ENDOTHELIAL DYSFUNCTION IN COVID-19 PATIENTS AND CLINICAL APPLICATION OF LASER THERAPY

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This review covers the published papers describing endothelial dysfunction pathogenesis and molecular mechanisms behind the effect of low-level laser therapy on regulation of the said pathogenesis. Herein, we present the current experience of using laser therapy to prevent development of endothelial dysfunction in the context of post-COVID-19 rehabilitation, as well as the accumulated data on the methods of combination of external or intravenous laser blood therapy and influence on the immunocompetent. We provide justification for practicing personalized approach at various stages of post-COVID-19 rehabilitation and treatment. The basis allowing greater efficacy of post-COVID-19 rehabilitation, including protocols making use of laser therapy, is the analysis of single-nucleotide polymorphisms of genes that determine adaptation processes, peculiarities of the immune response to infectious pathogens, predisposition to the development of respiratory distress syndrome, severe pneumonia, sepsis, multiple organ failure, development of endothelial dysfunction, thrombotic complications, the analysis that allows identification of patients running higher risk of critical conditions.

Keywords: COVID-19, endothelial dysfunction, rehabilitation treatment, laser therapy, personalized approach, genotyping

ЭНДОТЕЛИАЛЬНАЯ ДИСФУНКЦИЯ У БОЛЬНЫХ COVID-19 И КЛИНИЧЕСКОЕ ПРИМЕНЕНИЕ ЛАЗЕРНОЙ ТЕРАПИИ

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В обзоре литературы по патогенезу развития дисфункции эндотелия и молекулярным механизмам влияния низкоинтенсивного лазерного излучения на его регуляцию представлены актуальный опыт применения лазерной терапии для предотвращения развития эндотелиальной дисфункции в реабилитации больных COVID-19, методы сочетания наружного или внутривенного лазерного освечивания крови и воздействия на иммунокомпетентные органы. Обоснован персонализированный подход к лечению и профилактике на различных этапах реабилитации пациентов, перенесших COVID-19. Анализ однонуклеотидных полиморфизмов генов, детерминирующих процессы адаптации, особенности иммунного ответа на инфекционные возбудители, предрасположенность к развитию респираторного дистресс-синдрома, тяжелому течению пневмонии, сепсиса, полиорганной недостаточности; развитию эндотелиальной дисфункции, тромботическим осложнениям для выявления пациентов с повышенным риском критических состояний является основой повышения эффективности восстановительного лечения таких больных, в том числе с применением методов лазерной терапии.

Ключевые слова: COVID-19, эндотелиальная дисфункция, восстановительное лечение, лазерная терапия, персонифицированный подход, генотипирование

The global COVID-19 pandemic caused by the SARS-CoV-2 coronavirus is a challenge for the entire mankind, but the first to search for solutions thereto are scientists and doctors that are tasked with finding ways to curb incidence, effectively treat and rehabilitate COVID-19 patients and minimize the associated complications and mortality.

One of the many features of COVID-19 is the pronounced non-specificity of the observed pathological processes and its capacity to damage both organs and tissues and functional regulatory systems. At the same time, development of endothelial dysfunction can be named a factor that largely unites various disorders. There is an opinion gaining popularity that vascular endothelial damage is the cornerstone of organ dysfunction in severe SARS-CoV-2 infection cases [1].

The patients that died from respiratory failure resulting from COVID-19 had diffuse alveolar injury with perivascular T-cell infiltration as a specific histological pattern registered in the peripheral lung. The lungs of these patients have distinctive vascular features: severe endothelial damage associated with intracellular presence of the virus and fragments of destroyed cell membranes. Histological analysis of pulmonary vessels of COVID-19 patients revealed widespread thrombosis and microangiopathy. COVID-19 patients had alveolar capillary micro clots 9 times more often than influenza patients ($p < 0.001$). All this indicates that the disease also translates into a severe endothelial dysfunction [2].

Endothelial dysfunction (EnD) — a complex multifaceted process typically seen in the context of cardiovascular,

metabolic and immune disorders — is a current and serious challenge for clinical practitioners, even when considered outside of connection to COVID-19 [3]. And with a developing viral infection in the background, exploring the means to prevent this pathology is of paramount importance.

