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CASE REPORT

Rare Cadaveric Finding of a Horseshoe Kidney in the Presence of an Abdominal Aortic Aneurysm: A Case Report

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Abstract

Horseshoe kidney is a congenital malformation that occurs during gestational development as a result of the fusion of the parenchyma of both kidneys at their lower poles. This leads to the malrotation of the urinary tract and abnormal renal vasculature. The co-existence of horseshoe kidney and abdominal aortic aneurysm is rare; it is found in approximately 1 in 700 autopsied cases and only 0.12% of patients undergo surgical repair of the abdominal aortic aneurysm. Both horseshoe kidney and abdominal aortic aneurysm usually present asymptomatically, and diagnosis may be made by physical examination, incidental findings on imaging, or during post-mortem examination. Furthermore, physicians face a great challenge in the medical and surgical management of these conditions due to the lack of standard guidance. In this article, we present a cadaveric case of a horseshoe kidney accompanied by an abdominal aortic aneurysm. The case report will demonstrate and categorize the horseshoe kidney with the Eisendrath system as type II and III classification, as well as discuss anatomical variations and medical management of the co-existence of a horseshoe kidney and abdominal aortic aneurysm.

Key Words: *Horseshoe kidney; Abdominal aortic aneurysm; Co-existence; Management; Anatomical variation*

Introduction

Horseshoe kidney (HSK) is the fusion of the ascending kidneys that occurs during embryology at the lower pole, with functioning renal lobes present on both sides of the vertebral column [1]. The lobes are connected by a central solid renal parenchyma referred to as the isthmus [1]. Union of the renal masses prevent independent rotation, leading to variations in kidney position and vascular supply [1]. HSK is one of the most common congenital kidney anomalies, yet its prevalence is only 1 in 400 cases [2,3]. Furthermore, the presence of HSK and abdominal aortic aneurysm (AAA) is rarely reported, with an incidence of 1 in every 710 autopsied cases (0.14%) and only 0.12% of patients with this association undergo

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AAA repair [4]. This co-existence of HSK and AAA is rarely detected due to the absence of symptoms and is typically discovered through physical examination, imaging, or postmortem examinations [4]. Given the lack of standard guidance, clinicians face a special challenge in the medical and surgical management of this condition [4].

Case Report

During a routine dissection in a medical gross anatomy course, the authors discovered the co-existence of an AAA and HSK in the abdominal cavity of a 91-year-old male cadaver. Careful inspection and dissection demonstrated complex vasculature surrounding the HSK, resembling a combination of Type II and Type III classification based on the Eisendrath system (Figures 1-4). In a Type II classification, one renal artery supplies each lobe of the HSK, and one branch supplies the isthmus; this classification is the most common type with an incidence of 30% [5]. In a Type III classification, two renal arteries supply each lobe of the HSK with an isthmic branch off the aorta [5]. The present case consists of two right renal arteries, one left renal artery, and one isthmic artery, all branching off the aorta (Figure 2-4) (Table 1).

