

Dental status and oxidative homeostasis state in patients exposed to occupational vibration: superoxide dismutase and catalase content in oral fluid

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The aim of the research involved determination of catalase and superoxide dismutase content in oral fluid of patients exposed to occupational vibration depending upon their dental status.

Materials and methods. The assessment of dental status (DS) and superoxide dismutase (SOD) and catalase (CAT) content in oral fluid (OF) was performed in three groups of patients: control group ($n_0=129$) included the persons exposed to occupational vibration and whose results of combined medical examination excluded the presence of vibration disease (VD); the second ($n_1=63$ patients with VD stage I) and the third ($n_2=66$ patients with VD stage II) groups consisted of the patients, who underwent treatment at the clinical department of the Research Institute of Occupational Hygiene and Occupational Diseases of Kharkiv National Medical University of the Ministry of Health of Ukraine. DS determination was carried out according to the method of K. M. Kosenko (Patent No. 57512, Ukraine) for in-patients and controls (during medical checkups) using the following indices: PMA, OHI-S, DMFT, with assessment of vacuum-pressory resistance of gingival capillaries (VPRC) (according to V. I. Kulazhenko) and community periodontal index of treatment needs (CPITN). SOD content was determined by the nonenzymatic method; CAT content was revealed spectrophotometrically. Primary data were statistically processed with the determination of accuracy by Student's test.

Results. SOD content depending upon PMA intensity in VD patients ranged from 14.1 ± 0.2 U/min to 15.7 ± 0.5 U/min, was reliably ($p < 0.05$) lower in patients with VD versus the controls (17.8 ± 0.2 U/min and 14.2 ± 0.2 U/min respectively, when $PMA > 2.1$) and did not differ depending upon VD severity (15.7 ± 0.5 U/min in VD stage I and 15.3 ± 0.3 U/min in VD stage II, respectively). SOD content in OF in patients depending upon their OHI-S ranged from 13.5 ± 0.3 U/min to 16.3 ± 0.2 U/min and was reliably ($p < 0.05$) lower in patients with $OHI-S \geq 1.7$ U. A comparative analysis showed that the activity of the enzymatic protection of the periodontal membrane could be also determined by the state of hard tissues, in particular by such DS index as DFTM. The activity of SOD in VD stage II was found to be reliably ($p < 0.05$) reduced in patients with DFTM index exceeding 15 pts. A somewhat different pattern of SOD activity was found in OF in patients with VD stage I: SOD activity in OF was similar in all DFTM indices and it became reduced depending upon an increase of DFTM index. SOD content depending upon VPRC index in patients with VD ranged from 10.7 ± 0.5 U/min to 16.8 ± 0.3 U/min and was reliably ($p < 0.05$) lower in cases with VPRC index ≤ 40 sec. CAT content depending upon PMA intensity in VD patients ranged from 4.6 ± 0.4 U/min to 11.3 ± 0.3 U/min and was reliably ($p < 0.05$) higher in patients with VD stage I versus the controls and differed according to the severity. CAT content in OF in

Stan uzębienia i stan homeostazy oksydacyjnej u chorych narażonych na wibracje zawodowe: zawartość dysmutazy nadtlenkowej i katalazy w płynie z jamy ustnej

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Celem badań było określenie zawartości katalazy i dysmutazy nadtlenkowej w płynie z jamy ustnej chorych narażonych na wibracje zawodowe w zależności od stanu uzębienia.