Endothelium is a cardiovascular endocrine organ that, in critical situations, enables communication between blood and tissues [4]. It acts as a barrier between the blood and the vascular wall, helps adaptation to changing environmental conditions through local regulation of vascular tone, vascular wall integrity protection etc. Normally, endothelial cells, following alterations in blood flow rate, exposure to mediators or neurohormones, react by increasing the synthesis of substances that cause relaxation of the vascular wall's smooth muscle cells (nitric oxide (NO) and other relaxants). They also work to prevent thrombogenesis by blocking platelet aggregation, oxidating low density lipoproteins, expressing adhesion molecules, "sticking" monocytes and platelets to the vascular wall, producing endothelin etc. Compensatory mechanisms are activated under the influence of a damaging factor. In case of prolonged exposure to such a factor (hypoxia, intoxication, inflammation, hemodynamic overload, etc.), compensation fades and a pathological process develops. Endothelial dysfunction is an imbalance between biologically active substances synthesized by endothelial cells (potentially protective NO, endothelial hyperpolarization factor, prostaglandins) and damaging substances (endothelin-I, thromboxane A2, superoxide anion etc.) [5].

It is the genotype that shapes all these normal and pathological molecular mechanisms of endothelium's adaptive response to normal or excessive influences. Currently, there are over 1500 genes established to have an association with multifactorial human diseases.

The human genome investigation efforts in the context of the Human Genome Project, Hap Map project, The 1000 Genomes projects, The SNP Consortium, have revealed mutations and single nucleotide polymorphisms (SNP) in genes encoding protein molecules of the body's regulatory systems. Their associations with various pathologies were either confirmed or refuted [6–9].

For example, the public Online Mendelian Inheritance in Man database (OMIM) [10] and the single nucleotide polymorphisms database contain more than 3.5 million SNP markers [11].

One of the studies investigating the significance of polymorphism of various genes considered possible contributors to the development of cardiovascular diseases (CVD) identified 105 genes that most likely support pathophysiology of CVD [12]. The researchers focus on genes that determine endothelium's properties, its role in the development of local vasospasm/vasodilation, hemostasis, inflammation, atherosclerosis, angiogenesis, etc [13–14].

Long before the COVID-19 pandemic, significant individual characteristics of critical conditions observed dynamically triggered the analysis of the results of geno-phenotypic examinations of IC patients [15–19]. These studies presented comorbid conditions gene diagnostics, identified SNP markers associated with an increased risk of community-acquired and nosocomial pneumonia, risk of development of an acute respiratory distress syndrome, CVD-related thrombotic complications. The results of the analysis of identified gene polymorphisms the products of which shape regulation (hemostasis, renin-angiotensin system regulation, immune system regulation, i.e. individual response to infectious pathogens and production of cytokines, drug metabolism) provide justification to screening patients running a high risk of development of life-threatening conditions. Such patients need non-standard treatment approaches in critical situations.

Personalized approach is a strategy popular at various stages of rehabilitation. In particular, patients in cardio- and neurorehabilitation undergo genotyping enabled by various SNP panels [20–25]. In clinical practice, molecular markers of individual susceptibility to various patterns of CVD development (the most important of which is endothelial dysfunction) allow predicting sudden death of a patient or the development of catastrophic multiple organ complications, as well as choosing the most effective therapies, which may be pharmacotherapy and non-drug methods [26–27], including laser therapy.

It was observed that patients with different phenotypes respond to laser therapy differently. In particular, low level laser therapy (LLLT) was more effective in patients that exhibited domination of reactions of the sympathetic nervous system than in those whose parasympathetic responses were stronger [28]. The peculiarities of the endothelial function were found to be behind this difference. The said function is genetically determined by the cooperation of gene regulatory networks [26–27]; it needs to be studied further, same as the collation of geno- and phenotypical characteristics and individual responses to various therapies.

