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An accessory aberrant testicular artery branching from the inferior suprarenal artery: embryologic and genetic consideration

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ABSTRACT

During routine educational dissection of a cadaver (61-year-old, male, white descent), multiple aberrant right testicular artery and veins were discovered.

Detailed analysis of the abdominal vascular pattern showed that the accessory aberrant testicular artery branched off the right inferior suprarenal artery. It crossed the anterior pararenal space, gave several branches to the soft tissues of the posterior pararenal space, and descended towards the deep inguinal ring. The accessory aberrant testicular vein accompanied the aberrant artery and was drained by the right inferior suprarenal vein into the renal one. Two other testicular veins followed a typical course and opened into the inferior vena cava.

The anatomical variability reported in this paper requires attention of the related specialists, as it may significantly complicate surgical and minimally invasive procedures on the organs of the retroperitoneal space and inguinal region. Thorough preoperative investigation regarding the variability of the vascular pattern should be conducted in patients considered for intervention on the adrenal gland, kidney, or testis.

Keywords: testicular artery, testicular vein, inferior suprarenal artery, embryogenesis, molecular regulation of angiogenesis.

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Introduction:

The testicular arteries are paired branches of the abdominal aorta. They typically arise from the ventrolateral aspect of the abdominal aorta and descend towards the deep inguinal ring. The venous outflow from the testis is facilitated by the pampiniform venous plexus, which condenses to form four veins at the superficial inguinal ring, two veins at the deep inguinal ring and, finally, one testicular vein in the retroperitoneal space.

According to extensive cadaveric research of *Asala et al.*¹, testicular arteries do vary in their origin, course, and number in 4.7 percent out of 150 cases. Among the described variabilities, the testicular vessels may be missing, or one or both arteries may arise from the renal, suprarenal, or lumbar arteries. The most common congenital variation of the testicular vein is its partial or complete duplication, which was observed in 32 percent of cadavers on the left side, and in only 6 percent bilaterally¹.

Described in our case is a right sided aberrant triple testicular vein associated with accessory aberrant testicular artery which has not been previously reported in the literature. The accessory vessels anastomosed with the inferior suprarenal vessels, drained the posterior pararenal space, and crossed the anterior surface of the right kidney and descended medially. Such aberrant course of the atypical vessels represents a potential risk for both open and minimally invasive surgical procedures on the suprarenal glands, kidneys, and inguinal region.

In the present report, we discuss the macroscopic anatomy of a rare pattern of testicular vessels, and summarize possible embryologic and genetic preconditions of such development.

Case report:

During the routine educational dissection of a cadaver (61-year-old, male, white descent), two atypically arranged testicular arteries and three testicular veins were differentiated on the right side of the posterior abdominal wall.

One of two testicular arteries, with an outer diameter equal to 3.4 mm, branched off from the right renal artery (Figure 1). It descended vertically passing between the two medial testicular veins, reached the deep inguinal ring, and contributed to the formation of the spermatic cord. The aberrant accessory testicular artery, 2.4 mm in width, originated from the inferior suprarenal artery. It crossed the anterior surface of the right kidney, supplied branches to the soft tissues of the posterior pararenal space, and then joined the lateral testicular vein descending towards the deep inguinal ring.

The two medial testicular veins, 4.8 and 5.1 mm in width, ran upward and medially parallel to each other and joined the inferior vena cava 3 and 24 mm below the entrance point of the right renal vein, respectively (Figure 2). The lateral testicular vein ascended toward the right kidney deviating laterally. It received numerous tributaries from the right posterior pararenal space, then turned medially, and joined the right inferior suprarenal vein approaching the right renal vein. On its way, the lateral testicular vein anastomosed with the median vein via a vessel 4.2 mm in diameter.

On the left side of the body, the arrangement of testicular vessels did not show any peculiarities.

Discussion:

Although the presence of accessory testicular arteries originating from the renal, inferior colic arteries, and abdominal aorta is quite common²⁻⁴, there are only few reports showing possible developmental interplay between the inferior suprarenal and testicular arteries in the literature.

Thus, *Jyothsna et al.*, reported a double testicular artery with the accessory one arising from the abdominal aorta via a common trunk with the inferior suprarenal artery⁵. In the case reported by *Brohi et al.*, the reversed development took place and the inferior suprarenal artery branched off the left testicular artery, then progressed upward to supply the lower pole of the gland⁶. The branching of the

accessory testicular artery from the inferior suprarenal artery with the aberrant course through the posterior pararenal space is being

reported for the first time to the best of our knowledge (Schema 1).

