[Role of renin-angiotensin system in prostate cancer].

[Article in Japanese]
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Source
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Abstract

Although a low prevalence of cancer in hypertensive patients receiving angiotensin converting enzyme inhibitors was reported, the molecular mechanisms have not been elucidated. It is known that the Angiotensin- II (Ang- II) plays a fundamental role not only as a vasoconstrictor in controlling blood pressure and electrolyte/fluid homeostasis, but also as a mitogenic factor through the Ang- II type-1 (AT1) receptor in cardiovascular cells. Recently, there has been increasing evidence that the renin-angiotensin system (RAS) is implicated in the development of various cancers. Ang- II has been demonstrated to be a cytokine, especially acting as a growth factor. Of interest, the physiological function of Ang- II seems to be similar in prostate cancer and stromal cells as we previously reported. AT1 receptor blockers (ARBs), a class of anti hypertensive agent, have the potential to inhibit the growth of prostate cancer cells and tumors through the AT1 receptor. We conducted a pilot clinical study to examine whether ARBs were able to elicit an anti proliferative effect on prostate cancer clinically, resulting in a PSA decline of hormone refractory cancer or delaying PSA progression after radical prostatectomy. As a number of investigators have clarified that Ang- II induces oxidative stress in vascular cells, we reported the hypothesis that Ang- II generated in the prostate gland maybe a cause of oxidative stress linked to prostatic carcinogenesis. This review provides an insight into the key role of Ang- II and AT1 receptor, and the possibility of ARBs for molecular targeting of mitogenesis and angiogenesis in prostate cancer.

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