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Renal Development in the Pregnancy and the Preterm Child

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Introduction

Congenital abnormalities of the kidney are common and frequently detected during pregnancy. Premature birth and prenatal trauma may also affect renal development. Here, we go over the mechanisms underlying aberrant renal development and talk about how to assess the kidneys of foetuses and premature babies.

Description

Normal growth of the kidney

The first trimester of pregnancy is when the human kidney develops. The pronephros, mesonephros, and metanephros are the three phases of mammalian kidney development. As they develop, the pronephros and mesonephros effectively involute. The final functional mammalian kidney, the metanephros, develops.

At 9-10 weeks of gestation, the first glomeruli begin to develop. There is a substantial rise in nephrons in the late second and third trimesters [1]. Nephron development is finished between 32 and 36 weeks of pregnancy. At around 16-20 weeks of gestation, foetal urine production increases significantly and can reach 300 mL/kg foetal weight/day.

Congenital kidney and urinary tract anomalies (CAKUT)

One of the most common major birth defects, accounting for 20-30% of all major birth defects, are congenital abnormalities of the kidney and urinary system. Some estimates suggest the frequency of renal anomalies at roughly one in 500 live births, however the prevalence of anomalies frequently relies on the location (for example, tertiary care centres often have a greater prevalence of anomalies) [2].

Vesicoureteral Reflux (VUR) or upper or lower urinary tract obstruction may be the cause of foetal hydronephrosis, which may be temporary (discussed in detail below). Renal cysts, which can be bilateral or unilateral, are the second most common anomaly, followed by renal agenesis (unilateral > bilateral). Cysts or hydronephrosis may be present along with renal abnormalities [3].

Cellular processes causing CAKUT

When the ureteric bud fails to form or fails to reach/induce the metanephric mesenchyme, resulting to apoptosis, renal aplasia (agenesis), the most severe renal abnormality, occurs. If the metanephric mesenchyme does not receive growth factors generated by the ureteric bud, such as glialderived neurotrophic factor (GDNF), renal agenesis will result. Severe oligohydramnios (formerly Potter's) sequence and foetal or perinatal death are the results of bilateral renal agenesis [4].

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The upper ureter typically attaches to the bladder in an ectopic site and is blocked, while the lower pole ureter typically exhibits VUR. These are the hallmark features in full duplications. VUR, UVJ, and ureteropelvic junction (UPJ) blockage are all related to abnormalities in ureteric bud development and branching, even in the absence of duplications.

Genetic issues causing CAKUT

The most common monogenic aetiology of CAKUT is autosomal dominant mutations in HNF1B. A transcription factor called HNF1B aids in the segmentation of the nephron and the formation of the ureter. HNF1B genetic variations are linked to a wide range of CAKUT, from renal hypoplasia to non-functioning multicystic dysplastic kidneys, most likely because of its numerous roles in renal development. Hyperuricemia with hypomagnesemia and maturity-onset diabetes of the young (MODY), as well as renal cysts and diabetic syndrome, are also linked to HNF1B variations. The expression of sodium/potassium ATPase subunits [5], which alter renal absorption of magnesium in the distal convoluted tubule, is likely what causes the hypomagnesemia.

In sporadic CAKUT, autosomal recessive genetic mutations most likely happen at a low frequency. These included the Fraser syndrome (CAKUT)-related gene abnormalities in FRAS/FREM. Heparin sulphate, which binds most growth hormones, is modified by the protein products of these genes.

Negative effects of the prenatal environment on renal development

Another cause of kidney developmental abnormalities is maternal drug exposure. The renin-angiotensin system promotes the branching of the ureteric bud, and maternal usage of angiotensin-converting enzyme inhibitors or angiotensin receptor blockers is linked to renal agenesis and abnormalities. Glucocorticoids, mycophenolate mofetil, antiepileptic medicines, and cyclophosphamide are a few other pharmaceuticals that may be linked to renal abnormalities.