Concomitant diseases can synergistically activate pathophysiological pathways. Thus, inflammation activates vascular pathology through pro-inflammatory cytokines, endothelin-1 and nitric oxide, which contributes to long-term damage to fatty acids, proteins, DNA, and mitochondria. Dysfunctional energy metabolism (impaired production of mitochondrial ATP, the formation of amyloid-β), development of endothelial dysfunction and violation of the blood-brain barrier lead to the cerebral blood flow reduction and chronic cerebral hypoperfusion with oxygen and nutrient deficiency, metabolic and synaptic disorders, neurodegeneration and white matter atrophy, cognitive dysfunction and development of Alzheimer's disease [29]. Identification and assessment of the entire complex of pathogenetic mechanisms driving inflammation form basis for targeted therapies designed to remedy the reduced cerebral blood flow and hypometabolism.

Molecular-cellular and physiological mechanisms of vascular homeostasis regulation

The key manifestations of EnD are abnormal bioavailability of nitric oxide (NO), the main vasodilator, which results from suppression of endothelial NO synthase (NOS), with the NO synthesis decreasing consequently [30]. Under normal physiological conditions, there is a balance between vasoconstrictors secreted by the endothelium and vasodilators. Any violation of this balance leads to a local spasm and vascular tone growth. As a result, the compensatory capacity of endothelium deteriorates gradually, which translates into breakdown of a rather complex regulation of the natural vascular bed expansion and shrinking mechanisms [13]. Endothelium plays a key role in maintaining vascular homeostasis since it releases biologically active substances (Table 1), but is also susceptible to the effects of external regulators [31–33]:

– mast cells that release heparin and histamine;

– platelets containing vascular endothelial growth factors and blood coagulation factors, etc;

– hormones and neuropeptides (adrenaline, acetylcholine, histamine, bradykinin, etc).

Despite the fact that the regulation mechanisms are known (see Table 1), the ways to remedy endothelial dysfunction pharmacologically require further comprehensive study and

Table 1. Physiologically active substances, regulators of the circulatory system, synthesized in the endothelium

Vascular wall tone regulators	
Vasoconstrictors	Vasodilators
Endothelin I–II Angiotensin II Thromboxane (TXA$_2$) Prostaglandins H$_2$ and G$_2$	Nitric oxide (NO) Prostaglandin E$_2$ (PGE$_2$) Endothelial hyperpolarizing factor (EDHF) Bradykinin C-natriuretic peptide Adrenomedullin Endothelin III
Regulators of hemostasis and antithrombosis	
Prothrombogenic factors	Antithrombogenic factors
Platelet-derived growth factor (PDGF) tissue plasminogen activator inhibitor (PAI-I) Von Willebrand factor (coagulation factor VIII) Angiotensin IV Endothelin I	NO Tissue plasminogen activator (t-PA) Prostacyclin (PGI$_2$)
Leukocyte adhesion regulators	
Adhesion stimulants (E-selectin, P-selectin, intercellular adhesion molecule 1 (ICAM-I), vascular cell adhesion molecule 1 (VCAM-I)	
Vascular growth regulators	
Stimulants	Inhibitors of myocyte migration and proliferation
Endothelin-I Angiotensin-II Superoxide radicals Growth factors: fibroblast, platelet, insulin-like, transforming growth factor β (bFGF, PDGF, IGF, TGFβ)	NO Prostacyclin (PgI$_2$) C-natriuretic peptide
Regulators of inflammation, vascular permeability, apoptosis of vascular wall components	
Stimulants Tumor necrosis factor α (TNFα) Superoxide radicals (O$_2^-$, OONO$^-$) Protein kinase C	Inhibitors
	NO

evaluation, since currently they have negative side effects and deliver mediocre results [3]. Physiotherapeutic procedures are one of the options considered when the functional state of the endothelium needs to be normalized [34].

Primary and secondary mechanisms of the biomodulating action of low-level laser therapy (LLLT)

The current understanding of the biomodulating action of LLLT, which agrees with the clinical practice of laser therapy use, has the thermodynamic triggering of Ca^{2+} dependent processes as the primary mechanism. Once the various intracellular components have absorbed photon energy (laser light), the intracellular calcium store is activated, Ca^{2+} ions are released and the concentration in the form of two waves with half periods of 100 and 300 s is increased, which is followed by the cascade of responses on all levels, from cells to the entire body: activation of mitochondria, cellular metabolism and proliferation, normalization of the immune and vascular systems, inclusion of the autonomic and central nervous system into the process, etc (Fig. 1) [35–37].

