Morphological and clinical aspects of the occurrence of accessory (multiple) renal arteries

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Abstract

Renal vascularization variants vastly differ between individuals due to the very complex embryogenesis of the kidneys. Moreover, each variant may have implications for clinical and surgical interventions. The number of operating procedures continues to grow, and includes renal transplants, aneurysmorrhaphy and other vascular reconstructions. In any surgical technique, unawareness of the presence of multiple renal arteries may result in a fatal outcome, especially if laparoscopic methods are used. The aim of this review is to comprehensively identify the variation within multiple renal arteries and to highlight the connections between the presence of accessory renal arteries and the coexistence of other variants of vascularization. Another aim is to determine the potential clinical implications of the presence of accessory renal arteries. This study is of particular importance for surgeons, intervention radiologists, nephrologists and vascular surgeons.

Key words: kidney, renal artery, accessory, multiple, anatomical variation.

Introduction

In classic anatomic descriptions, typical renal vascularization is described as a single renal artery originating from the abdominal aorta at the level of the intervertebral disc between L1 and L2, just below the inception of the superior mesenteric artery. The renal artery enters the renal hilum, branches within the renal sinus and sends out *interlobar arteries*, which are situated between the renal pyramids in the cortex and take an arched course along the base of the pyramid, between the medulla and the cortex. Here, the interlobar arteries are designated *arcuate arteries*. *Interlobular arteries* branch from the arcuate arteries and ascend through the cortex towards the renal capsule. As they travel to the renal capsule, the interlobular arteries give off branches, the *afferent arterioles*, to each glomerulus.

In 2008, the simplest definition of *accessory renal artery* was introduced by Mir *et al.* [1]. While "normal" renal arteries were described as entering the kidney through its hilum, the accessory renal arteries might enter the renal artery through the hilum or through the surfaces of the kidney [1]. However, from the sixteenth century until today, accessory renal artery terminology has been controversial and unclear [2, 3]. The first description of accessory renal arteries was given in the sixteenth century. In 1564, Eustachi wrote about accessory renal arteries: "These arteries are end-arteries, they are not supplemental or accessory as they are frequently called in textbooks. They do not make anastomotic connections once they enter the kidney" [4].

This long held opinion, that there is usually only one renal artery vascularizing the kidney, has been rejected with a new thesis claiming that the most common type of renal vascularization contains accessory renal arteries, which are classified according to their origin, given by Merklin and Michels (1958) [5] as:
1) accessory renal arteries originating from the aorta,
2) accessory renal arteries originating from the main renal artery,
3) accessory renal arteries originating from other sources.

In 1956, Graves [6] considered that due to lack of standards, the term "multiple" could describe any additional vessel entering the kidney, either originating from the abdominal aorta or the main renal artery. In 1958, Merklin and Michels [5] used the designations *main renal*, *aortic superior/inferior polar* and *renal inferior polar* arteries. In 1962, Geyer and Poutasse [7] classified the additional vessels of the kidney as *supernumerary*, *accessory* or *aberrant*, and they also confirmed the sizes of these vessels to be different. In 1969, Poisel and Spängler [8] divided additional renal arteries according to the areas of kidney which they penetrate – *accessory*, *supplementary* and *supernumerary* renal arteries enter through the renal hilum, while *aberrant* renal arteries penetrate an area of the kidney other than its hilum.

In 1982, Stephens [9] claimed that as the vessels are not superfluous, but essential non-anastomotic arteries corresponding to the segmental branch of a single renal artery, the terms *accessory*, *supernumerary* or *aberrant* were not suitable for them. In 1992, Sampaio and Passos [10] introduced the terms *hilar* for the aortic branch penetrating the hilum, *extrahilar* for the branch of the renal artery with an extra-hilar penetration, *superior polar* for the aortic branch penetrating the superior pole and *inferior polar* for the aortic or common iliac artery penetrating the inferior pole of the kidney. They also claim that these vessels should be denominated as *multiple*, because they are segmental end-arteries.

In 2001, Satyapal *et al.* [2] denominated the artery arising from the abdominal aorta and ending in the kidney as *additional*. In the same year, Vilhova *et al.* [11] classified renal arteries originating from the aorta, or its major branches supplying the superior or inferior renal poles, on the basis of their point of entry to the renal parenchyma – via the hilum as *accessory* or outside the hilum as *perforated*.

