ORIGINAL ARTICLE

Electro - Ion Membrane Distress Syndrome induces Chronic Fatigue Syndrome/Myalgic Encephalomyelitis (CFS/ME)

Maria Vasilieva[1.1a,3,3a,3b,3d,3e,3g,3h,5,5a,5b,5c], Irina Vasilieva[1.1a,3,3b,3c,3d,3e,3f,3g,3h,5], I. Vasiliev[1.2,4.5,6,9,11], M. Malakhova[4,6], S. Groppa[2,3,3a,5,5a,5c], L. Tofan-Scutaru[3,3c], P. Vanevsky[4], M. Gavriliuk[2,3,3a,5], I. Moldovanu[2,3,3a,5], Tatyana Globa[1,1g,3,3e], Ninel Revenco[2,3k,5,5b], Mirta D'Ambra[1.1i,7], P. Globa[2,3,3g], A. Visnevschi[1,1c,2,3,3d], Elena Globa[3,3g], Larisa Spinei[1,1f,2,3,3h], I. Katereniuk[1,1e,2,3,3f], G. Karadja[3,3j], V. Naku[1,1b,2,3,3f], V. Diug[3,3i,5,5b], G. Stoleriu[1,1d,8], I. Stavrou[1,1j,10], V. Vartanov[1,1h,11], G. Litarczek[12]

[1]World Academy of Medical Sciences, Netherlands (WAMS), [1a]Junior Member of the WAMS; [1b]International Board of Human Tissue Engineering and Cell Cultures, WAMS; [1c]International Board of Laboratory Medicine, WAMS; [1d]International Board Dermatology, WAMS; [1e]International Board of Human and Pathological Anatomy, WAMS; [1f]International Board of Biostatistics, WAMS; [1g]International Board of Human Histology, WAMS; [1h]International Board of Anaesthesiology an Intensive Care, WAMS; [1i]WAMS Argentinian Committee, Public Health UBA, Minister Health UN Coordinator; [1j]International of Board Pain Management, WAMS; [2]Academy of Sciences of the Republic of Moldova, [3]State University of Medicine and Pharmacy "Nicolae Testemitanu", Republic of Moldova; [3a]Department of Neurology, State University of Medicine and Pharmacy "Nicolae Testemitanu", Republic of Moldova; [3b]Departament of Pharmacology and Clinical Pharmacology, State University of Medicine and Pharmacy "Nicolae Testemitanu", Republic of Moldova; [3c]Deparament of Gastroenterology, State University of Medicine and Pharmacy "Nicolae Testemitanu", Republic of Moldova; [3d]Departament Medical Laboratory, State University of Medicine and Pharmacy "Nicolae Testemitanu", Republic of Moldova; [3e]Departament Histology and Embryology, State University of Medicine and Pharmacy "Nicolae Testemitanu", Republic of Moldova; [3f]Departament Normal and Pathological Anatomy, State University of Medicine and Pharmacy "Nicolae Testemitanu", Republic of Moldova; [3g]Department of Biochemistry, Organic and Inorganic Chemistry, and Colloidal Physics, State University of Medicine and Pharmacy "Nicolae Testemitanu", Republic of Moldova; [3h]Departament Biostatistics Science Center, State University of Medicine and Pharmacy "Nicolae Testemitanu", Republic of Moldova; [3i]Departament Obstetrics and Gynecology, State University of Medicine and Pharmacy "Nicolae Testemitanu", Republic of Moldova; [3j]Departament Endocrinology, State University of Medicine and Pharmacy "Nicolae Testemitanu", Republic of Moldova; [3k]Department of Pediatrics, State University of Medicine and Pharmacy, "Nicolae Testemitanu", Republic of Moldova, [4]Department of Anesthesiology and Reanimatology, "V.L. Vanevsky", State Institute for Postgraduate Advanced Training of Doctors, Saint Petersburg, Russia, [5,5a]Institute of Neurology and Neurosurgery of the Republic of Moldova; [5,5b]Insitute of Mother and Child of the Republic of Moldova; [5,5c]Institute of Emergency Medicine, Republic of Moldova, [6]Laboratory of Experimental Pulmonology, State Institute for Postgraduate Advanced Training of Doctors, Saint Petersburg, Russia, [7]Department of Medical Sciences, Medicine University of Buenos Aires, Argentina, [8]Faculty of Medicine and Pharmacy, University of the Lower Danube, Galati, Romania, Enna-Italy, [9]Private Healthcare Institution Ltd. "Via - Intosana", Republic of Moldova, [10]Department of Disciplinary Committee of the Hellenic Society of Pain Management and Palliative Care, Athens Medical School; National and Kapodistrian University of Athens, University Hospital Aretaieion, Athens, Greece, [11]Department of Anaesthesiology of Reanimatology, State Medical University of Samara, The Russian Federation, Russia, [12]Fundeni Institute Bucharest, Romania