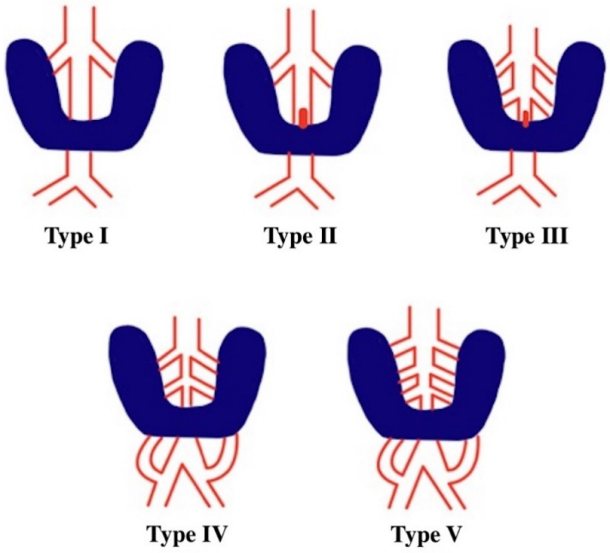

Figure 1) *Eisendrath's five classifications of the horseshoe kidney [6].*

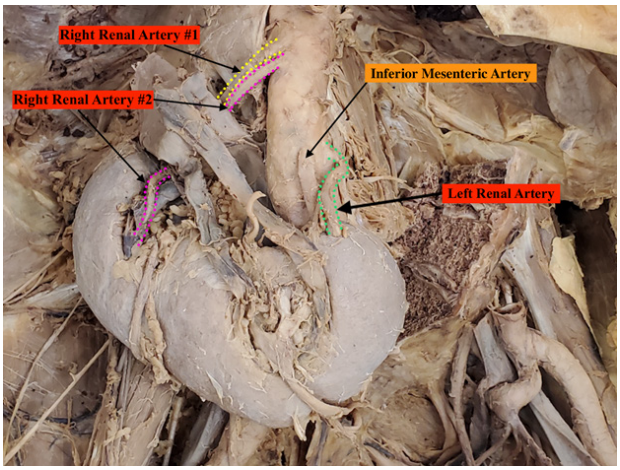

Figure 2) *Anterior view of the horseshoe kidney, illustrating a type II and III Eisendrath's classification [6]. The isthmic artery is not shown in this figure.*

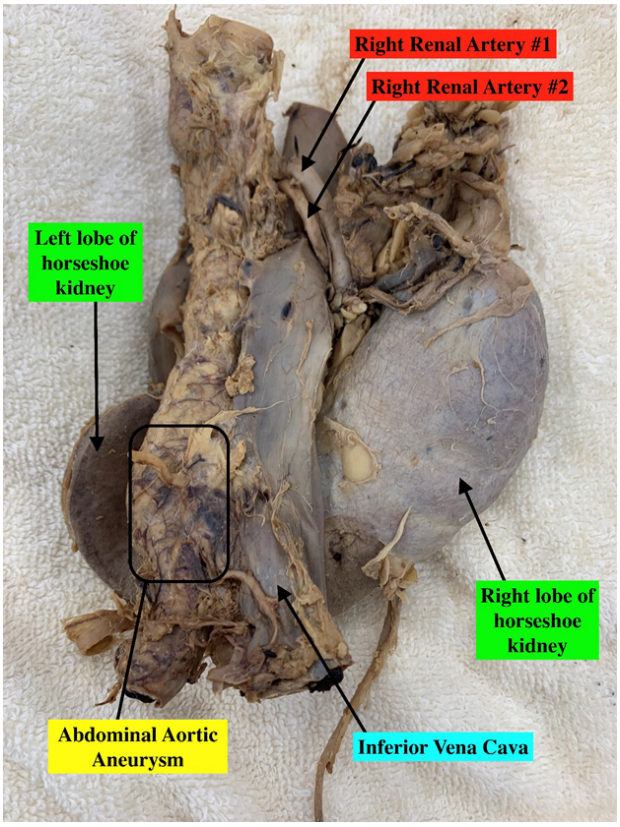

Figure 3) *Posterior view of the horseshoe kidney and abdominal aortic aneurysm with relation to the inferior vena cava. The horseshoe kidney illustrates a type II and III Eisendrath's classification [6]. The isthmic artery and left renal artery are not shown in this figure.*

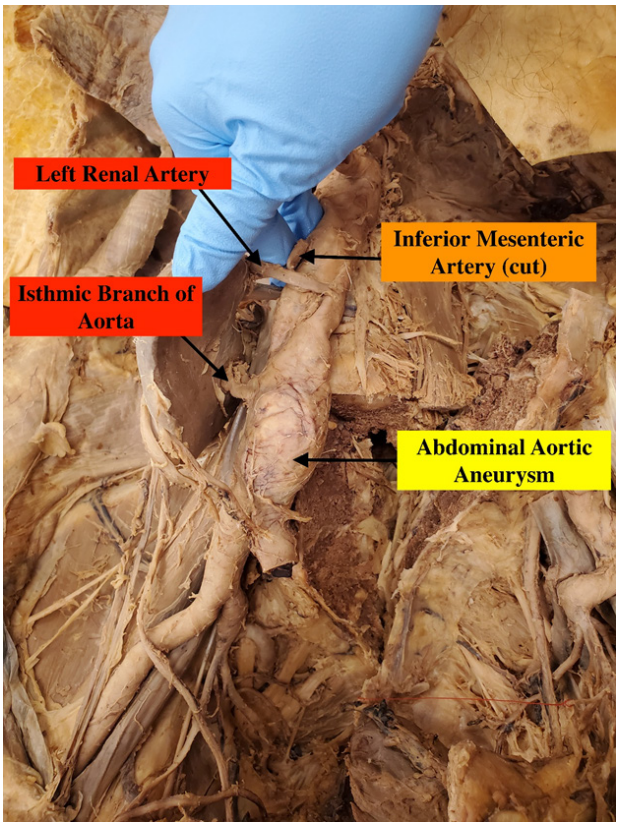

Figure 4) *Anterior view of the left renal and isthmic artery branching off the aorta with the abdominal aortic aneurysm posterior to the left lobe.*