Materiał i metody. Ocenę stanu uzębienia (DS) oraz zawartości dysmutazy nadtlenkowej (SOD) i katalazy (CAT) w płynie z jamy ustnej (OF) przeprowadzono w trzech grupach: grupa kontrolna ($n=129$) obejmowała osoby narażone na wibracje zawodowe i u których wyniki połączonego badania lekarskiego wykluczyły obecność choroby wibracyjnej (VD); grupa druga ($n=63$ chorych z I stadiem VD) i trzecia ($n=66$ chorych z II stadiem VD) składały się z chorych, którzy byli leczeni w Oddziale Klinicznym Instytutu Higieny Pracy i Chorób Zawodowych Charkowskiego Narodowego Uniwersytetu Medycznego Ministerstwa Zdrowia Ukrainy. Oznaczenie DS przeprowadzono według metody KM Kosenki (patent nr 57512, Ukraina) dla hospitalizowanych i z grupy kontrolnej (podczas badań lekarskich) przy użyciu następujących wskaźników: PMA, OHI-S, DMFT, z oceną podciśnienia – oporność ciśnieniowa naczyń włosowatych dziąseł (VPRC) (według VI Kulazhenki) oraz wspólny wskaźnik potrzeb leczenia przyzębia (CPITN). Zawartość SOD oznaczono metodą nieenzymatyczną. Zawartość CAT została ujawniona spektrofotometrycznie. Dane pierwotne zostały przetworzone statystycznie z określeniem znamienności testem Studenta.

Wyniki. Zawartość SOD w zależności od intensywności PMA u chorych z VD wahała się od $14,1 \pm 0,2$ U/min do $15,7 \pm 0,5$ U/min i była znamienne ($p < 0,05$) niższa u chorych z VD w porównaniu z grupą kontrolną ($17,8 \pm 0,2$ U/min i $14,2 \pm 0,2$ U/min, gdy $PMA > 2,1$) i nie różniły się w zależności od stanu ciężkości VD (odpowiednio $15,7 \pm 0,5$ U/min w I stopniu VD i $15,3 \pm 0,3$ U/min w II stopniu VD). Zawartość SOD w OF chorych w zależności od OHI-S wahała się od $13,5 \pm 0,3$ U/min do $16,3 \pm 0,2$ U/min i była znamienne ($p < 0,05$) niższa u chorych z $OHI-S \geq 1,7$ U. Analiza porównawcza wykazała, że aktywność ochrony enzymatycznej błony ozębnej może być również determinowana stanem tkanek twardej, w szczególności takim wskaźnikiem DS jak DFTM. Stwierdzono, że aktywność SOD w II stopniu zaawansowania VD jest znamienne niższa ($p < 0,05$) u chorych z indeksem DFTM powyżej 15 pkt. Nieco inny wzorzec aktywności SOD stwierdzono w OF chorych z I stadiem VD: aktywność SOD w OF była podobna we wszystkich wskaźnikach DFTM i ulegała zmniejszeniu w zależności od zwiększania się wskaźnika DFTM. Zawartość SOD w zależności od wskaźnika VPRC u chorych z VD wahała się od $10,7 \pm 0,5$ U/min do $16,8 \pm 0,3$ U/min i była wiarygodnie ($p < 0,05$) niższa w przypadkach ze wskaźnikiem VPRC ≤ 40 sek. Zawartość CAT w zależności od intensywności PMA u chorych z VD wahała się od $4,6 \pm 0,4$ j./min do $11,3 \pm 0,3$ j./min i była wiarygodnie ($p < 0,05$) wyższa u chorych z I stadiem VD w porównaniu z grupą kontrolną i różniła się w zależności od ciężkości. Zawartość CAT w OF

patients depending upon OHI-S ranged from 5.2 ± 0.2 U/min to 10.1 ± 0.3 U/min, was reliably ($p < 0.05$) lower in cases with $OHI-S \geq 1.7$ U, did not differ from the indices observed in the control group and was also found to be reliably lower in patients with VD stage II versus those with VD stage I (7.3 ± 0.3 U/min and 8.6 ± 0.2 U/min, respectively, when OHI-S ranged within 0.7 ± 1.6 U). CAT content in OF depending upon VPSC index in patients with VD ranged from 5.8 ± 0.2 U/min to 8.6 ± 0.6 U/min and was reliably ($p < 0.05$) lower in cases with VPSC index ≤ 40 sec. Thus, CAT activity in OF in patients was reliably ($p < 0.05$) reduced (in $VPRC > 40$ sec it was equal to 7.8 ± 0.2 U/min, and in $VPRC \leq 40$ sec it was 8.6 ± 0.1 U/min) in VD stage I with decreased VPRC.