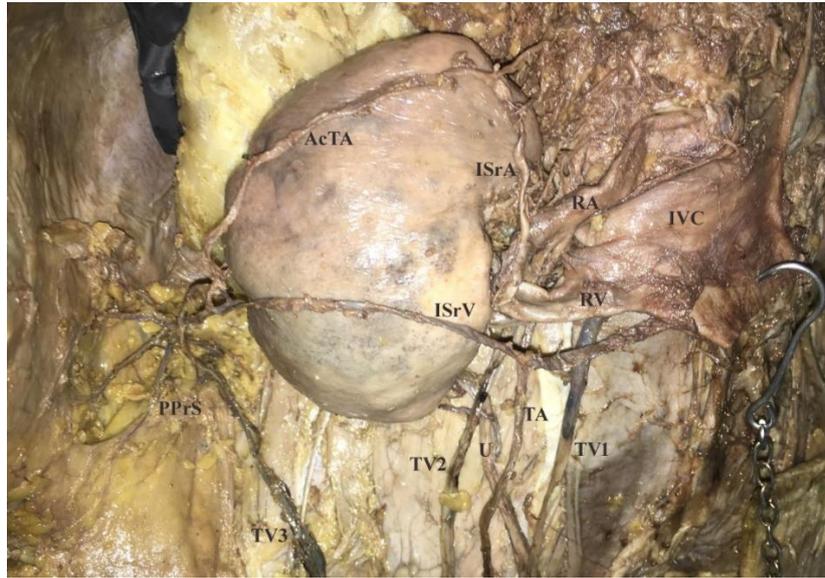
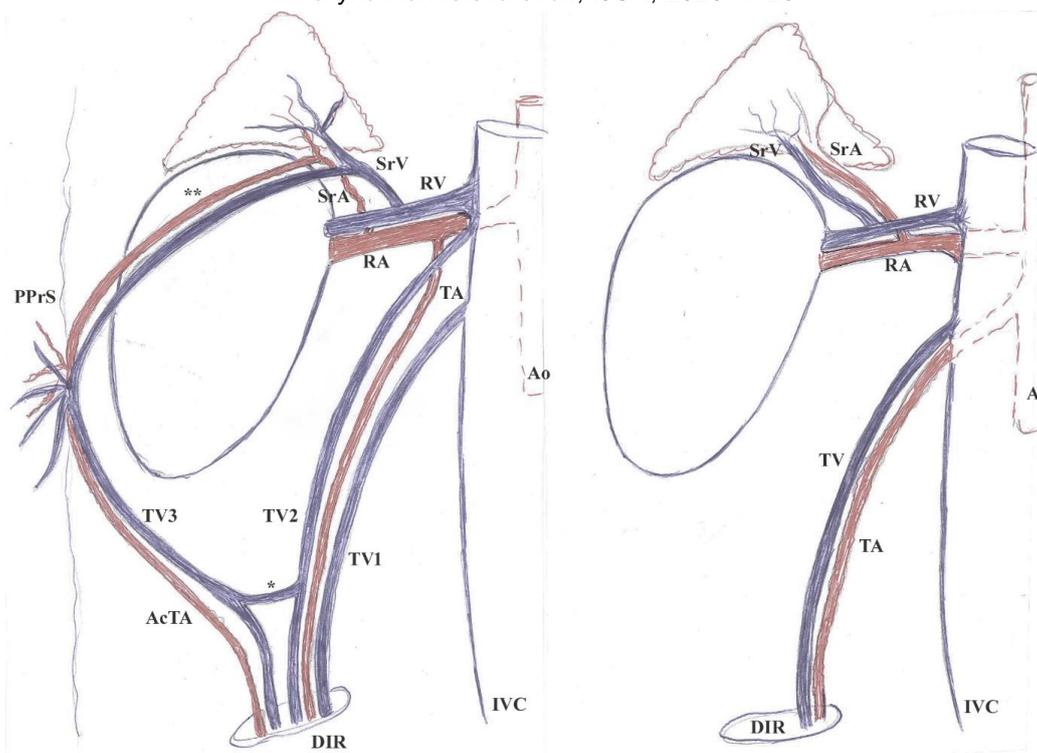


Figure 1. The right testicular artery (TA) branches off the right renal artery (RA); the accessory testicular artery (AcTA) originates from the inferior suprarenal artery (ISrA), and runs laterally through the anterior pararenal space giving off branches to soft tissues of the posterior pararenal space (PPrS); then it descends downwards and medially along with the lateral testicular vein (TV3). Other abbreviations: ureter (U); inferior suprarenal vein (ISrV); renal vein (RV); medial and median testicular veins (TV1 and TV2, respectively); inferior vena cava (IVC, reflected).



