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Clinical Manifestations of Temporal Lobe Epilepsy: An Overview

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Abstract Temporal lobe epilepsy (TLE) is the most common type of epilepsy in adults, encompassing both mesial TLE (MTLE) and neocortical TLE (NCTLE). It accounts for around 30% of all epilepsies and 60% of focal epilepsy and nearly 30% of TLE patients become resistant to medication. Despite the crucial role of clinical manifestations in diagnosing and pinpointing the location of TLE, research in this area remains surprisingly scarce. This article delves into the various facets of TLE, unraveling its history, types, causes, and seizure semiology. Finally, we'll explore the potential of resection surgery as a treatment option.

Keywords Temporal lobe epilepsy, Seizure, Aura, Automatism, Surgery

1. Introduction

Temporal lobe epilepsy (TLE) is the most common type of epilepsy in adults, encompassing both mesial TLE (MTLE) and neocortical TLE (NCTLE). It accounts for around 30% of all epilepsies and 60% of focal epilepsy cases seen in specialized centers [1]. Worryingly, nearly 30% of TLE patients become resistant to medication. This highlights the complex nature of TLE, often originating in the highly epileptogenic temporal lobe area of the brain [2]. The most prevalent cause of TLE is mesial temporal sclerosis (MTS), a syndrome characterized by hippocampal sclerosis (HS) visible on Magnetic Resonance Imaging (MRI) alongside a specific electro-clinical profile. Other causative factors include various focal lesions within the temporal lobes, some yet undetectable with current technology. Additionally, familial forms linked to genetic mutations have been identified [3] [4].

During epileptic episodes, TLE patients often exhibit a fixed gaze with unresponsiveness, frequently accompanied by automatisms involving the mouth or hands. These features are present in roughly two-thirds of individuals with refractory TLE who require surgical intervention [5].

This review has aimed to provide a comprehensive overview of temporal lobe epilepsy, highlighting its prevalence, causes, and clinical manifestations. Understanding the multifaceted nature of ELT is essential to improving its management.

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2. Historical Perspectives on Temporal Lobe Epilepsy

John Hughlings Jackson: In the 19th century, neurologist John Hughlings Jackson pioneered the link between "dreamy state" seizures and lesions near the uncus in the temporal lobe. This connection led to the term "unciform seizure." [6].

Gibbs and Lennox: Expanding on Jackson's work, Gibbs and Lennox coined the term "psychomotor epilepsy" to describe the characteristic EEG pattern and the emotional, mental, and autonomic phenomena associated with temporal lobe seizures [7].

Montreal Neurological Institute: Researchers at the Montreal Neurological Institute (MNI) further refined the understanding of psychic phenomena in TLE. Based on clinical observations and intraoperative stimulation studies, they defined these experiences as "experiential hallucinations." [8]

Gastaut and "complex partial seizures" (CPS): Henri Gastaut introduced the term CPS to categorize partial seizures involving altered consciousness [9].

Technological advancements: Videotape and computer technology revolutionized the study of TLE. By closely examining captured seizures and their EEG data, detailed descriptions of temporal lobe seizure characteristics became possible [5].

3. Types of Temporal Lobe Epilepsy

The International League Against Epilepsy (ILAE) recognizes two primary subtypes of TLE: mesial TLE (MTLE), affecting the medial and internal structures of the temporal

lobe (including the hippocampus) and accounting for around 80% of all TLE cases, and neocortical TLE (NCTLE), involving the external parts of the lobe. MTLE is therefore the dominant form of TLE, with NCTLE making up the remaining 20% [9].

4. Causes of Temporal Lobe Epilepsy

The underlying cause of TLE can be either structural (visible on Magnetic Resonance Imaging (MRI)) or non-structural and may or may not have a familial (genetic) component [10]. The most common cause of TLE is hippocampal sclerosis (HS), accounting for over 80% of cases.

Other causes include structural lesions: perinatal lesions (brain injuries occurring around the time of birth), cortical developmental malformations (abnormalities in brain development), arteriovenous malformations (tangles of abnormal blood vessels), central nervous system infections, glial tumors (brain tumors arising from supporting cells), hamartomas (non- cancerous growths), and head trauma. Also, some cases of TLE have a genetic basis, with mutations in certain genes increasing the risk of developing the condition [11] [12].

5. Seizure Semiology in Temporal Lobe Epilepsy

Seizure semiology refers to the objective signs and subjective symptoms experienced during epileptic episodes. Its accurate portrayal is crucial for the proper diagnosis and classification of both seizures and epileptic syndromes [13]. While valuable information can be gleaned from patients and witnesses, video recordings of seizures offer even more detailed and objective data. Advancements in high-resolution video technology allow for capturing subtler nuances, enhancing our understanding of TLE seizures. The synergy of synchronized Electroencephalogram (EEG) and Video-EEG recordings enables a comprehensive analysis of the electro-clinical correlation throughout the entire seizure event. However, subjective symptoms reported by patients, invisible on video, remain essential for precise characterization, localization, and lateralization of the seizure focus [14].

5.1. Auras in Temporal Lobe Epilepsy

Certain auras are particularly associated with TLE seizures and can provide valuable clues for diagnosis and seizure localization. These auras include viscerosensory symptoms such as an ascending epigastric sensation. Also, gustatory, olfactory, auditory or visual hallucinations have been reported. Significantly, psychic aura such as a strange sensation. These can be emotional symptoms (fear, anxiety), or déjà vu or jamais vu [15] [16].

