https://doi.org/10.15360/1813-9779-2022-3-38-44



# Parameters of the Blood Oxidant/Antioxidant System in Elderly Patients with Acute Poisoning

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# Оценка оксидантно-антиоксидантной системы крови у гериатрических пациентов с острыми отравлениями

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**For citation:** *Anastasia Yu. Simonova, Kapitolina K. Ilyashenko, E.V. Klychnikova, Anatoly K. Evseev, Mikhail M. Potskhveriya, Maria V. Belova, Elizaveta V. Tazina, Aslan K. Shabanov, Artem N. Kuzovlev.* Parameters of the Blood Oxidant/Antioxidant System in Elderly Patients with Acute Poisoning. *Obshchaya Reanimatologiya = General Reanimatology.* 2022; 18 (3): 38–44. https://doi.org/10.15360/1813-9779-2022-3-38-44 [In Russ. and Engl.]

## Summary

**The aim of the study** was to assess the oxidant/antioxidant status in elderly patients in the early period of acute poisoning by psychotropic drugs or corrosive substances.

**Material and methods.** An open prospective observational study with retrospective control was conducted in 80 patients (age ⩾60 years) with acute poisoning, of which 49 patients aged 72.1±9.55 years had psychotropic drug poisoning (PDP) and 31 subjects aged 73.0±10.3 years had corrosive substance poisoning (CSP). Patients with mild poisoning were excluded from the study. The control group consisted of 39 volunteers aged 68.3±6.3 years. Total antioxidant status (TAS), blood levels of malondialdehyde (MDA), stable nitric oxide metabolites (nitrite/nitrate, NOx), and oxidative stress index (MDA/TAS) were measured on days 1, 3 and 5 after hospital admission.

**Results.** When analyzing the changes in the parameters of the oxidant/antioxidant system, we observed lower values of the studied parameters in patients with both PDP and CSP compared to the control group. In patients with PDP, several parameters were reduced: MDA by 1.2 times on days 1 and 3 ($P$=0.002; $P$=0.008, respectively), NOx by 1.7 times ($P$<0.001) at all stages of the study, MDA/TAS by 2.4–2.9 times ($P$<0.001). In patients with CSP, MDA level decreased by 1.1–1.2 times at all study timepoints ($P$=0.003; $P$=0.010; $P$=0.046, respectively), NOx dropped 1.4–1.6-fold ($P$=0.012; $P$=0.004; $P$=0.023, respectively), and MDA/TAS decreased by 2.3–2.4 times ($P$<0.001). While comparing patients with favorable and fatal outcome, we found that in survived patients an increase of MDA/TAS along with growing NOx level was seen by day 5 with no significant changes of MDA and TAS, while in non-survivors MDA/TAS dropped continuously due to progressive fall of NOx level, reaching values 2.8–2.9 times ($P$<0.001) lower than those of the controls.

**Conclusion.** In elderly patients with acute poisonings due to psychotropic drugs or corrosive substances, an inadequate response of the oxidant/antioxidant system occurs manifesting as a reduced blood level of peroxidation products with simultaneous normal or slightly decreased concentration of antioxidant protection system components. Thus, the oxidative stress develops, which contributes to the death of the patients.

***Keywords: acute poisoning; oxidative stress; elderly patients; lipid peroxidation; antioxidant activity***

**Conflict of interest.** The authors declare no conflict of interest.

The full text version of the paper is available at www.reanimatology.com.

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## Introduction

Acute chemical poisonings are among current medical challenges impacting the demographic situation in Russia, as they are associated with considerable medical and socio-economic costs [1, 2]. The pathogenesis of acute chemical poisonings involves disorders of various body functions and systems, including peroxide homeostasis [3, 4].

Currently, researchers focus their attention on the concept of «oxidative stress» as a key pathogenetic factor in the development of various chronic and acute diseases [5–8]. It leads to disruption of cellular structures, their functional activity change and ultimately to their death [3, 7, 8].

