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Case Report

Antenatal Diagnosis of Cystic Renal Dysplasia: A Case Report

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Abstract

Multicystic dysplastic kidney (MCDK) is a prevalent renal condition in children, marked by numerous cysts within the kidney that do not communicate with each other and contain minimal to no functional renal tissue. Prenatal diagnosis relies on ultrasound and MRI. Abnormalities detected prenatally, especially those affecting the contralateral kidney, are associated with adverse neonatal outcomes, emphasizing the importance of accurate prenatal assessment for prognosis determination. We present the case of a 42-year-old patient in whom the prenatal diagnosis of unilateral cystic renal dysplasia was made based on antenatal ultrasound and fetal MRI.

Keywords: *Multicystic; Kidney; Dysplastic; Antenatal; MRI; Ultrasound*

Introduction

Multicystic dysplastic kidney (MCDK) is a prevalent renal condition in children, marked by numerous cysts within the kidney that do not communicate with each other and contain minimal to no functional renal tissue. It stands as the most frequent cause of renal cystic disease in neonates [1].

MCDK arises as a developmental anomaly triggered by urinary tract obstruction during embryonic development. This obstruction disrupts normal metanephric-mesenchymal differentiation, resulting in MCD [1].

MCDK can manifest unilaterally or bilaterally, with the bilateral presentation being extremely rare (less than 1 in 10,000 cases) and incompatible with life due to the absence of functioning renal tissue and associated anomalies [2].

Prenatally identified findings, especially with contralateral renal abnormalities, are associated with adverse neonatal outcomes, emphasizing the importance of accurate prenatal assessment for prognosis [3]. Prenatal ultrasound (US) is the primary method for visualizing fetal MCDK, while fetal magnetic resonance imaging (MRI) has been increasingly utilized for diagnosing urinary tract anomalies [3].

Case Report

We report the case of a 42-year-old patient, G2P2, with no particular medical history, no known genetic history, and no specific symptoms. An obstetric ultrasound performed at 28 weeks of gestation as part of routine pregnancy monitoring revealed an oligohydramnios (Figure 3), with the left renal compartment showing small, well-defined rounded formations with regular contours, pure anechoic echostructure,

non-communicating with each other and without identification of renal parenchyma (Figure 1). The contralateral kidney appeared normal (Figure 2).

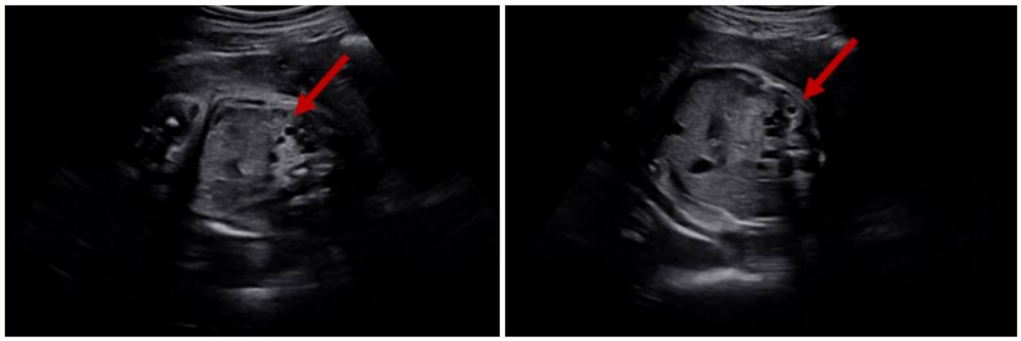

Figure 1: *Fetal ultrasound showing a left renal compartment with cystic formations: Cystic renal dysplasia (Red arrow).*

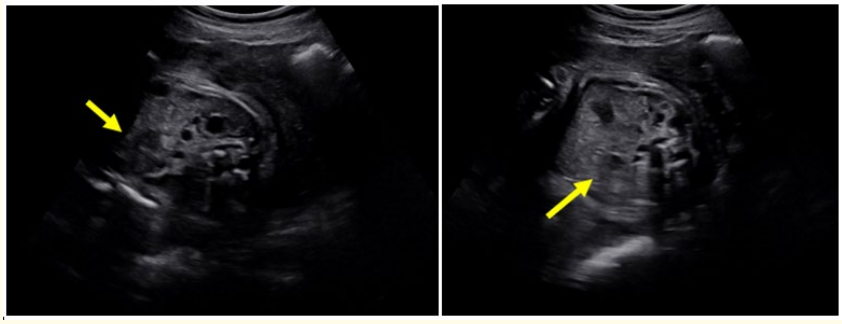

Figure 2: *Fetal ultrasound showing the right kidney, which is without anomaly (Yellow arrow).*

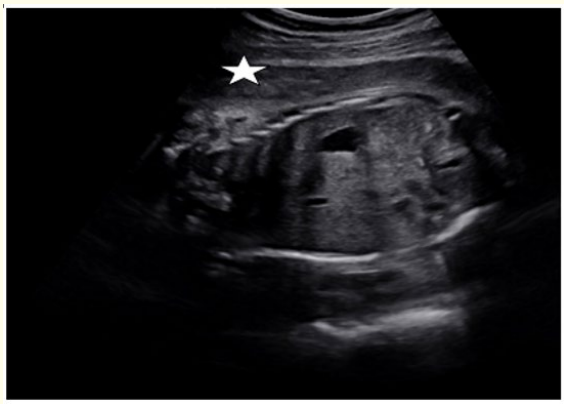

Figure 3: *Fetal ultrasound showing oligohydramnios, with the placenta located posteriorly (Star).*

The findings were consistent with left-sided cystic renal dysplasia.

