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Abnormal Reactions of Free Radicals and Oxidative Damages in the Bodies of Patients With Chronic Glomerulonephritis

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Objective To study the abnormal reactions of a series of free radicals and the oxidative damages induced by free radical abnormal reactions in the bodies of patients with chronic glomerulonephritis. **Methods** Eighty chronic glomerulonephritis patients (CGNP) and eighty healthy adult volunteers (HAV) were enrolled in a random control study, in which concentrations of nitric oxide (NO) in plasma, lipoperoxides (LPO) in plasma and in erythrocytes, and vitamin C (VC), vitamin E (VE) and beta-carotene (β-CAR) in plasma as well as activities of superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPX) in erythrocytes were determined with spectrophotometric assays. **Results** Compared with the average values of the above biochemical parameters in the HAV group, the average values of NO in plasma, and LPO in plasma and erythrocytes in the CGNP group were significantly increased ($P = 0.0001$), while those of VC, VE and β-CAR in plasma as well as those of SOD, CAT and GPX in erythrocytes in the CGNP group were significantly decreased ($P = 0.0001$). Pearson product-moment correlation analysis showed that with increase of the concentration of blood creatinine as well as prolongation of the course of disease in the CGNP, the concentrations of NO in plasma, and LPO in plasma and erythrocytes in the CGNP increased gradually, while the concentrations of VC, VE and β-CAR in plasma as well as the activities of SOD, CAT and GPX in erythrocytes in the CGNP decreased gradually ($P = 0.002454 - 0.000001$). The relative risk ratio (RR) of the above biochemical parameters reflecting oxidative damages in the bodies of CGNP ranged from 6.061 to 72.429. The reliability coefficient (alpha) that the above biochemical parameters were used to reflect the oxidative damages of the CGNP was 0.8137, standardized item alpha = 0.9728, Hotelling's T-Squared = 1135680.191, $F = 53274.6478$, $P = 0.000001$. **Conclusions** The findings in this study show that in the bodies of CGNP a series of free radical chain reactions result in severe pathological aggravation and induce oxidative damages in their bodies. Therefore, suitable dose of antioxidants should be supplemented to them so as to alleviate oxidative damages in their bodies.

Key words: Chronic glomerulonephritis; Free radicals; Oxidation; Lipoperoxidation; Nitric oxide; Lipoperoxides; Antioxidant; Antioxidase; Oxidative stress; Oxidative damage

INTRODUCTION

Chronic glomerulonephritis (CGN) is a set of renal diseases induced by pathologic change of glomerulus, and its inducing factors include a variety of infectious agents such as

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upper respiratory and dermal streptococcal infections and bacterial endocarditis as well as the deposition of immune complexes in autoimmune diseases such as SLE, or the damaging effect of circulating antibodies against the GBM as in Goodpasture's syndrome[1-4]. In studying pathophysiology of glomerulonephritis, some authors reported that the content of nitric oxide in the exhaled air of CGN patients (CGNP) markedly increased[5], and that in the bodies of CGNP or experimental animals with CGN serum levels of lipoperoxides or malondialdehyde significantly increased, while superoxide dismutase, catalase and glutathione peroxidase significantly decreased[6-12]. However, up to now, there is neither report on changes of a free radical chain reactions in the bodies of CGNP, nor report about relationship between CGNP and oxidative damages. To investigate the abnormal reactions of a series of free radicals and the oxidative damages induced by free radical abnormal reactions in the bodies of patients with chronic glomerulonephritis, 80 chronic glomerulonephritis patients (CGNP) and 80 healthy adult volunteers (HAV) were enrolled in a random control study, in which concentrations of nitric oxide (NO) in plasma, and lipoperoxides (LPO) in plasma and erythrocytes, and vitamin C (VC), vitamin E (VE) and beta-carotene (β-CAR) in plasma as well as activities of superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPX) in erythrocytes were determined with spectrophotometric assays. At the same time the differences between average values of the above biochemical parameters in the CGNP group and in the HAV group were compared. The Pearson product-moment correlation between blood creatinine concentration in the CGNP and the above biochemical parameters were analyzed. Additionally, relative risk ratio (RR) of the above biochemical constituents reflecting oxidative damages in the bodies of CGNP, and 95 % CI of RR as well as the reliability coefficient (alpha) used for the above biochemical parameters to reflect the oxidative damages in the bodies of CGNP were estimated.