In 2004, Bordei *et al.* [12] introduced three new definitions: *superior/inferior polar arteries* which distribute blood to the renal poles, *main renal artery* as the largest hilar artery and *hilar supplementary artery* as the smaller one. In 2005, Holden *et al.* [13] defined the accessory arteries as vessels entering the kidney from the hilum along with the main renal artery, whereas the aberrant arteries enter the kidney directly from the capsule outside the hilum. These accessory/aberrant renal arteries usually originate from the abdominal aorta or iliac arteries; however, they can, on rare occasion, arise from the lower thoracic aorta or from the lumbar or mesenteric arteries.

Finally, in 2010 Daescu *et al.* [14] imposed a new classification: the renal arteries can be hilar and polar (superior/ inferior). The polar arteries were divided into four groups: (1) *solitary*, (2) *pedicular*, if the second one is accompanied by a polar vein and a nerve plexus, (3) *false supernumerary*, if it replaces the segmental artery and (4) *true supernumerary artery*, if the respective segmental artery emerges from the renal artery.

The relevance scores for nomenclature variants based on the numbers of articles published on PubMed, which contains the most popular terms used for naming renal arteries, are given in Table I.

It is also worth mentioning that the occurrence of accessory renal arteries differs according to side of the body and the part of the kidney where the supernumerary vessel enters its parenchyma. This information is included in Table II [10].

Embryogenesis of renal vessels

The multiple renal artery (MRA) is a vestigial structure caused by failure to degenerate during the ascent of metanephros. The development of the kidney and its vessels is complex, and the development of kidney arterioles is poorly understood [15]. In an 18 mm fetus, the developing mesonephros, metanephros, suprarenal glands, and gonads are supplied by nine pairs of lateral mesonephric arteries arising from the dorsal aorta [16]. Felix divides these nine pairs of arteries into three groups: cranial (1^{st} and 2^{nd} pair), middle (3^{rd}–5^{th} pair) and caudal (6^{th}–9^{th} pair) [16]. The renal arteries develop from a single pair from the middle group. The remaining arteries of the middle group give rise to the accessory or the aberrant renal arteries [17]. Renal vascular segmentation was originally recognized by Hunter in 1974 [18]. Further

Table I. Relevance of different nomenclature of renal arteries when more than one renal artery is present

Variable	Artery	Arteries
Accessory	486/6103 = ~7.96%	439/4048 = ~10.84%
Multiple	3130/6103 = ~51.29%	2036/4048 = ~50.3%
Supernumerary	60/6103 = ~0.98%	57/4048 = ~1.41%
Aberrant	192/6103 = ~3.15%	152/4048 = ~3.75%
Supplementary	37/6103 = ~0.61%	25/4048 = ~0.62%
Hilar	223/6103 = ~3.65%	158/4048 = ~3.9%
Additional	1744/6103 = ~28.57%	1001/4048 = ~24.73%
Perforated	28/6103 = ~0.46%	19/4048 = ~0.47%
Polar	203/6103 = ~3.33%	161/4048 = ~3.98%
Total	6103	4048

Table II. Differences in frequency of presence of accessory renal arteries due to side of the body and area of kidney in which supernumerary vessels enter its parenchyma

Arterial feature	Right kidney	Left kidney
Two hilar arteries	37.5%	53.6%
Three hilar arteries	23.2%	14.5%
Four hilar arteries	1.79%	4.4%
One superior polar	7.14%	11.6%
One inferior polar	3.57%	2.9%
Extra-hilar superior polar	28.6%	11.6%
Extra-hilar inferior polar	0%	1.4%

studies revealed that on the basis of vascular supply, the renal parenchyma is divided into five segments: apical, upper, middle, lower and posterior. The main renal artery separates initially into anterior and posterior branches. The anterior branch almost always supplies the upper, middle and lower segments of the kidney. The posterior branch nourishes the posterior and lower segments. The lower renal segment is often fed by an accessory vessel [19].

According to Zahoi [20], kidneys with one renal artery can be classified morphologically on the basis of segmentation type. The renal parenchyma was divided into 5 segments in 57% of studied cases, into 6 segments in 31.64%, into 7 segments in 5.7%, into 4 segments in 3.8% and finally, into 8 segments in 1.9% of cases. When accessory renal arteries were present, 24 morphological types were described. In 57.14% of studied cases, 5 renal segments were present, 6 renal segments in 33.33%, 7 segments in 7.14% and 8 renal segments in 2.38%. On the basis of renal corrosion casts of kidneys with 2 renal arteries (88%) and 3 renal arteries (12%), Matusz *et al.* [21] found the renal parenchyma to be divided into 5 segments in 68% of cases, into 6 segments in 25.33%, into 7 segments in 4% and 8 renal segments in 2.67% of cases.

