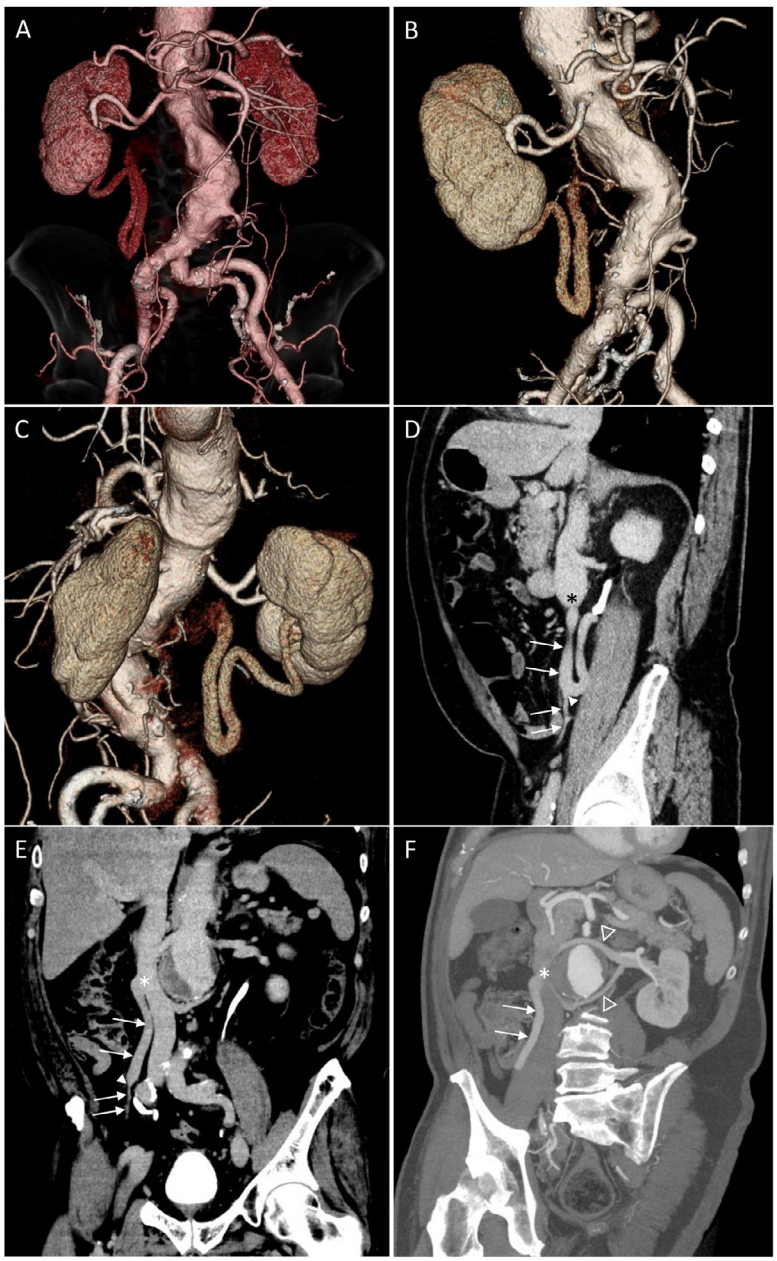

Fig. 1 – CT scanning. (A-C) Angiographic phase: coronal, oblique anterior view, oblique posterior view Volume volume rendering (VR) reconstructions; (D and E) urographic phase: parasagittal and paracoronal maximum intensity projection (MIP) reconstructions; (F) angiographic phase: paracoronal MIP reconstruction. Juxtarenal aortic aneurysm (A, B, E, F) with thick parietal thrombus (E and F). There is 1 renal artery per side (A). Two branches coming from right kidney hilum join to form 1 renal vein (C) that goes caudally until aortic carrefour level (A-C) and drains into the gonadal homolateral vein. In this point (D and E: full arrowhead), gonadal vein (B-F: arrows) shows an abrupt diameter increase, until its confluence into the inferior vena cava (D-F: *). Furthermore, duplication of left renal vein with circumaortic configuration is recognizable (F: empty arrowheads).