Address for correspondence:
I. Vasiliev, World Academy of Medical Sciences, Netherlands, The Republic of Moldova; Department of Anesthesiology and Reanimatology, "V.L. Vanevsky" and Laboratory of Experimental Pulmonology, State Institute for Postgraduate Advanced Training of Doctors, Saint Petersburg; Russia State University of Medicine and Pharmacy "Nicolae Testemitanu", Republic of Moldova; Hospital Medical Institution "Via-Intosana" Republic of Moldova. E-mail: ilievasiliev@gmail.com

ABSTRACT

Primary/secondary morphofunctional damage to pores/channels of biomembranes provokes broken to ion-electrogeneration, 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.Immuno Compromise(IC) CHAOS dissonance creates a disorder of electro-storm/electro-paralysis 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/²H, "heavy water" which inhibits some cleavage reactions.

Key words: Sindrom maria and irina vasilieva, electro-ion membrane distress syndrome, chronic fatigue syndrome/myalgic encephalomyelitis, less deformed red blood cells, flow deformation, increased densified "rigid" cell membrane, cryobio xenoperfusion (mielo-timo-spleen), deuterium D/²H, synairesis

INTRODUCTION

Pharmacology acting on the membrane electrical potential (MEP), interrupting or stimulating the transmission and conversion of histochemical signals into electrical and vice versa, thereby MEP modifying the effects of cells. Example of chronic electro-ion membrane distress syndrome (EIMDs) is membrane electroparalysis of motor neurons of the spinal cord, the genes SMN1 and 2, observed in spinal muscular atrophy.

Membrane proteins are the most communicative biomolecules since the genome[1] encodes many important proteins that line up in the cell biomembrane and signal with both neighboring and distant cells. Cellular homeostasis is provided by a membrane electrochemical its bioelectromagnetic field, quantum energy and quantum electromagnetic radiation, transfer signal-induced mitochondrial (Mch) redox potential, of DNA and RNA synthesis, enzymatic correction of excision repair of bases of damaged DNA, and expression of anti-apoptotic gene blocking BAX protein (BCL-2 family) protecting telomeres of DNA chromosomes. The preclinical phase indicates many syndromes with dissonant MEP ↔ EIMDs ↔ MED (Mitochondrial Energy Deficiency), [2] provoking of pathology.

Chronic fatigue syndrome/myalgic encephalomyelitis (CFS/ME) has become relevant.[3,4] The development of CFS/ME due to EIMDs is considered in this article through the prism of IC CHAOS dissonance.[5]

In the US, was created, the Surviving Sepsis Campaign, the success of the treatment of sepsis, has led to a reduction and rapid death in non-septic patients. However, in survivors, both post-septic and post-non-septic (fungal,[6] oncogenic,[7,8] after massive injuries and bleeding,[9,10] anaphylactic shock,[11] burns that have overcome the syndromes multiple organ dysfunction syndrome (MODs); persistent inflammation, immunosuppression and catabolism syndrome[12]), more often developed CFS/ME syndrome. Since these patients are in a constant stressful state of "emergency", with the development of inflammatory processes and social inability to persistent CFS/ME syndrome.

