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Original Articles

A study on relationship of nitric oxide, oxidation, peroxidation, lipoperoxidation with chronic cholecystitis

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Abstract

AIM To study relationship of injury induced by nitric oxide, oxidation, peroxidation, lipoperoxidation with chronic cholecystitis.

METHODS The values of plasma nitric oxide (P-NO), plasma vitamin C (P-V C), plasma vitamin E (P-VE), plasma β-carotene (P-β-CAR), plasma lipoperoxides (P-LPO), erythrocyte superoxide dismutase (E-SOD), erythrocyte catalase (E-CAT), erythrocyte glutathione peroxidase (E-GSH-Px) activities and erythrocyte lipoperoxides (E-LPO) level in 77 patients with chro nic cholecystitis and 80 healthy control subjects were determined, differences of the above average values between t he patient group and the control group and differences of the average values bet ween preoperative and postoperative patients were analyzed and compared, linear regression and correlation of the disease course with the above determination values as well as the stepwise regression and correlation of the course with th e values were analyzed.

RESULTS Compared with the control group, the average values of P -NO, P-LPO, E-LPO were significantly increased ($P < 0.01$), and of P-VC, P-VE, P-β-CAR, E-SOD, E-CAT and E-GSH-Px decreased ($P < 0.01$) in the patient group. The analysis of the lin ear regression and correlation s howed that with prolonging of the course, the values of P-NO, P-LPO and E-LPO in the patients were gradually ascended and the values of P-VC, P-VE, P-β-CAR, E-SOD, E-CAT and E-GSH-Px descended ($P < 0.01$). The analysis of the stepwise regression and correlation indicated that the correlation of th e course with P-NO, P-VE and P-β-CAR values was the closest. Compared with the preoperative patients, the average values of P-NO, P-LPO and E-LPO were significantly decre ased ($P < 0.01$) and the average values of P-VC, E-SOD, E-CAT and E-GSH-Px in postoperative pa tients increased ($P < 0.01$) in postoperative patients. But there was no signif icant difference in the average values of P-VE, P-β-CAR preope rative and postoperative patients.

CONCLUSION Chronic cholecystitis could induce the increase of nitric oxide, oxidation, peroxidation and lipoperoxidation.

INTRODUCTION

Chronic cholecystitis is a frequently encountered disease of the digestive syste m. Some studies point out that in blood of patients with acute cholecystitis the levels of inducible nitric oxide (*i* NO) and lipoperoxides are markedly incre ased, while the level of vitamin C, the activities of superoxide dismutase and g lutathione enzyme are significantly decreased[1-5]. However, up to now , there has been no reports on the above in patients with chronic cholecystit is. In order to observe the metabolic state of nitric oxide and other free radi cals in patients with chronic cholecystitis, and the degree of injury induced by oxidation, peroxidation, lipoperoxidation due to the chronicity of cholecystiti s, we determined nitric oxide (P-NO), vitamin C (P-VC), vitamin E (P-VE), β-carotene (P-β-CAR) and lipoperoxides (P-LPO) levels in the pla sma as well as superoxide dismutase (E-SOD), catalase (E-CAT), glutathione peroxidase (E- GSH-Px) activity and lipoperoxides (E-LPO) level in the erythrocytes in 77 patients with chronic cholecystitis and 80 healthy controls.

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We also an alyzed and compared differences of the above average determination values betwee n the patient and the control group, and between preoperative and postoperative patients. Additionally, we analyzed the relationship between the course of the d isease and the above values in the patients by the linear regression and correla tion as well as the stepwise regression and correlation.

SUBJECTS AND METHODS

Subjects

Patients Seventy-seven patients suffering form chronic cholecystitis with gallsto nes who were confirmed diagnostically through abdominoscopy and biopsy in the People's Hospital of Jinhua City were randomly sampled. Their ages ranged fro m 31 to 69 years (52.4 a ± 10.3 a), and their courses of disease were from 2 to 20 years (5.2 a ± 5.6 a). Of them, 32 were male and 45 were female. No patients had abnormality in the routine examination of blood, urine, feces, ECG and X -rays, and medical history about heart, brain, lung, liver, kidney, diabetes, a utoimmune disease, peripheral vascular disease, cataract, tumor, and so on. The gallbladders of all the patients with chronic cholecystitis were removed by abdo minoscopy.