Versatility and high efficacy of laser therapy, which is unique type of physiotherapy, relies on the action at cellular level, with the maximum frequency of optical band electromagnetic waves and laser light coherence (monochromaticity).

Influence of LLLT on the vascular homeostasis regulation factors and immunity

It is well known that almost all of the above regulators (Table 1) are, to a certain degree, associated with changes in Ca^{2+} concentration; we will cite only a few reviews [38–39].

From the point of view of the subject researched, we should be primarily interested in nitric oxide, the synthesis and release of which is Ca^{2+} dependent [40]; therefore, it is not surprising that many studies confirm that LLLT can stimulate the release of NO, thus enabling regulation of the vascular homeostasis [41–47].

Moreover, there are studies demonstrating a direct relationship between intracellular Ca^{2+} concentration increase and NO release intensity and subsequent vasodilation [48–50].

Endothelial system normalization in children with bronchial asthma was confirmed by changes in various parameters of blood plasma, including endothelin-1 and nitric oxide [51–52]. The capacity of LLLT to effectively stimulate the release of PGE$_2$ has been proven both in experimental [53–55] and clinical studies [56–58].

In arterial hypertension patients, regimens of both external laser therapy (ELT) pulsed infrared LLLT) and intravenous laser blood therapy (IVLBT) improve a number of biochemical, hemorheological and hormonal parameters (C-peptide, insulin, angiotensin, bradykinin, aldosterone, cortisol), and the improvements persist for at least 6 months [59–61].

Many researchers have shown the role of the kallikrein system in hemovascular regulation and the possibility of its correction through illumination of blood with red laser (wavelength of 635 nm) and/or incoherent ultraviolet (UV) light [62–65].

Current laser therapy techniques actively exploit the well-known anti-inflammatory effect of LLLT. Numerous studies have shown that LLLT can activate phagocytes (which absorb foreign particles of bacteria, viruses, and dying cells) and the synthesis of cytokines, including interferons (IFNs), which spearhead the first line of defense against viruses
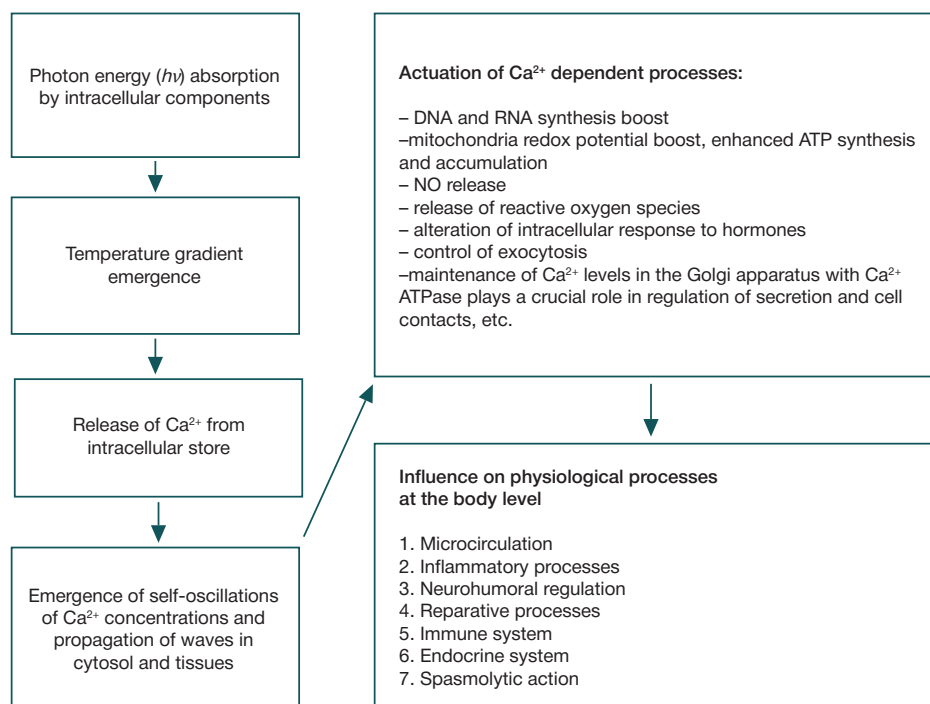
Fig. 1. Molecular-cellular mechanisms of the biomodulating action of LLLT