Figure 2. The medial (TV1) and median (TV2) testicular veins drain into the inferior vena cava (IVC), while the lateral testicular vein (TV3) takes an aberrant course via the posterior pararenal space (PPrS) and continues with the inferior suprarenal vein (SrVs) that opens into the right renal vein (RV). The black arrow shows the anastomotic vessel connecting the TV3 with TV2. Other abbreviations: testicular artery (TA); accessory testicular artery (AcTA); ureter (U); deep inguinal ring (DIR); psoas major (PsM).



Schema 1. Comparison of typical appearance of the gonadal vessels (B) with the vascular pattern uncovered in our case (A). Abbreviations: **IVC**, inferior vena cava; **Ao**, aorta; **TV**, testicular vein; **TV1,2,3** – medial, median, and lateral testicular veins, respectively; **TA**, testicular artery; **AcTA**, accessory testicular artery; **SrA**, suprarenal artery; **SrV**, suprarenal vein; **PPrS**, posterior pararenal space; **DIR**, deep inguinal ring.

From the embryologic point of view, the suprarenal and testicular arteries have common origin as they all develop from the multiple lateral splanchnic arteries nourishing the mesonephric ridge in a 2-4 mm embryo. One gonadal and three suprarenal arteries persist on each side of the developing kidney, gonad, and the adrenal gland⁷. The inferior phrenic artery branches off the most cranial suprarenal artery, the middle one continues to supply the adrenal gland becoming the middle suprarenal artery, while the renal artery arises from the most caudal inferior suprarenal artery. Typically, the gonadal artery develops independently from the suprarenal vessels and extends down following the descending gonads.

The sprouting of the accessory testicular artery from the inferior suprarenal artery may be particularly explained from the position of molecular regulation of angiogenesis. The proliferation and free migration of the endothelial cells require an interplay between the various

forms of vascular endothelial growth factors (VEGF), angiopoietins (Ang), fibroblast growth factors (FGFs) and their receptors⁸. Modern genetics provides clear evidence that the receptor for VEGF-C is highly expressed during embryonic vascular remodeling⁹. Although the molecular mechanisms determining the exact localization of endothelial sprouting is mostly a mystery, we can assume that duplication and shifting of the codons responsible for the expression of these receptors could alter their following number and localization in the endothelial wall, thus leading to the development of the aberrant accessory vessels.

Another assumable molecular mechanism that may also be involved in the formation of excessive branching vascular patterns is the downregulation of ICAP1 (integrin cytoplasmic domain-associated protein 1). ICAP1 increases cell motility and the initial formation of capillary sprouts by inhibiting Rho kinase activity and ERK phosphorylation, and by inducing

expression of p21 and p27 (cell cycle inhibitors)
¹⁰. Downregulation or reduced expression of ICAP1 therefore may be implicated in cases of excessive vascular development triggering the accelerated endothelial proliferation.

Different regulatory factors are involved in the developmental reconstruction of the venous network in the mesonephric region. At the stage of development of the somatic veins, the renal and gonadal veins on both sides join the supracardinal-subcardinal anastomosis. On the right side these vessels form the inferior vena cava and its tributaries, while on the left side they largely degrade with the exception of the left renal, suprarenal, and gonadal veins ⁷. According to *Boon-Huat Bay et al.*, the transforming growth factor beta-3 (TGF- β 3) encoded by the TGFB3 gene is explicitly involved in downregulation of this regression ¹¹. Thus, the overexpression of TGF- β 3 prevents regression of the supracardinal veins and lead to persistence of the subcardinal anastomotic veins. Such influence could lead to the development of numerous testicular vessels as reported in our case.

The anatomical variability of the testicular vessels is of high importance for testicular function, as the thermoregulation and effective spermatogenesis heavily depend on intensity of the blood circulation. The detailed knowledge of normal morphology and possible vascular patterns is an essential prerequisite for the related specialists such as surgeons working on the organs of the retroperitoneal space and inguinal region, and radiologists interpreting angiographic images of the abdomen and pelvis.

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