Abstract

The pathogenesis of preeclampsia is still unclear. One popular hypothesis is that the altered vascular reactivity in preeclampsia is due to a relative or absolute deficiency of vasodilating prostaglandins (PGs). Cyclooxygenase (COX) exists in at least two isoforms. The amounts of each PGs synthesized by COX-1 and COX-2 was different. COX-2, therefore, may deviate the amounts of each PGs in preeclampsia. Here we have investigated whether COX-2 is expressed in human umbilical vein endothelial cells (HUVEC) from preeclampsia. HUVEC were obtained from babies born to preeclampsia (n=12) and normal pregnancy (n=12). Cells were grown to confluent in 6-well culture plate until use and replaced with fresh medium for 24h. The expression of COX-1 and COX-2 protein was detected by immunoblot using specific antibodies. HUVEC from normal pregnancy contained COX-1 protein but not COX-2 protein. In contrast, HUVEC from preeclampsia contained both COX-1 and COX-2 protein. Interestingly, equal amounts of COX-1 and COX-2 protein were detect in both preeclampsia and normal pregnancy. Thus, COX-2 expressed in HUVEC from preeclampsia but not in normal pregnancy suggesting the involvement of COX-2 in the pathogenesis of preclampsia. Further investigating on COX activity and mechanism of induction should be elucidated.