A study performed in 2014 showed that within the embryonic kidney, several putative progenitors marked by the expression of either winged-forkhead transcription factor 1 (Foxd1+ progenitor), aspartyl-protease renin (Ren+ progenitor), and/or hemangioblasts (Scl+ progenitor) are likely to differentiate and endow most of the cells of the renal arterial tree [15]. While such surveys may better clarify the trigger in the process of developing MRAs, it is currently only possible to explain how this course takes place, but the factor or factors which directly initiate it remain unknown.

Morphological and demographic aspects

The frequency of occurrence of MRA varies in a wide range of studies. On average, the MRA has been observed in about 30% of normal subjects [22–24]. However, the frequency of presence of MRA varies widely with ethnicity (Table III) [2, 10, 12, 13, 24–73]. Therefore, knowledge of anatomic variants in each population is valuable. The incidence of MRA varies widely with ethnicity, ranging from 4% in Malaysians [58] to 61.5% in Indians [32, 34]. The frequency of supernumerary renal arteries was found to be 39.2% in a North Indian population. It tends to be more common in Africans (37%) and Caucasians (35%), and less common in Indian citizens (17%) [64, 74]. In a Chinese population frequency of occurrence of accessory renal arteries was found to be 14.5% [35] In a Caribbean population, accessory renal arteries were present in 36.1% of patients [34]. In a Greek population, supernumerary renal arteries occurred in 11.2% of subjects [41]. Nearly a third of the Colombian population presents one additional renal artery

Table III. Frequency of multiple renal arteries in different populations

Population	Researcher	MRA %	N/(Total)
American	Lloyd (1935) [25]	24.2	73/306
	Satyapal *et al.* (2001) [2]	27.7	122/440
Austrian	Janschek *et al.* (2004) [26]	19.8	47/238
Bosnian	Talović *et al.* (2004) [27]	25.8	55/213
	Talović *et al.* (2013) [23]	46.2	18/39
Brazilian	Santos Soares *et al.* (2013) [28]	12	6/50
	Costa *et al.* (2011) [29]	18.5	47/254
	Aragão *et al.* (2012) [30]	21.7	13/60
	Tyson *et al.* (2011) [31]	23	(510)
	Sampaio and Passos (1992) [10]	30.4	81/266
	Palmieri *et al.* (2011) [32]	61.5	123/200
Canadian	Kapoor *et al.* (2011) [33]	12	21/171
Caribbean	Johnson *et al.* (2013) [34]	36.1	107/302
Chinese	Tao *et al.* (2013) [35]	14.5	55/378
Colombian	Saldarriaga *et al.* (2008) [36]	25	97/390
Dutch	Kok *et al.* (2008) [37]	21	60/288
Egyptian	Harraz *et al.* (2013) [38]	14.5	108/731
French	Gerard (1911) [39]	22	63/287
	Laouad *et al.* (2012) [40]	27.02	70/259
Greek	Natsis *et al.* (2014) [41]	11.2	23/206
	Giavroglou (1982) [42]	17.6	327/1855
	Papaloucas *et al.* (2007) [43]	27.4	59/215
Indian	Aristotle *et al.* (2013) [44]	13.3	4/30
	Budhiraja *et al.* (2010) [45]	15	15/100
	Gupta *et al.* (2011) [46]	28.3	17/60
	Budhiraja *et al.* (2013) [47]	59.5	44/74
Iranian	Amirzargar *et al.* (2013) [48]	15.1	76/502
	Tarzamni *et al.* (2008) [49]	24.8	58/234
	Shoja *et al.* (2008) [50]	43.2	35/81
Italian	Levi (1909) [51]	25.5	49/1921
Japanese	Kadotani *et al.* (2005; 2005) [52, 53]	14.1	48/340
	Adachi (1928) [54]	22.8	77/338
	Iijima (1925) [55]	25	15/60
Kenyan	Sungura (2012) [56]	11.3	23/204
	Ogeng'o *et al.* (2010) [57]	14.3	51/356
Korean	Choi *et al.* (1997) [58]	13	65/500

Table III. Cont.

