Renal Vascular Changes and Uterine Artery Resistance in Pregnant Women with Renal Disease

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Dear Sir,

Pregnant women with primary renal disease, impaired renal function and hypertension are at higher risk for the development of fetal and maternal complications [1]. However, pregnancy outcome in normotensive women with preserved renal function is less clear. It has been postulated that the vascular changes in the renal biopsy may be useful in predicting pregnancy-related complications [2]. Preeclampsia and intrauterine growth retardation, two major complications of pregnancy, are associated with an increased resistance index in the uteroplacental circulation [3]. It may be speculated that preexisting renal vascular changes may be related to the increased vascular resistance in uteroplacental circulation during pregnancy. To test this hypothesis, we analysed the relationship between vascular changes in the renal biopsy and uterine artery resistance index in 20 normotensive pregnant women with primary kidney disease and normal renal function. All women had a renal biopsy performed within the 12 months prior to pregnancy. The vascular changes were semiquantitatively classified as normal, mild, moderate and severe. Each blood vessel was evaluated for the presence or absence of intimal thickening, medial thickening, medial hyalinosis and intimal hyalinosis by an independent investigator. A blood vessel was regarded as normal when none of the above abnormalities was present. Vessel changes were considered mild when 1 out of 4 of the above-mentioned abnormalities was present; moderate when 2 out of 4 abnormalities were present and severe when 3 out of 4 abnormalities or severe intimal thickening were present. The score used was 1 for normal, 2 for mild, 3 for moderate and 4 for severe. Uterine artery resistance index was derived from uterine artery waveform obtained by Color Doppler ultrasound between 19 and 24 weeks' gestation [3]. The resistance index was calculated on the placental side of the uterus from the peak systolic (A) and late diastolic (B) flow velocities and expressed as $(A-B)/A$ [4].

Of the women studied, 2 had normal renal vessels (10%), 11 had mild changes (55%), 5 had moderate changes (25%) and 2 severe changes (10%). Among the 13 women who had no or mild renal vascular changes only 1 developed preeclampsia (7%), while 5 out of 7 (71%) with moderate or severe vascular changes developed preeclampsia and/or intrauterine growth retardation ($p < 0.05$; by χ^2 test). The semiquantitatively classified renal vascular changes correlated with the uterine artery resistance index obtained from the placental side of the uterus ($r=0.54$; $p < 0.05$; by Spearman's correlation) (fig. 1).

0.3 0.4 0.5 0.6 0.7 0.8 0.9 Uterine artery resistance index

Fig. 1. Relationship between semiquantitative assessment of vascular change severity and placental side uterine artery resistance index in 20 pregnant women with renal disease. 1 = No vessel change; 2 = mild vessel change; 3 = moderate vessel change; 4 = severe vessel change. The presence of a link between renal vascular changes prior to pregnancy with uterine artery resistance index in early gestation suggests that there may be an excess or a deficiency of a renal product influencing the uteroplacental circulation. Since a dysfunction of endothelial cells has been implicated in the pathogenesis of certain renal vascular lesions [5], circulating vasoactive substances, dependent upon the integrity of the endothelium, may contribute to an abnormal development of uteroplacental circulation and hence to an increased vascular resistance in early gestation [6]. Putative factors include an excess in the vasoconstrictor endothelin or a deficiency in the vasodilator nitric oxide.

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