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Role of Acute Vascular Distress Syndrome in the development of Multisystem Inflammatory Syndrome in SARS-CoV-2 and modern views on the research and treatment of critical Coronavirus.

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Keyword. Acute Vascular Distress Syndrome; Multisystem Inflammatory Syndrome; Capillary Leak Syndrome; Microcirculatory-Mitochondrial Distress Syndrome; Lysosomal Mitochondrial Clearance of Autophagy (Mitophagy); Abnormal/extreme myelopoiesis.

Abbreviation IC CHAOS: Immunocompromised dissonance (IC) CHAOS-[**C**]ardiovascular Compromise: shock; [**H**]homeostasis; [**A**]poptosis; [**O**]rgan dysfunction; [**S**]suppression of the immune system.

Abstract

Background: The COVID-19 pandemic, caused by SARS-CoV-2, has highlighted the complex pathology of the virus, which extends beyond respiratory symptoms to include multisystem inflammatory syndrome (MIS). This review explores the role of Acute Vascular Distress Syndrome (AVDS) in the development of MIS in both adults (MIS-A) and children (MIS-C), providing a comprehensive overview of modern research and treatment approaches for severe coronavirus infections.

Methods and Materials: This review synthesizes findings from multiple studies and clinical reports to analyze the mechanisms by which SARS-CoV-2 induces AVDS and subsequently MIS. Key materials include molecular and cellular research data, clinical case studies, and treatment protocols. Diagnostic tools such as PCR and serological testing, as well as various biomarkers like neurofilament light chain and galectin-3, are discussed to elucidate their roles in identifying and managing severe COVID-19 cases.

Results: The interaction between SARS-CoV-2 and host endothelial cells, mediated by ACE2 receptors, triggers a cascade of inflammatory responses leading to AVDS. This syndrome is characterized by severe endothelial dysfunction, cytokine storms, and mitochondrial distress, which collectively contribute to the pathogenesis of MIS. Clinical evidence indicates that AVDS is a critical factor in the severity of COVID-19, with widespread implications for multiple organ systems, including the central and peripheral nervous systems.

Conclusion: Understanding the role of AVDS in MIS development offers valuable insights into the pathophysiology of severe COVID-19. Effective management requires a multifaceted approach, combining antiviral therapies, immunomodulators, and supportive treatments like extracorporeal membrane oxygenation (ECMO). Future research should focus on targeted therapies to mitigate endothelial damage and improve patient outcomes in critical COVID-19 cases.

Maria Vasilieva, Irina Vasilieva, Stanislav Groppa & Ilie Vasiliev in 2022 described recurrent COVID-19 infection with meningitis without lung damage.

SARS-CoV-2 causes coronavirus disease 2019 (COVID-19), which has killed ~7 million people worldwide. SARS-CoV-2 was initially considered respiratory pathology. However, it turned out that SARS-CoV-2 causes Multisystem Inflammatory Syndrome (MIS), which the authors Irina Vasilieva, Maria Vasilieva & Ilie Vasiliev (2023) wrote in the book "Molecular Pathological Biology of Coronavirus infection SARS-CoV-2" published in the UK [1]. The described as the Multisystem Inflammatory Syndrome in children (MIS-C) [2,3] as well as Multisystem Inflammatory Syndrome in Adults (MIS-A) [4], in the context of the global community [5], of SARS-CoV-2 confirms polysystemic, with multi-inflammation of many other organs, affecting the lungs, heart, liver, pancreas, gastrointestinal tract, and others. And also about coronavirus neuroinvasion as a recurrent COVID-19 infection with meningitis without lung damage, reported by Maria Vasilieva, Irina Vasilieva, Stanislav Groppa & Ilie Vasiliev (2022), published by WHO, PubMed Central, Euro PubMed Central, International Congress of Clinical Neurophysiology Geneva, Switzerland, Harvard Library, USA, Hungary. Eötvös Loránd University (ELTE), and others [6]. Damages to the central nervous system, which we will write about in this book "Neuro COVID-19", where the leading role in the spread of multi-inflammation belongs to the Immunocompromising (IC) CHAOS - [C]ardiovascular Compromise: shock ; [H]omeostasis; [A]poptosis; [O]rgan Dysfunction; [S]uppression.Disorder immuno-inflammatory syndromes, Local Inflammatory Response Syndrome (LIRS), Systemic Inflammatory Response Syndrome (SIRS), and Compensatory Anti-inflammatory Response Syndrome (CARS) in turn generate another syndrome, with damage to the endothelial system - Acute Vascular Distress Syndrome (AVDS). Thus, coronavirus with IC CHAOS provokes AVDS, which are responsible for MIS, including the central nervous systems (CNS) and peripheral nervous systems (PNS). Cytokine Release Syndrome (cytokine storm) and SIRS associated with COVID-19 remain a leading mechanism in the development of MIS, including CNS and PNS associated with SARS-CoV-2 involving the endothelial system infection during the pandemic.

The Coronavirus genome is single-stranded, ~ 29,903 bp (β-coronavirus), positive polarity (+)ARN, capable of direct conjugation with ribosomes for translation coding of nucleoprotein, membrane protein, envelope protein, spike glycoprotein (SP), polymerase. Coronaviruses are characterized by glycoprotein S, the transmembrane proteins M (type III transmembrane glycoprotein) and E (the smallest structural protein in the viral particle with a length of 74–109 amino acids and a molecular weight of 8.4–10.9 kDa), and nucleoprotein combines with the viral RNA to form a viral ribonucleoprotein complex. Severe acute respiratory syndrome coronavirus type 2 (SARS-CoV-2) membrane protein is a glycosylated structural protein that localizes to the endoplasmic reticulum and Golgi and is essential for the production of viral particles. Membrane protein inhibits synthesis of type I and III Interferons, IFNs.

Coronaviruses cause mild to fatal respiratory infections in humans and birds. COVID-19, the disease caused by Severe acute respiratory syndrome SARS-CoV-2, has caused significant morbidity and mortality worldwide. SARS is a viral respiratory disease caused by a coronavirus associated with SARS-CoV-1, which was first identified in 2002 - 03 as the source of SARS. This was the 2002–04 SARS outbreak caused by SARS-CoV or SARS-CoV-1. They cause diarrhea in cows and pigs and hepatitis and encephalomyelitis in mice. Deadly variants can cause SARS, MERS, and COVID-19.

According to the World Health Organization (WHO), coronavirus infection (COVID-19, (COronaVIrus Disease 2019) is an infectious disease caused by the Severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) virus. SARS-CoV-2 virus, Severe Acute Respiratory Syndrome-related coronavirus 2. α-Coronavirus (HCoV-229E) was discovered in the mid-1960s, β-Coronavirus A (HCoV-OC43) in 1967, β-Coronavirus B (SARS-CoV-1) in 2002, and HCoV-NL63 in 2004. In 2015, Middle East respiratory syndrome was caused by β-Coronavirus C (MERS-CoV), and in 2019, the COVID-19 pandemic was caused by β-Coronavirus B (SARS-CoV-2) [1-11].

Role of Acute Vascular Distress Syndrome in the Development of Multisystem Inflammatory Syndrome in SARS-CoV-2.

