Letter to the Editor

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Hematological Side Effects of ACE Inhibitors and a Hypothetical Local Renin-Angiotensin System in the Bone Marrow

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Dear Sir,

The renin-angiotensin system (RAS) was originally thought to be active only in plasma as a circulating system that controls blood pressure and electrolyte homeostasis. It is now known that components of the RAS are also present in specific tissues, such as the walls of blood vessels and heart, kidney, adrenal gland, brain, pituitary gland, ovary, testes, uterus, chorion-amnion, placenta, gut (jejunum), and salivary glands [1]. Angiotensin II produced locally has an autocrine/para-crine effect on angiotensin-mediated function(s) in these tissues [1-6]. Consequently, it has now become clear that the RAS must also include tissue-specific systems that have complex paracrine and autocrine functions within many organs [1,6]. We have read with interest the letter by Teruel et al. [7], reporting that angiotensin-converting enzyme (ACE) inhibitors have a worsening effect on anemia in hemodialysis patients. It has also previously been implied that ACE inhibitors have various hematological side effects, including the effects on erythropoiesis which cause anemia and erythropoietin resistance [8]. The bone marrow is a highly organized and complex organ, and it is the principal hematopoietically active tissue in adults. For the division of hematopoietic cells, increases occur in the rates of production of cellular constituents, including DNA, RNA, and proteins [9]. Angiotensin II is capable of inducing cellular hyperplasia and growth by directly stimulating the synthesis of DNA, RNA, and protein in specific tissues [1,6, 10]. Thus, angiotensin(s) may be accepted as locally active growth factors. We have already published a hypothesis that there may be a locally active RAS in the bone marrow [11].

A local bone marrow RAS might influence pharmacological activities of various drugs which are used in different diseases. Future progress might be possible, if the putative local bone marrow RAS could be blocked by inhibitors of RAS, such as ACE inhibitors. Familiar hematopoietic side effects of present ACE inhibitors in hypertensive patients may be accepted as an initial clue to this intention. The exact localization of tissue RAS, including bone marrow RAS, and their autocrine and paracrine effects seem to be worthy of further study.

References


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