Conclusions. A trend ($p > 0.05$) towards an increase in SOD activity in VD stage I versus the controls was revealed, whereas VD stage II demonstrated a reliable ($p < 0.05$) reduction of the above activity. At the same time, an unsatisfactory state of oral hygiene was shown to promote inhibition of the enzymatic protection of their periodontal membrane in patients with VD stage I. A trend ($p > 0.05$) towards an increase of SOD activity in VD stage I versus the controls was revealed, whereas VD stage II demonstrated a reliable ($p < 0.05$) reduction of the above activity. The assessment carried out in cases requiring combined treatment with surgical or non-surgical debridement and also in patients with supra- or subgingival dental calculus found out that SOD activity was reliably reduced only in cases with VD stage II. CAT activity assessment in OF in VD patients having different levels of CPITN showed that the above activity in persons requiring combined treatment (including prosthodontic treatment; $CPITN \geq 3$, 1 points) was markedly and reliably reduced. All the above facts determine peculiarities in oral treatment strategies for this group of patients.

Key words: dental status, periodontal membrane, vibration disease

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Only few researches, which contain some clinical recommendations, have been dedicated to problems in the clinical picture, diagnosis and treatment of oral diseases in patients with vibration disease (VD) [16,17,20]. At the same time, slightly higher levels of periodontal morbidity have been registered in people exposed to occupational vibration [1,28,6,22,23]. The research of oxidative homeostasis [4,10,15,27] is the actual aspect in patients with concomitant pathology of the internal organs [5,9,14,18,21,26]. Such patients have revealed desadaptative disorders on the level of functional and morphological changes, based on those of nervous reception, microcirculatory disorders and changes in the activity of some enzymatic systems [2,7,9,12]. A number of researchers define the presence of systemic disorders in the periodontium of the patients, who are occupationally exposed to vibration, as “*vibroperiodontal syndrome*”, thereby emphasizing the pathogenetic dependence of periodontal changes in such patients [6,12]. Superoxide dismutase (SOD) and catalase (CAT) of the oral fluid (OF) can be regarded as indicators of the state of antioxidant protection and its reserve abilities. Moreover, in case of progression of catabolic processes (for example, in chronic diseases of the periodontium), these components of enzymatic provision of oxidative homeostasis are informative too [11].

The aim of the research consisted in study of catalase and superoxide dismutase levels in the oral fluid of the patients, who have an occupational contact with vibration, depending upon their dental status.

MATERIALS AND METHODS

The study of the dental status (DS) and content of SOD and CAT in OF was conducted in patients of three groups: the control group ($n_0=129$) contained the persons, who were exposed to occupational vibration and results of their complete health examination excluded the presence of VD; the second ($n_1=63$

u chorych w zależności od OHI-S wahała się od $5,2 \pm 0,2$ U/min do $10,1 \pm 0,3$ U/min, była znacząco ($p < 0,05$) niższa w przypadku OHI-S $\geq 1,7$ U, nie różniła się od stwierdzonej w grupie kontrolnej, a także była niższa u chorych w II stadium VD w porównaniu z chorymi w I stadium VD (odpowiednio $7,3 \pm 0,3$ U/min i $8,6 \pm 0,2$ U/min, gdy OHI-S mieścił się w zakresie $0,7 \pm 1,6$ U). Zawartość CAT w OF w zależności od wskaźnika VPSC u chorych z VD wahała się od $5,8 \pm 0,2$ U/min do $8,6 \pm 0,6$ U/min i była znacząco ($p < 0,05$) niższa u chorych z indeksem VPSC ≤ 40 sek. W ten sposób aktywność CAT w OF była znacząco obniżona ($p < 0,05$) (w $VPRC > 40$ s była równa $7,8 \pm 0,2$ U/min, a w $VPRC \leq 40$ s wynosiła $8,6 \pm 0,1$ U/min) w VD w I stadium z obniżonym VPRC.