The gateway of infection of the Coronavirus SARS-CoV-2 in the body is due to interaction with angiotensin-converting enzyme 2 receptors (ACE2) and Toll receptors (TLRs), activating the factor kappa B (NF-κB) of broncho-alveolar pneumocytes type II epithelial cells, vascular endothelial cells, and intestinal epithelial cells. Research [12] has shown that the SARS-CoV-2 nucleocapsid protein, rather than the spike protein, triggers lung epithelial A549 cells (adenocarcinomic human alveolar basal epithelial cells) to express a number of biologically active substances: IP-10 (C-X-C motif chemokine ligand 10 known as Interferon gamma-induced protein 10); RANTES (Regulated upon Activation, Normal T Cell Expressed and Presumably Secreted), IL-16 (Pro-inflammatory pleiotropic ”one gene influences two or more unrelated phenotypic traits” cytokine); MIP-1α (Macrophage Inflammatory Protein-1 Alpha); FGF (basic Fibroblast Growth Factor); Eotaxin, are a CC chemokine subfamily of eosinophil chemotactic proteins that attract eosinophils and have a direct relationship with inflammation; IL-15 (nflammatory cytokine with structural similarity to Interleukin-2); PDGF-BB (Platelet-Derived Growth Factor-BB human); TRAIL (tumour necrosis factor-related apoptosis-inducing ligand); VEGF-A (Vascular endothelial growth factor A) , and IL-5 (Interleukin produced by type-2 T helper cells and mast cells). IC CHAOS SARS-CoV-2 "Cytokine storm" develops Acute Respiratory Distress Syndrome and MIS due to the manifestation of Acute Vascular Distress Syndrome (AVDS). With Coronavirus SARS-CoV-2, the alveoli are perfused but not ventilated; blood passes through unventilated areas and is not oxygenated, as blood comes from the right to the left heart through arteriovenous shunts - an intrapulmonary right-to-left shunt. Thus, a case of AVDS associated with Covid-19 has been described [13]. Blood flow from right to left through the cardiac openings or in the pulmonary arteriovenous defect is considered right-to-left a pulmonary shunt leading to hypoxic non-oxygenated blood flowing from the lungs to the heart through the pulmonary veins. It is worth noting that extrapulmonary forms of SARS-CoV-2 coronavirus damage to other organs and systems and MIS manifestations have expanded the concept of AVDS. Since generated by vascular endothelial cells and distributed by the bloodstream, cytokine storm is an overreaction of the immune system IC CHAOS SARS-CoV-2 in which the protective function of cytokines reaches the harmful effect of attacking healthy tissue, including brain damage. After SIRS COVID, disruption of the blood-brain barrier permeability.

Thus, the essence of AVDS comes down to an inflammatory-immune disorder of the endothelial system. With a violation of numerous functions of endothelial cells, including transcapillary metabolism between the microcirculation and cells with the

development of MIS. The basis of which is confirmation respiratory distress (ARDS) and/or microcirculatory-mitochondrial distress (MMDS) syndromes.

SARS-CoV-2 proteins bind to mitochondrial proteins in infected cells and destroy the mitochondrial gene distributed along the mitochondrial DNA (mtDNA) within each mitochondrial. When studying animal models and determining the time of peak viral load in the lungs, it was found that genes responsible for mitochondrial function were suppressed in the cerebellum, although the SARS-CoV-2 virus itself was not detected in the brain. These data suggest that when viral titer first peaks, a systemic host response occurs, followed by viral suppression of mitochondrial gene transcription and induction of glycolysis, leading to the deployment of antiviral immune defenses. Even when the virus was eradicated and lung mitochondrial function was restored, mitochondrial function in the heart, kidney, liver, and lymph nodes remained disrupted, potentially leading to severe COVID-19 pathology [14]. It has been proven that microRNA 2392 (miR-2392), which regulates mitochondria, could be a potential target for therapy [15]. Dysfunction of mitochondria, cellular energy generators, with the development of energy deficiency establishes MMDS with dysfunction of many organs and the development of MIS. We consider the leading role of IC CHAOS dissonance accompanied by Acute Vascular Distress Syndrome, Microcirculatory-Mitochondrial Distress Syndrome, aggravated by Electro-Ion Membrane Distress Syndrome, accompanied by Abnormal/extreme myelopoiesis and generates MODS, creates extreme genomic, transcriptomic, proteomic, metabolic and phenomenal functional structural disorders myelopoiesis, are reduced, incl. lysosomal mitochondrial clearance of autophagy (mitophagy) [16-18].

COVID-19 Molecular markers. Testing for respiratory disease (coronavirus disease 2019 (COVID-19) and the related virus SARS-CoV-2 is possible by two main methods: molecular detection and serological testing. Molecular methods use polymerase chain reaction (PCR) along with nucleic acid tests and other advanced analytical methods to detect the genome of the virus. The Center for Disease Control (CDC) has developed real-time reverse transcription polymerase chain reactions for diagnostic purposes. Serological testing uses ELISA antibody test kits to detect the presence of immune system antibodies against the virus [19].

Standard laboratory examination for COVID-19 infection. Detailed general blood test. Coagulogram. D-dimer analysis, with COVID-19, the level of these proteins often increases, indicating the presence of blood clots in the lungs and other organs. Test for ferritin, this is a protein that transports iron in the body, necessary for red blood cells that transport oxygen. In those infected with COVID-19, the level of this protein in the body is very high, which indicates anemia, and transport is affected oxygen and red blood cell function. Analysis for C-reactive protein: this is a blood plasma protein that is very sensitive to inflammatory processes. With COVID-19, CRP acts as the main marker of the intensity of inflammation in the lung и other organs. Test for interleukin-6, an anti-inflammatory cytokine, the level of which increases during certain inflammations, including inflammation due to COVID-19. A strong increase in interleukin-6 causes a Cytokine Storm, an excessive reaction, manifesting itself as a complication, the degree of danger of which is much greater than the actual one coronavirus.

Irina Vasilieva (2024) describes it as a biomarker Neurofilament light chain (NF-L), representing a cytoplasmic protein highly expressed in large-caliber myelinated axons. For diagnostic purposes in Alzheimer's dementia, motor neuron disease, Parkinson's syndrome, diseases of small vessels of the brain, and intracellular neuronal disorders [20]. NF-L may also be a good biomarker for diseases with a neuronal component, such as some lysosomal storage diseases (the general name for a group of very rare inherited diseases caused by dysfunction of intracellular organelles of lysosomes) [21]. Irina Vasilieva [22] describes Biomarker Galectin-3 has a diagnostic role in cardiac conditions such as atrial fibrillation, myocardial infarction, cardiac insufficiency, congenital heart disease, and atherosclerosis. It is expressed in the kidney, blood vessels, and macrophages, and it is overexpressed in the heart. Activated macrophages secrete Galetctin-3, which is bound with the matrix. As a result, the heart is fibrosis. The SARS-CoV-2 spike (S) protein (as first reported for other β-coronaviruses) possesses a galectin fold within the N-terminal S1 subunit domain (S1-NTD) that exhibits structural homology identical to human galectin-3 (Gal-3). Gal-3 bound to epithelial cells promotes activation of innate immune cells, including basophils, dendritic cells (DCs), and monocytes [23]. Increased acute phase inflammatory protein A2AP, which is part of the plasmin-antiplasmin system, plays a key role in blood coagulation and fibrinolysis [24]. Hyaluronic acid (HA) has been associated with SARS-CoV-2 infection and is recommended as an accessible biomarker to predict the progression of SARS-CoV-2 infection and aid early clinical decision-making in the post-COVID-19 era. In addition, HA levels and the proportion of severe infections increased significantly in early COVID-19 infections [25].

In the COVID-19 Intensive Care Unit, critical patients are hospitalized only with the positive confirmed diagnostic test RT-PCR (Coronavirus) (RNA, qualitative) in the nasopharyngeal exudate were also examined for RPR, HIV, etc. The determination of severity of SARS-Cov2 / COVID / 19 was determined according to the scales: HScore (P. Mehta et al.), high CRP, ESR, ferritin, fibrinogen, D-dimer, lymphopenia, thrombocytopenia; Thromboembolic risk (PADUA); Disseminated intravascular coagulation, DIC (ISTH); level of Consciousness (FOUR) in intubated patients; Organ failure (SOFA); Liver failure (Child - Turcotte - Pugh). Also determined LDH ferments, Troponins, Creatinine phosphokinase, INR, Ionogram, Oxygenation Index SpO2/FiO2 ratio, pCO2 (AV gap), and so on [26].