Control Eighty healthy adults confirmed through the comprehensive he alth examination by the 2nd Affiliated Hospital of Zhejiang University were randomly sampled, their ages were from 31 to 70 years (52.7 a ± 9.6 a), and 40 were male and 40 were female. The healthy adults were all normal in the routine examination of blood, urine, feces, ECG and X-rays, with no medic al history regarding heart, brain, lung, liver, kidney, cholecystic disease, diabetes, autoimmune disease, peripheral vascular disease, cataract, tumor, etc .

All the patients and the healthy adults had neither exposure to kind of radiation, nor contacted any kind of pesticide and poison. Within a month prior to the study they had not taken any antioxidants such as vitamin C, vitamin E, g inkgo leaf agents, tea-polyphenol etc. There was neither any significant di ffer ence ($P > 0.05$) between the average age of the patient and the control group a s determined by *t* test, and nor any significant difference ($P > 0.05$) betw een the gender proportion of the patient and the control group as determined by χ^2 test.

Methods

Blood samples Fasting venous blood samples were collected in the morning for all the subjects with heparin sodium as an ant icoagulant. The separated plasma and erythrocytes were stored immediately at 4℃[6].

Plasma NO (P-NO) level Colloidal aluminium hydroxide without nitrite was used to absorb yellow pigments and to cause protein sedimentation in the plasma. The nitrite in the supernatant, which contained sodium acetate (0.20 mol/L) an d sulphanilic acid (3.30 mmol/L), reacted with β-Naphthyla mine and formed a colored product, which was detected spectrophotometrically, using sodium nitrit e (2.50 μmol/L) as the standard and at a wavelength of 520 nm. The P-NO concentration was expressed in nmol/L[6,7].

Plasma LPO (P-LPO) level Trichloroacetic acid (TCA) solution (20.0 g%, w /v) was used to cause protein sedimentation in the plasma. The protein sediment reacted with thiobarbituric acid (TBA) solution (0.67 g%, w/v) and produced red colored compounds following incubation in a water bath at 100℃. This was detect ed spectrophotometrically at 532 nm, using tetraethoxypropane (TEP, 5.0 μmol/L) as the standard. The P-LPO concentration was expressed as μmol/L[6,8].

Plasma VC (P-VC) level TCA (5.0 g%, w/v) was used to cause protein sedim entation in the plasma, and ferric trichloride was added to the supernatant. Vit amin C in the supernatant reduced Fe^{3+} in ferric trichloride to Fe^{2+} . Fe^{2+}, on reacting with ferrocene, produced a colored product which was detected spectrophotometrically at 563 nm, using vitamin C as the standa rd. The P-VC concentration was expressed as μmol/L[6,9] .

Plasma VE (P-VE) level Absolute ethyl alcohol was used to cause protein sedimentation in the plasma and to extract vitamin E. Vitamin E in the supernata nt reduced Fe^{3+} in ferric trichloride to Fe^{2+}. Fe^{2+} reacted wi th ferrocene to form a colored product that was detected spectrophotometrically at 563 nm, using vitamin E as the standard. The P-VE concentration was expressed as μmol/L[6,10].

Plasma β-CAR(P-β-CAR)level A mixture of a bsolute ethyl alcohol and petroleum ether was used to cause protein sedimentati on in the plasma and to extract β-carotene. The petroleum ether extract containing β-carotene was an alyzed colorimetrically, using β-carotene as the standard at a wav elength se tting of 440 nm. The P-β-CAR concentration was expressed as μmol/L[6,11].

Erythrocyte LPO (E-LPO) level A mixture of absolute ethyl alcohol and t richlorom ethane (5 : 3) was used to precipitate hemoglobin (Hb) from a hemolytic solution (HS) of RBC without WBC and platelets. Hb level was determined in the HS. LPO in the extracted solution reacted with TBA-glacial acetic acid solution (1.0 g%, w/v) in a water bath at 100℃ and produced red colored compounds. These were det ected using TEP (5.0 μmol/L) as the standard at 532 nm. The E-LPO concentration was expressed as nmol/g Hb[6,12].