MRI was performed for better lesion characterization, and to search for any associated malformations, using a protocol involving sections through the three planes of space with T1-weighted, T2-weighted, and fat-suppressed sequences. The MRI also revealed a left renal compartment occupied by multiple fluid-filled formations with T1 hyposignal, T2 hypersignal, not suppressed on fat suppression, rounded, well-defined contours, non-communicating with each other, measuring 32x35x30 (HxTxAP) (Figure 4-6). These formations come into contact with the anterior abdominal wall. The contralateral kidney was without anomaly (Figure 4), measuring 32x35x30 (HxTxAP) Oligohydramnios was also associated with it. There were no other associated malformative abnormalities intra- or extrarenal.

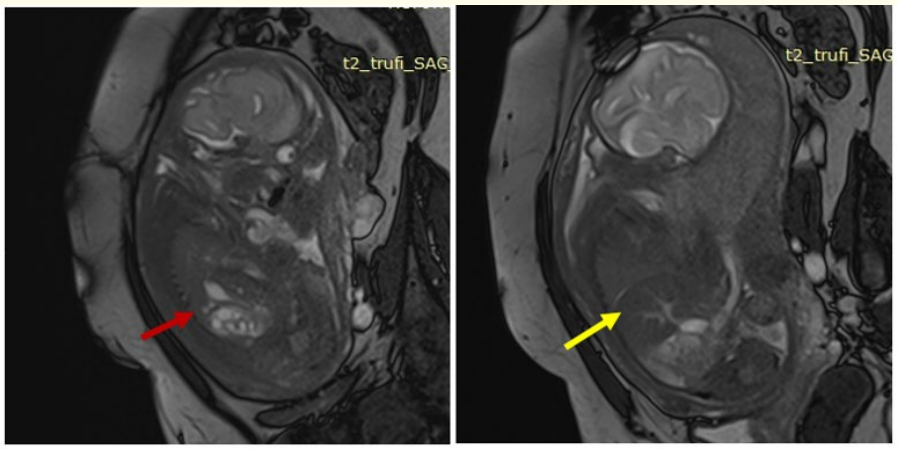

Figure 4: *MRI T2-weighted sequence, sagittal sections showing left renal dysplasia (Red arrow), with a right kidney of normal appearance (Yellow arrow), and oligohydramnios.*

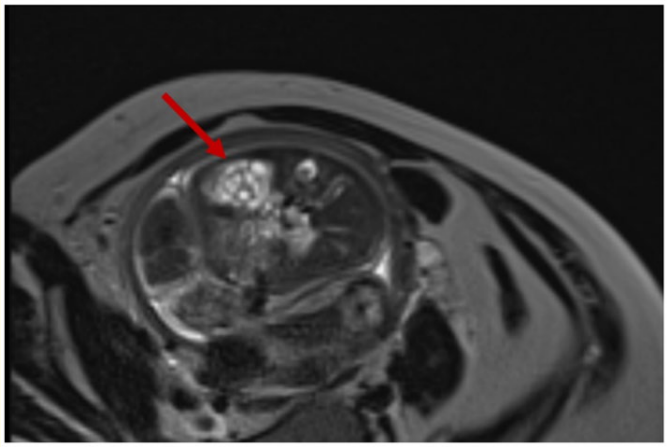

Figure 5: *MRI T2-weighted sequence, axial section showing left renal dysplasia (Red arrow).*

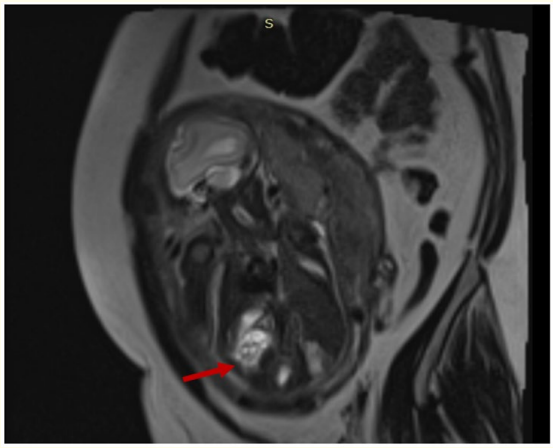

Figure 6: *MRI T2-weighted sequence, coronal section showing left renal dysplasia (Red arrow).*

The diagnosis of left cystic renal dysplasia was confirmed.