MATERIALS AND METHODS

Study Design

The random control design was used in this research. In this design and its study, the principles of random sampling, control, replication and equilibrium, and the management factor, the experimental effect and the subjects were taken into full consideration in order to obtain a comparative objective and right research conclusion[13-16].

Subjects

CGNP. With "Select Cases–Random Sample" of "SPSS 10.0 for Windows", 80 chronic glomerulonephritis patients (CGNP) were randomly sampled from 160 CGNP whose diagnoses were confirmed by the diagnostic criteria[1-4] and the inclusion criteria[1-4] in the Second Affiliated Hospital, Medical College of Zhejiang University. Their ages ranged from 21 to 30 (25.6±2.4) years, 56 were males and 24 were females, and their course of disease ranged from 2 to 11 (6.6±2.4) years. Their blood creatinine contents ranged from 259.32 to 370.45 (315.81±30.04) μmol/L and 95 % confidence intervals were 309.12 – 322.49 μmol/L. These CGN patients were within normal ranges in their routine feces tests, radiographs and electrocardiograms, and had no disorders associated with heart, brain, lung, liver or other organs, or other medical problems such as hyperlipaemia, chronic bronchitis, diabetes, atherosclerosis, and tumors.

HAV. With "Select Cases–Random Sample" of "SPSS 10.0 for Windows", 80 healthy

adult volunteers (HAV) were randomly sampled from 200 healthy adult volunteers who were confirmed by an overall physical examination at the Second Affiliated Hospital, Medical College of Zhejiang University. Their ages ranged from 21 to 30 (25.7±2.4) years, 50 were males and 30 were females. They were within normal ranges in their routine blood, urine and feces tests, radiographs and electrocardiograms, and had no disorders associated with heart, brain, lung, liver, kidney and other organs, or other medical problems such as hypertension, hyperlipaemia, chronic bronchitis, autoimmune disease, diabetes, atherosclerosis, and tumors.

There was no significant difference between the average values of their age in the CGNP group and in the HAV group by t test ($t = 0.2287$, $P = 0.8194$), and between the sexual proportions in the CGNP group and in the HAV group by χ^2 test ($\chi^2 = 1.0063$, $P = 0.3158$).

All the above subjects had never been exposed to radiation and intoxicating materials or pesticides. Within one month before recruitment they had not taken any ginkgo biloba, tea-polyphenols or other antioxidative agents.

Methods

Measurement of blood creatinine content. Blood creatinine content was measured by an automatic biochemistry analyser, and expressed as μmol/L.

Methods of other biochemical parameters. Fasting venous blood samples were collected in the morning from all the subjects and heparin sodium was added as anticoagulant, and the promptly separated plasma and erythrocytes were stored at −50℃ immediately[13,14]. The coloration of α-naphthylamine and nitrite was used to determine the plasma NO concentration expressed as nmol/L. The spectrophotometry of thiobarbituric acid reactive substances (TBARS) was used to determine the plasma LPO concentration expressed as μmol/L. The spectrophotometry of TBARS was used to determine the erythrocytic LPO concentration expressed as nmol/g.Hb. The ferrozine coloration was used to determine the plasma VC and VE concentrations expressed as μmol/L. The plasma β-CAR concentration was extracted with a mixture of ethanol and petroleum ether, and assayed with spectrophotometry, and expressed as μmol/L. The spectrophotometry of inhibiting pyrogallol auto-oxidation was used to determine the erythrocytic SOD activity indicated as U/g.Hb. The spectrophotometry of coloration of hydrogen peroxide and acetic acid-potassium dichromate was used to determine the erythrocytic CAT activity indicated as K/g.Hb. The improved Hafeman's spectrophotometry was used to determine the erythrocytic GPX activity expressed as U/mg · Hb.

