Comparison between Different Techniques for Management of Renal Stones in Horseshoe Kidneys

Thesis

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Abdelrahman Nazmy Abbas Hatata

List of Contents

List of Tables

List of Figures

List of Figures cont...

INTRODUCTION

Horseshoe kidney (HSK) is the most common renal fusion anomaly, affecting about 0.25% of the newborn ***(G.V. et al., 2003).*** In most cases, fusion occurs in the lower pole of the kidney, leading to failure of ascent, and malrotation of the kidneys. Ureter insertion is usually superior and lateral, leading to impaired drainage of the collecting system, as well as urinary stasis, and concomitant stone formation. Urolithiasis is the most common complication of horseshoe kidney, with a reported incidence of 21–60% ***(Yohannes and Smith, 2002).*** However, various methods are indicated for the management of stones associated with HSK, including percutaneous nephrolithotomy (PCNL) and extracorporeal shock wave lithotripsy (ESWL), which are the most commonly used methods ***(G.V. et al., 2003; Yohannes and Smith, 2002; K.Z. et al., 2003)*** More recently, flexible ureteroscopy (URS), laparoscopic pyelolithotomy, laparoscopic assisted PCNL and robotic pyelolithotomy are also effective ***(E.S. et al., 2015; Ölçücüoğlu et al., 2014).***

The majority of the published papers that concern the treatment of HSK stones emphasize on one modality of management, discussing its safety and efficacy. Therefore, we present our single tertiary center experience of >15 years involving different management modalities for HSK stones, as a means to demonstrate and emphasize the outcomes of treatment with these modalities.

We will compare between laparoscopic pyelolithotomy, laparoscopic assisted PCNL, PCNL and flexible ureteroscopy for management of renal stones in horse shoe kidneys.

AIM OF THE WORK

To report the operative management, intra and post operative complications and subsequent stone-free rates of patients with urolithiasis in a horseshoe kidneys.

Chapter 1

EMBRYOLOGY OF THE HORSESHOE KIDNEY

There are two theories about HSK embryogenesis. The classic theory of mechanical fusion proposes that during the metanephric stage (4th week of gestation, 5–12 mm caudal to rostral CR length) and while the kidneys are still in the pelvis and in close proximity, their lower poles enter in contact and fuse in the midline, forming a HSK with a fibrous isthmus. This fusion can be attributed to abnormal flexion or growth of the developing spine and pelvic organs that causes the fusion of the nephrogenic blastemas of the immature kidneys that do not possess renal capsule. Normally, during the 7th and 8th weeks, the kidneys migrate out of the pelvis and at the same time rotate so that the anteriorly facing renal pelvis turns medially ***(Yoshinaga et al., 2002).***

In contrast, when HSK ascends, at the level of the lower lumbar vertebrae, the inferior mesenteric artery (IMA) prevents the ascent and the kidney is trapped in the midabdomen. The isthmus is therefore blocked under the IMA and as a result HSK is malrotated and each renal pelvis remains anteriorly at a lower lumbar position ***(Sadler et al., 2010)*** (Fig.1). It has also been proposed that HSK can result as a teratogenic event that involves the abnormal migration of posterior nephrogenic cells that form a parenchymal isthmus ***(Yoshinaga et al., 2002).***

This could possibly explain the increased incidence of malignancies associated with HSK, such as Wilms' tumor and HSK carcinoid ***(O'Brien et al., 2008).***