Population	Researcher	MRA %	N/(Total)
Malaysian	Hlaing *et al.* (2012) [59]	4	2/50
New Zealanders	Holden *et al.* (2005) [13]	26	52/200
Polish	Wozniak (2000) [60]	11.2	17/152
	Goscicka (1996) [61]	12.1	34/280
Romanian	Bordei *et al.* (2004) [12]	20.9	57/272
Russian	Seldowitsch (1909) [62]	17.7	53/300
Taiwanese	Hung *et al.* (2012) [63]	17	17/100
Thai	Khamanarong *et al.* (2004) [64]	18.4	98/534
Tunisian	Chabchoub *et al.* (2011) [65]	21.2	44/208
Turkish	Sezer *et al.* (2012) [66]	14.1	35/249
	Ozkan *et al.* (2006) [67]	14.5	248/1710
	Gürkan *et al.* (2004) [68]	14.8	17/115
	Bakirtas *et al.* (2006) [69]	15	28/187
	Ciçekcibaşi *et al.* (2005) [70]	25	45/180
	Gümüş *et al.* (2012) [71]	27	443/1640
	Zağyapan *et al.* (2009) [72]	42	63/150
Ukrainian	Vlihova *et al.* (2001) [73]	31.8	21/66

MRA – multiple renal arteries, N – number of cases where MRA were present, Total – all cases included in study.

and about 3% of the same population presents two additional renal arteries, most of them reaching the kidney through its hilar region [36]. In a Turkish population, accessory renal arteries were found in 42% of subjects [72].

However, in our opinion, detection of the MRA may also depend on the sensitivity of diagnostic procedures, for example, cadaveric or radiological examination. A few other methods can be used to reveal the presence of supernumerary renal arteries. These include dissection of cadavers, angiography and magnetic resonance angiography. While many studies are based on dissected cadavers or specimens from an autopsy, other angiographic studies use live patients. It is argued that cadaver dissection probably affords a more accurate determination of the number of renal arteries than aortography [7]. In angiographic studies, MRA were detected less frequently due to their thickness (diameter < 2 mm). The MRA are often not detectable when originating from the abdominal aorta, and the arteries entering the kidney outside the hilum are frequently confused with the adrenal or capsular arteries [67, 75]. It is noteworthy that magnetic resonance angiography failed to predict the anatomy of renal arteries in 10% of patients with MRA compared to angiography, in which the relative incidence was 3% [37].

Volume rendering (VR), maximum intensity projection (MIP), and multiplanar reformatted reconstruction (MPR) images may accurately demonstrate accessory renal arteries [76].

The arterial phase of three-dimensional CT angiography is used to depict the renal arteries and is the most sensitive phase for detection of MRA and other possible arterial abnormalities [77]. 3D CTA correctly defines renal arterial anatomy (caliber, number, arterial branching pattern and accessory renal arteries) in 97.6% of patients and provides better detail of the venous anatomy compared with routine angiography [78]. Rubin *et al.* [78] showed 3D CT angiography to be 100% sensitive in the visualization of accessory renal arteries.

Since the average renal diameter is approximately 4–5 mm and accessory arteries are considered to be smaller, up to 15% of vessels can be missed by 1–4 detector row CTA [79].

The most common origin of MRA was the abdominal aorta (Figures 1–4). They may be observed unilaterally (Figure 2) and ipsilaterally (Figure 3). Multiple renal arteries rarely originate from the external iliac, lumbar, spermatic, ovarian, inferior mesenteric, superior suprarenal, inferior phrenic, right colic, subcostal, contralateral renal, splenic or thoracic aorta [80] (Figure 5). The most

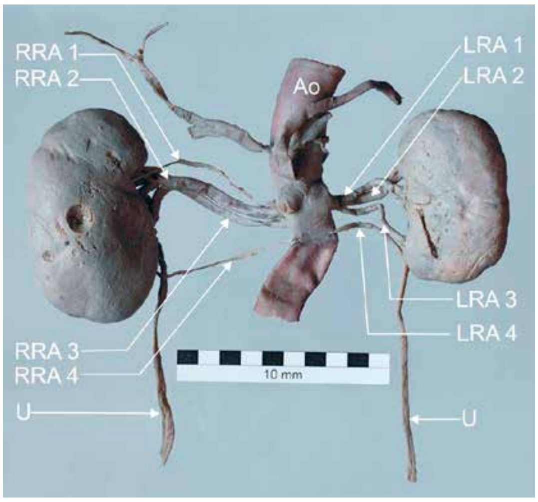

Figure 1. Structures of the retroperitoneal region

Ao – abdominal aorta, RRA 1 – right additional superior hilar renal artery, RRA 2 – main right renal artery, RRA 3 – right additional inferior hilar renal artery, RRA 4 – right additional inferior polar renal artery, LRA 1 – left additional superior hilar renal artery, LRA 2 – main left renal artery, LRA 3 – left additional inferior polar renal artery, LRA 4 – left additional inferior polar renal artery, U – urether.

