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Fetal renal artery blood velocimetry in multicystic kidney disease

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ABSTRACT

Color flow mapping and Doppler velocimetry were used to measure impedance to flow in both renal arteries of 11 fetuses with unilateral multicystic kidney disease at 19–34 (mean = 26) weeks' gestation. In all cases, the pulsatility index in the renal artery of the multicystic kidney was higher than that of the contralateral normal kidney; the mean pulsatility index in the arteries of the affected kidneys was significantly higher than the normal mean for gestation.

INTRODUCTION

Color flow mapping and pulsed wave Doppler velocimetry studies have established that, in normal pregnancy, impedance to flow in the fetal renal arteries decreases with gestation[1]. It was suggested that the decrease in impedance is a consequence of renal angiogenesis and an increase in the total arteriolar cross-sectional area with gestation.

The term 'multicystic kidney' is generally used to describe a specific entity in which the normal renal parenchyma is replaced entirely by multiple cysts of varying size. The normal pelvic and calyceal architecture is absent and the ureter is almost invariably atretic[2]. In unilateral multicystic kidney disease, postnatal angiographic and radionucleotide studies have demonstrated that perfusion and isotope uptake of the abnormal kidney are reduced or even absent. Furthermore, morphological studies have shown that, although some multicystic kidneys have a definite renal pedicle, the majority show only multiple small vessels or no vessels at all[3 7]. The aim of the present study was to determine whether abnormal perfusion of multicystic kidneys is present antenatally.

PATIENTS AND METHODS

In 11 fetuses with postnatally confirmed unilateral multicystic kidney disease, impedance to flow in both renal arteries was measured using color flow mapping and pulse wave velocimetry (Aloka color Doppler SSD-680; Aloka Co, Japan, with 3.5 MHz curvilinear transducer) at 19–34 (mean = 26) weeks' gestation. In all cases the contralateral kidney was normal and there were no other defects.

Real-time ultrasonography was used to obtain a longitudinal view of the fetal kidneys. Subsequently, with color flow mapping, the renal arteries could be visualized branching from the abdominal aorta above the origin of the common iliac arteries[1]. The Doppler gate was placed on the proximal part of each renal artery and flow velocity waveforms were obtained (Figure 1). The high-pass filter was set at 100 Hz and the angle of insonation was always less than 30°. The pulsatility index was measured in four consecutive flow velocity waveforms, and the values were averaged[8].

The Wilcoxon test was applied to examine whether the difference in pulsatility index between the multicystic

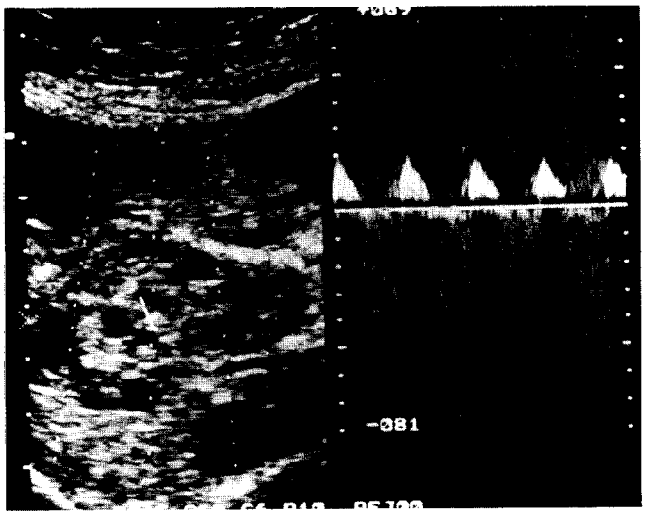

Figure 1 Flow velocity waveforms from the renal artery of a multicystic kidney, demonstrating high impedance to flow with complete absence of diastolic frequencies

