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Review
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THE DIFFERENTIAL DIAGNOSIS OF VERTIGO AND EPILEPSY

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Summary

Vertigo is most commonly a feeling of spinning, usually due to the disturbance in the balance (vestibular) system. It may result from a dysfunction of the vestibular system at any point from the ear to the cerebral cortex.

Epileptic vertigo is a rare form of partial seizures, due to epileptic activity in parts of the cortex that represent the vestibular system: the parietal, temporal and frontal cortex. The episodes usually last no more than seconds or minutes. Unconsciousness will follow only if the seizure becomes generalized.

Diagnostic tests include EEG and MRI scans of the head. Further diagnostic evaluation in the case of medical intractability would include: video EEG monitoring, intracarotid amytal testing, ictal SPECT, neurophysiogical evaluation and intracranial EEG monitoring. An abnormal EEG is a major criterion for diagnosis. In most patients the abnormality consists of temporal or bitemporal sharp or slow wave foci. In some cases there are associated generalized seizure discharges. Treatment of epileptic vertigo is usually successful with traditional anticonvulsants such as carbamazepine and its relatives. If, after a reasonable trial with appropriate antiepileptic drugs, seizures remain inadequately controlled, a surgery can be considered.

Differential diagnosis of epileptic vertigo includes: a basilar type migraine, confusional migraine, benign paroxysmal vertigo of childhood and an aura without a headache. The main differential diagnosis of neocortical temporal lobe seizures is a mesial temporal lobe seizure. Ictal SPECT scanning and MRI can provide diagnostic data not otherwise obtain-

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able. In addition, proton MR spectroscopy and PET studies can be helpful to distinguish the mesial from neocortical temporal lobe seizures.

Key words: epilepsy, epileptic vertigo, partial epilepsy, neocortical epilepsy

INTRODUCTION

Although dizziness as a manifestation of epilepsy was recognized 100 years ago by Hughlings Jackson and later by Gowers, the possibility that brief episodes of dizziness may be due to epilepsy was not recognized. Today it is well known that epilepsy is an important cause of transient dizziness [1].

True vertigo, from the Latin „vertere“, to turn, is a distinct, often severe form of dizziness that is a movement hallucination. Vertigo is most commonly felt as a spinning, usually due to a disturbance in the balance (vestibular) system. The balance apparatus provides us with a sense of head position in the space and is linked via the nervous system to the eyes, spine and various parts of the brain. The connection between the balance system and the eyes serves to maintain visual stability during head movement, while connections with the spine assist in the maintenance of postural stability. Connections to other parts of the brain provide conscious awareness of head position and movement. A patient may experience severe vertigo for days or weeks. Nausea, vomiting and involuntary eye movements are common. The condition gradually improves, but symptoms can persist for weeks and months.

Epileptic vertigo is vertigo due to epileptic activity in parts of the cortex that represent the vestibular system: the parietal, temporal and frontal cortex. It is a rare form of partial seizures. Specific areas include the superior lip of the intraparietal sulcus, the posterior superior temporal lobe and the temporal parietal border regions [2] as well as the V5 area [3]. If there is full consciousness during the seizure, the clinical symptoms and signs are considered relatively simple and the seizure is termed a simple partial seizure. If consciousness is impaired, the seizure is more complex and is termed a complex partial seizure.

Pathophysiology

There is no unique pathophysiology of the neocortical temporal lobe. Any destructive, neoplastic, vascular, or congenital epileptogenic lesion can result in seizures from this region. Potential spread patterns may be mesial structures, producing seizures similar to mesial temporal lobe seizures. Other potential spread patterns have not been extensively studied [4]. A head trauma can produce focal lesions that involve the temporal or parietal association cortex which receives vestibular projections. These lesions can occasionally form seizure foci which can lead to a simple or partial complex sensory

manifestation in episodic vertigo. The episodes usually last no more than seconds or minutes. They can be associated with nausea, but not vomiting. Nystagmus can be seen due to stimulus of the contiguous cerebral oculomotor nerve. Tinnitus can be associated at times. Contralateral paresthesias and/or olfactory and gustatory symptoms are occasional. Unconsciousness will follow only if the seizure becomes generalized.

History

Although nonconvulsive seizures with complex behavior have been recognized since antiquity [5), their relationship to temporal lobe origin was more recently recognized, being first described in the late 1800s by Jackson [6,7]. Bladin wrote in 1998 [8]: «In the 19th century it was believed that epileptic vertigo could come over the affected person, changing that person into an uncontrollable psychopathic beast of lethal potential». Because of the lack of technology with which the various forms of vertigo could be differentiated, every onset of dizziness might have had potential to evolve into epileptic seizures. In stage one both disorders were attributed to different degrees of brain involvement [8]. Stage two started in 1861 with Menier's inner localization of vertigo, followed soon by the empathic recantations from Charcot, Jackson and Gowers, who realized that vertigo was essentially otic, and that they had overlooked ear diseases. Considerable evidence linking epilepsy, vertigo and the ear needs explanation [9]. Lehman wrote in 1999 [9]: «The Van Gogh shows how muddled this area still is. Many, including Gastaut, considered him to be epileptic, yet Arenberg, a Meniere disease expert, was convinced that Van Gogh had Menier's».