Peroxidation processes were shown to play a critical role in the pathogenesis of acute chemical injury [3, 4, 9]. The trigger mechanism is the entry of a toxicant into the body with subsequent oxidative stress maintained by endotoxemia [10]. Currently, there are sufficient data on the role of peroxide homeostasis disorders in middle-aged patients with acute toxicosis.

Only a few studies (especially comparative ones with different age groups) on acute chemical poisoning in geriatric patients have been available. The elderly and senile patients, in contrast to the middle-aged ones, are characterized by higher levels of malondialdehyde (4.2 (3.74–4.59) vs. 2.27 (2.11–2.47) μmol/l) and lower values of total antioxidant status (1.5 (1.28–1.59) vs. 1.61 (1.56–1.68) μmol/l) [9]. In poisoning with corrosive substances, the lack of adequate response to acute chemical injury in elderly and senile patients was also shown, which manifested by primary activation of venous blood lymphocyte apoptosis, and only then by peroxidation processes [9]. The structure and functional capabilities of the body organs and systems decrease with age [11].

The aim of this study was to examine the oxidant/antioxidant status in geriatric patients in the early phase of acute poisoning by psychopharmacological drugs and corrosive substances.

## Material and Methods

An open prospective observational study with retrospective control was conducted in the department of acute intoxications and somatopsychiatric disorders of the N.V. Sklifosovsky Research Institute of Emergency Medicine during 2015–2020 after obtaining approval of the biomedical ethics committee. The inclusion criteria were moderate to severe psychopharmacological drug poisoning (PDP) according to the classification of E. Luzhnikov widely accepted in Russia [1], moderate to severe corrosive substance poisoning (CSP) according to the classification of S. Volkov et al. modified by Pinchuk [12, 13], and age 60 years and older. All patients with CSP underwent esophagogastroduodenoscopy to determine the severity and extent of chemical burns of upper gastrointestinal tract (GIT). Patients with mild PDP and CSP were not included in the study.

Eighty patients with acute poisoning were enrolled and divided into 2 groups including 49 patients aged 72.1±9.55 years with PDP and 31 patients aged 73.0±10.3 years with CSP. Each of these groups was divided into subgroups with favorable and lethal outcomes. The parameters of oxidant/antioxidant system were also studied in 39 volunteers aged 60 to 85 years (mean age 68.3±6.3 years) (control group). Of the 119 patients included in the study, 82 (68.9%) were women and 37 (31.1%) were men. Women (86%) were predominant among patients with PDP, and men (71%) were predominant among patients with CSP. This was probably due to the fact that men more commonly experience accidental poisonings with corrosive substances when intoxicated with alcohol.

Patients with PDP received forced diuresis, intestinal lavage, fluid and symptomatic therapy. Patients with CSP were treated with fluid, analgesic, antispasmodic, hormonal and local therapy.

The primary study endpoints were level of malondialdehyde (MDA) as a product of lipid peroxidation (LPO), total blood antioxidant status (TAS) to assess the antioxidant protection status, the blood levels of stable nitric oxide metabolites (NOx), and oxidative stress index (MDA/TAS ratio). The secondary endpoint was mortality. The serum MDA level was measured according to method by Gavrilov [14], TAS was determined using spectrophotometry on Olympus AU2700 biochemical analyzer (Beckman Coulter, USA) with the TAS kit (Randox, UK). Blood NOx was measured according to P. Golikov and N. Nikolayeva method [15]. MDA/TAS for each patient was calculated as the ratio of serum MDA to the serum TAS. The studied parameters were recorded at an early phase of acute poisoning, on days 1, 3, and 5 after hospital admission.

Statistical analysis was performed using the IBM SPSS Statistics 27.0 software. The data distribution was assessed using the Shapiro–Wilk test (for $n \leqslant 50$). In normal distribution, the arithmetic mean (M) and standard deviation (*SD*) were calculated. For nonparametric data, median (*Me*) and 25$^{th}$ and 75$^{th}$ percentiles were reported (as *Me* (*Q25–Q75*)). Intergroup comparisons of quantitative data were performed using Student's *t*-test (at normal distribution of variables) or Mann–Whitney test and Wilcoxon test with Bonferroni correction (related groups) (if distribution of variables was not normal). Relative variables were compared using Fisher's exact test. Spearman's correlation coefficient (rho) was calculated to assess the strength of relation between various parameters. Differences were considered significant at $P<0.05$.