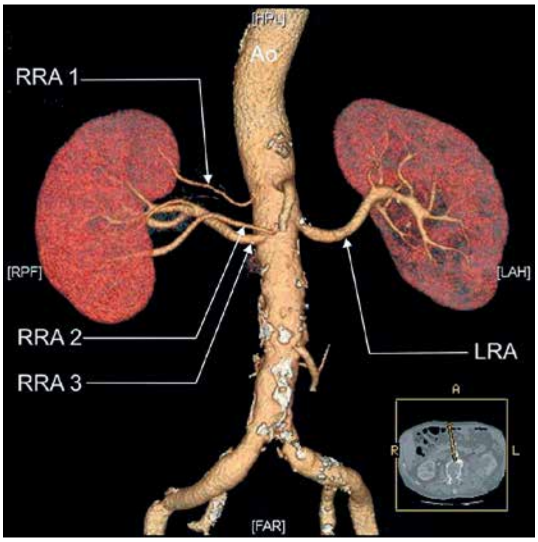

Figure 2. Three-dimensional computed tomography reconstruction of the arteries

Ao – abdominal aorta, LRA – main left renal artery, RRA 1 – right additional superior polar renal artery, RRA 2 – right additional superior hilar renal artery, RRA 3 – main right renal artery.

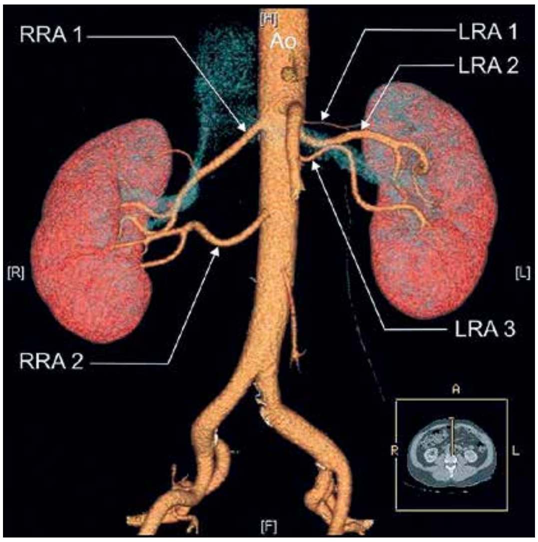

Figure 3. Three-dimensional computed tomography reconstruction of the arteries

Ao – abdominal aorta, RRA 1 – right additional superior hilar renal artery, RRA 2 – main right renal artery, LRA 1 – left additional superior polar renal artery, LRA 2 – main left renal artery, LRA 3 – left additional inferior hilar renal artery.

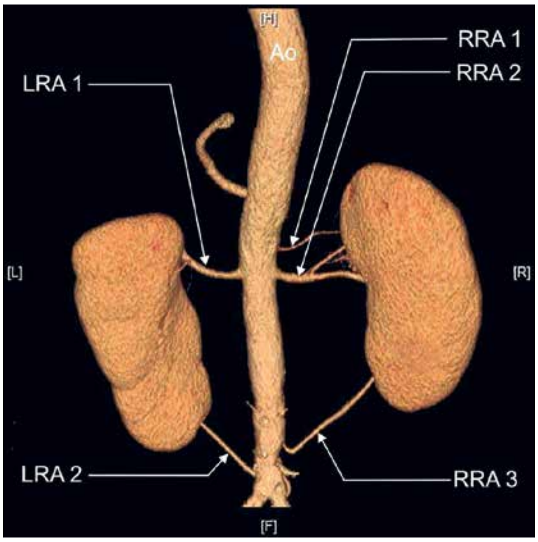

Figure 4. Posterior view of the kidneys and abdominal arteries on MDCT angiographies with 3D reconstruction

Ao – abdominal aorta, LRA 1 – main left renal artery, LRA 2 – left additional inferior polar renal artery, RRA 1 – right additional superior hilar renal artery, RRA 2 – main right renal artery, RRA 3 – right additional inferior hilar renal artery.

common type of MRA was accessory renal inferior polar arteries [81], as confirmed by Bordei *et al.* [12] and our own previous anatomical (Figure 1) and radiological observations (Figure 4). Bordei *et al.* [12] note that the additional renal arteries mainly entered the kidney through the hilum, together with the main renal artery in 61.11% of the cases, the superior pole in 9.26%, and the inferior pole in 29.63%.

Natsis *et al.* [41] reported no statistically significant relationship ($p > 0.05$) between gender and the side on which the supernumerary renal artery was situated. However, several authors have observed a higher incidence in males than females [3, 61, 64, 82, 83], whereas others conclude the opposite [56, 66, 84, 85] (Table IV).