IC CHAOS SARS-CoV-2 provoking Endothelial Disorders and Endotheliosis as predictors of Acute Vascular Distress Syndrome and the development of MIS confirmed and MMDS syndromes. The vascular endothelium is an active paracrine, endocrine, and autocrine organ essential for the regulation of vascular tone and maintenance of vascular homeostasis.5 Endothelial dysfunction is the main determinant of microvascular dysfunction, shifting the vascular equilibrium towards greater vasoconstriction with subsequent organ ischemia, inflammation with associated tissue edema, and a procoagulant state [26,27].

COVID-19, caused by SARS-CoV-2, is an MIS with multiinflammation, thrombosis, and endothelialitis. The conversion of angiotensin I to angiotensin 1-9 and angiotensin II to angiotensin 1-7 is catalyzed by Angiotensin-converting enzyme 2, a membrane protein exopeptidase, which is a zinc-containing metalloenzyme.

Consisting of 805 amino acids, molecular weight 92.5 kDa, containing 7 N-glycosylation sites. Separable producers are formed due to proteolytic cleavage by protease ADAM17, as well as serine proteases TMPRSS2, TMPRSS11D, and

HPN/TMPRSS1. Cellular receptor ACE2 is a gateway for the causative agent of severe acute respiratory syndrome, SARS-CoV. ACE2 is a type I transmembrane protein with an extracellular N-terminal domain containing the active site and a short intracellular C-terminal tail. Daniel W Lambert et al. (2005) showed that a soluble form of ACE2, lacking its cytosolic and transmembrane domains, has been shown to block the binding of the SARS-CoV spike protein to its receptor. The involvement of ADAM17 in the regulated ectodomain shedding of ACE2 has been proven that ACE2, heterologously expressed in HEK293 cells and endogenously expressed in Huh7 cells, undergoes metalloproteinase-mediated, phorbol ester-inducible ectodomain shedding. Confirmed ADAM17 as the protease responsible for ACE2 [28].

The development of MIS in SARS-CoV-2 is possible due to the fact that ACE2 is expressed in most tissues. The protein is primarily located on the membranes of type II pneumocytes, enterocytes of the small intestine, endothelial cells of arteries and veins, as well as smooth muscle cells in most organs. In addition, mRNA for ACE2 was found in cells of the cerebral cortex, striatum, hypothalamus, and brain stem. The presence of ACE2 in brain neurons and glia makes these cells susceptible to infection by the SARS-CoV-2 virus, which can lead to the loss of smell and the development of neurological deficits observed in COVID-19 disease. An immunocompromised neurological deficit also develops with other infections due to the spread of the infectious agent, cytokines, and other endogenous toxins in the bloodstream through the vessels with endothelial dysfunction [29,30].

In addition to respiratory symptoms, multiple extrapulmonary manifestations were observed, such as MIS, where endothelial dysfunction and immunothrombosis were found to be key pathogenetic mechanisms in COVID-19. Expressed (synthesized) in vascular endothelial cells, Piezo-type mechanosensitive ion channel component 1 (PIEZO1), a member of the piezo-ion channel family, is responsible for mechanical stimuli of blood flow and blood pressure. PIEZO1 has proven to be a dual physiological regulator and pathogenic factor in endothelial cells, modulating endothelial function and barrier integrity [31,32].

As a result, we arrived at the universal Immunocompromised mechanism (IC CHAOS) of endothelial dysfunction. Which is considered Acute Vascular Distress Syndrome, unable to liquidate Local Inflammatory Response Syndrome (LIRS), and manifested as a trigger for spreading and initiating SIRS, with immune paralyzed Compensatory Anti-Inflammatory Response Syndrome (CARS) [33-39].

The endothelium, with a total mass of ~1.5-2 kg with a monolayer length of endothelial cells of more than 7 km, is considered an active homeostatic metabolic organ. Participates in the localization of LIRS, local inflammation supported by CARS with the release of pro- and anti-inflammatory cytokines, preventing its generalization to SIRS, mediating immune processes. Influences capillary permeability by controlling colloid osmotic and hydrostatic pressure, preventing capillary leak syndrome. Maintains homeostasis by regulating the vasomotor function (vasomotion) of vascular tone (vasodilation/vasoconstriction) and hemostasis, the production and inactivation of fibrinolysis factors and platelets aggregation, as well as the synthesis and inhibition of vascular angiogenesis proliferation factors. One of the many producers produced by the endothelium is nitric oxide (NO), which reduces the adhesion of leukocytes to the endothelium and maintains its normal permeability. NO maintains and optimal perfusion pressure. Inhibits trans endothelial migration of monocytes, the proliferation of smooth muscle cells and collagen, inhibits adhesion and aggregation of platelets, activates tissue plasminogen activator, and serves as a powerful vasodilator and

antioxidant [40,41]. Endothelial dysfunction occurs under the name endotheliosis as a set of changes in the endothelium of blood vessels during hyperallergic reactions of the body in the form of swelling, proliferation, loosening, and desquamation. Endothelial dysfunction is considered a pathological condition of the endothelium, which is based on a violation of the synthesis of endothelial factors that are unable to ensure the hemorheological homeostasis of the blood, leading to dysfunction of organs and systems. We define the condition as Acute Vascular Distress Syndrome (AVDS).

Developing AVDS occurs due to IC CHAOS damage to endothelial cells [41]. Together with ACE2, for as the acceleration of SARS-CoV-2 cell entry, also play an important role, and by sialic acid, TMPRSS2, an inducer of extracellular matrix metalloproteinase (CD147), cathepsins B and L, expressed in endothelial cells [42].

Thus, SARS-CoV-2 hits all internal organs, including the pancreas [43], other systems, and the central nervous system, provoking MIS [1,15,44].

Mitochondrial Microcirculatory Distress Syndrome (MMDS) argues Acute Vascular Distress Syndrome (AVDS).

The most important component of AVDS is Mitochondrial and Microcirculatory Distress Syndrome (MMDS), which can occur during SIRS and is characterized by cytopathic tissue hypoxia uncorrected by oxygen transport optimization and associated with an acquired defect in the use of oxygen and energy production in mitochondria, leading to MODS [45]. The diagnosis of MMDS is based on a valid pCO2 marker of AV gap>6 mmHg. In tissue hypoxia, an increase in the valid pCO2 marker of AV gap>6 mmHg with the exclusion of acute respiratory distress syndrome confirms microcirculatory-mitochondrial distress syndrome as a mitochondrial collapse explaining the slowing/stopping of venous CO2 return from the periphery to the center, due to the disturbance and perfusion of the Systemic Perfusion Pressure (SPP), which is responsible for equilibrium macrocirculation-microcirculation. In MMDS, capillary leak syndrome indicates advanced AVDS with MODS [39,46]. This necessarily affects the membrane electrochemical potential (ΔμH) + and the mitochondrial membrane potential (ΔΨm) of an electrostorm or electro paralysis. Therefore, Immunocompromised dissonance (IC) CHAOS- [C]ardiovascular Compromise: shock; [H]homeostasis; [A]poptosis; [O]rgan dysfunction; [S] suppression of the immune system, mediates MODS and aggravates the disorder extreme/abnormal myelopoiesis and causes extreme genomic (G), transcriptomic (T), proteomic (P), metabolic (M) and phenomenal (F) functional and structural disorders. Optimal SPP in the absence of MMDS maintains cellular homeostasis, preventing Electro - ion membrane distress syndrome, and provides the membrane with electrochemistry, its bioelectromagnetic field, quantum energy and quantum electromagnetic radiation, signal transmission induced by the redox potential of mitochondria, DNA and RNA synthesis, enzymatic correction of base excision repair of damaged DNA and the expression of an anti-apoptotic gene that blocks the BAX protein (BCL-2 family), which protects the telomeres of DNA chromosomes. Immunotherapy with antibodies that block the PD-1 membrane receptors, as well as CTLA-4 and CAR-T cells, successfully restored damaged genes using nanoparticles for improved delivery of CRISPR-CAS9 in the treatment of cancer [47].