**Table 1. Changes in oxidant/antioxidant markers in geriatric patients with acute poisoning with psychotropic drugs.**

| Parameter | Values at study stages | | | |
|---|---|---|---|---|
| | Control | Day 1 | Day 3 | Day 5 |
| MDA, μmol/l | 4.2 (3.74–4.59) | 3.5 (2.9–4.22)<br>[1]*p*=0.002* | 3.52 (3.0–4.3)<br>[1]*p*=0.008*<br>[2]*p*=0.584 | 4.0 (3.12–4.8)<br>[1]*p*=0.356<br>[2]*p*=0.007*<br>[3]*p*=0.169 |
| TAS, mmol/l | 1.5 (1.28–1.59) | 1.47 (1.27–1.91)<br>[1]*p*=0.347 | 1.46 (1.24–1.7)<br>[1]*p*=0.256<br>[2]*p*=0.437 | 1.57 (1.29–1.83)<br>[1]*p*=0.237<br>[2]*p*=0.167<br>[3]p=0.182 |
| MDA/TAS, c.u. | 2.26 (1.86–2.76) | 0.78 (0.67–1.02)<br>[1]*p*<0.001* | 0.86 (0.73–1.06)<br>[1]*p*<0.001*<br>[2]*p*=0.004* | 0.93 (0.71–1.33)<br>[1]*p*<0.001*<br>[2]*p*=0.118<br>[3]*p*=0.520 |
| NOx, μmol/l | 27.2 (18.9–31.4) | 15.8 (12.4–21.9)<br>[1]*p*<0.001* | 15.6 (8.12–23.5)<br>[1]*p*<0.001*<br>[2]*p*=0.846 | 15.4 (10.5–26.5)<br>[1]*p*<0.001*<br>[2]*p*=0.745<br>[3]*p*=0.634 |

**Note.** C.u. — conditional unit. [1] — differences between variables vs the control values (*P*<0.05) (Mann–Whitney test); [2] — versus Day 1 (*P*<0.017) (Wilcoxon test with Bonferroni correction); [3] — versus Day 3 (*P*<0,017) (Wilcoxon test with Bonferroni correction). * — significant differences. The data are given as *Me* (*Q25–Q75*).

**Table 2. Changes in the oxidant-antioxidant markers in geriatric patients with acute poisoning with corrosive substances.**

| Parameter | Values at study stages | | | |
|---|---|---|---|---|
| | Control | Day 1 | Day 3 | Day 5 |
| MDA, μmol/l | 4.2 (3.74–4.59) | 3.48 (3.34–4.05)<br>[1]*p*=0.003* | 3.81 (3.34–4.07)<br>[1]*p*=0.010*<br>[2]*p*=0.312 | 3.92 (3.16–4.28)<br>[1]*p*=0.046*<br>[2]*p*=0.431<br>[3]*p*=0.644 |
| TAS, mmol/l | 1.5 (1.28–1.59) | 1.35 (1.19–1.65)<br>[1]*p*=0.103 | 1.38 (1.2–1.57)<br>[1]*p*=0.132<br>[2]*p*=0.312 | 1.19 (1.01–1.33)<br>[1]*p*<0.001*<br>[2]*p*<0.001*<br>[3]*p*=0.018 |
| MDA/TAS, c.u. | 2.26 (1.86–2.76) | 0.92 (0.78–1.18)<br>[1]*p*<0.001* | 0.99 (0.76–1.2)<br>[1]*p*<0.001*<br>[2]*p*=0.645 | 1.19 (0.94–1.4)<br>[1]*p*<0.001*<br>[2]*p*<0.001*<br>[3]*p*=0.265 |
| NOx, μmol/l | 27.2 (18.9–31.4) | 18.0 (11.2–23.4)<br>[1]*p*=0.012* | 16.7 (13.8–26.6)<br>[1]*p*=0.004*<br>[2]*p*=0.925 | 19.8 (13.3–24.1)<br>[1]*p*=0.023*<br>[2]*p*=0.728<br>[3]*p*=0.225 |

