Expression and Biological Activity of Parathyroid Hormone-Related Peptide in Pregnant Rat Uterine Artery: Any Role for 8-Iso-Prostaglandin F2α?

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Résumé en anglais	PTHrP is produced in vessels and acts as a local modulator of tone. We recently reported that PTHrP(1–34) is able to induce vasorelaxation in rat uterine arteries, but in pregnancy, this response is blunted and becomes strictly endothelium dependent. The present study aimed to get insights into the mechanisms involved in these changes because the adaptation of uterine blood flow is essential for fetal development. On d 20 of gestation, RT-PCR analysis of uterine arteries showed that PTH/PTHrP receptor (PTH1R) mRNA expression was decreased, whereas that of PTHrP mRNA was increased. This was associated with a redistribution of the PTHrP/PTH1R system, with both PTH1R protein and PTHrP peptide becoming concentrated in the intimal layer of arteries from pregnant rats. On the other hand, the blunted vasorelaxation induced by PTHrP(1–34) in uterine arteries from pregnant rats was specifically restored by indomethacin and a specific cyclooxygenase-2 inhibitor, NS 398. This was associated with an increase in cyclooxygenase-2 expression and in 8-iso-prostaglandin F2α release when uterine arteries from pregnant rats were exposed to high levels of PTHrP(1–34). Most interestingly, 8-iso-prostaglandin F2α itself was able to increase PTHrP expression and reduce PTH1R expression in cultured rat aortic smooth muscle cells. These results suggest a local regulation of uterine artery functions by PTHrP during pregnancy resulting from PTH1R redistribution. Moreover, they shed light on a potential role of 8-iso-prostaglandin F2α.
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