In determining the above biochemical substances and enzymes, the major analytical reagents, such as α-naphthylamine, 1,2,3-trihydroxybenzene, 1,1,3,3-tetraethoxypropane, 2-thiobarbituric acid, vitamin C, vitamin E, β-carotene, disodium 5,6-diphenyl-3-(2-pyridyl)-1,2,4-triazinedisulfonate, Cu/Zn-superoxide dismutase, catalase, were all purchased from SIGMA CHEMICAL COMPANY®, USA; and other analytical-grade reagents were all produced in China. The fresh quadruply distilled water was prepared with a quartz glass distilling apparatus. In determining the above biochemical substances and enzymes, the main analytical instruments included OLYMPUS AU2700-Automatic Biochemistry Analyser, Japan, *Hewlett Packard 8453-Spectrophotometer*, USA, *and UV-754-Spectrophotometer*, and 721-Spectrophotometer, Shanghai, China.

During determining process of the above biochemical substances and enzymes, standardization was strictly adhered to by using the same lot number of each reagent, the same quality control, the same lab assistant, and identical analytical apparatus for each

experiment in order to control and decrease its error and bias of the experiment as much as possible, and to ensure the accuracy of the measurements as much as possible[13-16].

Medical Statistic Analysis

All data were statistically analyzed with SPSS/10.0 for Windows and STATISTICA/6.0 for Windows statistic software using a Compaq Pentium III/1000 computer. The parameters in this study all presented normal distribution by Kolmogorov-Smirnov Z test. They were expressed as mean plus or minus standard deviation ($\bar{x} \pm s$) and 95% confidence interval (95% CI). Hypothesis testing methods used included independent samples *t* test, Chi-square test, Pearson product-moment correlation analysis and reliability analysis. In the statistical analysis of this study, the level of hypothesis testing (α) was $\leqslant$ 0.05 in order to avoid false positives (α-error), and the power of hypothesis testing (*power*) was $\geqslant$ 0.80 to avoid false negatives (β-error) [15,16].

RESULTS

1. Comparison Between the Average Values ($\bar{x} \pm s$) of Biochemical Parameters in the CGNP Group and HAV Group

Compared with those in the HAV group, the average values of concentrations of NO in plasma, and LPO in plasma and in erythrocytes in the CGNP group were significantly increased ($P = 0.0001$), and those of concentrations of VC, VE and β-CAR in plasma as well as those of activities of SOD, CAT and GPX in erythrocytes in the CGNP group were significantly decreased ($P = 0.0001$) (Table 1).

2. 95% CI of the Average Values of Biochemical Parameters in the CGNP Group and HAV Group

The lower limits of 95% CI of the average values of NO in plasma, and LPO in plasma and in erythrocytes in the CGNP group were greater than the upper limits of 95% CI of the same values in the HAV group. The upper limits of 95% CI of the average values of VC, VE and β-CAR in plasma as well as the activities of SOD, CAT and GPX in erythrocytes in the CGNP group were less than the lower limits of 95% CI in the HAV group (Table 1).

3. Pearson Product-moment Correlation Analysis of Blood Creatinine Content and Every Biochemical Parameter for 80 Chronic Glomerulonephritis Patients

The results of Pearson product-moment correlation analysis showed that the concentrations of NO in plasma, and LPO in plasma and in erythrocytes increased as the concentration of blood creatinine increased, and presented all linear positive correlations; while the concentrations of VC, VE and β-CAR as well as the activities of SOD, CAT and GPX in erythrocytes decreased as the concentration of blood creatinine increased, and presented all linear negative correlations (Table 2).

