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ABSTRACT

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BARIATRIC SURGERY IMPACT UPON OXIDATIVE STRESS MARKERS

Obesity is characterized by excessive fat accumulation, disruption of redox homeostasis, and metabolic changes that are often associated with self-catalyzed lipid peroxidation, which generates 4-hydroxynonenal, a pluripotent bioactive product of polyunsaturated fat peroxidation, and acids. Bariatric surgery leads to restoration of redox balance and improvement of body metabolism.

Aim. The primary objective was to assess the impact of laparoscopic sleeve gastrectomy, laparoscopic gastropliction, bariatric artery embolization on changes in redox balance in patients with metabolic syndrome.

Materials and Methods. The work is based on the materials of clinical examination and surgical treatment of 44 patients with metabolic syndrome (MS): 17 men and 27 women aged 47 ± 6.75 years with a body mass index > 40 kg/m^2. The control group included 12 people without manifestations of metabolic syndrome**.** Plasma levels of MDA, ox-LDL, TAA, catalase, total content of nitrates and nitrites were measured before and after (in 1, 3, 6 months) bariatric surgery (laparoscopic sleeve gastrectomy, laparoscopic gastropliction, bariatric artery embolization).

Results. We have established a tendency towards intensification of lipoperoxidation processes and reduction of protective resources of antioxidant protection. The indicators of the content of TBC-active products in the serum of MS patients were significantly higher ($p < 0.001$). The indicators of TAA activity were significantly lower (by 1.6 times) in patients with MS compared to controls ($p < 0.001$). However, catalase activity increased in our study. Thus, we found a 2.3-fold increase in catalase activity in MS patients compared to controls. The level of TBC-active products (MDA) increased significantly in patients with metabolic syndrome – by 1.64 times. After the bariatric interventions, we recorded a significant decrease

in the content of MDA – the main marker that characterizes the activation of LPO, but this indicator did not reach the level of the control group after all three types of surgical intervention (LSG, LGP, BAE).

Conclusions. Bariatric surgery reduces the intensity of lipid peroxidation, nitrooxidative stress, which prevents underoxidized decay products from entering the bloodstream. However, the state and activity of the blood antioxidant system does not always objectively reflect changes in organs and systems.

Keywords: metabolic syndrome, bariatric surgery, oxidative stress, lipid peroxidation, malondialdehyde.

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РЕЗЮМЕ

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ВПЛИВ БАРІАТРИЧНОЇ ХІРУРГІЇ НА МАРКЕРИ ОКСИДАТИВНОГО СТРЕСУ

Ожиріння характеризується надмірним накопиченням жиру, порушенням окислювально-відновного гомеостазу та метаболічними змінами, які часто пов'язані з самокаталізованим перекисним окисленням ліпідів, що генерує 4-гідроксиноненальні, плюрипотентні біологічно активні продукти перекисного окиснення поліненасичених жирів, кислоти. Баріатрична хірургія призводить до відновлення окисно-відновного балансу та покращення метаболізму всього організму.

Мета. Основною метою було оцінити вплив лапароскопічної рукавної гастректомії, лапароскопічної гастроплікації, баріатричної емболізації лівої шлункової артерії на зміни окисно-відновного балансу у пацієнтів з метаболічним синдромом.

Матеріали і методи. В основу роботи покладено матеріали клінічного обстеження та хірургічного лікування 44 пацієнтів з метаболічним синдромом (МС): 17 чоловіків та 27 жінок віком 47±6,75 років з індексом маси тіла > 40 кг/м2. Контрольну групу склали 12 осіб без проявів метаболічного синдрому. Плазмові рівні МДА, ОХ-ЛПНЩ, ТБК-активних продуктів, каталази, загальний вміст нітратів і нітритів визначали до і після (через 1, 3, 6 місяців) баріатричних операцій (лапароскопічна рукавна гастректомія, рукавна резекція шлунка.

Результати та їх обговорення. Встановлено тенденцію до інтенсифікації процесів ліпопероксидації та зниження захисних ресурсів антиоксидантного захисту. Встановлено статистично достовірно вищі показники вмісту ТБК-активних продуктів у сироватці крові хворих на РС ($p<0,001$). У хворих на РС зафіксовано статистично достовірно нижчі показники (в 1,6 раза) активності ТБК-активних продуктів порівняно з контролем ($p < 0,001$). Проте активність каталази в нашому дослідженні зростала. Так, ми виявили підвищення активності каталази в 2,3 рази у хворих на РС порівняно з контролем.