**Note.** C.u. — conditional unit. [1] — differences between variables vs the control values (*P*<0.05) (Mann–Whitney test); [2] — versus Day 1 (*P*<0.017) (Wilcoxon test with Bonferroni correction); [3] — versus Day 3 (*P*<0,017) (Wilcoxon test with Bonferroni correction). * — significant differences. The data are given as *Me* (*Q25–Q75*).

## Results

Serum MDA concentrations in patients with PDP on both days 1 and 3 of hospital stay were significantly lower than the control values (by 16%). On day 5, they were close to the control values (Table 1).

No significant differences in TAS level versus the control group were found at all stages of the study. However, MDA/TAS ratio in this category of patients was 2.4–2.9 times (*P*<0.001) lower than the age-specific reference values at all stages of the study. Blood NOx level during the whole period of observation was 1.7 times lower than in the control group (*P*<0.001).

Table 2 presents similar changes of lipid peroxidation and antioxidant protection parameters in geriatric patients with CSP.

A significant decrease of MDA/TAS ratio during the whole study was observed. In the first two stages, the ratios were 2.4 and 2.3 times lower, respectively, versus the control group (*P*<0.001); on day 5, it increased up to 1.19 (0.19–1.4) c.u., which was 1.9 times below the reference value (*P*<0.001). The indicated changes in the oxidative stress coefficient by day 5 were due to a slight increase in lipid peroxidation and a significant decrease in the antioxidant plasma activity. There was a significant difference in the serum level of nitric oxide metabolites compared to the controls throughout the study (1.5, 1.6 and 1.4 times higher).

The data presented in Table 3 show that with a favorable outcome of the disease, there were no differences vs the baseline values in the blood levels

**Table 3. The oxidant/antioxidant system parameters in favorable and lethal outcomes of acute poisonings with psychopharmacological drugs in geriatric patients.**

| Parameter | Control values (*n*=39) | Outcome | | | | | |
|---|---|---|---|---|---|---|---|
| | | Favorable (*n*=33) | | | Lethal (*n*=16) | | |
| | | Day 1 | Day 3 | Day 5 | Day 1 | Day 3 | Day 5 |
| MDA, μmol/l | 4.2 (3.74–4.59) | 3.73 (2.95– 4.42) [1]*p*=0.028* | 3.61 (2.92–4.31) [1]*p*=0.015* [3]*p*=0.276 | 3.69 (3.27–4.93) [1]*p*=0.026* [3]*p*=0.435 [4]*p*=0.835 | 3.47 (3.18–4.05) [1]*p*=0.002* [2]*p*=0.198 | 3.85 (3.33–4.24) [1]*p*=0.165 [2]*p*=0.276 [3]*p*=0.287 | 4.3 (3.7–4.43) [1]*p*=0.527 [2]*p*=0.165 [3]*p*=0.126 [4]*p*=0.476 |
| TAS, mmol/l | 1.5 (1.28–1.59) | 1.6 (1.33–1.92) [1]*p*=0.645 | 1.6 (1.29–1.77) [1]*p*=0.745 [2]*p*=0.894 | 1.5 (1.26–1.78) [1]*p*=0.832 [3]*p*=0.745 [4]*p*=0.834 | 1.4 (1.27–1.88) [1]*p*=0.672 [2]*p*=0.378 | 1.46 (1.29–1.77) [1]*p*=0.728 [2]*p*=0.498 [3]*p*=0.827 | 1.67 (1.47–2.0) [1]*p*=0.698 [2]*p*=0.892 [3]*p*=0.038 [4]*p*=0.049 |
| MDA/TAS, c.u. | 2.26 (1.86–2.76) | 0.77 (0.64–1.05) [1]*p*<0.001* | 0.81 (0.71–0.98) [1]*p*<0.001* [3]*p*=0.598 | 0.93 (0.71–1.66) [1]*p*<0.001* [3]*p*=0.049 [4]*p*=0.167 | 0.79 (0.68–2.76) [1]*p*<0.001* [2]*p*=0.823 | 0.89 (0.76–1.03) [1]*p*<0.001* [2]*p*=0.623 [3]*p*=0.276 | 0.79 (0.65–0.96) [1]*p*<0.001* [2]*p*=0.287 [3]*p*=0.923 [4]*p*=0.027 |
| NOx, μmol/l | 27.2 (18.9–31.4) | 15.7 (12.3–23.1) [1]*p*<0.001* | 18.8 (11.8–24.4) [1]*p*<0.001* [3]*p*=0.267 | 20.2 (13.1–28.3) [1]*p*=0.004* [3]*p*=0.105 [4]*p*=0.328 | 15.8 (12.8–19.3) [1]*p*<0.001* [2]*p*=0.834 | 11.7 (4.62–20.6) [1]*p*<0.001* [2]*p*=0.046* [3]*p*=0.083 | 11.0 (8.02–23.0) [1]*p*<0.001* [2]*p*=0.034* [3]*p*=0.113 [4]*p*=0.623 |