Impact of premature birth on renal development

Postnatal exposures to nephrotoxins may have an impact on renal development in addition to prematurity. According to a study of very low birth weight neonates in the neonatal critical care unit, 90% of the babies were exposed to at least one nephrotoxic substance, and the average baby was exposed to nephrotoxins for approximately two weeks. After birth, gentamicin was given to 30% and amphoteracin to 50% of the preterm infants. Indomethacin, another nephrotoxin, was given to 50% of patients as a non-surgical way to treat a patent ductus arteriosus. Neonatal renal blood flow severely depends on prostaglandin-induced vasodilation of afferent arterioles since preterm neonates have high renin-angiotensin levels [6].

Renal pathology can be diagnosed in utero

As early as 9 to 12 weeks of gestation, prenatal ultrasonography can detect foetal kidneys in the paraspinal area and can show that they are lobular in appearance. The average kidney is 1 cm long at 12 weeks, and by 20 weeks, it is 2.7 cm long on average. Additionally, corticomedullary differentiation takes place between 15 and 20 weeks, and by 20 weeks, an echogenic cortex with the hypoechogenic, dark renal pyramids of the medulla should be visible. By 20 weeks, the renal cortex is typically 20 weeks less echogenic than the liver. Though glomerular filtration starts at nine weeks into pregnancy, it doesn't make a major contribution to amniotic fluid until 16 weeks.

Dilation during the second trimester is typically a sign that a follow-up ultrasound should be performed during the third trimester. Grading scales take into account the presence of concomitant renal parenchyma thinning and the

presence of major e or, in more severe cases, minor renal calyces e dilatation due to hydronephrosis.

Fetal pyelectasis can be caused by the urethral/bladder outflow obstruction mentioned above, the VUR, the UPJ obstruction, the non-obstructed dilated ureters (mega-ureter), or temporary hydronephrosis. The majority (up to almost 90%), according to studies, of individuals with foetal pyelectasis in the second trimester have minor hydronephrosis, and 80% of them won't require surgery [7]. Most of these cases will have short-lived hydronephrosis that will go away.

A postnatal ultrasonography conducted in the first few days of life may understate the degree of hydronephrosis and be misleadingly encouraging if urine output is measured in the first 24 to 48 hours after birth. Therefore, between 7 and 10 days after birth is the ideal time for a postnatal ultrasound to evaluate the severity and duration of foetal pyelectasis, with the exception of the few cases mentioned above. It is advised to repeat the ultrasound at 4–6 weeks of age when the postnatal ultrasound was initially normal.

Conclusion

Leading congenital abnormalities include CAKUT. Problems in ureteric bud outgrowth and branching as well as/or defects in epithelial differentiation from mesenchymal renal progenitors are the main causes of CAKUT. CAKUT is likely influenced by both environmental and genetic factors. Genetic studies of animals and people have advanced our understanding of the cellular and molecular mechanisms regulating renal and genitourinary tract development, although single gene abnormalities are probably linked to a small percentage of non-syndromic CAKUT. Though not all birth abnormalities can always be found, prenatal ultrasonography is useful for finding CAKUT.

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Conflict of Interest

There are no conflicts of interest by author.

References

1. Lenz, Oliver, Sharon J. Elliot and William G. Stetler-Stevenson. "Matrix metalloproteinases in renal development and disease." *J Am Soc Nephrol* 11 (2000): 574-581.
2. Lancaster, Madeline A. and Juergen A. Knoblich. "Organogenesis in a dish: modeling development and disease using organoid technologies." *Sci* 345 (2014): 1247125.
3. Scolari, Francesco and Pietro Ravani. "Atheroembolic renal disease." *Lancet* 375 (2010): 1650-1660.
4. Thiery, Jean Paul, Hervé Acloque, Ruby YJ Huang and M. Angela Nieto. "Epithelial-mesenchymal transitions in development and disease." *Cell* 139 (2009): 871-890.
5. Rosenblum, Stacy, Abhijeet Pal and Kimberly Reidy. "Renal development in the fetus and premature infant." *Semin Fetal Neonatal Med* 22 (2017): 58-66.
6. Acloque, Hervé, Meghan S. Adams, Katherine Fishwick and Marianne Bronner-Fraser, et al. "Epithelial-mesenchymal transitions: the importance of changing cell state in development and disease." *J Clin Invest* 119 (2009): 1438-1449.
7. Ingelfinger, Julie R., and Lori L. Woods. "Perinatal programming, renal development, and adult renal function." *Am J Hypertens* 15 (2002): 46S-49S.

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