The constancy of ΔVP compliance of the brain is ensured according to the Monroe Kelly Doctrine, which is a balance between cerebral blood flow, cerebrospinal fluid, and the mass of the brain. Cerebral perfusion pressure not less than 100 mmHg,

designed to provide a metabolic rate in gray matter at 75 mL/100 g/min, in white at 30 mL/100 g/min, and an
average of 55 mL/100 g/min. In situations of falling blood flow up to 25 mL/100 g/min, there is a diffuse decrease in the electrical neural activity of the cerebral cortex. When the blood flow is ~15 mL/100 g/min, there is a slowdown/disappearance of the bioelectric nervous activity of the cerebral cortex <10 mL/100 g/min; irreversible, hypoxic, and ischemic cerebral lesions are observed stopped for 8-10 seconds the consciousness is lost [46,48].

Lysosomal mitochondrial clearance of autophagy. Mitophagy. Mitophagy: selective elimination of mitochondria.

Specific and nonspecific autophagy, a form of catabolism that occurs in autophagosomes, double-membrane vesicles that encapsulate non-functionally denatured, dying cellular components, amyloids splits and degrades them, thereby cleaning the intracellular environment. Supporting, thus, the intracellular homeostasis. Nonspecific autophagy appears when there is a deficiency of nutrients, that is, a lack of biologically active elements of food that determine the life support of the body. In such cases, autophagy breaks down its own intracellular biomaterial to provide nutrients. Specific or targeted autophagy lyses defective cellular organelles, denatured proteins, and accumulated toxic products, purifying the intracellular ecological environment and maintaining intracellular homeostasis. The role of autophagy as a cell-autonomous system for the removal of molecular patterns (molecular structures) of pathogens, toxins, and cellular stress products is highlighted. By protecting cells, autophagy stimulates cell survival. Autophagy appears to be a natural regeneration process that occurs at the cellular level. David Rubinstein discovered that autophagy protects against Parkinson's disease, Huntington's disease, and some forms of dementia and helps fight infection and inflammation. At the same time, cell necrosis is considered damage to an entire cell, which leads to premature death in living tissue through autolysis. In the context of which, it is necessary to describe the function of apoptosis, which genetically pursues the elimination of useless cells from the body with minimal damage to the ecosystem while maintaining adequately functioning surrounding tissues. There is also necroptosis, a programmed form of necrosis or inflammatory cell death. Inhibition of necroptosis and, in particular, Receptor-interacting serine/threonine-protein kinase 1 (RIPK1 kinase) activity reduces inflammation and leads to a significant increase in lifespan [49,50]. The IC CHAOS dissonance accompanied by AVDS, MMDS aggravates Electro-Ion Membrane Distress Syndrome, accompanied by Abnormal/extreme myelopoiesis and generates MODS, creating extreme genomic, transcriptomic, proteomic, metabolic, and phenomenal functional-structural disorders myelopoiesis, are reduced, incl. lysosomal mitochondrial clearance of autophagy (mitophagy) [46,51,52].

By screening known STING suppressors and proteins encoded by SARS-CoV-2, ORF3a was shown to be a potent inhibitor of STING-mediated autophagy, which facilitates viral replication. Bat-CoV ORF3a suppresses bat STING-induced autophagy, which can be neutralized by the small molecule inhibitor TPEN. Among human Coronaviruses, only SARS-CoV and SARS-CoV-2 encode ORF3a [53].

Mitophagy originated during endosymbiosis of the αproteobacterial ancestor of mitochondria. One type of specifically targeted autophagy is mitophagy, which selectively eliminates failed mitochondria that cannot cope with the role of "cell power plants" and energy production. As a result, mitophagy removes these mitochondria, allowing the regeneration of mitochondria capable of maintaining the role of energy and electrical supply to the cell. PTEN kinase-induced kinase 1 (PINK1) and Parkin E3 ubiquitin ligase support mitophagy. PINK1 and Parkin work together to identify and mark defective mitochondria for elimination while maintaining functioning mitochondria. PINK1 (PTEN-induced kinase 1) is a protein-coding gene. Associated pathways include gene expression (transcription) and selective autophagy. Parkinson's disease and other neurodegenerative processes are associated with PINK1 mutations.

Mitophagy is also important in severe infections. Genes determine autophagy. Mitophagy is supported by more than 25 ATG genes associated with autophagy in correlating mitophagy in various conditions of infectious and non-infectious (neurodegenerative processes) genesis. Thus, autophagic removal of damaged mitochondria is a conserved cellular process to maintain a healthy mitochondrion called Mitophagy [54].

Disruption of mitochondrial Ca+2 homeostasis. Inflammatory during SARS-CoV-2 infection is dependent on Ca2+ level and Damage the open-close function of the Mitochondrial Permeability Transition Pore (mPTP), the Mitochondrial Calcium Uniporter (MCU), and Voltage-dependent anion channels (VDAC)

Mitochondrial Ca 2+ uptake controls intraorganellar and cytosolic functions. In the mitochondria, increased Ca 2+ regulates the activity of tricarboxylic acid cycle enzymes, supporting energy-oxidative metabolism and ATP production. At the same time, reactive oxygen species are formed as by-products of oxygen consumption. In this case, mitochondria act as buffers for the increase in cytosolic Ca 2+, thereby regulating Ca 2+ -dependent cellular processes. In pathological conditions, mitochondrial Ca 2+ overload causes the opening of the mitochondrial permeability transition pore (mPTP) and the release of apoptotic cofactors. Mitochondrial Ca 2+ uptake occurs in response to a local increase in Ca 2+ between the endoplasmic reticulum and mitochondria and is mediated by the mitochondrial Ca 2+ uniporter (MCU), a highly selective inner mitochondrial membrane channel. Both channel and regulatory subunits form the MCU complex (MCUC). The MCUC is composed of pore-forming subunits, including MCU, the MCU-dominant negative b (MCUb), the essential MCU regulator (EMRE), and regulatory subunits including Mitochondrial Calcium Uptake 1 (MICU1), MICU2, MICU3 and MCU regulator 1 (MCUR1) [55-57].
Ion channels are pore-forming proteins (single or entire complexes) that maintain the potential difference that exists between the outer and inner sides of the cell membrane of all living cells. They belong to transport proteins. With their help, ions move according to their electrochemical gradients through the membrane. Such complexes are a collection of identical or homologous proteins tightly packed in the lipid bilayer of the membrane around an aqueous pore. The channels are located in the plasmalemma and some internal cell membranes. The ions passing through ion channels are Na+, K+, Cl−, and Ca2+. Due to the opening and closing of ion channels, the concentration of ions on different sides of the membrane changes, and a shift in the membrane potential

occurs. We have described electro-ion membrane distress syndrome. Primary/secondary morphofunctional damage to pores/channels of biomembranes provokes broken to ion-electro generation, transduction, and transmission of the membrane electric potential (MEP). The biomagnetic field and quantum energy/quantum electromagnetic radiation are also destabilized as a connecting signal of transmission and amplification pathochemical reactions of a pathophysiological cell. We consider this syndrome as electro-ion membrane distress syndrome (EIMDS), whose disorder generates with chronic fatigue syndrome/myalgic encephalomyelitis (CFS/ME). We also called this syndrome Maria & Irina Vasilieva, as a switch that destabilizes the homeostasis of intracellular and extracellular media due to membrane distress. IC CHAOS dissonance creates a disorder of electro-storm/electroparalysis of MEP due to cytokines whose disorder install EIMDs and CFS/ME, with diagnostic marker is less deformed red blood cells with a compacted “hard” cell membrane, by determining the permeability of the erythrocyte membrane and the sorption capacity of red blood cells. Standard therapy in surviving critical patients with IC CHAOS dissonance did not provide a stable decrease in CFS/ME, the effect of which was observed after MOST-ELSO and due to cryo-bio-xeno myelo-timo-spleeno perfusion. This success of cryotherapy expressed ↓toxic oxygen and nitrogen and heavy water effect of compression and reduction of “synairesis” of proteins, separation of liquid from the gel caused by a reduction in protein due to the release of water from the membrane and the release from of cell membranes and cells deuterium D/^{2}H, “heavy water” which inhibits some cleavage reactions [58].