Wnioski. Wykazano obecność trendu ($p > 0,05$) w kierunku zwiększenia aktywności SOD w I stadium VD w porównaniu z grupą kontrolną, podczas gdy w II stadium VD wykazywano znamienne ($p < 0,05$) zmniejszenie tejże aktywności. Jednocześnie wykazano, że niezadawalający stan higieny jamy ustnej sprzyja hamowaniu enzymatycznej ochrony błony przyzębnej u chorych z I stadium VD. Trend ($p > 0,05$) w kierunku zwiększenia aktywności SOD w I stadium VD w stosunku do grupy kontrolnej, natomiast w II stadium VD wykazano znamienne ($p < 0,05$) zmniejszenie tejże aktywności. Ocena przeprowadzona w przypadkach wymagających leczenia skojarzonego z chirurgicznym lub niechirurgicznym oczyszczeniem rany, a także u chorych z nad- lub podziąsłowym kamieniem nazębnym wykazała, że aktywność SOD była znacząco obniżona tylko w przypadkach VD w II stopniu zaawansowania. Ocena aktywności CAT w OF u chorych z VD o różnym poziomie CPITN wykazała, że powyższa aktywność u wymagających leczenia skojarzonego (w tym leczenia protetycznego; $CPITN \geq 3$, 1 pkt) była znacząca i znacząco obniżona. Wszystkie powyższe fakty determinują odmienność strategii leczenia doustnego tej grupy chorych.

Słowa kluczowe: stan uzębienia, błona przyzębia, choroba wibracyjna

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cases with VD stage I) and third ($n_2=66$ cases with VD stage II) groups included the patients, who were treated at the clinical department of the Research Institute of Occupational Hygiene and Occupational Diseases of Kharkiv National Medical University of the Ministry of Health of Ukraine [24].

DS was assessed according to K.M. Kosenko (Patent No. 57512, Ukraine) in in-patients and controls (during medical examinations), using: papillary-marginal-alveolar index (PMA), simplified oral hygiene index (OHI-S) and carious lesion intensity index (DMFT)] with assessment of the vacuum-pressory resistance of gingival capillaries (VPRC) (according to V.I. Kulazhenko) and community periodontal index of treatment needs (CPITN) [11]. The content of SOD was revealed by the nonenzymatic method [8], based on the ability of SOD to inhibit reduction of nitroblue tetrazolium. The content of CAT was determined spectrophotometrically with $\lambda=410$ nm; the principle of this method is based on the fact that CAT reacts with hydrogen peroxide, whose residual content was revealed in the reaction with ammonium molybdate.

Primary materials were statistically processed with determination of reliability according to paired t-test. Results of the research were statistically analyzed with help of variation statistics and assessment of normality of distribution and reliability of findings [3,13,19,25].

RESULTS AND DISCUSSION

The level of SOD content depending upon the expression of PMA in patients with VD ranged from 14.1 ± 0.2 U/min to 15.7 ± 0.5 U/min, was reliably ($p < 0.05$) lower in cases with VD versus the controls (respectively, 17.8 ± 0.2 U/min and 14.2 ± 0.2 U/min, when $PMA > 2.1$) and did not differ depending upon the severity of VD (respectively, 15.7 ± 0.5 U/min in VD stage I and 15.3 ± 0.3 U/min in VD stage II). The above facts support the diagnostic benefit of this index at early stages of VD manifestation. Even

in cases of minimum lesions of the periodontal membrane (PMA<1.1) under the presence of VD we registered a reliably ($p<0.05$) lower level of SOD content. It should be noted here that the control group revealed a decreased level of SOD content with an increasing PMA index, whereas cases with VD demonstrated a relative inhibition of SOD production, first of all in VD patients with PMA values exceeding 1.1 pts.

The level of SOD content depending upon the values of OHI-S ranged from 13.5 ± 0.3 U/min to 16.3 ± 0.2 U/min, was reliably ($p<0.05$) lower in cases with OHI-S values ≥ 1.7 U, did not differ from control values as well as proved to be reliably ($p<0.05$) lower in patients with VD stage II versus cases with VD stage I (respectively, 13.5 ± 0.3 U/min and 14.4 ± 0.2 U/min, when $OHI-S \geq 1.7$ U). The above facts support both an activation of the enzymatic chain of antioxidant protection in VD patients with low OHI-S values and, at the same time, an inhibition of the enzymatic activity of SOD in patients suffering from VD stage II with high OHI-S values (fig. 1). Thus, VD stage I revealed a trend ($p>0.05$) towards an increased SOD activity versus controls, whereas VD stage II demonstrated its reliable reduction ($p<0.05$). At the same time, an unsatisfactory state of the oral cavity hygiene contributed to inhibition of the enzymatic protection of their periodontium in patients with VD stage II.

