Oxidative Stress (OS) and PTH in Hemodialysis Patients (HDP)

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Introduction

It is now well established that free radicals, such as superoxide (O2), hydroxyl (OH), and nitric oxide (NO), and other reactive oxygen species (ROS) as Hydrogen peroxide (H2O2) are continuously formed in vivo (1). ROS originate from activated macrophages, endothelial cells, smooth muscle cells and various glomerular kidney cells (2). In healthy subjects there seems to be a balance between the production of ROS and the activity of antioxidant enzymes such as superoxide dismutases (SODs), glutathion peroxidase (GSH), vit.E, catalase. A disturbed balans between pro - and antioxidant mechanisms characterizes the OS. There are some arguments supporting the hypothesis of enhanced OS in uremic patients but a very few studies include sHPT as an additional factor, increasing OS evidence, despite the well known fact that PTH is suspected as one of the uremic toxins (3, 4, 5).

The aim of this study was to investigate whether sHPT plays an additional role for development of OS and which part of the system is disturbed or not.

Subjects and Methods

The tested patients and the included parameters were as follows: 1st GROUP – 15 pts. on RDT (mean duration 65 ± 32 months) with sHPT - PTH >200ng/ml; AP > 280E/l; 2nd GROUP – 17 pts. on RDT (mean duration 41 ± 24 months) without sHPT. SOD activity was measured in RBC according to Misra and Fridovich (6) in adrenaline units (U/gHb). MDA contents of RBC was quantified by HPLC - Young et al. (7) (pg/ml packing RBC). Vit.E was measured routinely (in mg/l of pellet). GSH was measured in nmol/mgHb in RBC. Hb was estimated routinely (Coulter, g/dl). Statistical analysis was made using Student's T-test.

Results

MDA was significantly increased in all patients independently of the group (mean value - 6.65±0.42 pg/ml).

All antioxidant enzymes were significantly lower in the 1st GROUP than in the 2nd, as it is showed on the table No 1:

Discussion

A significant increase in MDA content in RBC of patients on RDT was found compared with the reference levels which suggested permanent membrane alterations in this kind of pathology (1, 3, 4) and also increased production of reactive oxygen species in the circulation

Table 1

	SOD	Vit. E	GSH
1st group n = 15	0.43 +/- 0.02	23.5 +/- 0.12	4.2+/-0.5
2nd group n = 17	0.55 +/- 0.04	28.6 +/- 0.18	5.6+/- 0.1
p <	0.01	0.01	0.05

This was also demonstrated by other authors(5, 6, 7). The lack of a special difference of MDA between the group with sHPT and the other - without sHPT, suggests that PTH does not influence lipid peroxydation. But the obviously more suppressed levels of some antioxidant enzymes in the 1st Group - with sHPT - is a hint that PTH may disregulate the oxidative balance of the human body in the level of antioxidant systems. This fact is a news about the pts. on RDT, because the problem could be solved more directly, i.e. easier, by supplementation of some antioxidant medicines and suppressors of PTH production. But the study gives also a reason for the nephrologists to introduce in the near future a stronger treatment against OS in the pts. with sHPT than in the others.

References

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