Approximately one third of the patients treated with recombinant human erythropoietin (rhEPO) will experience either an aggravation of preexisting hypertension or will develop de novo hypertension [1]. But the underlying mechanism is unclear and various causes such as the increased blood viscosity and total red cell mass inducing an increase in peripheral resistance [2] and the reversal of compensatory vasodilatation induced by renal anemia [3] have been proposed by several studies before. Besides, some investigators have suggested that rhEPO itself has some effect on renin-angiotensin-aldosterone axis. So, it has been proposed that the alterations in this axis may contribute to the changes in blood pressure during rhEPO treatment [4,5]. In order to clarify the acute effect of rhEPO on plasma renin activity (PRA) and aldosterone secretion, we studied PRA and aldosterone levels after rhEPO injection in 9 predialysis uremic patients.

We included 9 patients with chronic renal failure, untreated by dialysis previously. The patients had never taken rhEPO, antihypertensives, and the drugs affecting the renin-angiotensin axis and aldosterone secretion such as aldosterone antagonists and angiotensinconverting enzyme inhibitors. Patients were maintained at bedrest in supine position for 6 h prior to rhEPO injection and they kept their supine position throughout the test. After the baseline sampling at 8.00 a.m. rhEPO was given intravenously at a dose of 50 U/kg. Blood samples for analysis were taken at 15, 30 and 60 min after injection and blood pressures were recorded simultaneously.
Fig. 1. Changes in plasma renin activity and aldosterone levels after intravenous application of rhEPO.

Blood pressure readings did not vary significantly during the study (p > 0.05). Figure 1 shows that the observed changes in PRA and plasma aldosterone levels after rhEPO injection were not found to be statistically significant (p > 0.05). In conclusion, our findings indicate that acute administration of rhEPO does not have any direct effect on PRA and aldosterone secretion in chronic renal failure.

References