The activity of the enzymatic protection of the periodontium, as it was revealed by results of our comparative analysis, also depended upon the state of hard tissues, particularly upon such an index of DS as DMFT. It was found out that in VD stage II the activity of SOD was reliably ($p<0.05$) reduced in patients with DMFT values larger than 15 pts (with $DMFT \leq 10$ pts the activity of SOD was 14.7 ± 0.5 U/min, whereas with $DMFT > 15$ pts it was 12.2 ± 0.4 U/min). The activity of SOD in OF patients with VD stage I was characterized by somewhat different regularity (fig. 1): with all values of their DMFT those patients revealed preservation of SOD activity in OF, and its reduction depended upon an increase of DMFT index. Thus, the presence of VD stage II caused a lower level of SOD activity in OF, an inhibition of this activity depending upon an increase of DMFT index; the above fact can be regarded as an additional indicator for choosing the tactics of prevention and treatment of periodontal pathology in this category of patients.

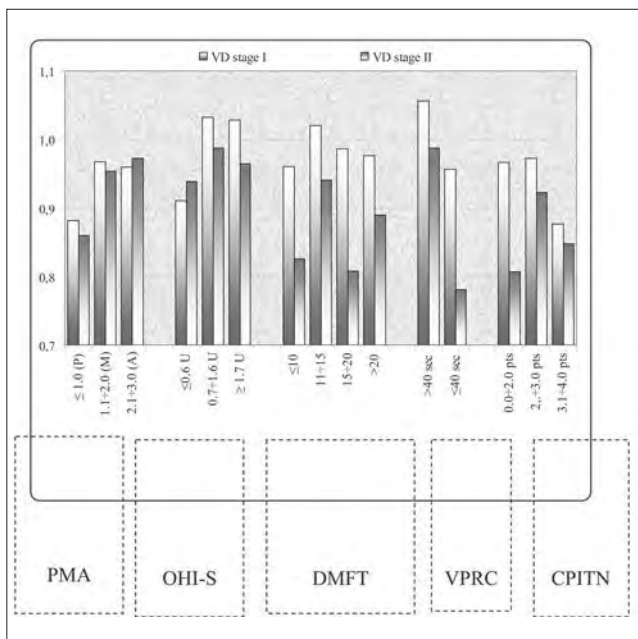


Figure 1. A relative level of superoxide dismutase (1.0 – the control group value) in the oral fluid of patients depending upon indices of their dental status and degree of severity of vibration disease

Rycina 1. Względne stężenie dysmutazy ponadtlenkowej (1,0 – wartość w grupie kontrolnej) w płynie z jamy ustnej chorych w zależności od wskaźników ich stanu uzębienia i stopnia nasilenia choroby wibracyjnej

The level of SOD content depending upon the value of VPRC in patients with VD ranged from 10.7 ± 0.5 U/min to 16.8 ± 0.3 U/min and was reliably ($p<0.05$) lower in patients with VPRC values ≤ 40 sec. For example, in VD stage I: if VPRC was decreased the SOD activity in the patients' OF was reliably ($p<0.05$) reduced (with $VPRC > 40$ sec it was equal to 16.8 ± 0.3 U/min, but with $VPRC \leq 40$ sec it was only 13.1 ± 0.5 U/min). A more expressed reduction of SOD activity was found out in OF in patients with VD stage II that manifested with both a reduced activity of SOD with normal values of VPRC and in case of a higher vacuum-pressory compliance of capillaries (respectively, 15.7 ± 0.3 U/min and 10.7 ± 0.3 U/min, $p<0.05$). The above facts are an additional support for the presence of pathogenetic relationships between the state of the periodontal microcirculatory bed and enzymatic activity of OF in patients with VD.