and contribute greatly to the development of adaptive immunity. IFNα and IFNβ, which are secreted by lymphocytes, macrophages, fibroblasts and some epithelial cells, stimulate the activity of macrophages and natural killer cells (NK). IFNγ, secreted by T-cells and EK, regulates the immune response, has antiviral and antitumor effects. In addition, LLLT improves micro- and macrocirculation by increasing the saturation of damaged tissues with oxygen and improving their trophic supply by boosting metabolism and proliferation, thus initiating

the development of recovery processes. These properties of LLLT make it an effective prevention and therapeutic tool that can be used to counter viral infection and its consequences and to prevent development of pulmonary fibrosis [37].

Laser therapy techniques

In the context of COVID-19 treatment, external laser therapy or intravenous laser blood illumination (ELD or IVLBT) are used
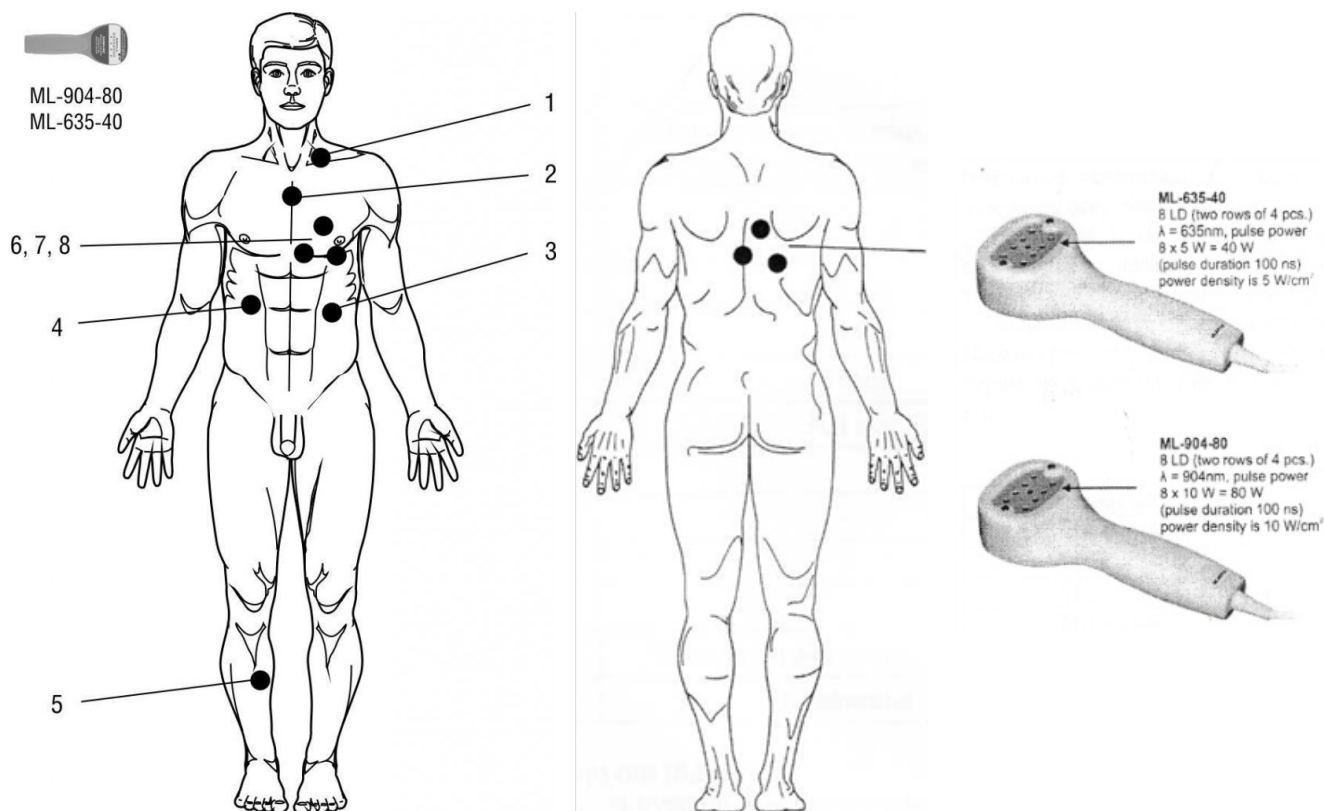

