Loss of vascular expression of nucleoside triphosphate diphosphohydrolase-1/CD39 in hypertension

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Résumé en anglais	Ectonucleoside triphosphate diphosphohydrolase-1, the major vascular/immune ectonucleotidase, exerts anti-thrombotic and immunomodulatory actions by hydrolyzing extracellular nucleotides (danger signals). Hypertension is characterized by vascular wall remodeling, endothelial dysfunction, and immune infiltration. Here our aim was to investigate the impact of arterial hypertension on CD39 expression and activity in mice. Arterial expression of CD39 was determined by reverse transcription quantitative real-time PCR in experimental models of hypertension, including angiotensin II (AngII)-treated mice (1 mg/kg/day, 21 days), deoxycorticosterone acetate-salt mice (1% salt and uninephrectomy, 21 days), and spontaneously hypertensive rats. A decrease in CD39 expression occurred in the resistance and conductance arteries of hypertensive animals with no effect on lymphoid organs. In AngII-treated mice, a decrease in CD39 protein levels (Western blot) was corroborated by reduced arterial nucleotidase activity, as evaluated by fluorescent (etheno)-ADP hydrolysis. Moreover, serum-soluble ADPase activity, supported by CD39, was significantly decreased in AngII-treated mice. Experiments were conducted in vitro on vascular cells to determine the elements underlying this downregulation. We found that CD39 transcription was reduced by proinflammatory cytokines interleukin (IL)-1β and tumor necrosis factor alpha on vascular smooth muscle cells and by IL-6 and anti-inflammatory and profibrotic cytokine transforming growth factor beta 1 on endothelial cells. In addition, CD39 expression was downregulated by mechanical stretch on vascular cells. Arterial expression and activity of CD39 were decreased in hypertension as a result of both a proinflammatory environment and mechanical strain exerted on vascular cells. Reduced ectonucleotidase activity may alter the vascular condition, thus enhancing arterial damage, remodeling, or thrombotic events.
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