Our analysis of SOD activity in OF of patients having VD with different levels of their generalized CPITN revealed that in the patients, who needed for combined therapy (including prosthodontic treatment, $CPITN \geq 3.1$ pts), the level of SOD was reliably and markedly decreased (versus the respective groups of patients, but with low CPITN values) both in VD stage I and VD stage II (respectively, 12.1 ± 0.4 U/min and 11.7 ± 0.3 U/min). It should be noted that in the cases, who needed for combined therapy with surgical or nonsurgical debridement, as well as in the patients who revealed presence of supra- or subgingival calculus, their SOD activity was reliably lower in cases with VD stage II, this fact determining peculiarities in the oral treatment for this category of patients.

The level of CAT content in patients with VD ranged depending upon the severity of PMA from 4.6 ± 0.4 U/min to 11.3 ± 0.3 U/min, was reliably ($p<0.05$) higher in patients with VD stage I versus the controls (respectively, 8.8 ± 0.2 U/min and 7.5 ± 0.6 U/min, when $PMA = 1.1\pm 2.0$) and differed depending upon the severity of VD (respectively, 8.8 ± 0.2 U/min in VD stage I and 6.3 ± 0.5 U/min in VD stage II). It should be noted here that the control group and the groups of patients with different VD severity revealed a decreased CAT level with an increase of PMA index, therewith demonstrating a close relationship between CAT level and PMA values. The above facts support the diagnostic benefit of this index both at early stages of VD manifestation and in case of its progression.

The level of CAT content in OF of patients (fig. 2) depending upon OHI-S values ranged from 5.2 ± 0.2 U/min to 10.1 ± 0.3 U/min, was reliably ($p<0.05$) lower in patients with OHI-S values

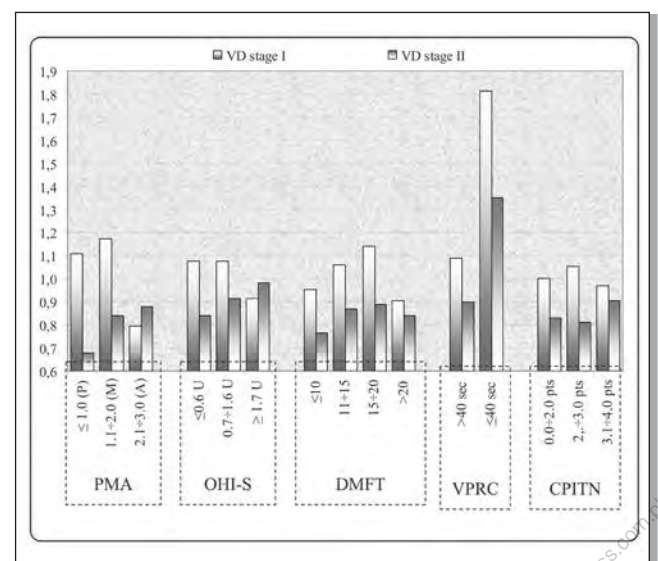


Figure 2. A relative level of catalase (1.0 – the control group value) in the oral fluid of patients depending upon indices of their dental status and degree of severity of vibration disease

Rycina 2. Względne stężenie katalazy (1,0 – wartość w grupie kontrolnej) w płynie z jamy ustnej chorych w zależności od wskaźników ich stanu uzębienia i stopnia nasilenia choroby wibracyjnej

≥ 1.7 U and did not differ from the control value as well as it proved to be reliably lower in patients with VD stage II versus those with VD stage I (respectively, 7.3 ± 0.3 U/min and 8.6 ± 0.2 U/min with OHI-S within 0.7 ± 1.6 U). The above facts reveal an increased CAT activity in OF of patients having VD with low OHI-S values and, at the same time, an inhibition of enzymatic activity of CAT in cases having VD stage I with high values of OHI-S (fig. 2).