The psychic and motor characteristics of these seizures first prompted the designation of psychomotor seizures [10, 11]. With the advent of electroencephalography and the increased interest in surgical intervention, because of their anatomical location, they were later termed as temporal lobe seizures [12]. Vertigo has been associated with epilepsy since ancient times, but it was almost certainly over-interpreted. True vertigo (tornado seizures) as a seizure symptom does occur, but only rarely. Over the years this symptom has been equated with seizure origin in the posterior temporal neocortex or the temporoparietal junction, others have reported it with frontal seizure onset. There are, overall, few well-documented examples.

Diagnosis

Epileptic vertigo is a diagnostic problem only when the person does not have a full seizure, in other words they do not have convulsions, psychomotor symptoms and twitching characteristics of classic partial or generalized seizures. In most cases it presents as a "quick spin" type symptom. The person notes that the world makes a quick horizon-

tal movement, lasting roughly 1-2 seconds at most. The quick spin must be differentiated from a variety of other conditions including vestibular neuralgia, due to microvascular compression, Menier's syndrome and BPPV [13].

Diagnostic tests that are particularly helpful include EEG and MRI scans of the head. When these tests are normal, a response to an anticonvulsant medication is suggestive of vestibular neuralgia. Vestibular epilepsy is diagnosed when the EEG is abnormal. It should be emphasized that many otherwise normal persons can have mildly abnormal EEG tests. When there is no response to medication, the probability of one of the other disorders mentioned above is increased.

All patients developing seizures should have a complete general and neurological examination. Focal neurological defects (impairment of fine finger movements, evidence of hemiatrophy indicating a cerebral lesion occurring in early life) should be sought, as well as specific signs which include the presence of any coetaneous stigmata that may indicate the cause of the epilepsy (cafe au lait spots, adenoma sebaceum or trigeminal capillary haemangiomas, suggesting the possibility of neurofibromatosis, tuberous sclerosis and Sturge-Weber syndrome).

The diagnosis of epilepsy is essentially clinical and relies on the description of the seizure provided by the patient and an eyewitness.

In the case of a new onset seizure, patients should have MRI scans early in the course of evaluation to look for structural lesions as the cause of seizures. Structural lesions, depending on their location and appearance, can have a significant impact on the management and evaluation. If the MRI is normal or nonspecific, no further evaluation is required and medical management should be initiated or continued. In the case of a documented medical intractability, a possibility of surgical intervention should be considered [14]. In addition, a magnetic resonance spectroscopy might help differentiate mesial temporal seizures from neocortical temporal lobe seizures [15]. Further diagnostic evaluation in the case of medical intractability would include video EEG monitoring, intracarotid amytal testing, ictal SPECT, neurophysiogical evaluation and possible intracranial EEG monitoring. Most cases of temporal neocortical epilepsy are associated with cortical lesions such as cortical dysplasias, neoplasms or vascular malformations and do not represent a specific syndrome.

Electroencephalography- an abnormal EEG is a major criterion for diagnosis. In most patients the abnormality consists of temporal or bitemporal sharp or slow wave foci. In some cases there are associated generalized discharges.

Treatment

Depending on the results of the evaluation, the management can take one of several directions. If the MRI is normal and neocortical temporal lobe seizures are suspected,

medical management should be undertaken. If, after a reasonable trial with appropriate antiepileptic drugs, seizures remain inadequately controlled, a surgery can be considered, although seizure onset localization can limit possibility of surgical treatment, particularly on the language-dominant side. However, with careful presurgical evaluation through experienced epilepsy surgery programs, surgical therapy can be successful even in patients with no obvious MRI abnormalities [16,17].

Differential diagnosis

Differential diagnosis of epilepsy includes: a basilar type migraine, confusional migraine, benign paroxysmal vertigo of childhood and an aura without a headache. The main differential diagnosis of neocortical temporal lobe seizures are mesial temporal seizures. When the MRI reveals potentially epileptogenic structural lesions in the mesial or neocortical temporal regions, the differential diagnosis is greatly simplified. Patients with a normal MRI and suspected mesial or neocortical temporal origin are much more challenging. Ictal SPECT scanning [18] and MRI can provide diagnostic data not otherwise obtainable [19]. In addition, a proton MR spectroscopy and PET studies can be helpful to distinguish the mesial from neocortical temporal lobe seizures [20].