TABLE 1

Classification of Horseshoe Kidney Variations adapted from Eisendrath et al. [6]

Types	Percentage of Occurrence	Description of Renal Artery
Type I	20%	One renal artery on each side of the horseshoe kidney.
Type II	30%	One renal artery on each side with an isthmic branch from the aorta.
Type III	15%	Two renal arteries on each side with an additional branch to the renal isthmus.
Type IV	15%	Two renal arteries on each side and an additional isthmic branch with one or more originating from the iliac arteries.
Type V	20%	Multiple renal arteries arising from the aorta, the mesenteric, and the iliac arteries.

The kidney's thickness was about 1.1 cm with an isthmus of 4.6 cm in width. The right lobe of the kidney was 1.5 cm in length and the left lobe was 3.8 cm in length. The AAA measured 3.5 cm in diameter and was posterior to the left lobe of the HSK (Figure 3). In relation to the body, the HSK is shifted to the right, with most of the lobe located laterally to the inferior vena cava (Figure 3). No symptomatic complications related to the AAA or HSK were reported by the medical donor program regarding the patient's cause of death.

Discussion

Anatomical variation of horseshoe kidney

At about six to eight weeks of gestation, the kidneys undergo a series of rotation and ascension into the posterior abdominal wall. HSKs are formed during the process of ascension where the kidneys come in close contact with each other, eventually fusing into one organ [6]. There are several hypotheses on the mechanism behind the fusion of HSK. Mechanical theory suggests that cephalic movement of the kidneys are restricted by other structures (i.e. umbilical arteries, inferior mesenteric artery (IMA), or growing fetal spine), altering the ascending pathway and allowing for compression of the mutually exclusive kidneys at the metanephric blastema [6,7]. Another theory states that the presence of a teratogen may induce an abnormality in the migration of nephrogenic cells, causing a fusion of kidneys as a parenchymal isthmus [6]. Teratogens affecting the development of kidneys in utero may include, but are not limited to, maternal consumption of alcohol, thalidomide, angiotensin converting enzyme (ACE) inhibitors, cocaine, and corticosteroids [1,8].

The presence of HSK is associated with vascular anomalies. Most HSKs are located around the vertebral levels of L3 to L5, near the origin of the IMA where the isthmus gets trapped during ascension [7]. A singular renal artery or

many accessory renal arteries may stem either from the abdominal aorta, the IMA, or the iliac arteries [7,9]. Most of the blood supply to the HSK is through the branches from the abdominal aorta below the isthmus [6,7]. The number of renal arteries to the HSK can have clinical significance during transplant surgery or AAA repair [6, 9, 10]. In the present case, the HSK was categorized as a Type II and III Eisendrath classification with two right renal arteries, one left renal artery, and one isthmic branch, all originating from the abdominal aorta (Figure 1-4).

Prevalence of abdominal aortic aneurysm and horseshoe kidney

AAA is defined as a pathological dilation of the abdominal aorta with a diameter > 3.0 cm that is commonly found between the renal arteries and the aortic bifurcation [11]. A meta-analysis of 56 studies ranging from 1988 to 2013 reported an AAA prevalence of 2.2% in America; gender difference of 6.0% in males and 1.6% in females; age difference of 1.3% in 55-64 years, 2.8% in 65-74 years, 1.2% in 75-84 years, and 0.6% in ≥ 85 years; aortic diameter of 3.3% in 30-39 mm, 0.7% in 40-49 mm, and 0.4% in ≥ 50 mm [12]. In addition, postmortem studies showed that 95% of deaths from ruptured AAA occurred in patients at or above age 65 years [12].