Рівень ТБК-активних продуктів (МДА) статистично достовірно підвищувався у пацієнтів з метаболічним синдромом – в 1,64 рази. Після баріатричних втручань ми зафіксували достовірне зниження вмісту МДА – основного маркера, що характеризує активацію ПОЛ, але цей показник не досягав рівня контрольної групи пацієнтів після всіх трьох видів оперативного втручання (ЛШГ, ЛГП, БАЕ).

Висновки. Баріатричні операції знижують інтенсивність перекисного окислення ліпідів, нітрооксидантного стресу, що запобігає надходженню недоокислених продуктів розпаду в кровотік. Однак стан і активність антиоксидантної системи крові не завжди об'єктивно відображає зміни в органах і системах.

Ключові слова: метаболічний синдром, баріатрична хірургія, оксидативний стрес, перекисне окиснення ліпідів, малоновий диальдигід.

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INTRODUCTION / ВСТУП

Obesity is characterized by excessive fat accumulation, disruption of redox homeostasis and metabolic changes, which are often associated with self-catalyzed lipid peroxidation [15, 16]. Since adipose tissue is known as a stress organ with a pronounced endocrine function, the cascade of pathological processes leading to adipose tissue dysfunction begins with the accumulation of fat, followed by changes in cellular composition, an increase in the number of infiltrated inflammatory cells, and an increase in the volume of adipocytes. The above leads to an increase in the secretion of pro-inflammatory, atherogenic and diabetogenic adipokines [11], as well as reactive oxygen species [6]. On the other hand, antioxidant defense mechanisms help maintain a healthy level of reactive oxygen species.

Surgery leads to restoration of redox balance and improvement of whole body metabolism [4, 5]. Thus, the source of bioactive lipids and fat-soluble antioxidants, as well as the complex pathophysiology of lipid peroxidation, should be considered from the perspective of personalized and integrative biomedicine for the appropriate treatment of obesity.

OBJECTIVE

The primary objective was to assess the impact of laparoscopic sleeve gastrectomy, laparoscopic gastropliction, bariatric artery embolization on changes in redox balance in patients with metabolic syndrome.

MATERIALS AND METHODS

The work is based on the materials of clinical examination and surgical treatment of 44 patients with metabolic syndrome (MS): 17 men and 27 women aged 47±6.75 years with a body mass index > 40 kg/m2. The control group included 12 people without manifestations of metabolic syndrome**.**

Blood samples taken for the study were obtained after patients fasted the night before the study. Plasma levels of glycated hemoglobin (HbA1c), glycaemia, as well as total cholesterol, low- and high-density lipoproteins (LDL, HDL), and triglycerides were measured using commercial kits (Roche Diagnostics) using Hitachi automatic analyzer. The insulin resistance index was calculated by the Caro method as the value of the ratio of glucose to insulin measured in the subjects on an empty stomach.

Malondialdehyde (MDA) was measured using a TBARS assay kit (Cayman Chemical Company, Ann Arbor, MI, USA) for assaying lipid peroxidation in plasma.

For measuring the plasma levels of leptin, ghrelin general, adiponectin, oxidized low-density lipoprotein cholesterol (Ox-LDL), resistin 10 ml of blood was collected in vacuum tubes. The samples were then kept at room temperature for 30 minutes and then centrifuged at 1670 g for 10 minutes. Isolated serum samples were stored in a freezer at −20°C.

The total antioxidant activity of blood serum (TAA) was determined by the method based on the

degree of inhibition of ascorbate-iron-induced oxidation of tween-80 to MDA and expressed as % inhibition of formation.

Catalase activity (CT) was assessed by the ability of hydrogen peroxide to form a stable colored complex with ammonium molybdate. Blood serum was tested. Catalase activity was determined by the photospectrometric method and expressed in μcat/l.