Discussion

The Potter classification of renal cystic diseases has been replaced by a classification focusing on genetic or nongenetic origins. Genetic conditions include well-known disorders like autosomal recessive (ARPKD) and dominant (ADPKD) polycystic kidney diseases, as well as newer ones like glomerulocystic kidney disease (GCKD), syndromic medullary cystic dysplasia and nephronophthisis-medullary cystic dysplasia complex. The previous classification divided cystic kidneys into four types: infantile polycystic kidney disease (now ARPKD), cystic dysplastic kidney disease (now MCDK), adult polycystic kidney disease (ADPKD), and obstructive dysplasia. Among nongenetic cystic diseases, obstructive dysplasia and MCDK are most common [4].

While ultrasonography (US) is the main diagnostic tool for MCDK, it has limitations. US may struggle with accurately diagnosing bilateral kidney anomalies in the presence of oligohydramnios. However, magnetic resonance imaging (MRI) isn't affected by these factors. MRI also isn't hindered by conditions like maternal obesity, oligohydramnios, or the presence of the fetal pelvic bone [3].

On ultrasonography, the fetal kidney becomes visible as early as the first trimester, appearing as an echogenic nubbin near the lumbar spine. Its size increases over gestation, measurable by its sagittal long axis. Normograms correlating gestational age with kidney size are available. Corticomedullary differentiation becomes evident during midtrimester sonographic evaluation, with the cortex becoming echogenic and the medulla hypoechoic. Throughout pregnancy, renal cortical echogenicity diminishes relative to the liver and spleen. Initially hyperechoic, it becomes isoechoic in late second and third trimesters until around 32 weeks when it should appear hypoechoic compared to the liver and spleen [4].

Sonographically, multicystic dysplastic kidney (MCDK) is characterized by kidneys filled with multiple cysts of various sizes, resulting in enlarged kidneys and increased abdominal circumference. The cysts obscure renal borders and appear as large anechoic structures. Additionally, increased echogenicity in the parenchyma is observed due to connective tissue proliferation. In unilateral cases, the contralateral kidney may also be enlarged without oligohydramnios, allowing proper lung development. However, bilateral MCDK may lead to oligohydramnios or anhydramnios, resulting in pulmonary hypoplasia due to reduced amniotic fluid levels [2].

MRI provides precise identification of bilateral kidney anatomy. It can detect various-sized cysts, minimal or absent renal parenchyma typical of MCDK on SSTSE sequences, and accurately diagnose associated urinary tract abnormalities, facilitating prenatal counseling. Unilateral MCDK often accompanies other urinary tract anomalies, including ipsilateral ureteral issues (megaureter, ureterocoele), contralateral kidney abnormalities (UPJO, renal agenesis, renal hypoplasia, bilateral MCDK), and lower urinary tract abnormalities [3].

Different cystic renal diseases may appear similar on fetal ultrasound. However, MRI can accurately distinguish multicystic dysplastic kidney (MCDK) from other common cystic renal diseases and hydronephrosis. In MRI's coronal plane, the connection between the renal pelvis and dilated ureter distinguishes hydronephrosis from MCDK, as MCDK cysts are not interconnected. Autosomal recessive polycystic kidney disease (ARPKD) typically involves both kidneys bilaterally and lacks dysplastic renal parenchyma. Fetal MRI shows enlarged kidneys with high signal intensity and tiny indistinct renal cysts in ARPKD, whereas MCDK cysts have a random distribution and irregular outline [3].

Published studies [5,6] suggest that utilizing diffusion-weighted imaging (DWI) sequences is feasible for assessing both normal renal function and various kidney diseases [3].

In DWI sequences, normal renal parenchyma presents a bright signal, facilitating differentiation between ectopic kidney and renal agenesis based on characteristic high signal intensity. Additionally, DWI can indicate declining function in a dysplastic kidney through a reduction in cortical signal compared to normal renal parenchyma [3].

Prognosis is influenced by the presence of extrarenal and contralateral renal abnormalities [1].

Up to 60% of multicystic dysplastic kidney (MCDK) cases can undergo spontaneous resolution, typically within the first three years of life but may take up to 10 years [1].

Most clinicians opt for conservative management, employing serial ultrasonography for follow-up until spontaneous involution occurs [1].

The risk of hypertension and malignant transformation in MCDK is minimal compared to the general population, questioning the necessity of preventive surgical removal. Surgery may be indicated in cases of recurrent urinary tract infections, sudden or progressive kidney enlargement, and persistent flank pain [1].

Conclusion

Currently, multicystic dysplastic kidney (MCDK) is primarily diagnosed antenatally [1]. Prenatal ultrasound (US) serves as the initial method for visualizing fetal MCDK [3].

Foetal MRI can supplement prenatal US by providing additional diagnostic insights, particularly in cases where ultrasound image quality is compromised or altered. This additional information from MRI can impact prenatal counseling and decision-making [3].

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