Blocking the calcium channels may be a suitable treatment option for SARS-CoV-2 since Ca2+ is required to enhance the fusion process of SARS-CoV-2. Inflammatory during SARS-CoV-2 infection is dependent on Ca2+ level. The process is determined by the opening/closing of the mitochondrial permeability transition pore-dependent Ca++uniporter (mPT pore), which we have described sufficiently in the Universal Medical Journal [59,60].

Exposure to the spike protein or receptor binding domain (S-RBD) of SARS-CoV-2 significantly affects endothelial cells and induces pulmonary vascular endotheliopathy. Blockage of the store-operated calcium channel (SOCC) and stretching of the right atrium against the background of hypoxia/reoxygenation develops ventricular fibrillation. SOCC is activated in response to the depletion of the calcium store - the endoplasmic reticulum. The SARS-CoV-2 spike protein receptor binding domain disrupts intracellular calcium homeostasis and damages pulmonary vascular endothelial cells. S-RBD leads to an acute or prolonged induction of intracellular free calcium concentration ([Ca 2+] through acute activation of the TRPV4 gene (Transient receptor potential cation channel subfamily V member 4 is an ion channel protein that in humans is encoded by the TRPV4) and long-term activation of the mechanosensitive channel Piezo1 and the store-operated calcium channel (SOCC). By mechanism, S-RBD interacts with ACE2, inducing the formation of clusters including Orai1 (Calcium release-activating protein 1 is a calcium-selective ion channel encoded by the ORAI1 gene in humans).), Piezo1 and TRPC1 (Transient receptor potential cation channel subfamily C member 1), facilitating activation of Piezo1 and SOCC channels and leading to increased apoptosis. These effects are blocked by cobophenol A, which inhibits the binding between S-RBD and ACE2 or the intracellular calcium chelator BAPTA. -AM (BAPTA - AM is a cell-permeant chelator, which is highly selective for

Ca2+). Blockade of Piezo1 and SOCC by GsMTx4 effectively protects S-RBD-induced pulmonary microvascular endothelial damage in hACE2 Tg transgenic mice by normalizing elevated [Ca 2+]. Transcriptomic analysis shows that the prototype S-RBD has more severe acute effects than Delta or Lambda S-RBD. The study provides compelling evidence that S-RBD can cause persistent damage to pulmonary vascular endothelium by binding to ACE2 and triggering [Ca 2+] through activation of Piezo1 and Orai1. Targeted Inhibition of the ACE2-Piezo1/SOCC-[Ca 2+] Axis Proves a Potent Strategy for the Treatment of S-RBD-Induced Pulmonary Vascular Diseases [61].

The PIEZO1 gene is also related to calcium channels. Greek piesi - “pressure” (piezo type mechanosensitive ion channel component 1), a homolog of the PIEZO2 gene, encodes a mechanosensitive ion channel, which is responsible for the response to mechanical damage. The PIEZO1 and PIEZO2 genes regulate blood pressure, respiration, and bladder activity. PIEZO1 is a mechanosensitive, nonselective cation channel that regulates the flux of calcium ions across the membrane. The activity of PIEZO1 is regulated by phosphatidylserine through its inhibition. The PIEZO1 gene contains 51 exons. The PIEZO1 ion channel protein has 36 transmembrane domains and is a homotetramer. PIEZO2 is also an ion channel. For the discovery of PIEZO1, PIEZO2, and other genes responsible for the response to cold and heat, the Nobel Prize in Physiology or Medicine was awarded to research teams from the USA led by Ardem Patapoutian) and David J. Julius (2021) [62].

Voltage-dependent anion channels (VDAC) are pore-forming proteins (approximately 30 kDa) associated with mitochondria, membrane-bound organelles found in almost all eukaryotes (cells that have a membrane-bound nucleus). Mitochondria, commonly referred to as the "energy generators of cells", are composed of binary membranes: the outer mitochondrial membrane (OMM) and the inner mitochondrial membrane (IMM). Although the latter is essentially impermeable to any hydrophilic solutions, the OMM is made "porous" by VDACs, which are integral membrane proteins that promote the free diffusion of molecules of approximately 5 kDa or less. "Pores" in OMM have been renamed VDAC, mainly due to their electrophysiological properties when reconstituted in vitro in phospholipid bilayers or liposomal vesicles [63]. The VDAC family of proteins consists of three isoforms from three separate genes (VDAC1, VDAC2, and VDAC3).

The mitochondrial calcium uniporter (MCU) is a conserved Ca 2+ transporter in the mitochondria of eukaryotic cells. Ectopically expressed MCU is localized in HeLa and primary cerebellar granule neurons (CGN). MCU has been shown to interact with VDAC1 and mediate VDAC1 overexpression-induced cell death in the CGN. This finding demonstrates that the MCU-VDAC1 complex regulates mitochondrial Ca 2+ uptake and oxidative stress-induced apoptosis, which may represent therapeutic targets for oxidative stress-related diseases [64].

Oxidative stress, Ca 2+ overload, hypoxia, and cytotoxic agents activate the permeability transition of the inner mitochondrial membrane of mitochondria; that is, they stimulate the Mitochondrial Permeability Transition Pore (mPTP) [65]. The mPTP opening level does not allow the passage of proteins, solutes, and metabolites up to 1.5 kDa in size, which freely pass through the normally impermeable inner membrane.

Water penetrates the mitochondrial matrix along an osmotic gradient, causing mitochondria to swell with mitochondrial rupture. mPTP plays a critical role in cell death, especially necrosis [66], and is involved in the development and progression of many diseases, including ischemia/reperfusion injury, muscular dystrophy, Alzheimer's disease, and cardiotoxicity. Mario Zoratti suggested that the electrical conductivity properties of VDAC are similar to those described for mPTP [67]. On the other hand, the GST-CypD fusion protein destroys VDAC in collaboration with adenine nucleotide translocase (ANT) as another component of mPTP in mitochondrial lysates. Restoration of this VDAC-ANT-CypD complex led to the formation of a Ca 2+ - dependent, cyclosporine-sensitive channel reminiscent of mPTP [68]. Researchers have not come to the final result of cell necrosis provoked by the predominance of mPTP or VDAC. Mitochondrially produced ROS are involved in cell death and promote the accumulation of Ca 2+ in mitochondria [69]. ROS generated by mitochondria induces the release of cytochrome c, which is inhibited by VDAC blockers or anti-VDAC antibodies [70]. Cytochrome c is a small heme-containing protein belonging to the class of cytochromes contained in the sequential heme type c. It performs two functions: it is a single-electron carrier that freely connects to the inner mitochondrial membrane and the front chain component.

Moreover, mitochondria lacking all VDACs with enhanced MPT responses did not demonstrate a death-promoting role for VDACs but instead a pro-survival role. [71]. In confirmation, mitochondria and cells lacking VDAC enhance the MPT response and death [72], indicating that VDAC protects against mPTP rather than promotes it.

