MIGRAINE AND NEUROLOGICAL DISORDERS COMORBIDITY CONSIDERATION OF SINUS HYPOXIC NITRIC OXIDE THEORY

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Sinus Hypoxic Nitric Oxide Theory (SHNOT)

- A large production of NO gas (s NO) takes place in the para nasal sinuses: synthesized by inducible NOS in the ciliated cells.

- Hypoxia is a powerful inducer of paranasal sinus nitric oxide synthetases (NOS).

- Factors - physiological and pathological conditions, anatomical anomalies, variation of the diameter of sinus ostia, duration of hypoxia and level of oxygen concentration.
SHNOT

10% \( \geq \) \( O_2 \)  

NOS  
Nasal & Sinus Hypoxia

Environmental  
Allergic  
Hormonal  
Other

Factors  
Nasal, Sinus, Ostial Oedema, Inflammation, Blockade

Factors

Psychological  
Physiological  
Pathological  
Anatomical
Sinus Hypoxic Nitric Oxide Theory (SHNOT)


Based on A NEW HYPOTHESIS
The New Hypothesis For Migraine

The diffused sinus NO (ds NO) in the para nasal sinus and nasal mucosa is a main causative and an initiative molecule for migraine headache.
Studies Supporting - SHNOT & Hypothesis for Migraine

Migraine treated by standardized surgical intervention that relives the sinuses hypoxia.

78% of patients with migraine were free of symptoms post operatively
Anomalies and other causes for PNS Hypoxia

1. conca bullae
2. paradoxical turbinate
3. large inferior & middle turbinate
4. nasal septal deviation
5. stenosis of nasal cavity
6. deviation of the uncinated process tip
7. nasal spur
8. uncinate bulla
9. Haller cells
10. ethmoidal bullae
Physiological and biological roles of NO

- A main neurotransmitter
- A potent vasodilator
- High ability to diffuse
- Release other Neurotransmitters - CGRP, Glutamate, n NO,

Migraine - about 88% of the sinus headache patients.

INTRANASAL

sNO .... ? +
CO₂ → (V)
NH₄ → (V)

INTRANASAL

B₁₂ – NO Scavenger
Lignocaine / cocaine

Pulmonary oxygen uptake enhanced by sNO
Conversion of ds NO into neuronal impulse

1. Activates free nerve endings and peri vascular nerves.
2. The nerves activated by vaso active substances.
3. Extra cranial vaso dilation and activation of the nociceptors.
4. The neurogenic inflammation.
Migraine co-morbid with Neurological Disorders and Evidences

- Stroke
- Epilepsy
- Motion sickness
- Vertigo
- Balance disorder
- BIH
- Narcolepsy

- Epidemiological evidence
- Pharmacological evidence
- Genetic
- Acquired mal-function of brain tissues
- Objective investigative evidence
- Neuro pathophysiological
Migraine Neuro pathophysiology

Cortical spreading depression (CSD)

1. Neuronal mediated vasodilatation
2. Inflammatory process
3. Vasoactive substances
4. Altered vascular permeability

1. ↓BF in major ICV & ↓ in Small ICV
2. Cytotoxic cell damage & gliosis
3. Activation of matrix metallo proteinases
4. Thrombosis in the blood vessel

- Dysfunction neurotransmitter projections
  1. (DA, 5HT, GLU, DA)
  2. Ab. sleep pattern, CS, stress to CNS

- Shear Stress in the BV - ↑e NO and ↓ e NO
- Melatonin ↓

Hyper excitability

1. CSD
2. ↑ responsiveness CC

- Excesses ↑ (n NO)
- Migraine aura ↓ Mg+, Glu ↑, 5HT ↓
Migraine and Neurological disorders explained by hypothesis and SHNOT
Objective investigative evidences

1. Migraine and BAD

2. Migraine and CVA
Conclusion

- The excess or unnecessary impulse generation and neurovascular activation primarily due to SHNOT in sinorhinological areas contribute for migraine and other neurological disorders.

- Rhino logical medical or surgical approach may be benefit of stop or prevent neuropathology affecting to cause or aggravate common neurological disorders.
Thank you