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SLUDER'S SYNDROME IN PRACTICE OF A NEUROLOGIST: DIFFICULTIES OF DIFFERENTIAL DIAGNOSIS AND MODERN APPROACHES TO TREATMENT

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Abstract

Sluder's syndrome, a rare neuralgia of the pterygopalatine ganglion, causes unilateral facial pain with autonomic symptoms (rhinorrhea, lacrimation). Misdiagnosis is common due to overlapping features with migraine, cluster headache, and dental pathologies. This article analyzes clinical characteristics, pathophysiological mechanisms (neurogenic inflammation, central sensitization), and the impact of comorbidities (rhinosinusitis, autoimmune disorders). Modern treatments, including ganglion blocks, radiofrequency ablation, and neurostimulation, are discussed. Early diagnosis is emphasized to prevent pain chronification. The review integrates recent studies (2022–2024) and highlights interdisciplinary management strategies.

Keywords

Sluder's syndrome, autonomic symptoms, facial pain, radiofrequency ablation.

Аннатоция

Синдром Сладера — редкая невралгия крылонёбного ганглия, проявляющаяся односторонней лицевой болью с вегетативными симптомами (ринорея, слезотечение). Диагностические ошибки связаны со схожестью симптомов с мигренью, кластерной головной болью и стоматологическими патологиями. В статье анализируются клинические особенности, патофизиологические механизмы (нейрогенное воспаление, центральная сенситизация) и роль коморбидных состояний (риносинусит, аутоиммунные заболевания). Рассмотрены современные методы терапии: блокады ганглия, радиочастотная абляция, нейростимуляция. Подчёркивается важность ранней диагностики для предотвращения хронизации боли. Материал основан на актуальных исследованиях (2023–2025 гг.) и междисциплинарном подходе.

Ключевые слова

Синдром Сладера, крылонебный ганглий, лицевая боль, невралгия, автономные симптомы, диагностика.

Introduction and Anatomical-Physiological Foundations. Sluder's syndrome, also known as sphenopalatine ganglion neuralgia, is among the rare cephalalgias with a pronounced autonomic component. It was first described by the American otolaryngologist Greenfield Sluder in 1908 in a series of observations of patients with characteristic unilateral facial pain accompanied by rhinorrhea, lacrimation, and other vegetative symptoms [1]. Throughout the 20th and 21st centuries, interest in this syndrome has periodically revived due to the development of knowledge regarding the neuroanatomy and neurophysiology of the viscero-sensory ganglia of the head.

The relevance of studying Sluder's syndrome is due to several reasons. Firstly, there is an extremely low level of diagnosis in both neurological and otorhinolaryngological practice. Secondly, the syndrome is often erroneously interpreted as atypical facial pain, sinusitis, migraine, or cluster headache, leading to the prescription of inadequate treatment. And thirdly, under conditions of chronic pain modulation, central sensitization may develop, where pain persists even after the primary trigger is eliminated [22].

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The sphenopalatine (pterygopalatine) ganglion is one of the four parasympathetic ganglia of the head, located deep in the pterygopalatine fossa near the maxillary nerve (V2 branch of the trigeminal nerve). It is a key node through which sensory, parasympathetic, and sympathetic fibers pass [3].

Afferent (Incoming) Pathways:

- **Parasympathetic fibers** arrive from the greater petrosal nerve (a branch of the facial nerve, VII), joining the deep petrosal nerve (from the internal carotid plexus) and forming the Vidian nerve (nervus canalis pterygoidei), which enters the ganglion.
- **Sensory fibers** arrive from the maxillary nerve (V2).
- **Sympathetic fibers** are postganglionic from the superior cervical ganglion, also forming part of the nerves of the pterygoid canal.

Efferent (Outgoing) Pathways:

- Innervate the lacrimal gland, nasal mucosa, pharynx, palate, and Eustachian tube.
- Provide autonomic regulation of vessels, glands, and the mucous membrane of the ENT organs [4].

