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The recruitment of microcirculatory-mitochondrial of critical obstetric situations in the complex multi-organ support therapy reduces pCO_2 (AV gap) and the development of the syndrome of acute multi-organ dysfunction

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A retrospective analysis of the 35-year absence of maternal mortality in critical obstetrics, in different countries, was due to the timely decentralization of macro-circulation, detoxification and analgesia. Macro-circulation was decentralized once the systemic perfusion pressure has been established; which is the difference between the mean blood pressure and the pressure of the capillary resistance, and what contribute to by decreasing the tissue hypoxia marker pCO_2 (pCO_2 AV gap >6 mm Hg) due to micro-circulatory-mitochondrial recruitment, through improved microcirculation at the level of the capillary-cell metabolic area: metabolic capillary cells mitochondria; with ameliorate of the venous return compliance and reduction (pCO_2 AV gap <6 mm Hg), and respectively, diminishes of the microcirculatory-mitochondrial distress syndrome (MMDs), and stopping expansion syndrome of acute multi-organ dysfunction. In cases of development of respiratory-pulmonary pCO_2 (ARDs), confirmed PaO_2/FiO_2 300 to Acute Respiratory Distress Syndrome (Berlin definition, 2012), thus also aggravates the MMDs (pCO_2 AV gap >6 mmHg), mitochondrial collapse and the recruitment of the microcirculatory-mitochondrial is supplemented with multi-organ support therapy (MOST), including detoxification: alveolar recruitment through respiratory support in specific ventilation modes, predominantly APRV, with permissive hypercapnia at a normal pH; MOST-extracorporeal with technical support. Extracorporeal life support organization-ELSO; modelling of extra-vascular pulmonary fluid index EVLWI; Th4-Th5 thoracic epidural block; active detoxification methods. The absence of decreasing of the pCO_2 tissue hypoxia marker at the pCO_2 AV gap 5.0 mm Hg after microcirculatory- mitochondrial recruitment, rejects the necrosis/apoptosis, hypo- (an) ergic cell and proves the mitochondrial eu-energetic metabolic remodelling with the elimination of the hypo-(an) ergic mitochondria performed by liposomal clearance (mitophagy), thus demonstrating eu-ergic mitochondria with the normalization of mitochondrial uniporter-Ca++ and mitochondrial permeability pore transition, which productively inactivate the toxic forms of oxygen and nitrogen.

Biography

Ilie Vasiliev, MD, is an Academy Professor of Medicine. The First Senior Vice-President the World Academy of Medical Sciences The Chairman of the General Council of the World Academy of Medical Sciences (World Council). The Chairman of the "WAMS Moldovan National Committee" Senior Executive Board Member of the World Academy of Medical Sciences Senior Fellow of the Academy of the World Academy of Medical Sciences Full Membership of the "Academy Faculty" Executive Board Membership of the WAMS' International Medical Research Council (IMREC).

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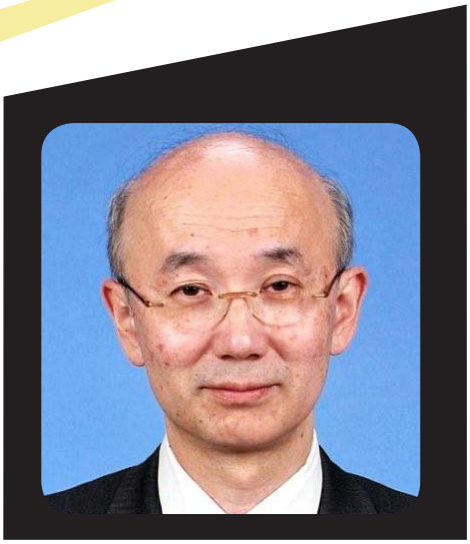
Annexin A5 and MFG-E8 as potential plasma biomarkers for Alzheimer's disease



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Biomarker study on dementia has developed and most reliable fluid markers are amyloid peptide (Aβ), TAU and phosphorylated TAU detected in cerebrospinal fluid. In addition, there is great interest in blood-based markers of Alzheimer's disease (AD) since blood extraction is much less invasive. Moreover, plasma biomarkers can be measured at relatively low expense once a standard system of measurement is established. However, there is not yet an established or validated diagnostic test for plasma biomarkers. Using a neuronal cell culture model we have found that annexin A5 and Milk fat globule-EGF factor 8 protein (MFG-E8), Ca^{2+} and phospholipid binding proteins were elevated in the cell culture medium by Aβ42 treatment. Immunohistochemical study using AD mouse model (APPPS1) brains revealed characteristic distributions of annexin A5 and MFG-E8: more intensive staining with anti-annexin A5 antibody was observed widely in APPPS1 mice compared with control; whereas staining with anti-MFG-E8 antibody was detected only in the central part of the anti-Aβ-antibody stained plaque in APPPS1 mice, while no-staining was observed in control. As both annexin A5 and MFG-E8 might cross the blood brain barrier due to their lipid binding property, it is plausible that both proteins might be plasma biomarkers for AD. For measuring plasma levels of them, we established ELISA systems with monoclonal antibodies against annexin A5 and MFG-E8, respectively. The concentrations of both annexin A5 and MFG-E8 were significantly higher in AD patients than in the healthy individuals ($P<0.0001$). From the ROC curve with plasma annexin A5 and MFG-E8 concentrations for the AD/control, the mean areas under the curve were 0.898 and 0.723, respectively. Interestingly, the level of plasma annexin A5 was also significantly higher in MCI patients than in control ($P<0.0001$). This suggest that annexin A5 was elevated an early stage of the onset of AD.

Biography

Hitoshi Sohma has completed his PhD in Biochemistry at Hokkaido University, Japan, focusing on Ca^{2+} signalling in cell-cell communications, and his Postdoctoral studies at the National Institute of Mental Health, NIH, USA. He is now a Professor in the Department of Educational Development, Sapporo Medical University Center for Medical Education, Sapporo, Japan. He is involved in both patho-biochemical research and the management of medical education at the university. Recently, he has engaged in community medical care program through inter-professional education. His research interest is dementia-related pathophysiology of Ca^{2+} signalling. The results of this research should prove to be of value in community health care education.

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