Ca2+ uptake via MCU triggers inner mitochondrial membrane, mPTP opening to induce outer mitochondrial membrane VDAC oligomerization. The non-selective calcium channel of the mitochondrial permeability transition pore (mPTP) - is a calcium-dependent, ion non-selective membrane pore with a wide range of functions. Mitochondrial Ca 2+ uniporter (MCU) is a transmembrane protein that allows the passage of calcium ions from a cell's cytosol into mitochondria. Anion-selective channel, Voltage-dependent anion channels (VDAC) is the most abundant protein in the outer membrane of eukaryotic mitochondria, is a protein weighing ~30 kDa, containing a channel with an internal diameter of ~3 nm, which allows the passage of molecules up to ~5 kDa. Optimization of the functions of MCU, mPTP, and VDAC in case of disruption of the pore opening-closing regime in order to avoid disruption of mitochondrial cellular calcium homeostasis with massive cell necrosis caused by the cytokine storm IC CHAOS COVID infection with pronounced MMD syndrome, carried recruitment of microcirculatory-mitochondrial [73].

Multi-organ Supportive Therapy (MOST) Extracorporeal Life Support Organization (ELSO) extracorporeal oxygenation ECMO and CO2 elimination by type ECCO2R.

Treatment of patients with SARS-Cov2 / COVID-19 was carried out in accordance with National Protocols, World Health Organization with the manifestation of MODS was guided by the “Surviving Sepsis COVID-19” using (ECMO) MOST-ELSO [74]. Mechanical ventilation with alveolar recruitment **[**35,73**]**. Initial treatment protocol for cardiogenic shock in patients with fulminant myocarditis: inotropes; vasopressors; artificial ventilation; artificial circulation support with extracorporeal membrane oxygenation; ventricular assist support; intra-aortic balloon pump [75]. Antiviral

treatment: RNA-dependent RNA polymerase inhibition (Remdesivir); Inhibition of coronavirus fusion with the cell membrane by altering the pH of the cell membrane surface (Hydroxychloroquine); Protease inhibitors (Lopinavir / Ritonavir). Systemic Corticosteroids as Anti-Inflammatories (Dexamethasone) [26]. Glucocorticoids, including dexamethasone, suppress the expression of inflammatory mediators and prevent neutrophil activation [76]. Hydrocortisone stabilizes the endothelial glycocalyx by inhibiting TNF-α and maintains the physiological barrier to endothelial permeability despite inflammatory processes [77]. Immuno modulators (Tocilizumab - Selective IL-6 Receptor Antagonist; Anakira - Selective IL-1 Receptor Antagonist). Other authors have also used immunomodulators: Siltuximab, Baricitinib, and Sarilumab [26]. Complement System Therapy. Eculizumab, a humanized monoclonal antibody targeting C5. Ravulizumab is a recombinant humanized anti-C5 monoclonal antibody. IFX-1 blocks the effect of C5a without interfering with C5b's function and keeping the membrane attack complex whole. Avdoralimab is an IgG1-kappa anti-C5aR1 blocking antibody. The MASP-2 inhibitor narsoplimab (OMS721) is applied to disturb the interaction between MASP-2 and the N-protein of SARS-CoV-2 [78]. C-reactive protein (CRP) disposes of the bacteria and host cells undergoing apoptosis or necrosis as the complement components of C1q-C4's work [79]. As high CRP levels are along with COVID-19, lowering CRP levels by therapeutic apheresis potentially reduces the pathological progression in the early stage [80]. The vaccines are now available, and issues such as declining efficacy against different SARS-CoV-2 variants and aging of vaccine-induced immunity highlight the importance of finding more antiviral drugs as a second line of defense against the disease. Drug repurposing has been used to rapidly find COVID-19 therapeutic options [81].

Medications, including Nirmatrelvir during the acute phase of COVID-19 and Metformin, are being evaluated and show promise for their ability to reduce the risk of adverse health outcomes associated with long-term COVID-19. The use of a drug repurposing strategy should be expanded to discover additional anti-COVID-19 drugs, especially since the development of resistance should be anticipated [82,83]. Stem Cell Therapy. Mesenchymal stem cells suppress the release of proinflammatory cytokines such as IL-6, IL-12, IL-1α, TNF-α, and IFN-γ, thereby reducing the frequency of cytokine storms [84]. Mesenchymal stem cells suppress and also secrete vascular endothelial growth factor and keratinocyte growth factor to relieve ARDS and regenerate injured lung tissues [85]. We also applied Convalescent plasma as an anti-COVID19 antibody carrier and active in IL-6 neutralization, with donor testing, including the absence of anti-HLA to prevent the risk of TRALI (Transfusion Related Acute Lung Injury) [26]. HNF (Unfractionated Heparin) Injectable Anticoagulant / HGMM (low molecular weight Heparin: Enoxaparin, Dalteparin, Nadroparin) in the treatment of Coagulopathies and for the prophylaxis of Venous Thromboembolism, Deep Venous Thrombosis, Pulmonary Embolism, DIC. We did not apply oral anticoagulants to intensive care patients, which are recommended only to outpatients Apixaban Rivaroxaban. [26]. Heparin/low molecular weight heparins neutralize chemokines and cytokines, reducing cytokine storm to protect the glycocalyx, inhibit heparanase activity from maintaining glycocalyx thickness, and reduce mortality in COVID-19 infection [86]. Antibiotics according to the de-escalation antibiotic therapy method for mono or multi-inflammatory syndrome. Enteral and parenteral nutrition by applying Glucose, Amino Acids and Intralipid.

Critically ill patients developing severe forms of multiple organ dysfunction syndrome (MODS) may not be adequately supported by pharmacologic management.

In these complex cases, a single form of extracorporeal organ support (ECOS) may be required, but multiple organ support therapy (MOST) is currently seen as a feasible approach. Severe renal dysfunction is a typical syndrome requiring renal replacement therapy (RRT) in the context of MODS. After more than a decade of RRT application in various intensive care settings, ECOS is no longer seen as an extraordinary or particularly aggressive techniques in MODS patients. Nowadays, a significant increase in the use of extracorporeal membrane oxygenation and extracorporeal carbon dioxide removal is occurring. [87]. With MODS, according to EVLWI, volemic resuscitation, hemostatic resuscitation in context Resuscitation of coagulation-anticoagulant-fibrinolytic balance is carried out. Back in 2007, we noted the successful stabilization of the "metabolic stage" of Coagulation Resuscitation, while we are still far from the methods of "no fluid resuscitation" or "low-volume resuscitation" [73,88].