Fig. 2. Zones exposed to laser radiation from the emitting heads

Table 2. Zones exposed to laser light as a coronavirus disease prevention measure

Emitting head type	Exposed area (Figure 2)	Exposure, min
ML-635-40	1 — left supraclavicular region	2
ML-904-80	2 — thymus	1
ML-904-80	3 — spleen	1

Table 3. Parameters of the LLLT technique for prevention of coronavirus disease

Parameter	Value	Notes
Laser light wavelength, nm (spectrum)	635 (red)	–
	904 (IR)	
Laser operating mode	Pulse	Matrix emitting head, surface area 10 cm^2
Light pulse duration, ns	100–150	–
Radiation power, W	35–40	635 nm
	60–80	904 nm
Power density, W/cm^2	4–5	635 nm
	8–10	904 nm
Frequency, Hz	80	
Exposure per zone, min	See table 2	–
Number of exposed zones	3	–
Localization	See table 2	–
Method	Contact	Through the transparent tip
Number of procedures per regimen	2–3	Daily

to reach immunocompetent organs and applied locally, to the lesion focus [66]. This approach, a combination of exposure to LLLT on the systemic and local levels, has shown great results in clinical practice [66–69]. The most common technique used for the purpose of endothelium function correction is the "classical" wavelength of 635 nm, 2–3 mW output power and 10–20 min of exposure [70–76]. However, recently a combined version of the technique that includes laser UV blood illumination (LUVBI) has been gaining popularity [77–79]. The specialists are also well aware of the degrees of efficacy achievable in combinations of laser therapy and other physiotherapeutic methods, which have been confirmed in the treatment of COVID-19 [80–82].

LLLR-based coronavirus disease prevention and treatment

Those who have come into contact with the sick or who have arrived from epidemiologically unsafe areas are prescribed 2–3 LLLT procedures as prevention.

The sick receive treatment in hospitals; the regimen includes 10–12 daily laser therapy procedures.

Two versions of LLLT methods have been developed, the first relying solely on non-invasive techniques (external illumination) and the second, more effective, which involves IVLBT.

Method 1: prevention

Before starting the procedure, it is necessary to remove the protective cover and mount the magnetostatic field (MF) tip. The tip should be subjected to preliminary chemical sterilization (disinfection).

Fig. 2A and Fig. 2B show the zones (points) of application; Table 2 and Fig. 2C prescribe the type of emitting head and the exposure; Table 3 contains the parameters of the laser light.

Method 1: treatment

Before starting the procedure, it is necessary to remove the protective cover and mount the MF tip. The tip should be subjected to preliminary chemical sterilization (disinfection).

Fig. 2A and Fig. 2B show the zones (points) of application; Table 4 prescribes the type of emitting head and the exposure. Table 5 contains the parameters of the laser light.

Combined method 2

Combined method: external irradiation of zones 6-8 as shown on Fig. 2; type of emitting head and exposure as given in Table 4;

Table 4. Zones exposed to laser light in coronavirus patients

Emitting head type	Exposed zone (Fig. 1)	Exposure, min
ML-635-40	1 — left supraclavicular region	2
ML-904-80	2 — thymus	1
ML-904-80	3 — spleen	1
ML-904-80	4 — liver	2
ML-635-40	5 — E36 (zu san li) — symmetrical	0.5 min per zone
ML-904-80	6–8 — lung injury projection (see Fig. 2 for localization example)	1.5 min per zone

Table 5. Parameters of the LLLT technique for treatment of coronavirus patients

Parameter	Value	Notes
Laser light wavelength, nm (spectrum)	635 (red)	–
	904 (IR)	
Laser operating mode	Импульсный	Matrix emitting head, surface area 10 cm²
Light pulse duration, ns	100–150	–
Radiation power, W	35–40	635 nm
	60–80	904 nm
Power density, W/cm²	4–5	635 nm
	8–10	904 nm
Frequency, Hz	80	Zones 1–5
	80–1500	Zones 6–8, frequency can be varied depending on symptoms and patient condition
Exposure per zone, min	See table 4	–
Number of exposed zones	8	–
Localization	See table 4	–
Method	Contact	Through the transparent tip
Number of procedures per regimen	10–12	Daily

aser light parameters as provided in Table 5. Next step: IVLBT-525 + LUVBI (Table 6; Fig. 3).