4. Pearson Product-moment Correlation Analysis of the Course of Disease and Every Biochemical Parameter for 80 Chronic Glomerulonephritis Patients

The results of Pearson product-moment correlation analysis showed that the concentrations

TABLE 1

Comparison Between the Average Values ($\bar{x} \pm s$) of Biomedical Parameters in CGNP Group and HAV Group

Group	*n*	Oxidative Substances			Antioxidative Substances					
		Plasma	Erythrocyte		Plasma			Erythrocyte		
		NO (nmol/L)	LPO (μmol/L)	LPO (nmol/g · Hb)	VC (μmol/L)	VE (μmol/L)	β-CAR (μmol/L)	SOD (U/g · Hb)	CAT (K/g · Hb)	GPX (U/mg · Hb)
CGNP	80	561.7±117.7	14.27±2.06	43.60±6.32	36.85±9.17	16.62±4.43	1.27±0.33	1725.8±132.6	227.2±57.0	20.13±5.37
		(535.5–587.9)	(13.81–14.73)	(42.19–45.00)	(34.81–38.89)	(15.64–17.61)	(1.20–1.34)	(1696.3–1755.4)	(214.5–239.8)	(18.93–21.32)
HAV	80	362.2±83.4	11.28±1.63	29.15±4.22	56.12±13.96	25.84±4.17	1.72±0.43	2106.6±150.2	305.7±76.1	29.06±7.23
		(343.6–380.7)	(10.92–11.64)	(28.21–30.09)	(53.01–59.23)	(24.91–26.77)	(1.62–1.82)	(2073.2–2140.0)	(288.8–322.7)	(27.45–30.67)
t^a		12.3686	10.1588	17.0122	10.3172	13.5535	7.4944	16.9974	7.3921	8.8667
P		0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001

Note. [a] independent-samples *t* test. The figures in parentheses are confidence limits.

of NO in plasma, and LPO in plasma and in erythrocytes increased as the course of disease was prolonged, and presented all linear positive correlations; while the concentrations of VC, VE and β-CAR in plasma as well as the activities of SOD, CAT and GPX in erythrocytes decreased as the course of disease was prolonged, and presented all linear negative correlations (Table 3).

TABLE 2

Pearson Product-moment Correlation Analysis of Creatinine and Every Biochemical Parameter

Correlative Item	*n*	Correlative Coefficient (*r*)	*t*	*P*
Creatinine and Plasma NO	80	0.6794	8.1783	0.000001
Creatinine and Plasma LPO	80	0.4086	3.9540	0.000168
Creatinine and Erythrocytic LPO	80	0.3371	3.1627	0.002228
Creatinine and Plasma VC	80	–0.3941	3.7875	0.000298
Creatinine and Plasma VE	80	–0.5376	5.6304	0.000001
Creatinine and Plasma β-CAR	80	–0.3732	3.5529	0.000650
Creatinine and Erythrocytic SOD	80	–0.3341	3.1307	0.002454
Creatinine and Erythrocytic CAT	80	–0.4213	4.1024	0.000100
Creatinine and Erythrocytic GPX	80	–0.4316	4.2258	0.000064

TABLE 3

Pearson Product-moment Correlation Analysis of the Course of Disease and Every Biochemical Parameter

Correlative Item	*n*	Correlative Coefficient (*r*)	*t*	*P*
Course of Disease and Plasma NO	80	0.5917	6.4828	0.000001
Course of Disease and Plasma LPO	80	0.4790	4.8197	0.000007
Course of Disease and Erythrocytic LPO	80	0.3688	3.5037	0.000763
Course of Disease and Plasma VC	80	–0.4468	4.4109	0.000033
Course of Disease and Plasma VE	80	–0.5142	5.2943	0.000001
Course of Disease and Plasma β-CAR	80	–0.4546	4.5076	0.000023
Course of Disease and Erythrocytic SOD	80	–0.3963	3.8124	0.000273
Course of Disease and Erythrocytic CAT	80	–0.4616	4.5962	0.000016
Course of Disease and Erythrocytic GPX	80	–0.4363	4.2824	0.000052

TABLE 4

The Estimate of Relative Risk Ratio (RR) of the Biomedical Parameters Reflecting Oxidative Damage of CGNP, and 95 % CI of RR