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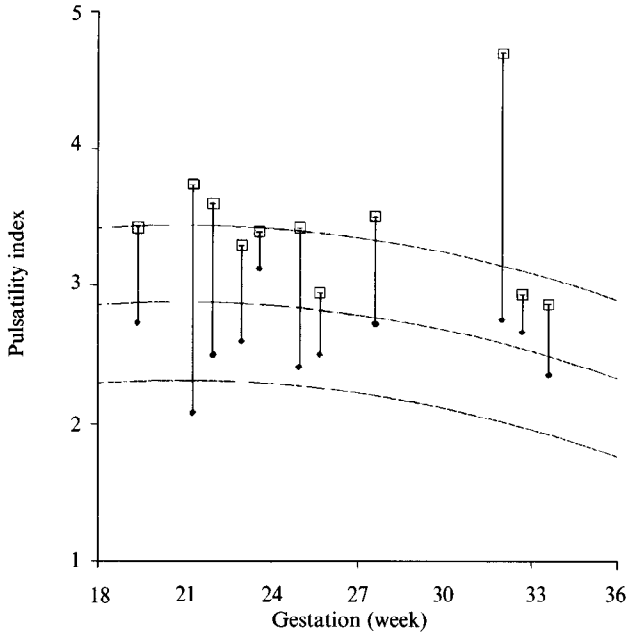

Figure 2 Pulsatility index (PI) in the fetal renal artery in multicystic (□) and normal (●) kidneys in fetuses with unilateral renal disease, plotted on the reference range (mean, 10th and 90th centiles) for gestation

kidneys and the contralateral normal kidneys was significant. In each case, the number of standard deviations (SDs) by which the measured renal artery pulsatility index differed from our normal mean for gestation was calculated (Δ values). Two-tailed *t*-test was used to examine whether the mean Δ pulsatility indices, in the affected and in the healthy kidneys, respectively, were significantly different from the normal mean for gestation.

RESULTS

In all 11 fetuses with unilateral multicystic kidney disease, the renal artery pulsatility index in the affected kidney was higher than the pulsatility index in the contralateral healthy one (Figure 2) (median difference = 0.70, Z = 2.89 SDs, $p < 0.01$). The mean pulsatility index for the affected kidneys was significantly higher than the normal mean for gestation (mean difference = 1.58 SDs, SEM = 0.36, $t = 4.34$, $p < 0.01$). Although the mean pulsatility index of the contralateral normal kidney was lower than the normal mean for gestation, the difference was not significant (mean difference = −0.40 SDs, SEM = 0.22, $t = -1.86$, $p = 0.09$).

DISCUSSION

This study has demonstrated that, in fetuses with unilateral multicystic kidney disease, impedance to flow in the renal artery of the affected kidney is increased. This presumably reflects the abnormal vascular development of such kidneys. The tendency for decreased impedance in the contralateral normal kidney may be an early manifestation of compensatory hypertrophy.

In normal renal development, the ureteric bud grows into the metanephric mass of mesoderm during the 5th week of fetal life. The ureteric bud dilates and branches to form the renal pelvis, calyces and collecting ducts. The glomeruli and the upper part of the nephrons develop from the metanephric blastema[9].

The etiology of multicystic kidney disease remains controversial. Attempts to correlate renal dysplasia with coincident ureteric abnormalities have led to the popularization of the 'bad bud' theory, in which it was suggested that the observed cystic dysplasia of the kidneys was a consequence of abnormal ureteric budding in early renal development[10,11]. It is probable that primary dysgenesis of the developing organ may distort subsequent development of the renal vascular tree.

An alternative hypothesis is that, during migration of the developing kidney from the sacral to the lumbar region, the shifting network of mesonephric vessels and capillaries may fail to maintain an adequate blood supply to the metanephros and ureteric bud. The ischemic insult to ureter and kidney could result in both the multicystic kidney and the associated ureteric atresia[12]. Even in neonatal life, renal ischemic necrosis may be followed by morphological alterations mimicking primitive ductules, tubules and glomeruli[13].

The extent to which increased impedance to flow in the fetal renal artery of multicystic kidneys is caused by renal dysgenesis, or whether multicystic kidney disease is a result of impaired perfusion of the developing kidney, remains to be resolved. Nevertheless, the present study has demonstrated the feasibility of prenatal diagnosis of abnormal renal vasculature.

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