**Note.** [1] - differences in parameters compared with the control values ($P<0.05$) (Mann–Whitney test); [2] — differences between groups (favorable and lethal outcome) ($P<0.05$) (Mann–Whitney test); [3] — versus Day 1 ($P<0.017$) (Wilcoxon test with Bonferroni correction); [4] — versus Day 3 ($P<0.017$) (Wilcoxon test with Bonferroni correction); * — differences are significant. The data are presented as *Me* (*Q25–Q75*).

of MDA and TAS on days 3 and 5. Meanwhile, a 1.3-fold increase in NOx level vs the initial value was seen by day 5.

The oxidative stress coefficient increased 1.2-fold from the baseline values by day 5 ($P=0.049$). In patients who died later, there was a trend toward increasing the MDA and TAS by day 5, whereas NOx level dropped 2.5 times lower than the control values ($P<0.001$). Meanwhile, the oxidative stress coefficient at all stages of the study was 2.9, 2.5 and 2.9 times lower versus control values ($P<0.001$).

In subjects with CSP and a favorable outcome, an increase in the initially low blood level of MDA and NOx and the corresponding «consumption» of TAS were observed by day 5 (Table 4).

A trend was detected toward an increase in the oxidative stress index: on Day 5 it was 1.12 times higher than the baseline values. In non-survivors we observed a decrease of oxidative stress index: by Day 5 it was 65% below the reference value ($P<0.001$) and 1.54 times lower than the baseline value ($P=0.063$). This was accompanied by a simultaneous decrease in blood MDA, NOx, and TAS levels at this stage of study.

In patients with PDP, a strong correlation between MDA and NOx was revealed at all stages of the study (on Day 1, $r=0.75$, $P<0.001$; on Day 3, $r=0.78$, $P<0.001$; on Day 5, $r=0.84$, $P<0.001$) and inverse intermediate and strong correlation between TAN and NOx (on Day 1, $r=-0.67$, $P<0.001$; on Day 3, $r=-0.74$, $P<0.001$; on Day 5, $r=-0.78$, $P<0.001$).

In patients with CSP, on Day 1 there was an intermediate strength correlation between NOx and TAN ($r=0.63$, $P<0.001$). At the second stage of the study, the correlation between these parameters was not significant ($r=0.23$, $P=0.025$). On Day 5, a significant intermediate strength correlation was found ($r=0.51$, $P=0.018$).

## Discussion

Currently, lipid peroxidation and disorders of antioxidant protection are considered essential in the progression and outcome of various diseases, including acute poisoning [3, 4, 9]. The reactive oxygen species and free radical reactions under stress are known to perform a regulatory function and, with their adequate production, increase the body's resistance. However, excessive accumulation of peroxide products causes imbalance in the lipid peroxidation and antioxidant protection system, which contributes to the disruption of cellular structures and changes in their functional activity [7–9, 16–23].