Erythrocyte SOD (E-SOD) activity A mixture of absolute ethyl alcohol and trichloromethane (5 : 3) was used to precipitate Hb from the HS of RBC without WBC and platelets. Hb level was determined in the HS. Pyrogallol (6.0 mmol/L) au to-oxidized in Tris-HCl buffer (50

mmol/L, pH 8.20), SOD was added to the buff er to inhibit its auto-oxidation and SOD activity was calculated according to t he auto-oxidation rate of pyrogallol and the rate of SOD-inhibited pyrogallol auto-oxidation. The WL of 420 nm was used and the E-SOD activity was indicated as U/g Hb[6,13].

Erythrocyte CAT (E-CAT) activity H_2O_2 (0.20 mol/L) was added to phos phate buffer (10 mmol/L, pH 7.0) containing HS of RBC without WBC and platelets.The Hb level was determined in the HS. After a reaction time of 60 s, a s olution of potassium dichromate (0.169 mol/L) and glacial acetic acid (1 : 3) was added to the reacting mixture to stop the reaction, and the reacting mixture was heated for 10 min at 100℃. Colorimetry was done at 570 nm. The E-CAT activity was indicated as K/g Hb[6,14].

Erythrocyte GSH-Px (E-GSH-Px) activity A mixture of absolute ethyl alc ohol and trichloromethane (5 : 3) was used to precipitate Hb from the HS of RBC without WBC and platelets. Hb level was determined in the HS. GSH-Px in the ext ract catalyzed the reaction of glutathione and 5, 5'Dithiobis-*p*-nitr obenzoic acid (DTNB) and produced yellow colored compounds which were detected at 422 nm, using glutathione (1.0 mmol/L) as the standard. The E-GSH-Px activity was expressed as U/g Hb[6,15].

Major analytical reagents such as Vitamin C, Vitamin E, β-Carotene , Superoxi de dismutase, Catalase, β-Naphthylamine, 1,2,3-Trihydroxybenzene (pyrogallol), 1, 1,3,3-Tetraethoxypropane, 2-Thiobarbituric acid were all purchased from SIGMA CHEMICAL COMPANY, USA; and the other analytical-grade reagents were all procured form China. The main analytical instruments were 721-spectrophotometer and UV-754-spectrophotometer.

Statistic analysis

All data were analyzed with SPSS/8.0 and Statistica/6.0 statistic software u sing Compaq Pentium III/600 computer. Statistical testing methods inc luded unpaired and paired *t* test and chi square test (χ^2 test), linear reg ression and correlation analysis, stepwise regression and correlation analysis, and confidence interval (CI) of 95%. The level of significance of hypothesis testing was $P < 0.05$ a nd the power of test (power) > 0.75.

Results

Comparison between the above mentioned determinations in the patien t and the control group The average values determined for P-NO, P-LPO and E-LPO in the patient group were significantly increased ($P < 0.01$) with respect to the control, whereas the average values of P-VC, P-VE, P-β-CAR, E-SOD , E-CAT and E-GSH- Px in the patient group were significantly decreased ($P < 0.01$) (Table 1).

Comparison between the above mentioned determinations in the preop erative an d the postoperative patients The average values of P-NO, P-LPO and E-LPO in the postoperative patients were significantly decreased ($P < 0.01$), whereas the average determination values of P-VC, E-SOD, E- CAT and E-GSH-Px in the postoperative patients were significantly increased ($P < 0.01$) , but there was no significant difference between the average values of P-VE, P-β-CAR in the pre- and postoperative patients (Table 2).

Linear regression and correlation analysis between the course of disease and the above mentioned values determined in the patients In pace with gradua l prolongi ng of the course of disease in the patients, the values of P-NO, P-LPO, E-LPO in the patients were gradually increased ($P < 0.01$), the values of P-VC, P-VE, P-β-CAR, E-SOD, E-CAT, E-G SH-Px were gradually decreased ($P < 0.01$) (Table 3).

Table 1 Comparison of various determinations between patient group and control group (CI 95%, $\bar{x} \pm s$)