Unfortunately, the prevalence of AAA in the presence of HSK is rarely reported. According to O'Hara et al., only 19 patients were reported with co-existence of HSK and AAA at Cleveland Clinic from 1960 to 1991 [13]. Of the 19 patients, 17 male and 2 female patients (mean age: 67 years; mean AAA diameter: 6.1 cm) underwent surgical repairs of the aneurysm via transperitoneal (16 patients) and retroperitoneal (3 patients) approaches [13]. Preoperative diagnosis with computed tomography (CT) and intravenous (IV) pyelography were most reliable in successfully identifying the HSK in 88% and 90% of patients, respectively [13]. These results showed 5 patients with two renal arteries in normal anatomic position while the rest had some form of anomalous bloody supply [13]. Prior to the diagnosis of this co-existing condition, patients had comorbidities of diabetes (11%), hypertension (37%), coronary artery disease (53%), and history of cigarette smoking (53%); moreover, 68% of the patients had normal preoperative renal function and only 3 of the 6 patients who had abnormal renal function (blood urea nitrogen level > 40 mg/dL and serum creatinine > 1.4 mg/dL) required postoperative dialysis [13].

Management of the co-existence of abdominal aortic aneurysm and horseshoe kidney

With few reported clinical cases, there are no established guidelines for the medical or surgical management of individuals with this rare co-existing condition. A proposed nonsurgical management is to slow the progression of the AAA. Current guidelines of the United States Preventive Services Task Force (USPSTF) recommend a one-time screening for AAA with ultrasonography (US) in men aged 65-75 years who have ever smoked [11,12]. Smoking cessation is beneficial as research shows that smoking causes an increased AAA diameter growth rate of 0.4 mm per year [14]. As for pharmacological treatment, antihypertensive medications, such as beta blockers and ACE inhibitors, can help control further vascular injury but provide little benefit in slowing the enlargement of the AAA [14].

After attempting nonsurgical interventions, a patient may undergo surgical repair of the AAA once it reaches a threshold of 5.5 cm in diameter [15]. This threshold is thought to provide the most successful outcome for both pre- and post-operative management [15]. The

goal of the surgery is to minimize the risks of renal insufficiency, vascular hypertension, and ruptured aneurysm [14,15]. Pre-operative blood work, including renal function, and a CT angiography or IV pyelography are recommended to provide information on any anatomical variations and blood supply to the HSK and AAA that may interfere with graft placement [13,14].

Two main approaches of surgical repair are available-open and endovascular. An open AAA repair consists of the insertion of a synthetic graft at the aneurysmal segment via a transperitoneal or retroperitoneal approach [16]. This open approach was the traditional standard for AAA repair until the introduction of endovascular aneurysm repair (EVAR), a minimally invasive procedure that inserts a stent graft into the aneurysm via femoral artery access [16]. The EVAR has shown marked improvement in peri-operative morbidity, mortality, and recovery compared to the traditional open repair; in fact, the EVAR is now considered the primary surgical repair for AAA [16]. However, long-term outcomes did not show significant difference in total mortality or aneurysm-related mortality between EVAR and open repair [15,16]. A large, randomized clinical trial conducted from 1999 through 2004 at 37 hospitals in the United Kingdom evaluated 1,252 patients with large AAA (≥ 5.5 cm in diameter) who underwent either an EVAR or open repair [16]. Patient outcome was followed until 2009 to evaluate rates of death, graft-related complications, and reinterventions. Overall, results showed that the EVAR group had a 30-day operative mortality of 1.8% versus the 4.3% in the open-repair group; however, the benefit of EVAR was lost after several patients required emergency open repair due to fatal endograft ruptures [16].

Based on these studies, the proposed medical treatment for the patient in this case report would be to prevent vascular injury through medications, slow the progression of the AAA via smoking cessation, if applicable, and monitor AAA diameter on imaging. Once the AAA reaches a diameter of 5.5 cm, the patient would benefit more from an open repair than an EVAR because an open repair provides better visualization and control of the anatomical variations of the HSK and AAA.

Conclusion

The co-existence of HSK and AAA is a rare and mostly asymptomatic condition. There is no standard protocol on the medical or surgical treatment for this condition. A proposed management includes early detection of the AAA with imaging and reduction of risk factors to slow the progression of the AAA before exploring surgical options. Nevertheless, more research is needed to evaluate the management of the co-existence for HSK and AAA to set a universal guideline.

Ethical Approval

Institute review board review and approval was not required for this cadaver case report. The donor at the focus of this case report donated their body to the University of Texas Southwestern (UTSW) Willed Body Program. Consent for anatomical dissection and research was obtained by UTSW as part of the donor application packet. Edward Via College of Osteopathic Medicine (VCOM) Division of Anatomical Sciences obtained permission for education and research from the UTSW Willed Body Program.

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