A review of studies reveals a great difference in the description of MRA incidence depending on

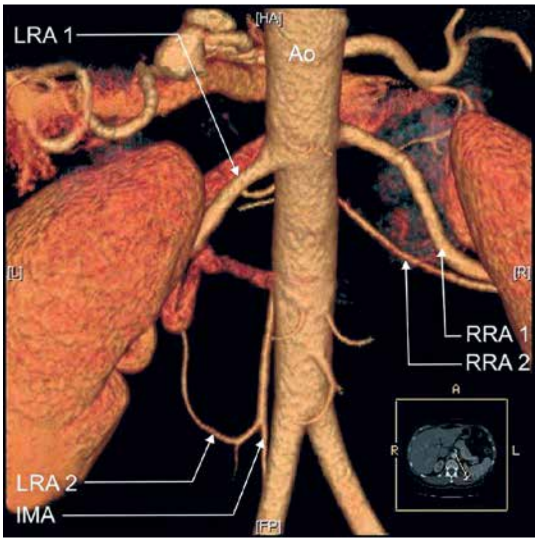

Figure 5. Three-dimensional computed tomography reconstruction of the arteries

Ao – abdominal aorta, IMA – inferior mesenteric artery, LRA 1 – main left renal artery, LRA 2 – left additional inferior hilar renal artery, RRA 1 – main right renal artery, RRA 2 – right additional inferior hilar renal artery.

side of occurrence of MRA. Some studies report that MRA are most frequently left-sided [12, 29, 32, 34, 36, 67, 72, 86–93], while others find that the right side predominates [13, 37, 49, 61, 64, 67, 71, 94–96] (Tables II, IV). More frequent occurrence on the left side of the body and in males is also characteristic for an ectopic kidney. Ectopic kidneys are rare (0.1%), and the incidence of pelvic ectopy ranges from approximately 1 in 2200 to 1 in 3000 [97]. An ectopic kidney may present with pain, hydronephrosis, pyelonephritis, fistulae between the gut and the kidney, renal calculi or obstetric problems in females [17].

Adachi studied a series of 1838 kidneys and found additional renal arteries to be present in 23.1% of cases. He also noted that the number of additional renal arteries ranged from 1 to 4 with a prevalence of 19.81%, 2.88%, 0.44%, and 0.05%, respectively [98]. In a later study conducted in 2011 by Matusz *et al.* [98] the number of additional renal arteries found ranged from 1 to 6 with a prevalence of 9% for one artery, 7% for two, 1.6% for three, 0.3% for four, 0.2% for five, and 0.1% for six.

Clinical implications of presence of multiple renal arteries

Multiple renal arteries have major clinical importance, because overlooking them during pre-operative preparations of patients for surgical procedures may have fatal consequences, especially if laparoscopic methods are used for the procedure. The increasing number of kidney transplants has led to an increase in use of laparoscopic surgical techniques, which, with all their advantages, also have failings. Using these methods reduces the operative field, whereby the risk is increased that variations in vascularization of the kidney may lead to a fatal outcome [99]. Here, it must be emphasized that the effects of the presence of MRA are still to be explored and classified, because accessory vessels are mainly found accidentally during pre-operative procedures or during the diagnostic process. In surgical terms, the upper pole artery represents a major risk, because it is frequently located high up the kidney, meaning that in most cases the surgeons may mistake it for surrounding connective tissue and unknowingly cut through it, thereby causing massive bleeding, which most often leads to a fatal outcome [5]. Furthermore, Rizzari *et al.* [100] note that individuals with kidneys with 2 or more arteries appear to have an increased incidence of hypertension.

However, in a study which took place from 1996 to 2002 on 185 hypertensive patients who underwent MR angiography of the renal arteries, Gupta and Tello [101] found no statistically significant

Table IV. Frequency of multiple renal arteries according to gender and side

Author(s)	Population	Gender		Side	
		F (%)	M (%)	L (%)	R (%)
Adachi (1928) [54]	Japanese	19	24	–	–
Aragão *et al.* (2012) [30]	Brazilian	7.2	6.25	10	11.7
Ciçekcibaşi *et al.* (2005) [70]	Turkish	13.3	28.8	17.6	32.1
Goscicka *et al.* (1996) [61]	Polish	12.8	9.3	7.7	11.5
Palmieri *et al.* (2011) [32]	Brazilian	58	65	67	56
Saldarriaga *et al.* (2008) [36]	Colombian	15.4	26.3	27.3	22.2
Satyapal *et al.* (2001) [2]	American	20.2	33.1	32	23.3
Vilhova *et al.* (2001) [73]	Ukrainian	33.3	30.6	35.3	28.1