Group	*n*	Plasma				Erythrocyte		Plasma						Erythrocyte					
		NO		LPO		LPO		VC		VE		β -CAR		SOD		CAT		GPX	
		+	–	+	–	+	–	+	–	+	–	+	–	+	–	+	–	+	–
CGNP	80	76	4	73	7	78	2	76	4	77	3	66	14	78	2	66	14	72	8
HAV	80	30	50	29	51	28	52	35	45	37	43	35	45	32	48	35	45	35	45
χ^2		59.1474		52.3597		69.8812		49.4503		48.8177		25.8030		61.5564		25.8030		38.6246	
P		0.0001		0.0001		0.0001		0.0001		0.0001		0.0001		0.0001		0.0001		0.0001	
Relative Risk Ratio (95% CI)		31.667 (10.513–95.382)		18.340 (7.460–45.089)		72.429 (16.450–317.170)		24.429 (8.146–73.255)		29.829 (8.681–102.493)		6.061 (2.931–12.533)		58.500 (13.409–255.223)		6.061 (2.931–12.533)		11.571 (4.928-27.171)	

Note. "+" indicates "number cases" that the values of NO in plasma, and LPO in plasma and in erythrocyte in CGNP group > the upper limits of 95 % CI of the values of the same biochemical parameters in HAV group, and that the values of VC, VE and β-CAR in plasma as well as SOD, CAT and GPX in erythrocyte in CGNP group < the lower limits of 95% CI of the values of the same biochemical parameters in HAV group, while "–" expresses opposite those.

5. Estimate of Relative Risk Ratio (RR) of the Above Biochemical Constituents Reflecting Oxidative Damages in the Bodies of Chronic Glomerulonephritis Patients, and 95 % CI of RR

Supposing the values of NO in plasma, and LPO in plasma and erythrocytes in the CGNP group > the upper limits of 95 % CI of the values of same biochemical parameters in HAV group to be the reference criteria of oxidative damage of positive number in the CGNP, and supposing the values of VC, VE and β-CAR in plasma as well as SOD, CAT and GPX in erythrocytes in CGNP group < the lower limits of 95 % CI of the values of same biochemical parameters in HAV group to be the reference criteria of oxidative damage of positive number in the CGNP, the relative risk ratio (RR) of the above biochemical parameters reflecting oxidative damage of the CGNP ranged from 6.061 to 72.429, and 95% CI of RR ranged from 2.931 to 317.170 (Table 4).

6. Reliability Analysis for the Above Biochemical Parameters Used to Estimate the Oxidative Damages in the Bodies of Chronic Glomerulonephritis Patients

The results of reliability analysis for the values of NO in plasma, LPO in plasma and erythrocytes, the values of VC, VE and β-CAR in plasma as well as the values of SOD, CAT and GPX in erythrocytes reflecting the oxidative damages in the bodies of CGNP were as follows: reliability coefficient (alpha) = 0.8137, standardized item alpha = 0.9728, Hotelling's T-Squared = 1135680.191, F = 53274.6478, P = 0.000001. Single measure intraclass correlation = 0.1953, 95% CI: 0.1420 – 0.2680, and average measure intraclass correlation = 0.8137, 95% CI: 0.7487 – 0.8682, F = 5.3686, P = 0.000001.