With MODS, an increase in ↑ pCO2, caused by pulmonary/ extrapulmonary Acute Respiratory Distress Syndrome, ARDS [89-92] and confirmed by the fall in the oxygenation index ↓ PaO2/FiO2 ↓ 300 in the context of the Berlin 2012 classification violations of pathologies of gas exchange are also taken into account: 1) Lung gas exchange, a) Acute respiratory failure FetCO2 ↓, SaO2 ↓, PaO2 ↓, FiO2 ↓, b) Parenchymal (endothelial-epithelial damage to alveolar and vascular tissue) FetCO2↓ / or normal, SaO2 ↓, PaO2 ↓. 2) Transportation of gas in the blood (minute volume) ↓, Hb ↓, SvO2 ↓, PvO2 ↓, avSO2, avPO2. 3) Gas exchange in tissues SvO2 ↑, BE ↑, PvO2 ↑, avSO2 ↓, avPO2 ↓ lactate/pyruvate ↑. At the same time the pressure/volume loop of the trachea is also considered which are presented in 4 types (cucumber, pod, pear, tomato) which means that the more the loop surface is expanded the more the respiratory pattern as well as the definition of the dynamic (Cdyn) and statistical (Cst) compliance confirming damage to the respiratory organs aggravating Mitochondrial Collapse Microcirculatory Mitochondrial Distress Syndrome and Recruitment of Microcirculatory-Mitochondrial in such cases are supplimented with MOST therapy in the EXTRACORPOREAL Life Support Organization (ELSO) with active detoxification methods:1) Alveolar recruitment with respiratory support in special modes of ventilation mainly APRV with permissive hypercapnia at normal pH, 2) Recruitment of Microcirculatory-Mitochondrial (RMM), 3) MOST Extracorporeal Life Support Organization (ELSO) extracorporeal oxygenation ECMO and CO2 elimination by type ECCO2R [74], 4) Active detoxification methods intra and extracorporeal electrochemical ultraviolet (laser) photomodulation of auto blood ultra diafiltration continuous intermittent filtering, hemodialysis, bioimmunoactivation and biodetoxification through the use of extracorporeal bio xenoperfusion (myelo-timo-spleen), enterosorption, volnerosorption, plasma sorption, plasma exchange, lympho sorption, liquoro sorption, peritoneal dialysis, oxygenation of the liver through the bougienized umbilical vein, hypothermia and others [33,35-38,93-96,107] 5) Modeling of the index of extravascular pulmonary fluid (EVLWI). If EVLWI is <10 mL/kg this indicates alveolar atelectasis, which requires volemic resuscitation, bronchoscopy, alveolar recruitment, and surfactant therapy. In situations where EVLWI is>10 mL/kg, which is a threat to pulmonary edema which requires a reduction in volemic resuscitation and the inclusion of diuretics ultrafiltration and MOST-ELSO, inotropic therapy and invasive monitoring, 6) Th4-Th5 thoracic epidural block. The level of catheterization of the epidural space should be Th4 -Th5 (thoracic epidural block) in hyper-eu-kinetic patients, especially with hypertension and hypervolemia (EVLWI>10 mL/kg), but without hypocoagulation coagulopathy. Epidural analgesia at the level of

the chest is favorable because it expands spastic coronary arterioles (cardio-coronary dilatation), increases the delivery of O2 to the myocardium, reduces myocardial oxygen consumption, reduces the risk of myocardial infarction and ischemia, improves lung function and contributes to the functioning of lung gas exchange; reduces pulmonary hypertension, accelerates intestinal motility, promotes bowel movement and conforms to the multimodal analgesia protocol.

Thus, decentralization, anti-shock therapy, detoxification and analgesia in the recruitment Microcirculatory - Mitochondrial strategy, supplemented by MOST-ELSO (ECMO and CO2 elimination ECCO2R, etc.) and in combination with antibacterial/antifungal [97] / antiviral treatment and surgical correction [98], counteracts the Microcirculatory - Mitochondrial distress syndrome mitochondrial collapse, and regression Acute Vascular Distress Syndrome and MODS [99-101]. Comprehensive life support and treatment capable of preventing organ transplantation and especially lung transplantation in advanced forms of SARS-Cov2 [102]. Respiratory cardiac using markers, including galectin-3 in heart disease [50] and cerebral support [103-121], has justified itself not only in critical-terminal obstetrics bleeding [122] but also with massive injuries and in oncological MODS and even with coronavirus infection SARS-Cov2 / COVID19, where Acute Vascular Distress Syndrome is significant for the development of MIS. In the prevention of the SARS-Cov2 / COVID-19 pandemic, vaccination plays a special role [123] based on quality management [124,125]. On the one hand, the benefits of SARS-CoV-2 spike mRNA vaccines are well known, including a significant decline in COVID-19 morbidity and a decrease in the mortality rate of SARS-CoV-2 infected persons. On the other hand, pharmacovigilance studies have revealed the existence of rare cases of cardiovascular complications after mass vaccination using such formulations as what we consider how Acute Vascular Distress Syndrome [126], which coincides with other data. The effect of SARS-CoV-2 Infection and BNT162b2 Vaccination on the mRNA Expression of Genes Associated with Angiogenesis contributes to the development of adverse cardiovascular effects [127].

Although nowadays the danger of acute SARS-CoV-2 infection has been mitigated by less aggressive viral variants and by mass vaccinations, adequate serum 25(OH)D3 concentrations in COVID-19 patients could be protective against systemic complications, including acute and chronic neurological manifestations (long-COVID). Calcitriol and hydroxyderivatives of L3 and T3 show interesting neuroimmunoendocrine effects in the course of SARS-CoV-2 infection and can play an adjuvant role in neuroprotection, reducing blood-brain barrier (BBB) endothelial damage, antagonizing vascular immunothrombosis, and downregulating neuroinflammation. From the multiple neurovegetative correction (NVC) schemes of cerebral insufficiency (CI) of various causes (traumas, onco, stroke, neuro infections, in this case, COVID-19 SARS-CoV-2, metabolic, hypoxic, etc.), ongoing 41 years, retrospectively, arranged the pattern, with three essential syndromes manifestation of CI [128] with neurostimulation [129].

The anti-inflammatory, antioxidant, and cytoprotective effects of melatonin on viral infections have been studied in hyperinflammation, cytokine storm, and oxidation associated with COVID-19. Encouraging results, including the virucidal effect of melatonin and increasing the effectiveness of vaccines against SARS-CoV-2, have been published [130]. In addition, melatonin also had a positive effect on sleep quality in adults with respiratory and metabolic diseases and primary sleep disorders.

COVID-19 is associated with psychotic disorders in a variety of ways, including hyperinflammation, neurotoxicity, and psychological stressors [131]. The etiology of psychosis associated with COVID-19 is caused by hyperinflammation and activation of the immune system [132-137].
Brain damage in COVID is primarily neurological, as a material signaling pathway of inflammatory COVID-19 infection of cerebral vessels. This is important, from the release of these patients from additional stress, suffering, stigmatization in society, their isolation, intimidation, coercion (bullying) with violation and restriction of their rights. Since, concomitantly with inflammatory lesions of many organs, cognitive deficits are a component of the post-acute consequences of COVID-19 (PASC), where the role of the kynurenine pathway is significant with temporary cognitive impairment [41,138]. The main pathway for the breakdown of tryptophan is an amino acid, without which the synthesis of serotonin is impossible, which controls the cycles of sleep and wakefulness, which is the precursor of the sleep hormone melatonin.

SARS-CoV-2 uses the SARS-CoV receptor ACE2 for entry and the serine protease TMPRSS2 for S protein priming. A TMPRSS2 inhibitor approved for clinical use blocks entry and may represent a treatment option. Sera from convalescent SARS patients cross-neutralize entry caused by SARS-2-S. The serine protease inhibitor camostat mesylate, active against TMPRSS2, partially blocked SARS-2-S-driven entry into Caco-2, Vero-TMPRSS2, and Vero-TMPRSS2 cells [139,140]. At the same time, hydroxychloroquine-mediated inhibition of SARS-CoV-2 entry is attenuated by TMPRSS2 [141].
Due to the fact that ACE2 and TMPRSS2 are regulated by androgens and are involved in the entry of the virus into the cell infected with Coronavirus, antiandrogens, antigonadotropins, or 5-alpha reductase inhibitors (5-ARI) are considered possible treatments for SARS-CoV-2 [142]. Other proteases, such as cathepsin L and elastase expressed by neutrophils (ELANE), have the ability to trigger S protein and promote SARS-CoV-2 infection. The association of single nucleotide variants (SNVs) in ELANE with COVID-19 and biochemical markers was investigated. Genotyping of ELANE rs17216663C/T (Pro257Leu), rs17223045C/T (As1n30Asn), and rs3761007G/A was performed using a 5′ nuclease allelic discrimination assay (TaqMan assay). The results show that ELANE rs17223045C/T and rs3761007G/A provide protection against COVID-19 [143].
In theory, Fc-fusion decoy receptors could be used against any infectious disease once their receptors are identified. However, Fc fusion proteins may produce anti-drug antibodies, which may compromise safety and therapeutic efficacy [144]. A risk would be the development of immunological cross-reactivity against endogenous ACE2 or purified recombinant dipeptidyl peptidase 4 enzyme (DPP4) following treatment with recombinant Fc-fusion receptor decoys. To avoid such effects, the decoy receptor fusing ACE2 with immunoglobin Fc domain (ACE2-Fc) and DPP4 could be further improved by including only the protein fragment that interacts with the RBD of SARS-CoV-2 and MERS-CoV. [145]
Therapeutic interventions aimed at reducing neutrophil serine protease (NSP) activity may interfere with viral clearance and inflammation in patients with COVID-19 [146]. Chronic helminth infections may reduce the COVID-19 severity by reducing the SARS-CoV-2 entry points at ACE2(Angiotensin-converting enzyme 2)/DPP4 (is a serine exopeptidase that cleaves X-proline or X-alanine dipeptides from the N-terminus

