Neural Diseases, Their regulatory Mechanisms and Energy Metabolism

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CSF NEUROPEPTIDE/NEUROTRANSMITTER BLANCE AND MODULATION OF NEURODEGENERATIVE PROCESSES

Neurological symptoms and neurodegeneration are long term silent features of many neuropathological conditions. These include acute metabolic and physiological insult, chronic motor and dementia disorders as well as inflammatory diseases representing important cause of morbidity and mortality in man. Studies during the last decade have provided convincing evidence that numerous neuroactive molecules play a vital role in chemical signaling and neurodegenerative processes of the nervous system (1). These substances represented before mainly putative transmitters and neuropeptides. Investigations on oxidative stress involving reactive oxygen species (ROS) and reactive nitrogen species (NOS) are now largely believed to be in part responsible for the induction of neurogenic lesions not only by producing neurotoxins but also leading to DNA adduct formation (2). The interdependence of neuropeptide/neurotransmitter pathways and their co-existence in the same neuron is a tremendous advance to understand how neurosecretion can control the processes of transmitter synthesis, release and metabolism. Receptor active opioid peptides in human brain were

demonstrated more than 20 years ago (3). Regardless of our advance knowledge on the neurochemistry of neuropeptide/neurotransmitter and free radicals, it still remains extremely difficult to have a precise diagnosis of the early onset of neurodegenerative processes and only the post-mortem histopathological and laboratory tests can provide the true neuropathological picture. Despite the existence of several barriers between the blood and the brain, cerebro spinal fluid (CSF) can still be considered as an ultrafiltrate of plasma as well as a reflection media of cerebral metabolism. Involvement of neuropathological processes can be reflected by changes in CSF cellular and chemical composition. CSF obtained by lumbar puncture is normally employed to assess the cell counts, protein, IgG levels and among all these parameters, pleocytosis or increase of cells in CSF is the hall mark of all types of neuropathologies (4). Even more important is the possibility to follow each disease during its different stages, and to understand the metabolic and pathogen changes of the disease identified by neurochemical markers such as neuropeptides, neurotransmitter and free radicals.
The present investigations summarized in this chapter provide a brief reflection of the invited conference at Kyoto Joint Conference on natural variations in human CSF content of the neuropeptides, catechol and indole transmitters, excitatory and inhibitory amino acids and free radical NO. Due to limit of publishing space, we shall discuss in detail the true impact of neuropeptide Y (NPY) in different neurological diseases reflecting chronic and acute degenerative processes. Some clinical data on neuroprotective role of vitamin B-12 to reverse neurodegenerative processes has also been provided.

Our clinical studies were oriented to differentiate the effects of chronic long term neurodegenerative diseases such as multiple sclerosis, cerebro vascular stroke, Parkinson's disease from acute clinical situations such as aseptic meningitis and tuberculosis meningities.