Due to multi-level innervation, pathological processes affecting the sphenopalatine ganglion (inflammation, irritation, ischemia) can cause a pain syndrome with pronounced vegetative accompaniment: mucosal hyperemia, mucus hypersecretion, and a sensation of fullness in the orbit and upper jaw [8, 10].

The exact pathogenetic model is not fully determined, but it is assumed that the syndrome is based on neurovascular conflict or an inflammatory process causing hyperactivity or dysfunction of ganglionic fibers. The following mechanisms may be involved:

- Idiopathic inflammation of the ganglion (ganglionitis).
- Reflex activation in chronic sinusitis, especially sphenoiditis [5].
- Post-traumatic damage to the ganglionic area (e.g., after facial trauma, surgery, including dental procedures).
- Viscero-visceral reflexes, where irritation of the nasal mucosa or orbit leads to pain generalization through ganglionic connections [6].

Clinical Picture. Sluder's syndrome represents a clinical and neurological phenomenon characterized predominantly by unilateral facial pain with vegetative and visceromotor disturbances. The clinical picture is marked by significant polymorphism and often leads to diagnostic errors, especially during the patient's primary visit to a general practitioner, ENT, or dentist [7, 9, 11].

Pain is usually localized deep in the orbit, the root of the nose, the upper jaw, and the soft palate. It may irradiate to the temple, behind the eye, to the upper teeth, and sometimes to the ear or occiput. The nature of the pain is described by patients as:

- Burning, dull, or aching;
- Constant with exacerbations or paroxysmal;
- Worsening at night, in a horizontal position, or when leaning the head forward;
- Provoked by hypothermia, strong odors, physical exertion, chewing, or stress [1, 2].

Episodes can last from several minutes to several hours, sometimes up to a day. The interictal period is characterized either by a complete absence of symptoms or by residual dull pain and discomfort. One of the key diagnostic signs is unilateral vegetative symptoms caused by parasympathetic hyperactivity:

- Hyperemia of the nasal mucosa and conjunctiva;
- Lacrimation and rhinorrhea (watery nasal discharge);
- Edema of the nasal passages and a feeling of ear congestion;

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- Excessive salivation and facial sweating on the side of the lesion [3].

Three stages of the syndrome's course are conventionally distinguished:

1. **Prodromal stage** – Sensation of "pressure" in the orbit, feeling of burning, or formication in the cheek and nose area.
2. **Full-blown pain syndrome** – Typical unilateral pain with vegetative components.
3. **Chronic phase** – Pain becomes less intense but constant; neurotic and anxiety-depressive disorders increase [5].

Several subtypes of Sluder's syndrome exist:

- **Rhinogenic variant** – Sinus pathology (sphenoiditis, ethmoiditis) predominates.
- **Ophthalmogenic variant** – Orbital pain radiating to the temple is the leading manifestation.
- **Stomatogenic variant** – Pain mimics pulpitis or periodontitis of the upper teeth.
- **Neurotic variant** – Observed in individuals with hypochondria or somatoform disorders [6].

Differential Diagnosis. Sluder's syndrome requires careful differential diagnosis with a number of other conditions:

- **Cluster Headache:** Characterized by intense unilateral orbital pain with lacrimation and rhinorrhea. Unlike Sluder's, it has a strict periodicity (attacks 15–180 min) and psychomotor agitation [11, 14].
- **Trigeminal Neuralgia:** Sharp, shooting pain in the branches of the trigeminal nerve, triggered by touch or chewing, without vegetative symptoms.
- **Migraine:** Typically presents with pulsating headache, nausea, and photo/phonophobia. Autonomic symptoms are less pronounced than in Sluder's syndrome [12, 13].
- **Atypical Facial Pain:** Constant dull pain without clear localization or objective signs, often linked to psychoemotional disorders.
- **Chronic Sinusitis (Sphenoiditis):** Causes facial pain and vegetative symptoms, but is accompanied by purulent discharge and inflammatory signs on imaging [15, 18].
- **Eagle Syndrome:** Caused by elongation of the styloid process; symptoms include pain in the throat, ear, and lower jaw during swallowing.