of polypeptides)/CD147 (Basigin (BSG) also known as extracellular matrix metalloproteinase inducer (EMMPRIN) or cluster of differentiation 147) axis in the initial phase and immunomodulation in the late phase of the disease by suppressing TLR4/NF-kB signaling pathway [147].

The cytokine removal is done by means of two resin membranes (HA330 and Mediasorb). Researchers explored the importance of cytokine removal by means of two resin membranes (HA330 and Mediasorb) in COVID-19 patients treated in ICUs. Although considering from a pathophysiological basis, the possibility of utilizing cytokine adsorption techniques to modulate the immune response in critically ill COVID-19 patients is achievable. It is too early to assert that the result is good [148]. The CytoSorb is a hemoadsorption column designed to eliminate inflammatory mediators from the circulation [149].

The artificial liver support system and blood purification. The artificial liver support system and blood purification can rapidly remove inflammatory mediators, eliminate cytokine storms, and prevent shock, hypoxemia, and respiratory distress syndrome. Artificial liver technology wipes out inflammatory factors on a large scale. Based on the previous use of artificial liver technology on H7N9 bird flu, it is now being used in COVID-19, with progress. Artificial liver technology wipes out inflammatory factors on a large scale. Based on the previous use of artificial liver technology on H7N9 bird flu, it is being used in COVID-19 with progress now [150-151].

Exhaled Low doses of NO (10 ppm) are useful as adjunctive therapy to enhance the efficacy of antibiotics. Exhaled NO is strongly related to the type-2 inflammatory response found in asthma, which has been suggested to be protective against SARS-CoV-2 infection. Low doses of NO (10 ppm) are useful as adjunctive therapy to enhance the efficacy of antibiotics; the use of inhaled NO has been an effective therapy during this pandemic since the ventilation-perfusion ratio in COVID-19 patients improved subsequently, and they did not require mechanical ventilation. It has been beneficial to use a low dose of NO (10 ppm) as adjunctive therapy to enhance the efficacy of antibiotics used to treat acute P. aeruginosa exacerbations in cystic fibrosis patients. At the same time, the authors are looking for the question if the endogenous NO (in ppb) is rapidly metabolized by ROS in an extreme environment generated by the virus, what degree of danger, if any, exists with the inhalation of NO (in ppm) [152].

Monocarboxylate transport pumps lactate and H+ ions simultaneously from the extracellular area to the cytosol to lower the elevated lactate level. Monocarboxylate transport (MCT) pumps lactate and H+ ions simultaneously from the extracellular area to the cytosol to lower the elevated lactate level. Three important structures maintain cell pH. These ion regulators are lactate/H+ ion symporter (also called monocarboxylate transporters), Na+/H+ exchanger (NHE), and Cl−/HCO3− exchangers. NHE becomes active as a reflex due to the increase of H+ ions in the cell. After the activation of NHE, Na+ and Ca+2 are introduced into the cell, while H+ ion is pumped out of the cell. As this reaction continues, the cell continues to swell and lose its functions, and eventually dies [41, 153,154].

In case of complicated forms of COVID-19 SARS-CoV-2 in the form of irreversible respiratory failure, lung transplantation is performed. Indications for corticosteroids are considered [155], which is a severe SARS-CoV-2 infection leading to acute respiratory distress syndrome, with indications for oxygen therapy, mechanical ventilation, and ECMO. In the case of complicated forms of COVID-19 SARS-CoV-2

in the form of irreversible respiratory failure and the ineffectiveness of artificial ventilation, lung transplantation is performed [95,102].

Fractal Signatures in the Dynamics of an Epidemiology. An Analysis of COVID-19 Transmission. Integrated Perspective of Fractal Time Series Analysis for Infected Cases of COVID-19 (Geometrically - Mathematical Research Model COVID-19).

The recent Covid-19 pandemic threw the world into complete chaos with its rapid and devastating spread. Scientists are still trying to obtain a better understanding of the patterns of COVID-19 and a deeper understanding of mutant strains and their pathogenicity by performing genomic sequencing of more samples. Fractal-based analysis provides its unique forecasting policy to reduce the spread of COVID-19 and, in general, any outbreaks. The book presents fractal and multifractal models of COVID-19 and reviews the impact of the pandemic, including epidemiology, genome organization, transmission cycle, and control strategies based on mathematical models for developing an immune intervention. Also, it covers non-clinical aspects such as economic development with graphical illustrations, meeting the needs of onlookers outside the sector who desire additional information on the epidemic. The fractal signatures describe the fractal textures in the patterns of Coronavirus. Studies on the epidemiology of COVID-19 in relation to the fractals and fractal functions serve to exhibit its irregular, chaotic nature. Moreover, the book, with its wide coverage of the Hurst exponent analysis and the fractal dimension estimation, greatly aids in measuring the epidemiology [156,157].

The virus's attack on humanity met with heroic resistance from medical personnel around the world, life-saving countless SARS-CoV-2 infected people and H3N2 Influenza.

The SARS-CoV-2 virus causing coronavirus disease 2019 (COVID-19), killed ~7 million persons worldwide. 2020 was a health devastating year. Pandemics do not die-they fade away.COVID-19 disease appears to have been associated with significant mortality among doctors and healthcare workers globally. The forfeit that they are making for the safety and welfare of humans is invaluable. The frontline warriors like the doctors, Health Department officials, tahsildars, executive officers, etc, have been working relentlessly to save lives, thereby keeping the death rate at the lowest. Death and grief have made us numb. The sleep-abated champions encompass doctors, nurses, medical cleaners, pathologists, paramedics, ambulance drivers, and healthcare administrators [158]. A special role belongs to medical students and medical residents who, in addition to providing practical medical care, studied the scientific features of Coronavirus infection under the guidance of brilliant and talented Professors and Academicians [159-167]. The viruses attack on humanity met with heroic resistance from medical personnel around the world, life saving countless SARS-CoV-2 infected people and H3N2 Influenza [168,169].

It is not possible to honor all of the physicians who have died from COVID-19, but in telling the stories of a few of the doctors from different specialties and various countries who lost their lives to the disease, these short obituaries serve as a tribute to the many other doctors who have died in the pandemic. These lives are also a reminder of the ongoing dedication and service of those who continue to care for patients at a time when COVID-19 cases and deaths are increasing in many countries. Doctors who lost their

lives in the war against the COVID-19 pandemic should be recognized as ‘Covid martyrs’ with due acknowledgment of their sacrifice. These physicians died in the line of duty and should be forever admired as the heroes they are. The World Academy of Medical Sciences (WAMS) acknowledges the importance of Dr. Rotaru's loss. WAMS needs to respect you and have an attitude of gratitude. Thank you, Dr. N. Rotaru, for everything you did for our WAMS [170].

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