The point of view that we share in the emergence of EIMDs, due to the generation of IC CHAOS dissonance in response to an infectious/non-infectious pathological agent (PA), at the level by genomic (G), transcriptomic (T), proteomic (P), metabolic (M), and phenomenal (F) functional-structural disorders, thereby installing MODs.

Prolonged fatigue for CFS/ME is characterized cognitive impairment of thinking and memory, insomnia, impaired kinetics, myalgia, general soreness,[13-16] and vegetative changes aggravated after mental, emotional, and physical stress, and high susceptibility to infections and problems of social adaptation.

In formation EIMDs and CFS/ME with a deterioration in erythrocyte flux deformation in the presence of less deformed ones, as a result of which, resulting in general hypoxia disrupts and cerebral perfusion[17-19] as a consequence of the following

factors. Damage open-close function Mitochondrial (Mch) permeability transition pore-dependent Ca uniporter, mPT pore.[20] Disruption of NO homeostasis, with the establishment of myo-vasodilation[20,21] and the failure of antioxidant[22] effects. Ascertainment Microcirculatory - mitochondrial distress syndrome, MMDs, with an increase in pCO2 (AV gap) > 6 mmHg,[23,24] as a valid marker of tissue hypoxia. Abnormal/extreme myelopoiesis of IR CHAOS dissonance accompanied by MMDs[25,24] aggravates EIMDs, establishes MODs, creates extreme G, T, P and M myelopoiesis,[12] are reduced, incl. lysosomal Mch clearance of autophagy (mitophagy).[12] Assertion Spanish and Dutch endocrinologists that high iod diets soften CFS / ME.

MATERIALS AND METHODS

Given that in patients with CFS/ME, generally accepted tests were within normal limits, Davis *et al*. from the US laboratory confirmed that this syndrome has of signaling molecules provoking inflammation and proving the presence of CFS/ME syndrome.

It turned out to be a real discovery that patients with CFS/ME have less deformed red blood cells in relation to non-suffering ME/CFS.[26] The increased rigidity of cell membranes during *less deformed red blood cells (erythrocyte)*, LDE in relation to non-suffering ME/CFS.[27] is devoid of flow deformation with a violation of the sigma effect, the Phareus-Lindquist phenomenon, which provides a high degree of erythrocyte deformation in a flow the destabilization of which gives rise to tissue ischemia, EIMDs, and MMDs.

In this direction, we have observed 60 patients with MODs since 1984. The level of endogenous intoxication, EI, was also determined by markers,[28,29] as well as indicators of osmotic resistance of red blood cells, permeability of the erythrocyte membrane, PEM, as well as the sorption capacity of erythrocytes, SCE by the method based on urea erythrocyte hemolysis.[30] PEM and SCE characterizes the degree of dynamic resistance of the membrane to PA, expressed in increased permeability similar to capillary leak or increased compacted "hard" cell membrane characteristic of CFS/ME, due to more than LDE. The degree of dynamic stability of the membrane made it possible to evaluate the effectiveness of therapeutic tactics due to the decentralization of macrocirculation, microcirculation of the mitochondrial recruitment (MMR), analgesia - sedation and detoxification of the supplemented MOST-ELSO in combination with antibacterial/antiviral treatment and surgical correction.[31,32,33] MOST included artificial ventilation of the lungs of the corresponding modes.[20,33] Extracorporeal hemoxygenation and CO2 elimination by type extracorporeal carbon dioxide removal.[20,34,26] Hemo-liquoro-plasma-entero-voluno-lympho-sorption; lymph stimulation — drainage of the lymphatic duct.[35] Plasmapheresis (cryo); leukocytes pheresis; thrombocyta pheresis. Cryo-, bio-xeno myelo- timo- spleeno and hepatotransfusion extracorporeal perfusion. Ultraviolet (laser) photomodification of auto blood. Ultrafiltration. Electrochemical detoxification with sodium hypochlorite. Peritoneal dialysis; intrahepatic perfusion through the umbilical vein and hypothermia.[20,33]

RESULTS AND DISCUSSION

All patients with critical IC CHAOS dissonance in the presence of EI with the development of MODs had high PEM 71.5 ± 9.8 units and low SCE of 45% ± 3.2, which were improved with regression of MMDs and MODs after application of MOST-ELSO. An improvement in the general condition of patients with the proven Figure 1, which argues a decrease in the density of the "rigid" erythrocyte membranes and an increase in their sorption activity after applying the recruitment of MMR and MOST-ELSO, where the extracorporeal was connected cryo bio-xeno perfusion (myelo-timo-spleen).[12] It was in these patients that CFS/ME was not symptomatic.