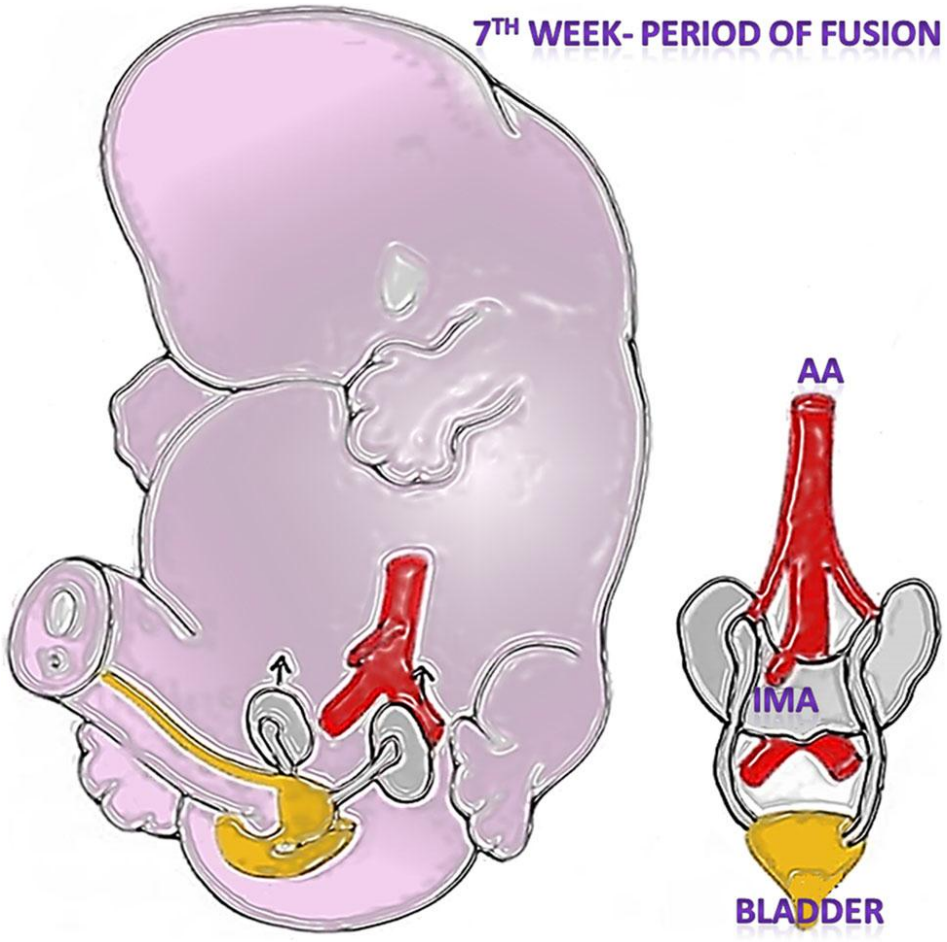

Figure 1: Schematic of the embryogenesis of the horseshoe kidney. The lower poles of the two kidneys touch and fuse as they cross the iliac arteries. The ascent (arrows) is stopped when the fused kidneys reach the inferior mesenteric artery (IMA), AA abdominal aorta ***(Natsis et al., 2013).***

Congenital anomalies associated with horseshoe kidney:

Glenn (1959) reported an incidence of 78.9% of associated congenital abnormalities in stillborn fetuses and infants and lower incidences of 28.5 and 3.5 % in children and adults, respectively.

This finding suggests that HSK is incompatible with long-term survival and patients may die at birth or in early infancy. Patau and Gardner syndromes (trisomy 13 and deletion q15q22) have been reported to coexist with HSK ***(Kravtzova et al., 1975; Herrera et al., 1986).***

It has also been mentioned that 20 % of Down and Edwards (trisomies 21 and 18, respectively) and 60 % of Turner syndromes possess a HSK ***(Araki et al., 1987; Kravtzova et al., 1975).***

The HSK has rarely been related to oral–cranial–digital syndrome and other skeletal abnormalities such as kyphosis, scoliosis and macrognathia, as well as neurological abnormalities, which include encephalocele, meningomyelocele and spina bifida ***(Mandell et al., 1996).***

Other associated abnormalities include supernumerary kidneys with horseshoe elements, undescended testis, septate vagina and hypospadias ***(O'Brien et al., 2008).***

Chapter 2

ANATOMY OF THE HORSESHOE KIDNEYS

Morphological classification of HSK is based on the renal shape based on the appearance of two fused renal masses ***(Pawar et al., 2018).*** The U-shaped HSK is formed by medial fusion. In this configuration, kidneys may be placed in asymmetrical position, U-shaped kidneys are observed in 42 % of incidences of HSK. Another type, which is a result of lateral fusion relative to the vertebral column, is called L shaped HSK. This type occurs in 58 % of incidences of HSK ***(H. et al., 2008).*** The asymmetry is more commonly left dominant (70 %) ***(B et al., 2009).***

In over 90 % of cases, fusion between the kidneys occurs at the lower pole; however, we can also observe upper pole fusion resulting in "inverted HSK" (5-10 %).