The total content of nitrates and nitrites was determined by the Griess method after the reduction of nitrates to nitrites with the help of cadmium. Calculations were performed according to the calibration schedule, using sodium nitrite as a standard. The content of nitrates and nitrites was expressed in mmol/l of blood serum.

Bariatric surgery: laparoscopic sleeve gastrectomy (LSG), laparoscopic gastropliction (LGP), bariatric artery embolization (BAE) were performed according to the methods [10].

LSG – after treating the surgical field with 10% betadine solution, a carboxyperitoneum of 15–17 mm Hg was created in the abdominal cavity, the esophagocaridal junction was crossed, the left pedicle of the diaphragm was isolated, and the stomach floor was partially mobilised using LigaSure. After identifying the pylorus, short vessels are transected 6–8 cm up the great curvature of the stomach to the left pedicle of the diaphragm and the stomach is mobilised. A probe (34 Fr) is inserted into the stomach and gradually, by applying cassettes (Endo GIA stapler, 45 and 60 mm long, 4.8 mm staples), a tubular stomach with a volume of up to 120–150 ml is formed. An invasive seromuscular suture was placed from the cardiac to the astral stomach using monofilament (V-Loc).

LGP – after treatment of the surgical field with 10% betadine solution, a carboxypneumoperitoneum was created at the level of 15 mmHg. The lig. gastrocolica was opened using high-energy devices. Mobilisation of the great curvature of the stomach was performed in the direction of the oesophagus until the left pedicle of the diaphragm was visualised. Next, the mobilisation of the large curvature of the stomach was performed in the direction of the duodenum, not reaching 2–3 cm to the pylorus. During the mobilisation of the large curvature of the stomach, special attention was paid to the disconnection of the gastro-splenic ligament. After mobilisation of the gastric curvature, VLock 2.0 (Covidien) suture was inserted into the abdominal cavity using a 1/2-gauge barbed needle, size 26. The first suture was placed 2–3 cm proximal to the pyloric veins. In the gap between the pylorus and the corner of the stomach, the sutures were placed inverted, capturing the posterior and anterior stomach walls with one stitch and a separate stitch. After passing the corner of the stomach, sutures were placed on the anterior and posterior surfaces of the stomach, capturing each wall with 2 or 3 stitches. Thus, the stomach wall from the side of high curvature is corrugated and pulled to the low curvature of the stomach, preventing the creation of excess tissue in the area of its bottom. The second row of sutures is applied by grabbing the anterior and posterior walls of the stomach with a single stitch. If necessary, a third row of sutures was performed in certain areas of the stomach. After that, the gastric lumen was reduced to 100–150 ml.

BAE – CT angiography was used to exclude vascular anomalies before embolisation. The intervention was performed through a transfemoral approach using a 5 F guidewire catheter. After that, digital subtraction imaging of the celiac disease was performed. The microcatheter was inserted into the target artery, and selective embolisation of the left gastric artery was performed with a high-flow microcatheter. Embolisation of one or more arteries was performed with spherical emboli of 300–500 mm in size (Embosphere (Merit Medical), until angiographic symptoms of "stasis" appeared. A perfusion study was performed to confirm the effectiveness of embolisation. After the intervention, patients were prescribed a proton pump inhibitor for 6 weeks after the intervention. Patients underwent mandatory endoscopy of the stomach and duodenum at 1 and 3 months.

Statistical processing of the obtained research data was processed using the software Excel (“Microsoft”, USA) and Statistica.10.1 (Statsoft, USA) with variation and correlation analysis. Data are expressed as mean ± S.D. p-value <0.05 signified statistical significance.

Results and discussion. It has been reported that hyperlipidaemia increases oxidative damage, contributing to the development of insulin resistance, altered endothelial dysfunction and energy metabolism [9]. Oxidative and nitrosative stress and, as a result, lipid peroxidation play a crucial role in the development of MS (Table 1).

The existence of an imbalance between antioxidant defence mechanisms such as CAT and SOD enzymes and the production of ROS increases the risk of oxidative stress establishment [8].