Figure 2 shows that standard therapy of MODs, without the use of the MMR and MOST-ELSO, using indicators PEM and SCE, as markers of EIMDs became critical, forming "hard" cell membranes and decreases in SSE. Depending on their role, if it is: neurons, cause cognitive impairment; if the cells of blood vessels, atherosclerosis develops; retinal cells

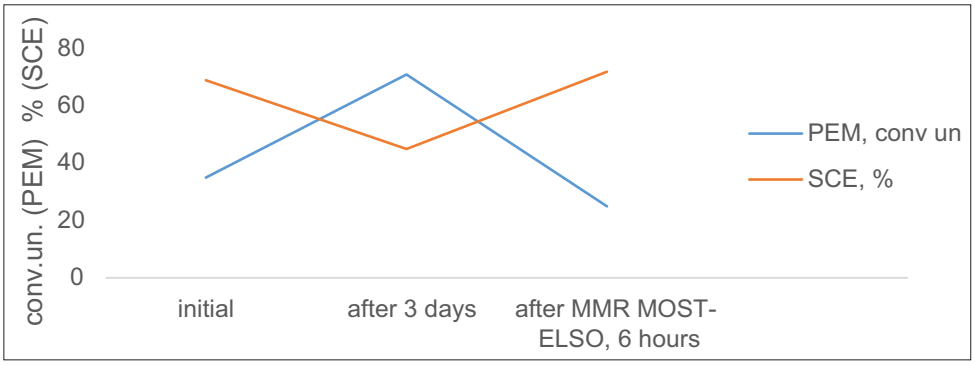

Figure 1: The permeability of the erythrocyte membrane, PEM, as well, sorption capacity of red blood cells SCE – markers of EIMDs after applying the MMR and MOST-ELSO

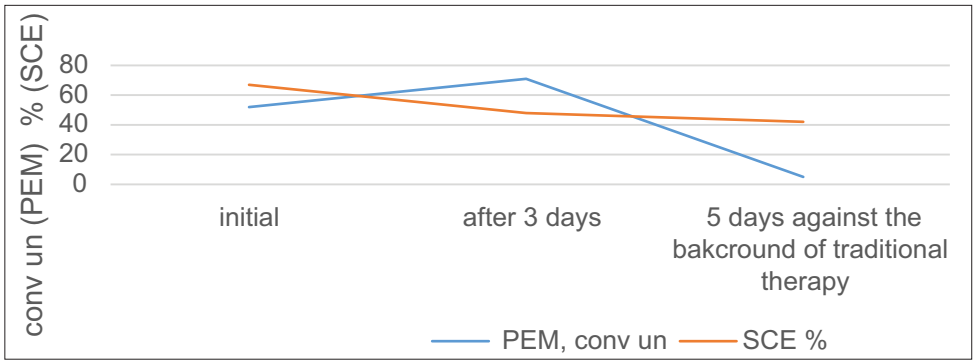

Figure 2: The permeability of the erythrocyte membrane, PEM, as well, sorption capacity of red blood cells, SCE - markers of EIMDs after application standard (traditional) therapy