Group	*n*	P-NO nmol/L	P-VC μmol/L	P-VE μmol/L	P-β-CAR μmol/L	E-SOD U/g Hb	E-CAT K/g Hb	E-GSH-Px U/g Hb	P-LPO μmol/L	E-LPO nmol/g Hb
Patient	77	514 ± 142	44.3 ± 10.9	19.1 ± 4.6	1.35 ± 0.38	1813 ± 249	233 ± 57	22.7 ± 4.8	13.6 ± 1.9	38.2 ± 7.2
		482-546	41.8-46.8	18.0-20.1	1.26-1.44	1757-1869	220-246	21.6-23.8	13.2-14.0	36.6-39.8
Control	80	365 ± 157	55.2 ± 12.8	25.4 ± 5.3	1.72 ± 0.45	2057 ± 212	309 ± 61	27.2 ± 5.5	11.3 ± 1.7	29.4 ± 6.7
		330-400	52.4-58.0	24.2-26.6	1.62-1.82	2010-2104	295-323	26.0-28.4	10.9-11.7	27.9-30.9
t		6.2290	5.7344	7.9415	5.5559	6.6197	8.0588	5.4535	8.0001	7.9316
P		<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01

Table 2 Comparison of various determinations between preoperative and postoperative patients (CI 95% $\bar{x} \pm s$)

Group	*n*	P-NO nmol/L	P-VC μmol/L	P-VE μmol/L	P-β-CAR μmol/L	E-SOD U/g Hb	E-CAT K/g Hb	E-GSH-Px U/g Hb	P-LPO μmol/L	E-LPO nmol/g Hb
Postoperative	77	514 ± 142	44.3 ± 10.9	19.1 ± 4.6	1.35 ± 0.38	1813 ± 249	233 ± 57	22.7 ± 4.8	13.6 ± 1.9	38.2 ± 7.2
		482-546	41.8-46.8	18.0-20.1	1.26-1.44	1757-1869	220-246	21.6-23.8	13.2-14.0	36.6-39.8
Postoperative	77	436 ± 139	48.5 ± 11.3	19.3 ± 5.1	1.34 ± 0.36	1915 ± 242	274 ± 59	25.1 ± 5.3	12.4 ± 1.8	34.9 ± 6.9
		404-468	45.9-51.1	18.1-20.4	1.26-1.42	1860-1970	261-287	23.9-26.3	12.0-12.8	33.3-36.5
*t**		8.9773	7.5376	0.8739	0.5732	7.9814	10.8058	9.5453	9.2384	10.3493
P		<0.01	<0.01	>0.05	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01

*Paired *t* test

Table 3 Linear regression and correlation analysis between the course of disease and the values determined in the patients

Correlative item	*n*	Regression equation	*r*	t_r	*P*
Course with P-NO	77	Y = 368.6527+18.8688X	0.7162	8.8874	<0.01
Course with P-LPO	77	Y = 11.8836+0.2212X	0.6958	8.3888	<0.01
Course with E-LPO	77	Y = 31.5301+0.8807X	0.6369	7.1553	<0.01
Course with P-VC	77	Y = 53.7124-1.1505X	0.6495	7.3982	<0.01
Course with P-VE	77	Y = 23.3849-0.4532X	0.5572	5.8114	<0.01
Course with P-β-CAR	77	Y = 1.7887-0.0486X	0.7428	9.6093	<0.01
Course with E-SOD	77	Y = 2075.87-29.3446X	0.6239	6.9146	<0.01
Course with E-CAT	77	Y = 282.0238-6.7284X	0.7227	9.0545	<0.01
Course with E-GSH-Px	77	Y = 28.6710-0.7006X	0.7233	9.0702	<0.01

Stepwise regression and correlation analysis for the course of disease and the a bove-mentioned values determined in the patients Supposing the course of disease in the patients to be y, the determination valu es of P-NO, P-VC, P-VE, P-β-CAR, E-SOD, E-CAT, E-GSH-Px, P- LPO and E-LPO in the patients to be x_1, x_2, x_3, x_4, x_5, x_6, x_7, x_8 and x_9 respectively, after stepwise regression and corre lation, the stepwise regression equation was $y = -0.2706 + 0.0203x_1 + 0.6844x_3 - 11.3731x_4$, $r = 0.7902$, $F = 40.4440$, $P < 0.01$. The e quation suggested that the correlation of the course of disease was the closest with the values determined for P-NO, P-VE and P-β-CAR.

