Plasma Renin Activity and Plasma Aldosterone in Acute Renal Failure

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Plasma renin activity and plasma aldosterone were determined by radioimmunoassay methods in 20 patients in oliguric phase, in 11 patients in polyuric phase and in 7 patients in convalescent phase of acute renal failure of various origin. The oliguric phase of acute renal failure was characterized by significant increase of plasma renin activity and plasma aldosterone. There was no direct dependence between them. Direct dependence was found between plasma aldosterone and serum potassium in the oliguric phase of acute renal failure, indirect dependence between plasma aldosterone and serum sodium was found before as well as after haemodialysis. These findings prove a direct influence of hyperkalemia and depletion hyponatremia upon aldosterone secretion in the oliguric phase of acute renal failure. Haemodialysis led to a further increase of plasma renin activity caused by ultrafiltration as well as successive dehydration and application of some drugs. The mean value of plasma aldosterone was not significantly changed after haemodialysis. Plasma renin activity decreased very slowly in the polyuric and convalescence phase of acute renal failure, while plasma aldosterone concentration was already in polyuric phase non-significantly different from the control group. There was no direct dependence in the various phases of acute renal failure between plasma renin activity, plasma aldosterone, systolic and diastolic pressure.

The renin-angiotensin-aldosterone system significantly participates in the pathogenesis of acute renal failure in man, but various causes of acute renal failure, different drugs, as well as therapeutic procedures do not make it possible to quantify it in detail.

Charcoal haemoperfusion in acute poisonings led only to non-significant increase of plasma renin activity and decrease of plasma aldosterone.

Aldosterone is a steroid hormone (11β,21-dihydroxy-3,20-dioxo-4-pregnene-18-al-18-11-hemiacetal) with a molecular weight of 360.4. This essential “mineralocorticoid” is formed in the superficial zone of the adrenal cortex (zona glomerulosa) in response to stimulation by angiotensin II, potassium ions and adrenocorticotropic hormone. The most significant physiologic aldosterone functions are as follows: maintenance of sodium balance, potassium homeostasis and hydrogen ion excretion. The most important target of the aldosterone effect is the distal tubule, nevertheless its effect upon other membranes and glandular tissues can have secondary influence on other parts of the nephron. The area of aldosterone effect is bound to specific protein receptors present in certain tissues.
These proteins in complex with aldosterone stimulate the formation of DNA-dependent RNA that facilitates synthesis of new proteins. The final mechanism of aldosterone effect is not known in detail [4].

The influence of plasma renin activity (PRA) upon plasma aldosterone (PA) in chronic renal failure has been examined by several authors [10, 13]. Vetter et al. [13] found positive influence in these patients solely before haemodialysis during regular dialysis treatment; after haemodialysis direct dependence between PRA, adrenocorticotrophic hormone, serum potassium, serum sodium and PA was not observed.

The activity of the renin-angiotensin-aldosterone system was examined in acute renal failure in people rather rarely as can be found in the literature available to us [5, 6, 11]. These authors found in the oliguric phase of acute renal failure significantly increased PRA, but the PA value was only slightly increased in some patients. Kokot et al. [5, 6] found indirect dependence between PA, serum sodium and serum bicarbonate. According to these authors the absence of direct dependence between PRA and PA proves the presence of factors other than renin that regulate aldosterone secretion in acute renal failure.

Charcoal haemoperfusion, which is used in the treatment of acute poisoning caused by less dialysable drugs and poisons, decreases the serum values of some proteins, hormones and vitamins with high molecular weight. There are no significant data in the literature about this influence on PRA and PA [7, 8].

**Material and methods**

Plasma renin activity and PA were examined in 20 patients aged 18 to 71 years. All patients were in the oliguric phase of acute renal failure, 11 patients were examined in the polyuric phase and 7 patients in the convalescent phase. Plasma renin activity and PA were examined between the 3rd and 7th days from the onset of disease. The causes of acute renal failure were various (Table 1). 20 patients were examined before and 4 hours after haemodialysis.

Five patients with acute poisonings, aged 12 to 67 years, were examined before and 5 hours after charcoal haemoperfusion. Five patients underwent 6 charcoal haemoperfusions. The causes of acute poisonings were various (Table 2). One patient with mushroom poisoning was in the oliguric phase of acute renal failure. Charcoal haemoperfusion was performed 2 to 4 days after acute poisoning.

Plasma renin activity was determined by the radioimmunoassay method of Haber et al. [2] in the modification of the CEA-IRE-SORIN test. The control group consisted of 20 healthy individuals; blood removal was performed in the morning after night sleep in the horizontal position without pressing the upper extremity. Plasma renin activity in the control group was $1.389 \pm 0.068$ ng/ml/hour. Plasma aldosterone was determined by the radioimmunoassay method of Malvano et al. [9] in the modification of CEA-IRE-SORIN test. The control group consisted