Kidneys ascend from the pelvis to the mesogastrium during the seventh week of fetal development and reach their final position by the end of the eighth or ninth week ***(T. K., J., and S.A. 2016).***

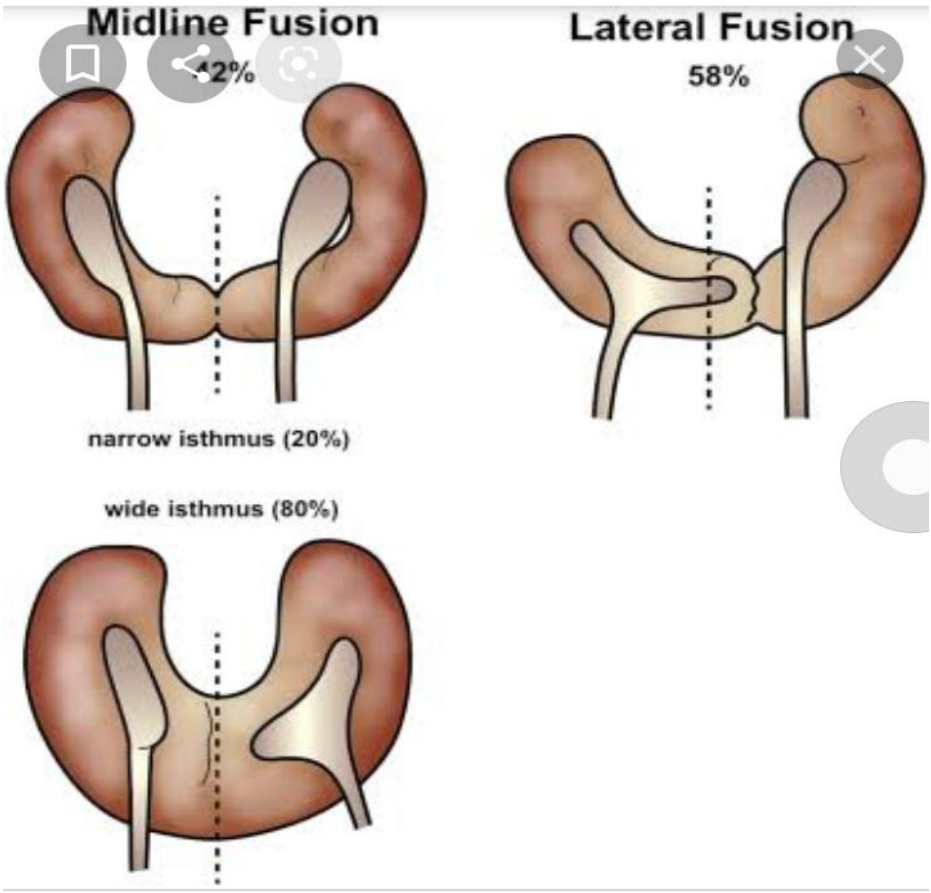

Figure 2: Morohological classification of horseshoe kidney ***(N. K. et al., 2014).***

HSK is found more commonly in a lower position than a normally located kidney because the isthmus is prevented from ascending to its normal position by the inferior mesenteric artery (IMA) ***(O'Brien et al., 2008).*** The isthmus usually lies under the IMA origin anterior to the large vessels at the level of the third to the fifth lumbar vertebra ***(Decter 1997).*** Less commonly, the isthmus is situated posterior to those vessels or runs between them.