DISCUSSION

The metabolic status of nitric oxide and functional status be tween oxidation and antioxidation systems in human body are in close relationship with health[6-10,12-73]. If the metabolism of nitric oxide is abnorm al and the dynamic balance between oxidation and antioxidation is disturbed, free radicals (FRs) concentration will unusua lly increase and a series of FRs chain reactions will pathologically aggravate i n human body. This status can speed senility of human cell s, and induce many diseases[6-10,12-73]. Vitamin C (VC), vitamin E (VE) and β-carotene (β-CAR) are the most imp ortant antioxidants in human body, and they play an important role in scavenging superoxide anions ($O^{\cdot}_2$), hydxoy l radic al (• OH), hydroperoxyl radical ($HO •_2$), lipid FRs, lip ox yl FRs, alkyl FRs, alkoxyl FRs, singlet oxygen ($^1O_2$), hydrogen perox ide (H_2O_2) and others, thereby protecting biological membranes a gainst oxidation, peroxidation and lipoperoxidation[6,9,10,44-54, 56-69] injury. And they can promote synthesis and stabilization of immunoglobulin in human body and obstruct formation of carcinogens such as nitrosamine [6,9,1 0,44-54,56-69]. Superoxide dismutase (SOD), catalase (CAT) and glu tathione peroxidase (GSH-Px) are the most important specific antioxidases in hu man body, SOD is able to clean $O^{\cdot}_2$, obstruct and preven t the pathological aggravation of a series of FRs chain reactions induced by $O •_2$, CAT enables toxic active mass H_2O_2 to degrad e into non-toxic O_2 and H_2O, GSH-Px may decompose toxic active mass LPO[6,13-15,29-39,41,42,46-54,56-69]. LPO and its metabolic products such as mal ondialdehyde (MDA), conjugated diene (CD) and others are important po i sonous residual products that enable biological membranes to be injured by lipop eroxidation. Marked increase in LPO level in h uman body can strongly attack DNA, proteins, enzymes, biological membranes and so on, which leads to the lipoperoxidation injury of the biological membranes, etc [5-8,39-43,48-53,62-71]. Nitric oxide (NO) is a neurotransmitter and en dothelium-derived factor that reduces tone of vascular smooth muscle, and disor der of NO metabolism can induce many diseases[6,7,16-28,46-49,55,62-66].

In this study the results that the average values of P-NO, P-LPO and E-LPO in the patient group were significantly higher than those in the control group ($P < 0.01$), and that the average values of P-VC, P-VE, P-β-C AR, E-SOD, E-CAT and E-GSH-Px in the patient group were significantly lower than those of the cont rol group ($P < 0.01$) showed that there was a severe disorder o f the NO metabolism and imbalance between oxidation and antioxidation, and there was the pathological aggravation of the oxidation, peroxidation, lipoperoxidat ion reactions in the bodies of the patients with chronic cholecystitis and galls tones. The causes probably were as follows. The cytokines, particularly interleukin -I (IL-1), which were released out by inflammatory cells such as phagocytes namely lymphocytes, neutrophilic granulocytes, macro-phagocytes in the cholec ystic inflammatory reaction, can activate inducible nitric oxide synthase (iNOS). The iNOS enables NO to be produced excessively in the body of p atien ts, thereby resulting in a significant increase in the P-NO value in the patients[6,7,16-28,46-49, 55,62-66]. Excessive NO was diffused into near by tissues and cells, thus furthe r leading to injury of the tissues and cells[6,7,16-28,46-49,55,62-66]. The excess NO can combine with iron ions in heme group, with activated guany late cyclase and lipoperoxidation reaction. The excess NO also inactivated antioxidases such as SOD, CAT, GSH-Px by means of the reaction of NO and hydrosul-fide group (-SH) in the enzymes, which furthe r resulted in marked decrease in SOD, CAT, GSH -Px activi ties and further injured cells and biologic membranes. Excessive NO in the body was able to be speedily oxidated into nitrogen dioxide (NO_2). Both NO and NO_2 thems elves are extremely active FRs, NO_2 was still able to react with the organ ic molecules in cystic bile, and activate the neutrocytes and phagocytes in the cho lecystic focus, thereby releasing out a vast amount of $O •_2$, • OH, $HO •_2$, and H_2O_2 etc. Meanwhile, the phagocytes such as polymorphonuclear leukocyte were speedily activated, and a large number of $O •_2$, • OH, $HO •_2$ etc were released out, and continuously got into the blood stream i n the patients, thereby inducing the pathological aggravation of a series of FRs c hain reactions[6,7,16-28,46-49,55,62-66].

