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Migraine and neurological disorders comorbidity-consideration of sinus hypoxic nitric oxide theory for migraine

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Migraine is an extremely common disorder and has co morbidity with many neurological illnesses. After reviewing the neurophysiological and biochemical basis of the research findings and hypotheses of migraine and other neurological disorders I present to the best of my knowledge the first para nasal sinus nitric oxide mediated neurobiophysiological explanation for many neurological disorders. The etiology of neurological illnesses is mainly due to neurotransmitter imbalance, neurodegenerative changes acute and chronic inflammation, effects of hypo and hyper endothelial and neuronal NO levels and genetic predisposition. According to this para nasal sinus nitric oxide based description those effects are mainly brought on by the sinorhinogenic impulse distribution of the central nervous system except genetic predisposition. Moreover, avoidance of the central neuronal influence and stress to the brain in early childhood or young age caused by migraine would help to prevent the progression or aggravation of the neurological illnesses. Indeed this article explains a new pathophysiological initiation between sino rhinogenic nitric oxide effects and neurological disorders and provides an etiologically important Neuro vascular impulse generating pathway to cause or aggravate neurological disorders. Therefore the patients who are clinically suspected of having migraine headache and neurological disorders or along with susceptible neurological disorders should receive comprehensive sinorhinological examination and evaluation based on the sinus hypoxic nitric oxide phenomena. A standard surgical and medical management of migraine that links with the sinus hypoxic nitric oxide theory are suggested to be used for even neurological disorder as a new treatment of neurological illnesses and to prevent the dysfunction of central specific neural circuits.

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Spinal mechanisms underlying acute pain syndrome induced by paclitaxel

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Paclitaxel (taxol) is a first-line chemotherapy-drug used to treat many types of cancers. Neuropathic pain and sensory dysfunction are the major toxicities that are dose-limiting and significantly reduce the quality of life in patients. Pathological pain induced by taxol in patients includes pain that occurs immediately after taxol treatment (the so called paclitaxel-associated acute pain syndrome, P-APS), and pain that persists for weeks to years after cessation of paclitaxel treatment (the so called paclitaxel induced chronic neuropathic pain). There is no proven standard of care for the prevention or treatment of P-APS. In this study, we found that paclitaxel causes acute pain in rodents. The paclitaxel-induced acute pain occurs within 2 hrs after a single intravenous injection of paclitaxel (2 mg/kg). This is accompanied with low levels of paclitaxel penetrating into the cerebral spinal fluid and spinal dorsal horn. Paclitaxel directly acts on microglial TLR4 and increases $Ca2^{+}$ levels in microglia in the spinal dorsal horn, which in turn activates astrocytes through releasing IL-1βIL-1βincreases glutamatergic synaptic activities and reduces glial glutamate transporter activities in the spinal dorsal horn. Activations of TLR4 and IL-1β receptors are critically implicated in the genesis of the paclitaxel-induced acute pain behaviors. The cellular and molecular signaling pathways revealed in this study could provide rationales for the development of analgesics and management of P-APS in patients.

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