Table 1 – Indicators of the prooxidant-antioxidant system in patients with metabolic syndrome

Parameters	Obese patients (n=44)	Control (n=12)	p
BMI, kg/m^2	41,52 ± 6,78	20,89 ± 2,06	p=0.0052
Glucose, mmol/l	8,56 ± 0,54	5,58 ± 0,37	p<0,001
HbA1c, %	7,84 ± 0,42	5,43 ± 0,25	p<0,001
Insulin, μU/ml	24,56 ± 4,56	8,61 ± 2,78	p=0.0042
Caro index	0,24 ± 0,04	0,64 ± 0,03	p<0,001
MDA, μM	6,92 ± 0,41	4,21 ± 0,35	p<0,001
ox-LDL, U/l	105,41 ± 2,78	70,34 ± 1,89	p<0,001
Total cholesterol, mmol/l	6,21 ± 1,43	5,83 ± 1,12	p=0.8350
LDL, mmol/l	3,68 ± 1,04	3,47 ± 1,09	p=0.8896
HDL, mmol/l	1,18 ± 0,42	1,42 ± 0,89	p=0.8082
Triglycerides, mmol/l	1,76 ± 0,64	1,54 ± 0,75	p=0.8242
Atherogenic index	4,78 ± 1,34	3,21 ± 1,27	p=0.3989
TAA % formation inhibition	48,65 ± 2,36	75,43 ± 3,39	p<0,001
Catalase, mkat/l	2,03 ± 0,78	0,88 ± 0,34	p=0.1822
NO_x, mmol/l	8,87 ± 1,66	4,08 ± 0,49	p=0.0077

Vávrová, L. implicated an increased oxidative stress in MS and a decreased antioxidative defense that correlated with some laboratory (triglycerides, high-density lipoprotein cholesterol (HDL-C)) [12] Despite the evidence highlighting activation of NO, in MS-related disease its biological activities are in general downregulated due to a dysfunction of the radical metabolism and availability. As reported above, the activation of NOXs measured in the MS-related diseases could lead to increased ROS production [14].

We have established a tendency towards intensification of lipoperoxidation processes and reduction of protective resources of antioxidant protection. Statistically significantly higher indicators of the content of TBC-active products in the serum of MS patients were established (p<0.001). Statistically significantly lower indicators (by 1.6 times) of TAA activity were recorded in patients with MS compared to controls (p<0.001). However, catalase activity increased in our study. Thus, we found a 2.3-fold increase in catalase activity in MS patients compared to controls.

As the results of our research showed, the level of TBC-active products (MDA) increased statistically significantly in patients with metabolic syndrome – 1.64 times (Table 1). This is obviously related to the exhaustion of the pool of antioxidant defense enzymes and the negative course of MS in patients. The obtained data indicate that MS contributes to oxidative and nitrooxidative stress, exhaustion of the antioxidant defense system. When assessing nitric oxide indicators, we recorded the development of endothelial dysfunction in patients with MS. A statistically significant increase in the concentration of nitrates and nitrites (NOx) in the serum of patients with MS was revealed by 2 times compared to controls (p=0.0077). The dynamics of indicators established by us testifies to the development of nitrooxidative stress and dyslabance in the pro-oxidant-antioxidant system in patients with MS.

Over the past fifteen years, various authors have investigated oxidative markers after bariatric surgery, thus creating abundant evidence of reduced oxidative stress. In works of Kelly A.S. bariatric surgery produced robust improvements in markers of inflammation, oxidative stress and several adipokines among adolescents with severe obesity [5].

After the bariatric interventions, we recorded a significant decrease in the content of MDA – the main marker that characterizes the activation of LPO, but this indicator did not reach the level of the control group of patients after all three types of surgical intervention (LSG, LGP, BAE) (Table 2).

Table 2 – Indicators of the redox homeostasis in patients with metabolic syndrome after LSG, LGP, BAE surgery