are cataracts, which predetermine the symptoms of CFS/ME, where endogenous toxic substances also damaged the genetic material of DNA molecules, with G, T, P defective synthesis of hormones, immuno-nutrition, etc. Example, with heart failure and CFS/ME, the PPP1R3A gene with EIMDs caused by a G defect, causes cardiac arrest, which explains the sudden death in CFS/ME. Successfully restored damaged gene[36] using nanoparticles for improved delivery of CRISPR-CAS9 in the treatment of cancer. Immunotherapy with antibodies that block the PD-1 membrane receptors, as well as CTLA-4 and CAR-T cells (Paris[39]), turned out to be effective. The decrease in CFS/ME in our observations in survivors of critical IC CHAOS dissonance after applying MOST-ELSO, where bioxeno (myelothymosplen) perfusion was included,[20,23,33,5,12,37,38] is explained by metabolic effects of biodetoxification microcirculatory recruitment, stabilization of cell membranes proven by improved PEM and SCE, modification of cell membranes from compacted "hard" erythrocytes with less deformed erythrocytes with impaired flow deformation, in more deformed erythrocytes providing optimal flow deformation in the microcirculatory capillary, with a reduction in tissue hypoxia and a diminution in MMDs pCO2 AV gap > 6.0 mm Hg. Consequently, the regressions of acute and chronic MODs and delayed, genomic restorations, where cytokines myelo-timo-spleen perfusate (cytokine therapy) carried out immunomodulation, biodetoxication, biosorption[36-40] protected against damage to G and contributed to their useful remodelling and providing excision repair of the bases of the damaged DNA. The slightly symptomatic CFS/ME observed after cryobioxeno (myelotymosplen) perfusion was also considered as a universal bio mechanism of cryotherapy expressed by the effect of compression and reduction of "synairesis" proteins, by separating the liquid from the gel, caused by the reduction of water and the cell membranes and cells from deuterium D /²H, "heavy water" which inhibits some cleavage reactions.

CONCLUSION

1. The syndrome of surviving patients, from the post-critical states of IR CHAOS dissonance, with the development of CFS/ME, due to the timely optimal failure of the cell membranes of their metabolic functions between the inner and outer cell space due to damaged membrane pores, channels with ion destabilization with an electro-energetic storm or paralysis, defined as EIMDs (EICDs) or the syndrome Maria and Irina Vasilieva
2. Permanent stressed EICDs create less deformed red blood cells that are devoid of flow deformation and cause tissue hypoxia and MMDs, confirmed by the tissue hypoxia marker pCO2 AV gap > 6.0 mmHg
3. Persistent EICDs alter the cell membrane osmotic resistance with the establishment of increased membrane permeability or LDE, densified "rigid" membranes, which in the corresponding tissues of cell membranes, neuro-cardio muscles form the symptoms of CFS/ME
4. The observed higher incidence of CFS/ME in survivors of critical IC CHAOS dissonance is explained by the use of standard therapy, without MOST-ELSO and especially without bioxeno (myelotymosplen) perfusion, where PEM and SCE markers indicated the predominance of the membrane compaction phase in EI, in which there was a more pronounced less deformed red blood cells with the progression of EICDs
5. Addition to the standard therapy of MMR and MOST-ELSO, which included bio-xeno (myelo-timo-spleen) perfusion in survivors of critical IC CHAOS dissonance, the incidence of CFS/ME was insignificant and not symptomatic, due to the nonprolonged phase of compaction of "membrane stiffness" which was accompanied by an unexpressed state of less deformed red blood cells during EI and concomitant EIMDs
6. A special role in the reduction of CFS/ME due to EICDs in survivors of critical but regressive IC CHAOS dissonance due to MOST-ELSO is played by bioxeno (myelotymosplen) perfusion, which minimized extreme myelopoiesis due to the release of cytokines, and hematopoietins, which is expressed in reducing anemia and regression of PICs
7. EICDs induced the insignificant symptomatic CFS/ME in surviving critical patients with IC CHAOS dissonance after MOST-ELSO and especially due to cryobioxeno

(myelotymosplen) perfusion, where biosorption, biodetoxification, enzyme modulation, enzymatic excision of DNA defects, and immune modulation are important in reducing cryoglobulins, it is also explained by the universal biomechanism of cryotherapy expressed in the effect of compression and reduction of "synairesis" proteins by separating the liquid from the gel, releasing it from water, and ridding the cell membranes and cells of deuterium D/²H, "heavy water" which inhibits some cleavage reactions.

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†The distinguished Academician G. Litarczek Patriarch of the Scientific Anesthesia – Therapy Care of Romania (born in Boston, USA), he died on March 14, 2019.