Thus, VD stage I revealed a reliable increase in CAT activity versus the controls, VD stage II demonstrating a reliable reduction of the above activity ($p < 0.05$). At the same time, an unsatisfactory state of the oral cavity hygiene contributed to inhibition of CAT activity in OF and, respectively, enzymatic protection in patients with VD stage II. It was found out that in VD stage II the activity of CAT was reliably ($p < 0.05$) reduced (versus both the controls and cases with VD stage I) in all the patients irrespective of gradations of DMFT index. For example, with $DMFT > 20$: if CAT activity in the controls was equal to 6.2 ± 0.2 U/min, it was 5.6 ± 0.2 U/min in VD stage I and 5.2 ± 0.4 U/min in VD stage II. The activity of CAT in OF in patients with VD stage II was characterized by somewhat different regularity: CAT activity in OF in patients with $DMFT = 15 \pm 20$ was characterized by somewhat different regularity: in VD stage I the activity of CAT was slightly higher (8.8 ± 0.6 U/min) versus the controls (8.3 ± 0.2 U/min), whereas in VD stage II it was reliably ($p < 0.95$) lower (7.2 ± 0.3 U/min). Hence, the presence of the revealed stage of VD and level of DMFT is a significant factor for formation of CAT level in OF patients with VD.

The level of CAT content in OF depending upon the value of VPRC in patients with VD ranged from 5.8 ± 0.2 U/min to 8.6 ± 0.6 U/min and was reliably ($p < 0.05$) lower in cases with VPRC values ≤ 40 sec. For example, in VD stage I: if VPRC was decreased the CAT activity in the patients' OF was reliably ($p < 0.05$) reduced (with $VPRC > 40$ sec it was equal to 7.8 ± 0.2 U/min, but with $VPRC \leq 40$ sec it was 8.6 ± 0.1 U/min). A more expressed reduction of CAT activity was found out in OF of patients with VD stage II that manifested with both a reduced activity of CAT with normal values of VPRC and in case of a higher vacuum-pressory compliance of capillaries (respectively, 7.1 ± 0.1 U/min and 5.8 ± 0.2 U/min, $p < 0.05$). The above facts are an additional support for the presence of pathogenetic relationships between the state of the periodontal microcirculatory bed and the enzymatic activity of OF in patients with VD.

Our analysis of CAT activity in OF of cases having VD with different levels of their generalized CPITN revealed that in the patients, who needed for combined therapy (including prosthodontic treatment, $CPITN \geq 3.1$ pts), the level of CAT was markedly and reliably decreased (versus the respective groups of patients), first of all in the patients with VD stage II (down to 5.6 ± 0.4 U/min). It should be noted that in the cases, who needed for combined therapy with surgical or nonsurgical debridement, as well as in the patients who revealed presence of supra- or subgingival calculus, their CAT activity was reliably reduced only in cases with VD stage II, this fact determining peculiarities in the oral treatment for this category of patients.

CONCLUSIONS

Patients with VD stage I revealed a trend ($p > 0.05$) towards an increase of SOD activity versus the controls, whereas cases with VD stage II demonstrated a reliable reduction ($p < 0.05$) of the above activity. At the same time, an unsatisfactory state of the oral cavity hygiene contributed to inhibition of the enzymatic protection of their periodontium in patients with VD stage II. The groups of patients with different VD severity were characterized by a decrease of CAT level with an increase of PMA index, this fact supporting a direct relationship between the level of CAT and PMA values.

VD stage I revealed a trend ($p > 0.05$) towards an increase of SOD activity versus the controls, whereas VD stage II demonstrated a reliable reduction ($p < 0.05$) of the above activity. At

the same time, an unsatisfactory state of the oral cavity hygiene contributed to inhibition of the enzymatic protection of their periodontium in patients with VD stage II. An increase in the activity of CAT in OF in patients having VD with low OHI-S values was found out.

In the cases that needed for combined therapy with surgical or nonsurgical debridement as well as in the patients who revealed presence of supra- or subgingival calculus their SOD activity was reliably reduced only in cases with VD stage II. Our analysis of CAT activity in OF of cases having VD with different levels of their generalized CPITN revealed that in the patients, who needed for combined therapy (including prosthodontic treatment, $CPITN \geq 3.1$ pts), the level of CAT was markedly and reliably decreased (versus the respective groups of patients), first of all in the patients with VD stage II (down to 5.6 ± 0.4 U/min). The above facts determine peculiarities in the tactics of oral treatment for this category of patients.

Prospects of further researches are associated with study of correlation relationships between immunometabolic indices of oxidative homeostasis and its changes under the influence of treatment.

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