Option	LSG (n=15)	LGP (n=22)	BAE (n=7)
MDA, μM (before surgery)	6,46±0,62*	6,87±0,42*	6,48±0,38*
1 month after surgery	5,40±0,52	5,20±0,58	5,18±0,34
3 months after surgery	4,86±0,34	4,64±0,54	4,89±0,45
6 months after surgery	4,67±0,43 (*p=0.0224)	4,48±0,42 (*p=0.0002)	4,18±0,21 (*p=0.0002)
ox-LDL, U/l (before surgery)	107,56±2,03**	103,57±2,23**	104,45±2,09**
1 month after surgery	92,44±1,67	94,67±2,03	91,45±2,13
3 months after surgery	87,89±1,99	85,12±1,98	85,67±2,56
6 months after surgery	81,07±2,01 (**p<0,001)	78,32±2,02 (**p<0,001)	77,04±2,21 (**p<0,001)
Catalase, mkat/l (before surgery)	2,08±0,34***	2,03±0,24***	2,01±0,11***
Catalase, mkat/l (6 months after surgery)	1,76±0,60 (***p=0.6463)	1,49±0,53 (***p=0.3587)	1,56±0,71 (***p=0.5438)
NO_x, mmol/l (before surgery)	8,96±1,23****	8,45±1,16****	8,78±1,22****
NO_x, mmol/l (6 months after surgery)	8,16±1,48 (****p=0.6809)	7,18±1,27 (****p=0.4645)	6,99±1,34 (****p=0.3444)
TAA % formation inhibition before surgery	47,96±2,70*****	49,07±3,01*****	48,67±2,67*****
TAA % formation inhibition (6 months after surgery)	64,78±2,12 (*****p<0,001)	61,34±2,36 (*****p=0.0025)	63,23±2,42 (*****p=0.0019)

The monitoring of this studied indicator showed that only 6 months after the surgical intervention, it only approached the control level and was 4.67±0.43 μM, 4.48±0.42 μM and 4.18±0.21 μM in of operated patients with LSG, LGP, BAE, respectively, versus control (4.21±0.35 μM).

Similar results were described in Min T. et al. research study. They observed an initial decrease in TBARS at 1 month and 6 months after LSG but not statistically significant [7]. However, Uzun et al. showed a significant decrease in MDA and oxidized LDL (ox-LDL) 6 months after laparoscopic adjustable gastric banding [11].

The decrease in manifestations of increased free radical oxidation and nitrooxidative stress had a positive effect on the activity of antioxidant defense enzymes. The indicators of the antioxidant system (TAA, catalase activity) that we studied tended to normalize after the surgical interventions.

Study design Joao-Cabrera et al., [3], measured in addition different markers of oxidative stress in plasma (e.g. MDA, superoxide dismutase, catalase, and total radical antioxidant parameter). A significant reduction 12 months post-surgery ($p < 0.01$) could be detected in MDA, superoxide dismutase. In contrast, plasma levels of glutathione and total radical antioxidant parameters showed a significant increase.

On the other hand, 6 months after LSG Cătoi A.F et. al, besides a significant reduction of BMI and weight, no significant changes of nitrooxidative stress markers (NOx, total oxidant status) were observed [1].

The activity of another enzyme of antioxidant protection, catalase, which we studied, increased in patients with MS, which can be explained by a compensatory increase in its activity in response to increased activity of lipoperoxidation processes. After the surgical intervention, the activity of the studied enzyme had a tendency to decrease. Depletion of indicators of antioxidant protection, namely general antioxidant activity in patients with MS can be explained on the one hand by the activation of lipoperoxidation processes caused by nitrooxidative

stress, and on the other hand by the depletion of the pool of antioxidant enzymes. This statement does not apply to the increase in catalase activity, which is considered to be acute-phase and usually increases with the inflammation inherent in MS.

CONCLUSIONS / ВИСНОВКИ

Bariatric surgery reduces the intensity of lipid peroxidation, nitrooxidative stress, which prevents underoxidized decay products from entering the bloodstream. However, the state and activity of the blood's antioxidant system does not always objectively reflect changes in organs and systems.

CONFLICT OF INTEREST / КОНФЛІКТ ІНТЕРЕСІВ

The authors declare no conflict of interest.

CONNECTION WITH THE RESEARCH WORK/ ЗВ'ЯЗОК З ІНШИМИ РОБОТАМИ

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AUTHOR CONTRIBUTIONS / ВКЛАД АВТОРІВ

The author confirms sole responsibility for the following: study conception and design, data collection, analysis and interpretation of results, and manuscript preparation.