The production of NO is an important link in the pathophysiology of oxidative stress. This active form of oxygen, which rapidly interacts with superoxide anion radical, forms the strongest oxidizing agent, peroxynitrite, which is involved in the initiation of oxidative stress. NO is also a powerful endogenous

**Table 4. Comparative assessment of the oxidant/antioxidant system parameters in geriatric patients with acute poisoning with corrosive substances depending on the outcome.**

| Parameter | Control values (*n*=39) | Outcome | | | | | |
|---|---|---|---|---|---|---|---|
| | | Favorable (*n*=22) | | | Lethal (*n*=9) | | |
| | | Day 1 | Day 3 | Day 5 | Day 1 | Day 3 | Day 5 |
| MDA, μmol/l | 4.2 (3.74–4.59) | 3.61 (3.39– 4.26) [1]*p*=0.019* | 3.84 (3.3–4.18) [1]*p*=0.218 [3]*p*=0.187 | 4.0 (3.43–4.31) [1]*p*=0.329 [3]*p*=0.176 [4]*p*=0.285 | 3.35 (3.23–3.57) [1]*p*=0.006* [2]*p*=0.327 | 3.56 (3.35–3.78) [1]*p*=0.009* [2]*p*=0.219 [3]*p*=0.295 | 3.16 (3.1–4.28) [1]*p*=0.003* [2]*p*=0.094 [3]*p*=0.385 [4]*p*=0.178 |
| TAS, mmol/l | 1.5 (1.28–1.59) | 1.36 (1.19–1.65) [1]*p*=0.179 | 1.37 (1.24–1.42) [1]*p*=0.139 [3]*p*=0.829 | 1.23 (0.99–1.4) [1]*p*=0.003* [3]*p*=0.003* [4]*p*=0.041 | 1.45 (1.29–2.43) [1]*p*=0.692 [2]*p*=0.132 | 1.38 (1.21–1.61) [1]*p*=0.193 [2]*p*=0.729 [3]*p*=0.149 | 1.13 (1.09–1.19) [1]*p*=0.004* [2]*p*=0.259 [3]*p*=0.167 [4]*p*=0.394 |
| MDA/TAS, c.u. | 2.26 (1.86–2.76) | 0.96 (0.78–1.33) [1]*p*<0.001* | 1.07 (0.85–1.33) [1]*p*<0.001* [3]*p*=0.828 | 1.08 (0.71–1.66) [1]*p*<0.001* [3]*p*=0.729 [4]*p*=0.839 | 1.22 (1.02–1.48) [1]*p*<0.001* [2]*p*=0.328 | 0.89 (0.76–1.03) [1]*p*<0.001* [2]*p*=0.428 [3]*p*=0.332 | 0.79 (0.65–0.96) [1]*p*<0.001* [2]*p*=0.182 [3]*p*=0.063 [4]*p*=0.628 |
| NOx, μmol/l | 27.2 (18.9–31.4) | 17.5 (11.8–31.7) [1]*p*=0.008* | 16.7 (13.9–26.1) [1]*p*=0.004* [3]*p*=0.628 | 20.4 (15.7–26.0) [1]*p*=0.078* [3]*p*=0.259 [4]*p*=0.217 | 22.2 (18.2–23.2) [1]*p*=0.176* [2]*p*=0.329 | 19.6 (14.2–26.6) [1]*p*=0.093* [2]*p*=0.294 [3]*p*=0.318 | 17.1 (13.3–19.5) [1]*p*=0.021* [2]*p*=0.145 [3]*p*=0.192 [4]*p*=0.584 |

**Note.** [1] - differences in parameters compared with the control values ($P<0.05$) (Mann–Whitney test); [2] — differences between groups (favorable and lethal outcome) ($P<0.05$) (Mann–Whitney test); [3] — versus Day 1 ($P<0.017$) (Wilcoxon test with Bonferroni correction); [4] — versus Day 3 ($P<0.017$) (Wilcoxon test with Bonferroni correction); * — differences are significant. The data are presented as *Me* (*Q25–Q75*).

vasodilator which alters tissue perfusion, decreases adhesion of leukocytes and platelets to the vascular endothelium, platelet aggregation, thus preventing the critical progression of the inflammatory response [10, 24–27].