5.2. Automatisms in Temporal Lobe Epilepsy

Automatisms are repetitive, involuntary, and often purposeless movements that occur during a seizure. While typically inappropriate for the situation, they can sometimes resemble everyday actions. Their presence can be a valuable clue for diagnosing and localizing TLE [13].

- Common Automatisms in TLE:

Oro-alimentary automatisms: These involve movements of the mouth and face, such as lip- smacking, sucking, swallowing, or chewing.

Gestural automatisms: These involve repetitive movements of the limbs or hands, like picking, groping, or fidgeting.

- Less Common Automatisms:

Vocalizations: Grunting, moaning, or other unarticulated sounds. Critical speech: Speaking in short, repetitive phrases or sentences.

Affective behaviors: Experiencing sudden emotions like fear or anxiety out of context.

- Rare Automatisms:

Dacrystosis: Excessive crying without apparent reason. Gelastic seizures: Uncontrollable laughter during a seizure.

Departure behaviors: Sudden attempts to leave the environment, such as running out of a room or onto the street [14] [17] [18] [19]

5.3. Motor Manifestations in Temporal Lobe Epilepsy

TLE exhibits various characteristic motor manifestations during seizures. These can include: Fixed gaze with behavioral arrest: The patient experiences a motionless stare and unresponsive state. Oro-feeding and bimanual automatisms: Involuntary, repetitive movements involving the mouth, face, and hands, such as lip-smacking, swallowing, chewing, picking, or fidgeting. Ipsilateral head rotation: A brief initial turning of the head towards the side of the seizure focus. Eye movements: Seizure-related ocular version: Forced, sustained, and coordinated deviation of both eyes, usually accompanying the head turning. Contralateral version before secondary generalization: Eye movement originating from the opposite hemisphere of the brain, often indicating potential spread of the seizure. Clonic movements: Twitching or jerking of body parts typically occur in TLE later in the seizure, when the patient loses consciousness, due to the seizure activity spreading to the motor cortex [13] [14] [20].

6. Surgical Treatment of Temporal Lobe Epilepsy

Temporal Lobe Epilepsy (TLE) is the most common form of pharmaco-resistant epilepsy in adults that can be treated with surgery. It is a safe procedure with low rates of postoperative complications and good long-term outcomes

[21]. The gold standard surgical modality for TLE is resection surgery, which should be proposed as a first-line treatment, allowing seizure control in 60 to 80% of cases. However, surgical resection is associated with neurological and neuropsychological deficits, including memory and language [21] [22] [23].

In recent years, several innovative techniques in the surgical treatment of TLE have been developed [22]. Stereotactic radiosurgery can have similar or slightly lower efficacy in some patients compared to invasive surgery and should therefore be considered as an alternative to open surgery for patients with contraindications or who are reluctant to undergo open surgery [24]. Laser interstitial thermal therapy (LITT) has also shown promising results as a curative technique in mesial TLE, but needs to be further evaluated [25]. Too, brain stimulation is a palliative treatment option for patients with unilateral or bilateral MTLE who are not candidates for temporal lobectomy or who have failed previous resection of the mesial temporal lobe [26] [27]. In the future, ultrasound therapies could become a credible therapeutic option for patients with refractory TLE [28].

6.1. Advantages of Surgical Treatment of TLE

Successful TLE resection surgery can improve patients' quality of life and offer many long- term benefits, including: (a) Prolonged seizure freedom (b) Reduced reliance on antiepileptic drugs (c) Reduced mortality (d) Improved psychosocial outcomes, enhancing social interactions and overall well-being (e) Improved cognitive functions affected by seizures [21] [23] [29].

6.2. Limitations and Considerations

Despite its potential benefits, TLE resection surgery is not a cure for all patients. On the one hand, it remains difficult to select patients eligible for surgery [21] [23]. On the other hand, even in carefully selected cases, about 30% may still experience seizures after surgery [30]. Surgical failures can be related to temporolateral epileptogenic zones or even to extra-temporal seizure zones [31]. Despite a possible individual deterioration, reoperation after failed resective epilepsy surgery has led to about 70% long-term seizure freedom and reasonable neuropsychological outcomes [32]. Another study also highlighted the improvement in seizure outcomes and that overall neuropsychological performance at long-term follow-up was not affected by the second resection in TLE. However, it remains difficult and essential to assess individual conditions to identify appropriate patients, as surgery still carries risks and complications [23].

Furthermore, despite advances and modifications in surgical resection techniques, memory deficits remain a persistent challenge. This is because long-term memory networks are predominantly located in the mesial temporal lobe and thalamus. The current trend is to minimize the surgical footprint on the brain to reduce postoperative deficits. Additionally, increased personalization of the resection, by limiting its posterior extent, can have a lesser impact on memory [33]. Minimally invasive therapies can also pave the way for more beneficial neuropsychological outcomes [25].

7. Conclusions

While significant progress has been made in understanding and managing TLE, the future holds exciting possibilities for further advancements. By recognizing its multifaceted nature and focusing on personalized care, novel therapeutic strategies, and deeper understanding of the underlying mechanisms, we can improve the lives of individuals living with TLE.

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