DISCUSSION

Nitric oxide (NO) plays an important role in renal hemodynamics and function[6]. Lipoperoxide (LPO) is a product of peroxidation (auto-oxidation) of lipids exposed to oxygen[6-14,17-29]. Lipoperoxidative reaction in plasma and tissues, especially in erythrocytic membranes containing a large number of polyunsaturated fatty acids (PUFAs), can lead to generation of a large number of free radicals [6-8,12-14,17-29]. LPO and its metabolic products, such as malondialdehyde (MDA), conjugated diene (CD), etc., are important poisonous residual products[6-8,12-14,17-29]. And they can strongly attack DNA, proteins, enzymes, biological membranes, PUFAs and others in the human body, leading to lipoperoxidative damage of cell membranes, lipoproteins and other lipid-containing structures [6-8,12-14,17-29]. Vitamin C (VC), vitamin E (VE) and β-carotene (β-CAR) are the most important antioxidants in the human body, while superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPX) are the most important antioxidases[13,14,18-20,26,27,29,30]. They play important roles in scavenging oxygen free radicals (OFRs), such as superoxide anion radical ($O_2^{\cdot-}$), hydroxyl radical (• OH), hydroperoxyl radical ($HO_2^{\cdot}$) and other free radicals (FRs) as well as singlet oxygen ($^1O_2$), hydrogen peroxide (H_2O_2) and other reactive oxygen species (ROS) which are excessive in the human body, and in preventing physiological and pathological aggravation of a series of free radical chain reactions induced by $O_2^{\cdot-}$, thereby protecting biological membranes of cells against oxidative and lipoperoxidative damages[13,14,18-20, 26, 27, 29, 30]. Marked decrease of the activities of the above antioxidants and antioxidases in the human body can cause metabolic disorders and pathological aggravation of a series of free radical chain reactions, thus inducing a variety of diseases related to the abnormal reactions of free radicals[1-14,18-20, 26-30].

The findings in the present study showed that in CGN patients' bodies the metabolism of NO resulted in serious disorders, the dynamic balance between oxidation and antioxidation produced grave imbalance, and the oxidative stress caused serious pathological aggravation, thereby inducing severe oxidative and lipoperoxidative damages in their bodies. There could be several interpretations. Nitric oxide plays an important role in renal hemodynamics and function and in pathogenesis of CGN [4, 5]. Evidence suggests that a variety of intraglomerular sources of nitric oxide (e.g., from macrophages and endothelial cells), in response to inflammatory cytokines, mechanical stress, and other mediators (e.g., acetylcholine or bradykinin), affect the contractile state and therefore the physiological function of the mesangial cells[5]. These inflammatory cytokines in the bodies of CGNP, especially interleukin-1 (IL-1), could activate immediately inducible nitric oxide synthase (iNOS), and stimulate the synthesis and/or release of NO, thus producing a large amount of NO[4, 5, 13, 14, 18-20, 27]. Excessive NO could inactivate antioxidases by combining with hydrosulfide group (-SH) which is a main active group of antioxidases, and excessive NO could combine with O_2^- to produce superoxide nitroso radical ($ONOO^-$), damaging the function of the mesangial cells and deactivating antioxidases with its extra-strong oxidative ability [4, 5, 13, 14, 18-20, 27]. Moreover, excessive NO could be rapidly oxidized into nitrogen dioxide (NO_2), and as a strong active catalyst in lipoperoxidation, NO_2 could aggravate the lipoperoxidation of PUFAs[13, 14, 18-20, 26, 27]. Excessive OFRs and ROS could also directly attack PUFAs, leading to lipoperoxidation of a large number of PUFAs, and subsequent production of a mass of LPO, MDA and CD, which strictly damage cell functions [13, 14, 18-20, 26, 27]. Additionally, significant decrease in the synthesis or regeneration of GPX decomposing LPO and the marked weakness or loss of the GPX activity could also result in significant increase of LPO level in CGN patients' bodies[13, 14, 18-20, 26, 27]. Meanwhile, these inflammatory cytokines, cells, phagocytes and other organic substances of the tissues in CGN patients also released a large number of OFRs, ROS and other FRs, which provoked pathological aggravation of a series of FRs chain reactions[13, 14, 18-20, 26, 27, 29, 30]. These abnormal metabolisms and disordered biochemical reactions resulted in marked increase of the values of NO in plasma, and LPO in plasma and erythrocytes, and the significant decrease of the values of VC, VE and β-CAR in plasma as well as SOD, CAT and GPX in erythrocytes[1, 2, 5-14, 18-20, 26-30]. At the same time, the above phenomena accelerated further the oxidative stress in the bodies of CGNP, thus leading to serious oxidative and lipoperoxidative damages in their bodies[1, 2, 5-14, 18-20, 26-30].