Thirty-one SARS-CoV2-induced pneumonia patients with comorbidities (CVD, metabolic syndrome, type 2 diabetes mellitus, COPD, etc.) received rehabilitation treatment in the Central Clinical Hospital for the Rehabilitation of FMBA of Russia. In this group, the degree of damage to the lung tissue varied from 25 to 92%. Both of the above laser therapy methods were used for the patients; they delivered good results in the treatment of COVID-19 patients with severe lung lesions.

Subjectively, all patients noted general condition improvement, relief of chest pain associated with coughing, better sputum discharge, less severe shortness of breath. Moreover, in all patients we registered better oxygen saturation (pulse oximetry data) with the mean improvement from 93 to 97%; stabilization of the external respiration function accompanied by the increase of the vital volume of lungs; improvements revealed by the repeated lungs computed tomography. It is important that in the process of rehabilitation, these patients had their psychoemotional status normalized and the number of asthenic and anxiety-depressive incidents decreased (as measured with the Beck Depression Inventory and the MPS test (multilateral personality study).

The use of laser therapy for COVID-19 patients for the first time in the Central Clinical Hospital for the Rehabilitation

of FMBA of Russia is mentioned as an example of the above promising non-drug therapies. As we gain experience, we shall report clinical data, more widely and in detail, with a statistical analysis of the results, evidence-based conclusions of the effectiveness of the method and personalized approaches in the complex treatment and prevention of complications.

CONCLUSION

This literature review demonstrates the capacities of laser therapy in the context of endothelial dysfunction treatment. The review cites positive experience of using laser therapy in the complex treatment and rehabilitation of patients with atypical pneumonia caused by various coronaviruses and the new SARS-CoV2.

LLLT is shown an absolutely safe, highly effective, simple and inexpensive method of prevention, treatment and rehabilitation of both chronic non-infectious cardiovascular and pulmonary pathologies and diseases caused by a viral infection, including COVID-19.

To enable personalized approach to rehabilitation of COVID-19 patients, it is necessary to search for informative biomarkers of genetic predisposition to endothelial dysfunction, hemostasis disorders, assess the individual characteristics of

Table 6. Parameters of the IVLBT 525 + LUVBI technique (basic)

Parameter	Value	Notes
Laser light wavelength, nm (spectrum)	365–405 (UV)	LUVBI
	520–525 (green)	IVLBT-525
Laser operating mode	Continuous	–
Radiation power *, mW	1,5–2	At the outlet of the disposable light guide
Exposure, min	3–5	LUVBI
	7–8	IVLBT-525
Localization	Vein ulnar median (v. mediana cubiti)	–
Method	Intravenously	Through the disposable sterile light guide KIVL-01 made by the Matrix R&D Center (TU 9444-005-72085060-2008)
Number of procedures per regimen	10–12	Daily, alternating every other day IVLBT-525 and LUVBI

innate immunity and adaptive immune response to infection, risks of hyperreaction, cytokine storm, multiple organ failure, delayed manifestation of complications in a particular patient. Determination of the contribution of these individual (hereditary and environmental) factors, consideration of their mutual influences are crucial for application of the results of such complex examinations in real practice and indispensable for the development of individual prognosis, prevention (primary and secondary) measures, targeted treatment regimens that, in particular, include LLLT.
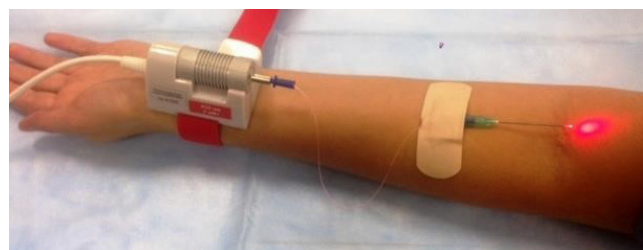

Fig. 3. IVLBT procedure

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