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Uterine Artery Blood Flow Volume in Patients with High Risk of Preeclampsia

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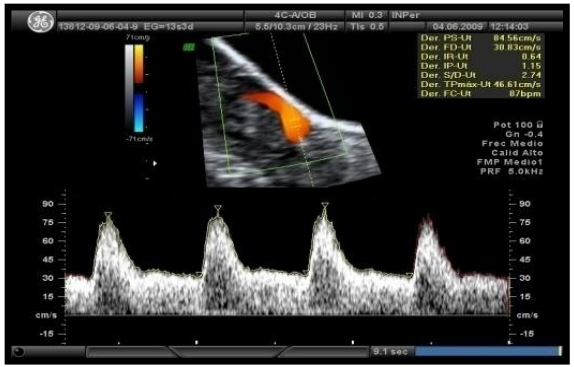
Uterine artery blood flow volume (UABFVt) indirectly quantifies how much blood reaches the uterus per unit of time. Changes in this flow between the first and second trimesters can reflect the quality of placentation. The objective of this study is to document the relationship between a decrease in this blood flow and increased risk of preeclampsia (PE).

The first stage included 389 healthy patients between 11 and 36 weeks of gestations (wog). UABFVt was measured with Doppler ultrasound to construct reference values. Using a linear regression model (r^2=0.71), UABFV was determined to be -3.035+0.87(wog). This increased 1 ml/s per week of gestation. A significant difference ($p<0.001$) in the ΔUABFVt was found between the first and second trimester, of 10.87ml/s (95% CI; 10.44-11.29).
During the second stage, 774 patients with risk factors for PE were studied and ΔUABFVt values were compared between those with PE (prevalence of 7%) and those without. The ΔUABFVt was less (19.4 ml/s) for those with PE than those without (25ml) ($p<0.001$), which supports the theory of placental hypoperfusion, the main theory in the physiopathology of PE.



Conclusion

The present study is based on Doppler measurements of the uterine arteries to indirectly study uterine-placenta perfusion in normal pregnancies and pregnancies with clinical and pathological factors associated with the risk of PE. The objective is to document hypoperfusion as a physiopathological mechanism in PE.

In the beginning of pregnancy, vascular resistance in the uterine arteries decreases, facilitating the flow of oxygen and nutrients through the maternal-fetal unit. This is secondary to the transformation of spiral arteries into low resistance vessels (Bird et al. 2003; Greiss 1996; Poston et al. 1995). Many theories exists about the cause of PE, with little agreement have being reached. It is likely that multiple early damages converge into one common physiopathology, or into two when considering early and late PE (Roberts 2009). All forms of this diseases are characterized by altered vascular remodeling and an anti-angiogenic response (Pennington 2012). The altering of vascular remodeling decreases flow and placental and fetal perfusion, playing a key role as an etiopathogenic mechanism in PE (Roberts and Cooper 2001).
Uterine artery blood flow volume (UABFV) is determined by average and maximum velocities which are obtained with Doppler velocimetry, taking into account the diameter of the artery and its imperfect tubular structure as well as the blood's viscosity (Boito S. 2006).