F – female, M – male, L – left, R – right.

difference in the prevalence of renal artery stenosis between patients with MRA and those without MRA. Assuming that the presence of two separate causes of hypertension in the same patient would be unlikely, this finding implies that MRA are a vascular anomaly and not a direct cause of hypertension. Of 185 hypertensive patients, 45 (24%) showed accessory renal arteries. Of these 45 patients, 9 (20%) showed renal artery stenosis and 36 (80%) showed no significant stenosis. Of the 140 patients with a single renal artery, 42 (30%) showed renal artery stenosis and 98 (70%) showed no stenosis.

The awareness of the existence of a MRA is very important, as it might complicate reconstructive surgery of abdominal aortic aneurysms and uroradiological procedures [7, 102]. Also hemorrhagic complications, renal artery thrombosis, and warm ischemia time are significantly higher in patients with MRA [23]. For renal transplants, kidneys with accessory renal arteries had a longer mean warm ischaemia time (35.3 vs. 29.2 min, p = 0.0003) and more urethral complications (6/36 vs. 10/312, p = 0.0013) [103].

Opinions are divided as to whether the presence of an accessory renal artery is associated with undesirable consequences of kidney transplants. Some studies consider the presence of accessory renal arteries as a contraindication to their use in transplant surgery: since these are end-arteries, the end-arteries must be re-implanted, and this would require several anastomoses and a prolonged ischemic time, leading to a theoretically higher incidence of renal failure, graft rejection and reduced graft function [104]. On the other hand, a study from the National Institute of Kidney Diseases in Lahore, Pakistan reported no association between multiplicities of renal arteries and the risk of vascular complications. They concluded that kidney transplantation using grafts with MRA is as safe as using grafts with a single renal artery, regarding vascular and urological complications and patients and graft survival [23]. Several other studies also confirm this thesis [105–109].

In 2001 Mizoguchi *et al.* [110] reported an association between anomalies in renal vascularisation and galactosemia, so any patient with galactosemia should be carefully investigated for the presence of any renal vascular anomalies. Bergman *et al.* [111] found inferior renal polar arteries to be implicated as an etiologic factor in a form of hydronephrosis correctable by surgery.

In 2013, the technique of using epigastric arteries in renal transplantation was re-described [48]. This technique was in use already in 2001 [112]. Anastomosing lower polar arteries to the inferior epigastric artery and upper polar arteries to the superior epigastric artery provides backflow from accessory renal arteries, which help to prevent thrombosis. Moreover, when using this technique, both ischemic and operating times are reduced. By this technique, the ischemic time and the operating time are reduced. Both in live and cadaver donor kidneys, the lower polar arteries were anastomosed to the inferior epigastric artery and upper polar arteries were anastomosed to the superior epigastric arteries. An injection of papaverine and ablation of the sympathetic nerves of these arteries dilate them and prevent post-operative spasm. In conclusion, this technique can be safely and successfully used for renal transplantation with kidneys possessing MRA, and may be associated with a lower complication rate and better graft function than existing techniques [48].

The diseases related to the renal accessory artery include:

(1) *hypertension*: the tenuous and tortuous shape of an accessory renal artery usually results in ischemia in the related area. The local blood flow reduction in the kidney stimulates the macula densa cells and juxtaglomerular cells to increase the synthesis and release of renin, and the subsequent renal vascular [2]. Also Mendelsohn [113] claims that patients with accessory renal arteries constitute 27% of patients with resistant hypertension, and reinforces the importance of denervation of MRA, if present, as a treatment for persistent hypertension. Even though the fall in systemic blood pressure will be smaller than in patients with only the main renal artery, the reduction is still noteworthy: he reports a drop of systemic blood pressure in patients with MRA of 21 mm Hg in 6 months.

(2) *bleeding*: non-traumatic renal bleeding is very rare. As an accessory renal artery is tenuous, the diagnosis and localization of the hemorrhage are difficult. New techniques, such as multidetector CT angiography (MDCTA), which can clearly show the accessory renal arteries, provide better approaches for the accessory renal artery disease [24].

(3) *hydronephrosis*: an accessory renal artery going to the lower pole of the kidney in front of the ureter may compress the ureter and cause hydronephrosis [9, 24].