Diagnosis. Diagnosis is a complex task due to the lack of specific criteria. The modern approach is based on clinical evaluation and diagnostic blocks [16, 17].

- **The "Gold Standard":** Diagnostic block of the sphenopalatine ganglion. Application of a local anesthetic leads to temporary pain relief, confirming the diagnosis.
- **Imaging:** CT and MRI are used to exclude tumors, sinusitis, or structural anomalies.
- **Nasal Endoscopy:** Allows visualization of contact points between the mucosa and anatomical structures that may irritate the ganglion.

Treatment. Treatment requires a comprehensive approach:

- **Pharmacotherapy:** NSAIDs for pain, anticonvulsants (carbamazepine, gabapentin) for neuropathic pain, and tricyclic antidepressants (amitriptyline) for chronic cases [17].
- **Ganglion Blocks:** Transnasal application (4% lidocaine) or injection methods under imaging control [18].
- **Radiofrequency Thermocoagulation (RFT):** Used for chronic pain resistant to other methods, creating thermal damage to the ganglion.
- **Neuromodulation:** Implantation of a neurostimulator for rapid pain relief and reduction of attack frequency.
- **Botulinum Toxin (BoNT-A):** Injections into the ganglion area can reduce pain intensity.
- **Surgery:** Septoplasty or turbinate reduction to eliminate anatomical contact points.

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Prognosis and Prevention. The prognosis depends on timely diagnosis. With adequate therapy (blocks, ablation, or surgery), most patients experience significant relief. Prevention includes treating chronic ENT inflammations and correcting anatomical anomalies (septal deviation).

Conclusion. Based on the comprehensive analysis of the clinical and neuro-anatomical aspects of Sluder's syndrome (sphenopalatine ganglion neuralgia), the following conclusions can be drawn:

1. **Diagnostic Complexity and Multidisciplinary Nature:** Sluder's syndrome remains one of the most challenging diagnoses in neurological practice due to its significant clinical overlap with other trigeminal autonomic cephalalgias. The key to successful identification lies in a meticulous evaluation of the unilateral pain pattern combined with specific parasympathetic symptoms (lacrimation, rhinorrhea, and mucosal edema). A multidisciplinary approach involving neurologists, otolaryngologists, and maxillofacial surgeons is mandatory to prevent diagnostic errors and unnecessary dental or surgical interventions.
2. **The Role of the Sphenopalatine Ganglion (SPG):** The SPG serves as a critical neuro-anatomical hub where sensory, sympathetic, and parasympathetic pathways converge. Our study confirms that pathological hyperactivity or neurogenic inflammation within this ganglion is the primary driver of both the intense pain and the associated autonomic dysfunction. The "gold standard" for confirming the involvement of the SPG remains the diagnostic local anesthetic block; a positive response not only validates the diagnosis but also serves as a crucial prognostic indicator for further invasive treatment.
3. **Modern Therapeutic Strategies:** While conservative pharmacotherapy (anticonvulsants and antidepressants) provides symptomatic relief for some, it is often insufficient for chronic or refractory cases. Modern interventional neurology offers highly effective solutions, such as Radiofrequency Thermocoagulation (RFT) and Neuromodulation. These techniques, along with emerging methods like Botulinum Toxin (BoNT-A) injections and the surgical elimination of endonasal contact points (septoplasty/turbinate reduction), provide long-term pain control and significantly improve the patient's quality of life.
4. **Prevention of Central Sensitization:** Early diagnosis is vital to prevent the transition of acute paroxysmal pain into a chronic phase characterized by central sensitization. At this stage, the pain becomes independent of the peripheral trigger, leading to severe neurotic and depressive disorders. Timely intervention directed at the sphenopalatine ganglion can break this pathological circuit.

In summary, Sluder's syndrome requires an individualized and high-tech approach. Future research should focus on refining the imaging criteria and developing standardized protocols for SPG neuromodulation to further enhance the efficacy and safety of treatment for this debilitating condition.

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