Horner syndrome

Disruption of cranial sympathetic tone leads to the symptom complex of miosis, ptosis, and hemifacial anhidrosis. It is widely believed that this phenomenon was discovered in 1869 by the Swiss ophthalmologist Johann Friedrich Horner, and as a result, the term Horner syndrome has become synonymous with the clinical presentation.

However, the syndrome that would become Horner syndrome had actually been described several times before his report.

François Pourfour du Petit documented the ocular effects of sympathetic trunk lesions in animal studies in 1727.

Claude Bernard identified the full clinical triad in animal studies in 1852, and as a result, the condition is sometimes called Bernard syndrome.

There were also 2 previous reports of ptosis and miosis resulting from sympathetic nerve damage in humans: 1 by Edward Selleck Hare in 1838 associated with brachial plexus tumor, and the other by Silas Weir Mitchell in 1864 associated with a gunshot wound to the neck. Although Horner was the first to objectively characterize the co-occurrence of vasomotor and ocular changes in a human patient, he did not identify the etiology of the condition, discuss its relationship to the sympathetic nervous system, or reference any of the previous studies in animals or humans. It is possible that a lack of familiarity with previous investigations delayed the full appreciation of the mechanism underlying this disorder [1].

Enophthalmos is also a frequent symptom. It indicates a problem with the sympathetic nervous system, a part of the autonomic nervous system. Medical imaging and response to particular eye drops may be required to identify the location of the problem and the underlying cause.

Evidence from computerized tomography (CT), magnetic resonance imaging (MRI), positron emission tomography (PET), and single-photon-emission computerized tomography (SPECT) studies have confirmed that reciprocally connected centers in the insular cortex, central nucleus of amygdala, hypothalamus, mesencephalic and pontine tegmentum, nucleus of tractus solitarius, and the ventrolateral medulla form the central pathway. The nucleus of tractus solitarius is probably the main reflex center for the sympathetic system, whereas the ventrolateral medulla serves as the pathway through which the central neurons influence the preganglionic neurons of the thoracolumbar outflow. Emotional and sensory inputs from the frontal and somatosensory cortices provide the inputs needed by the insula to drive the sympathetic nervous system to produce appropriate responses [2].

1)
Abbas A, Manjila S, Singh M, Belle V, Chandar K, Miller JP. Johann Friedrich Horner and the Repeated Discovery of Oculosympathoparesis: Whose Syndrome Is It? Neurosurgery. 2015 Sep;77(3):486-91. doi: 10.1227/NEU.0000000000000832. PubMed PMID: 26280826.
2)
Amonoo-Kuofi HS. Horner's syndrome revisited: with an update of the central pathway. Clin Anat. 1999;12(5):345-61. PubMed PMID: 10462732.

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