Spread patterns to mesial structures would produce seizures similar to those beginning medially. As the mesial temporal lobe seizures mainly occur in association with a mesial temporal sclerosis, the presence or absence of certain risk factors such as complicated febrile seizures can help differentiate the two conditions [21] as can the results of intracranial amytal testing [22]. A recent study suggested some of the lateralizing findings associated with the mesial temporal lobe seizures (contralateral dystonic posturing, ipsilateral automatisms, etc.) present in a mirror image fashion in aura of any type, complex visual auras, or complex memory flashbacks should suggest neocortical temporal seizure onset, but this needs better documentation [23]. Dizziness may also occur in patients with multiple sclerosis. Hyperventilation associated by anxiety or emotional distress may cause dizziness, with or without tetany, but in such patients EEG shows epileptic activity.

Conclusion

Dizziness is a common symptom, but its diagnosis is not always easy. It may result from a dysfunction of the vestibular system at any point from the ear to the cerebral cortex. In these disorders the onset occurs later than in patients with epileptic dizziness, where the mean age, according to the study of Kogeorgos et al. [1], is about 25 years. Epileptic dizziness may often appear as a part of an aura in generalized seizures, but is then usually poorly defined. It is more commonly a component of temporal lobe sei-

zures, in 19% of the patients [1]. In temporal lobe epilepsy dizziness is not simply an aura, but constitutes a part of the seizure, and may be its only manifestation. The dizziness itself is often characteristic, consisting of sudden very brief episodes followed by rapid recovery without sequelae. The person notes that the world makes a quick horizontal movement, lasting one to two seconds at most. These quick spins must be differentiated from a variety of other conditions including vestibular neuralgia, Menier's disease, BPPV, etc. Epileptic dizziness is caused by abnormal stimulation of the part of the cortex that represents the vestibular system: parietal, temporal and frontal. Specific areas include the superior lip of the intraparietal sulcus, the superior temporal lobe, and the temporal-parietal border regions [2]. These episodes usually occur without the associated symptoms of epilepsy, in other words, there are no convulsions, psychomotor symptoms or twitching characteristic of classic partial or generalized seizures. The diagnosis of epileptic dizziness is often suspected because of the occurrence of separate symptoms of temporal lobe epilepsy which can be found only by careful questioning [1]. Diagnostic tests that are particularly helpful include EEG and MRI. Treatment of epileptic vertigo is usually successful with traditional anticonvulsants such as carbamazepine and its relatives [24].

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Sažetak

Diferencijalna dijagnostika vertiga i epilepsije

Vertigo je najčešće osjećaj vrtnje, uobičajeno kao posljedica poremećaja osjetila ravnoteže (vestibularnog sustava). Može nastati zbog disfunkcije vestibularnog sustava na bilo kojem mejstu od unutarnjeg uha do kore mozga.

Epileptički vertigo je rijetki oblik parcijalnih napada koji nastaje zbog epileptičke aktivnosti u dijelovima korteksa koji predstavljaju vestibularni sustav: parijetalni, temporalni i frontalni korteks. Napadi obično traju nekoliko sekundi do nekoliko minuta, a gubitak svijesti nastaje samo kada se napad generalizira.

Dijagnostičke pretrage uključuju EEG i MRI glave. Daljnja dijagnostička obrada u rezistentnim slučajevima uključuje: video EEG monitoriranje, intrakarotidno primjenu amitala, SPECT tijekom napada, neurofiziološku procjenu i intrakranijsko EEG monitoriranje. Poremećeni EEG je glavni kriterij za dijagnozu. U većine bolesnika poremećaji EEG-a uključuju temporalna ili bitemporalna žarišta šiljastih i sporih valova. U nekih slučajeva pridružuju se generalizirana epileptička izbijanja. Terapija epileptičkog vertiga obično je uspješna primjenom tradicionalnih antiepileptika poput karbamazepina i njemu sličnih lijekova. Ukoliko, nakon razumnog roka primjene adekvatnih antiepileptičkih lijekova, napadi nisu adekvatno kontrolirani, mogu se razmotriti i kirurške metode.

Diferencijalna dijagnostika epileptičkog vertiga uključuje: bazilarnu migrenu, konfuzijsku migrenu, benigni paroksizmalni vertigo u djetinjstvu i auru bez glavobolje. Najznačajnija ke diferencijalna dijagnoza neokortikalnih temporalnih napada od mezijalnih temporalnih napada. Iktalni SPECT i MRI mogu pružiti dijagnostičke podatke koji se ne mogu dobiti na drugi način. Protonska MR spektroskopija i PET mogu biti od pomoći u razlikovanju mezijalnih od neokortikalnih temporalnih napada.

Ključne riječi: epilepsija, epileptički vertigo, parcijalna epilepsija, neokortikalna epilepsija