It is speculated that accessory renal arteries may provide nourishment to renal cell carcinoma. In a CT angiography investigation of 107 patients with renal cell carcinoma, Guan *et al.* [81] found MRA in 11 (10.3%) cases and MRA acting as feeding arteries for the renal cell carcinoma in 5 (4.7%). Catalano *et al.* [114] report that atypical variants of renal vascularization were found in 5 of 42 patients with renal cell carcinoma: 1 patient with triple renal artery and four with double renal arteries, one of whom also presented with

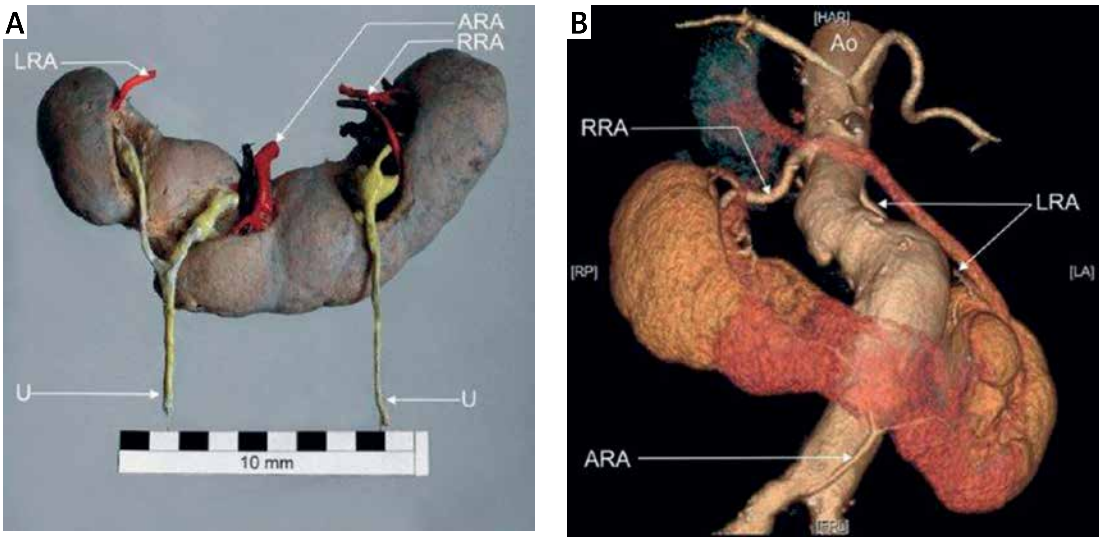

Figure 6. Horseshoe kidney with abdominal arteries: **A** – anatomical dissection (posterior view), **B** – volume rendering technique (VRT) image (anterior view from MDCT angiographies with 3D reconstruction)

Ao – abdominal aorta, ARA – additional renal artery (passing to the isthmus), LRA – main left renal artery, RRA – main right renal artery, U – ureter.

a double renal vein [107]. Moreover, awareness of the presence of accessory renal arteries is also important for the procedures prior to laparoscopic nephrectomy in renal cell carcinoma, which is one of the fundamental methods of cancer treatment; this surgical intervention under such circumstances should be more carefully planned to reduce serious potential surgical complications [81]. Undeniably, knowledge of anatomic variants is of great importance for all procedures in the abdominal region [115].

Coexistence of other atypical variants of vascularization

The occurrence of MRA may also be associated with coexistence of other atypical variants of vascularization, especially connected with the genito-urinary system. For example, MRA can be accompanied by double testicular arteries [116]. Moreover, the presence of accessory renal arteries can also be associated with dislocation of the kidney and its hilum: an ectopic kidney [17]. Yakeishi *et al.* [117] described a case of horseshoe kidney with four surplus renal arteries: one branching from the main renal artery and three following from the abdominal aorta. Furthermore, an accessory renal artery can have a common origin with the inferior mesenteric artery [118]. Based on our experience in the field of anatomical dissection (Figure 6 A) and radiological investigation (Figure 6 B), the horseshoe kidney usually has three renal arteries: two main (left and right renal artery) and one additional, which passes to the isthmus (Figure 6).

Conclusions

The origin, number and topography of accessory (multiple) renal arteries are complex are associated with renal embryogenesis. Knowledge of the variations of the renal arteries has grown in importance with the increasing number of renal transplants, vascular reconstructions and various surgical, urological and radiological techniques performed in recent years. Accessory renal arteries may coexist with other atypical vascularization variants, especially in the genito-urinary system – the testicular arteries, for example. The potential implications of the coexistence of accessory renal arteries and other atypical vascularization and finding the direct factor leading to these anatomical variants require further study.

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Conflict of interest

The authors declare no conflict of interest.

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