REFERENCES/СПИСОК ЛІТЕРАТУРИ

1. Cătoi AF, Pârvu AE, Mironiuc A, et al. Chemerin, Inflammatory, and Nitrooxidative Stress Marker Changes Six Months after Sleeve Gastrectomy. *Oxid Med Cell Longev.* 2018;2018:1583212. https://doi.org/10.1155/2018/1583212
2. Fejfer K, Buczko P, Niczyporuk M, et al. Oxidative Modification of Biomolecules in the Nonstimulated and Stimulated Saliva of Patients with Morbid Obesity Treated with Bariatric Surgery. *Biomed Res Int.* 2017;2017:4923769. https://doi.org/10.1155/2017/4923769
3. João Cabrera E, Valezi AC, Delfino VD, Lavado EL, Barbosa DS. Reduction in plasma levels of inflammatory and oxidative stress indicators after Roux-en-Y gastric bypass. *Obes Surg.* 2010;20(1):42-49. https://doi.org/10.1007/s11695-009-9988-2
4. Horn RC, Gelatti GT, Mori NC, et al. Obesity, bariatric surgery and oxidative stress. *Rev Assoc Med Bras (1992).* 2017;63(3):229-235. https://doi.org/10.1590/1806-9282.63.03.229
5. Kelly AS, Ryder JR, Marlatt KL, Rudser KD, Jenkins T, Inge TH. Changes in inflammation, oxidative stress and adipokines following bariatric surgery among adolescents with severe obesity. *Int J Obes (Lond).* 2016;40(2):275-280. https://doi.org/10.1038/ijo.2015.174
6. McMurray F, Patten DA, Harper ME. Reactive Oxygen Species and Oxidative Stress in Obesity-Recent Findings and Empirical Approaches. *Obesity (Silver Spring).* 2016;24(11):2301-2310. https://doi.org/10.1002/oby.21654
7. Min T, Prior SL, Dunseath G, Churm R, Barry JD, Stephens JW. Temporal Effects of Bariatric Surgery on Adipokines, Inflammation and Oxidative Stress in Subjects with Impaired Glucose Homeostasis at 4 Years of Follow-up. *Obes Surg.* 2020;30(5):1712-1718. https://doi.org/10.1007/s11695-019-04377-3
8. Monserrat-Mesquida M, Quetglas-Llabrés M, Capó X, et al. Metabolic Syndrome is Associated with Oxidative Stress and Proinflammatory State. *Antioxidants (Basel).* 2020;9(3):236. https://doi.org/10.3390/antiox9030236
9. Oguntibeju OO. Type 2 diabetes mellitus, oxidative stress and inflammation: examining the links. *Int J Physiol Pathophysiol Pharmacol*. 2019;11(3):45-63.
10. Prodan A, Dzhyvak V. Comparative evaluation of different types of bariatric surgery. *Journal of Education, Health and Sport*. 2022;12(4):186-192. https://doi.org/10.12775/JEHS.2022.12.04.016.
11. Taylor EB. The complex role of adipokines in obesity, inflammation, and autoimmunity. Clin Sci (Lond). 2021;135(6):731-752. https://doi.org/10.1042/CS20200895

12. Uzun I, Fuat G. Kinetics and thermodynamics of the adsorption of some dyestuffs and p-nitrophenol by chitosan and MCM-chitosan from aqueous solution. *Journal of colloid and interface science*. 2004;274(2):398-412.
13. Vávrová L, Kodydková J, Zeman M, et al. Altered activities of antioxidant enzymes in patients with metabolic syndrome. *Obes Facts*. 2013;6(1):39-47. https://doi.org/10.1159/000348569
14. Vona R, Gambardella L, Cittadini C, Straface E, Pietraforte D. Biomarkers of Oxidative Stress in Metabolic Syndrome and Associated Diseases. *Oxid Med Cell Longev*. 2019;2019:8267234. https://doi.org/10.1155/2019/8267234
15. Whaley-Connell A, McCullough PA, Sowers JR. The role of oxidative stress in the metabolic syndrome. *Rev Cardiovasc Med*. 2011;12(1):21-29. https://doi.org/10.3909/ricm0555
16. Prodan A, Dzhyvak V. Metabolic syndrome: Correlation between main hormones and oxidative stress parameters. *Romanian Journal of Diabetes, Nutrition and Metabolic Diseases*. 2022;29(2):214-219. https://doi.org/10.46389/rjd-2022-1095

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