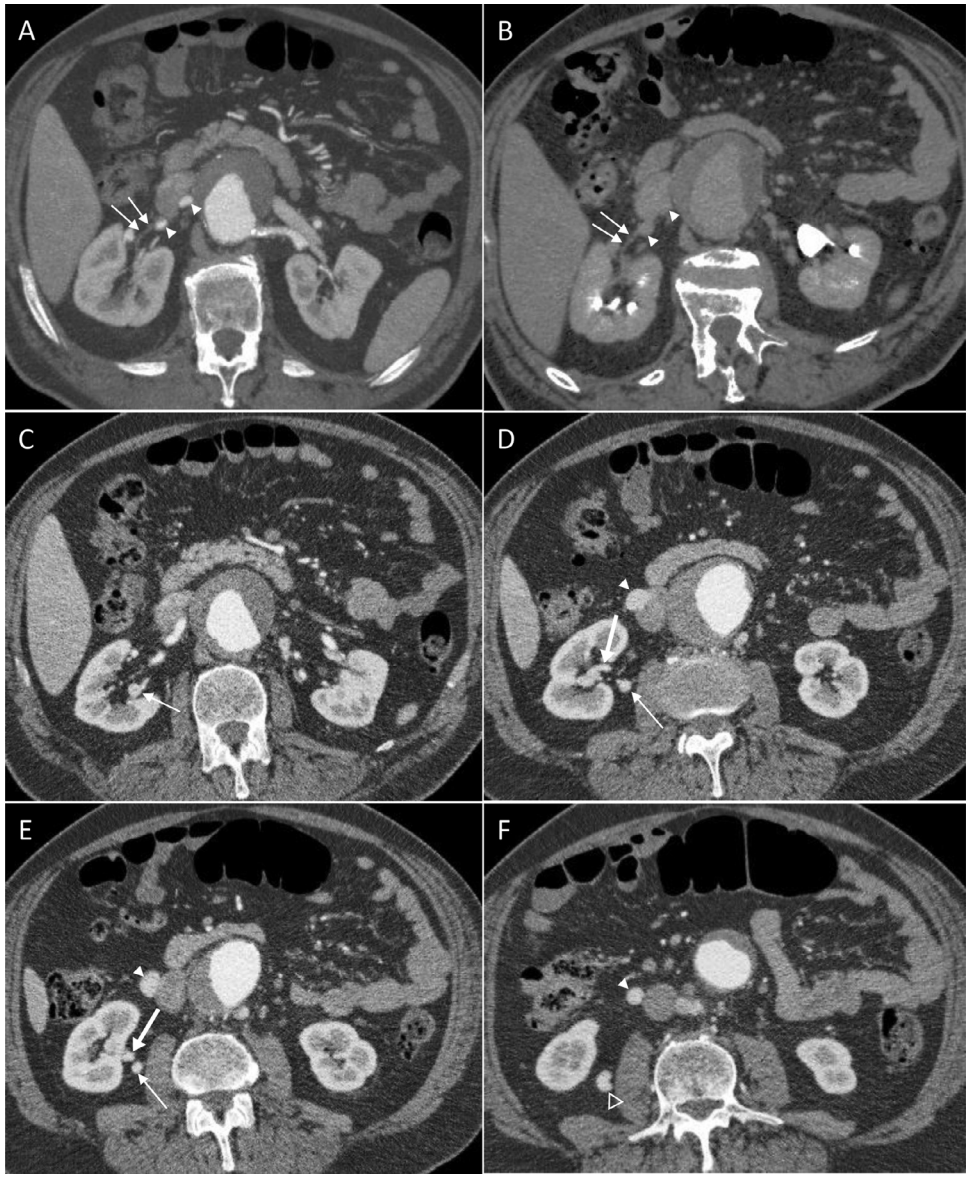

Fig. 2 – CT scanning. (A) Angiographic phase, axial MIP reconstruction. (B) Urographic phase, axial image at the same level of (A). (C-F) Angiographic phase, craniocaudal subsequent axial slices.
A tiny vein (A and B: arrows) with mild contrast enhancement goes from right kidney hilum to inferior vena cava. Right renal artery is discontinuously recognizable (A and B: arrowheads). Two distinct branches (C-E: thick and thin arrows, respectively)—coming from kidney hilum—join to form 1 renal vein (F: empty arrowhead), located behind gonadal vein (D-F: full arrowhead); confluence of gonadal vein into inferior vena cava (C).

ness 1 mm) and from diaphragm to pubis bone (120 kVp, other parameters similar to angiographic acquisition). Angiographic acquisition was triggered by "Sure Start" technique with region of interest at midthoracic descending aorta level (threshold Δ100 HU and diagnostic delay of 5 seconds), during a single 80-mL iodine bolus (Iobitridol - Xenetix 350 mgI/mL, Guerbet, Villepinte, France) followed by 40 mL of saline solution, injected into a right antecubital vein through a catheter using a 4 mL/s flow rate.