It must be stressed that excessive NO was capable of reacting speedily with $O •_2$, thereby forming another kind of free radical, i.e. sup erox ide nitroso free radical ($ONOO^-$) which possessed still more strong oxidative

properties. $ONOO^-$ can further attack and injure the various cells in the body, and deact ivate the antioxidases such as SOD, CAT and GSH-Px. Excess NO and NO_2 in hu man body injured DNA by way of th e deamination of the base and the chain scission[6,7,46-49,55,62-66]. So, on one hand the P-NO level in the body of patients was significantly increased, and on the other hand the body had no choice but to put to goo d us e a great quantity of antioxidants and antioxidases in the body so as to catch and clear these excess $O \cdot_2$, $\cdot OH$, $HO_2 \cdot$ and others [6-10,12-73], which resulted in significant decrease of the levels of P-VC, P-VE, P-β-CAR and the act ivities of E-SOD, E-CAT, E-GSH-Px in the patients. Besides the gallbladder calculi such as bilirubin, cholesterol and other organic substances themselves also produce a large number of FRs[2-5,29,34,35,70-78].

NO_2 is a very active catalyst, and NO_2 can aggravate lipoperoxidation of the polyunsaturated fatty acids (PUFAs) through hydrogen-extractive process. The excess NO, NO_2, $O \cdot_2$, $\cdot OH$, $HO \cdot_2$ also can attack upon directly PUFAs, aggra vate significantly the lipoperoxidation, thereby resulting i n a large number of PUFAs which get lipoperoxidated, and subsequently form LPO. With the addition of the significant reduction in th e synthesis or regeneration of GSH-Px decomposing LPO, and the marked lose of the GSH Px activity, it goes without saying that finally th is status resulted in significant increase of P-LPO and E-LPO levels[6,7,46-49, 5 1,52,55,62-66].

In general most anti-oxidative vitamins such as VC, VE, β-CAR, etc, must b e acquired from dietary sources because they cannot be synthesized in the body. It is generally recognized that the chronic chole cystitis patients have poor appetite because their diets are controlled, and digestion of VC, specially digestion of fat-soluble vitamin s such as VE and β-CAR, markedly reduced. And the anti-oxidative vitamin-poor diets cannot provide sufficient free radical scav engers to keep the balance between oxidation and antioxidation. For this reason, the values of P-VC, P-VE, P-β-CAR in the bodies of the patients were further significantly decreased[6,9,10,47-54,56 -68].

In this study the average values of P-NO, P-LPO and E-LPO in the pos toperative patients were significantly decreased, the average values of P-VC, E-SOD, E-CAT, E-GSH-Px were significantly increased, but there was no significant difference in the average values of P-VE and P-β-CAR between the preoperative and postoperative patients. The findings showed that the series of FRs chain reactions in the body of patients were marked lysis, and the dynamic balance between oxidation and antioxidation, obtained resumption to a very marked degree because of the elimination of inflammatory focus after operati on. However, before the compensation of common bile was established the absorpti on of fats and lipids was still limited, thus the absorption of fat-solubl e VE and β-CAR obviously reduced[6,9,10,47-54,56-68] . Therefore, the normal levels of P-VE, P-β-CAR in the patients were diffi cult to be resumed shortly after operation.

In this study there was the linear correlation between the course of disease and the above determined values, specially the stepwise correlation with the course with P-NO, P-VE, P-β-CAR values was the closest. This st atus suggested t hat in chronic cholecystitis, a large amount of NO p roduced by iNOS induced and activated by the long-time infection and stimulatio n of the calculi in gallbladder provoked the pathological aggravation of a serie s of FRs chain reactions[2-5,16-29,34,35,46-54,56-78]. As a result, the patients with chronic cholecystitis over a long time were in the state of serious imbalance between oxidation and antioxidation as well as injuries induced by oxidation, peroxida tion and lipoperoxidation[2-5,29,34,35,70-78]. The findingss also sho wed that the metabolic status of nitric oxide and the changes in vitamin E and β-carotene levels in the body played an important part in chronic cholecystitis. Therefore, the above values, particularly the dynamic determination of P-NO, P-VE and P-β-CAR values, to a g reat degree, contribute to wards monitoring the condition and course in patients with chronic cholecystitis.

We think that in treating preoperative and postoperative patients with chronic cholecystitis with suitable dosage of antioxidants such as vitamin C, vitamin E, β-carotene, ginkgo leaf agents, tea-polyphenol daily to the patients may alleviate the injuries induced by oxida tion, peroxidation and lipoperoxidation.

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