The level of stable NO metabolites (NOx) has been found to increase due to elevated inducible NO-synthase (NOS) activity in systemic inflammatory response, sepsis, thoracic and abdominal trauma, as well as in rheumatoid arthritis, systemic lupus erythematous, etc.

In contrast, several studies have reported a decrease in blood NOx levels. Thus, in patients with preeclampsia, the blood NOx concentration is significantly lower than in women with normotensive pregnancy. The authors attributed this to the inhibition of endothelial constitutive NO synthase (NOS) [26, 28, 29]. In patients with myocardial infarction, a low blood level of NOx on day 1 was considered as a marker of severe disease and unfavorable outcome [24]. Thus, the contradictory data on NOx levels cannot be always definitively explained.

A decreased blood level of NOx compared with the control values was found in the patients with PDP and CSP at all stages of the study. However, a strong correlation between MDA and NOx was detected at all stages only in those with PDP. Thus, NO deficiency could be associated with inhibition of NOS activity. Previously, a significant increase in NO generation due to NOS activation was found in working-age patients with clozapine poisoning. In contrast, methanol poisoning was associated with a decrease in NO production by leukocytes (by 2 times) and platelets (by 6.4 times) compared to controls, as well as with a 16.5-fold decrease in blood nitrite level [24]. Currently there is no consensus on the interaction of the above-mentioned markers in various conditions and their role in the disease development.

In this study, abnormal parameters were assessed in relation to the control values. In elderly and senile patients with PDP and CSP, there was an inadequate response from the lipid peroxidation and antioxidant system manifested as a low peroxide potential due to a reduced level of NO and MDA at all stages of the study with low or normal values of TAS. The oxidative stress index during the study period was low. In our opinion, this indicates the development of oxidative stress, which contributes to a more severe disease in this age group compared with younger adults. Also, this could be due to the general low adaptive body potential, which we have previously found in geriatric patients with PDP [30].

A comparative assessment of peroxide homeostasis parameters in patients with various outcomes of PDP showed that in the survivors the level of

MDA and TAS during the study period did not differ from the initial values. At the same time, an increase in NOx level was detected by day 5. This situation resulted in an increase in the oxidative stress index, whose baseline value was 2.9 times lower than normal by day 5. In non-survivors, an increase in MDA and TAS by this stage of the study with low NOx values were found, which probably could not provide an increase in the initially low MDA/TAS ratio. This could suggest impaired self-regulation of the peroxide homeostasis system and worsened oxidative stress.

In patients who survived in CSP, an increase in the oxidative potential and MDA/TAS ratio was observed indicating physiological functioning of the lipid peroxidation and antioxidant system. In those who did not survive oxidative stress was obvious, as in patients with PDP. This was evidenced by a decrease in blood MDA, TAS, and NOx by day 5 associated with a drastic reduction of oxidative stress index.

Thus, in geriatric patients with acute poisoning by psychopharmacological drugs and corrosive substances, an inadequate response of the oxidant-antioxidant system is observed, which is manifested by reduced blood level of peroxidation products with normal or slightly reduced level of antioxidant protection components. Oxidative stress develops, and its progression results in death.

The limitation of this study consists in a small size of patient sample. Further research with a larger number of patients is required.

## Conclusion

In elderly patients with acute poisoning by psychopharmacological drugs and corrosive substances, lipid peroxydation and antioxidant system occur manifesting as reduced blood levels of MDA and NOx with reduced or subnormal antioxidant protection parameters.

Reduction of oxidative stress index values in poisoning with psychopharmacological drugs and corrosive substances by 2.4–2.9 times and 1.9–2.4 times, respectively, at all stages of the study demonstrates the development of strong poison-induced oxidative stress.

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**Received 2021.12.02**
**Accepted 2022.03.24**