CT scan confirmed US findings consistent with a juxtarenal aortic aneurysm extending from the origin of the renal arteries to aortic carrefour, with a maximum diameter of about 6 cm; true lumen was reduced by irregular parietal thrombosis with a maximum depth of about 17 mm; no proximal neck was present. Furthermore, CT scan demonstrated normal left and right kidneys (morpho-volumetry, parenchymal contrast enhancement and pyelocalyceal system opacification) and normal left and right renal arteries (Fig. 1A-C).

An abnormal right kidney venous drainage was revealed, occurring by a dilated and tortuous abnormal vein that originated at the hilar region; this vein, tortuously directed caudally until L4-L5 intersomatic disc level, drained into an aberrantly dilated right spermatic vein (Fig. 2C-F). A thin (about 1 mm in main diameter) vein—located more cranially with mild

and late contrast enhancement—was also demonstrated from right kidney hilum to inferior vena cava, probably representing a remnant of the right main renal vein (Fig. 2A and B). Furthermore, duplication of left renal vein with circumaortic configuration was observed (Fig. 1F).

Color Doppler ultrasonography (5-1 MHz Curved array probe; iU22 Ultrasound System, Philips, Best, the Netherlands) was also performed, demonstrating venous blood flow from right kidney hilum to right spermatic vein and absence of flow in the thin more cranially located vein. Renal arterial resistive indexes were in normal range.

The serum creatinine and Blood Urea Nitrogen (BUN) levels of the patient were normal.

Discussion

Variation of the right renal veins is more common than that of the left side [6]. To our knowledge, only 3 cases of congenital absence of the right renal vein have previously been reported.

Pinggera et al. [5] first described a case of congenital absence of the right renal vein with anomalous venous drainage through right ovarian vein: in this case, there was a right renal vein originating from renal hilum with drainage into an ovarian venous plexus, from which right ovarian vein emerged to flow into inferior vena cava.

Bozlar et al. [3] described a case of congenital absence of the right renal vein with an aberrant venous drainage through the lower pole of the kidney into the inferior vena cava, interpreted as an ascending lumbar collateral vein (a persistent caudal end collateral of embryonic supracardinal venous plexus). In this case, neither fibrous band nor stricturered and/or thrombosed vein was identified at right renal vein level, but a suddenly interrupted right main renal vein (“bud”) was noted.

Lastly, Kim et al. [4] reported a case of absence of the right main renal vein with venous drainage starting from kidney hilar region and draining into an aberrant right adrenal gland venous plexus. Also in this case, any fibrous band or remnant stalk was not observed at the expected level of the renal vein.

Our case is similar but not equal to the other 3 reported in literature. In particular, as in that reported by Pinggera et al. [5], kidney venous drainage was directed into the gonadal vein; however, abnormal renal vein flowed directly into the spermatic vein, without interposition of a gonadal plexus. However, a special consideration should be done about the small vein—going from renal hilar region to inferior vena cava—that was present in our case and was interpreted as a markedly hypoplastic main renal vein.

It is well known that there are many acquired causes of renal vein obstruction, such as primary vein thrombosis, hypovolemia, primary renal disease, and trauma [7]. Nevertheless, in our case, renal morpho-volumetry and function were normal and our patient never suffered from any of the above-mentioned conditions. In this setting, the intrauterine developmental defect hypothesis is more plausible rather than the end result of an acquired renal vein injury occurred during the fetal age.

Our case report suffers from a limitation: the lacking of the basal and venous CT phases, which could better demonstrate the contrast enhancement of the hypoplastic right main renal vein; however, it was not considered ethical to repeat the examination when the anomaly was detected.

Furthermore, we cannot exclude that in some of the cases described as “absence of the main renal vein” [3–5], the vein was markedly hypoplastic, as in our case, but it was overlooked because of technical limits of the scanner employed in the CT studies.

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