O_2^-, ·OH, $HO_2^·$ and other FRs as well as $^1O_2$, H_2O_2 and other ROS possessing strong oxidizing ability as well as their reactive products and/or metabolic products could interact directly with DNA, thereby causing DNA damage, and inhibiting or depressing DNA replication[13, 14, 18-20, 26, 27, 29, 30]. And they could also linearize circular DNA, which lead to significant decrease in synthesis or regeneration of SOD, CAT and GPX as well as marked weakness or loss of SOD, CAT and GPX activities in CGN patients' bodies[13, 14, 18-20, 26, 27, 29, 30]. Additionally, they could strongly attack the molecular structures of SOD, CAT, GPX and other antioxidases, inactivate the antioxidases by combining with –SH, thus resulting in significant decrease of the activities of SOD, CAT and GPX in the bodies of CGNP[13, 14, 18-20, 26, 27, 29, 30].

It must be emphasized that most antioxidative vitamins such as vitamin C, vitamin E and β-carotene have to be acquired from dietary sources because these vitamins cannot be synthesized in the human body[13, 14, 18-20, 26-30]. In general, the everyday diets of CGN patients are strictly restricted, their diets are very simple and lack of nutrition, especially VC, VE and β-CAR, because of the needs of dietotherapy[1-4, 13, 14, 18-20, 26-30]. Nevertheless, *antioxidative*-vitamins-poor diets cannot provide sufficient scavengers of FRs to keep the dynamic balance

between oxidation and antioxidation, which leads to physiological and pathological aggravation of a series of FRs chain reactions in the bodies of CGNP[13, 14, 18-20, 26-30]. In this condition, CGN patients have to make use of a great quantity of antioxidants and antioxidases in their bodies in order to scavenge these excessive OFRs and other FRs and ROS, thus resuming and keeping dynamic balance between oxidation and antioxidation, and decreasing oxidative damages in their bodies[13, 14, 18-20, 26-30]. Therefore, we think that this is the main reason why in the bodies of CGNP the values of NO in plasma, and LPO in plasma and erythrocytes markedly increased, and the values of VC, VE and β-CAR in plasma as well as SOD, CAT and GPX in erythrocytes significantly decreased[13, 14, 18-20, 26-30].

In the present study, with increase of the blood creatinine content and prolongation of the disease course in CGNP, their contents of NO in plasma, and LPO in plasma and erythrocytes increased gradually, while their contents of VC, VE and β-CAR in plasma as well as the activities of SOD, CAT and GPX in erythrocytes decreased gradually. These findings showed that the values of the above biochemical parameters were closely related to the state of illness and course of disease in the CGNP. The above biochemical parameters determined dynamically and timely would be beneficial to CGN patients[13, 14, 18-20, 26-30].

The findings of the relative risk ratio (RR) estimation in this study showed that compared with the healthy adult volunteers, the RR of oxidative damages in the bodies of CGNP would be increased by 5.1 – 71.4 times. These evidences further showed that oxidation stress induced by NO, OFRs and other FRs as well as ROS played very important roles in the oxidative and lipoperoxidative damages in the bodies of CGNP[1, 2]. At the same time, in the reliability analysis of the present study the reliability coefficient was highly relative, and satisfactory, and showed that excessive NO, OFRs and other FRs as well as ROS in the bodies of CGNP would lead to severe injuries in CGN patients.

In summary, the findings of the present study suggested that the dynamic balance between oxidation and antioxidation in the bodies of CGN patients resulted in severe disorder, and oxidative stress in their bodies was gravely aggravated, and the chain reactions of a series of free radicals were seriously exacerbated, thereby leading to oxidative and lipoperoxidative damages in their bodies. We, therefore, recommend that antioxidant vitamins at suitable doses, such as vitamin C, vitamin E and β-carotene, should be given to CGN patients daily in order to alleviate oxidative and lipoperoxidative damages in their bodies because these vitamins are free-radical scavengers and can protect chronic glomerulonephritis patients against further oxidative damages[13, 14, 18-20, 26, 27, 30-39].

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