ARTIGO DE REVISÃO/REVIEW ARTICLE

Musicogenic Epilepsy in Adults: Epileptogenesis and Management Update

Epilepsia Musicogénica no Adulto: Epileptogénese e Atualização da sua Abordagem

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Abstract

Musicogenic epilepsy (ME) is a rare form of reflex epilepsy in which seizures are triggered by exposure to musical stimuli, in particular, those with high emotional content. Despite rare and still very unknown, this disease can be very limiting and easily underdiagnosed. It is therefore important to increase clinicians' knowledge about ME, enabling better management of these patients.

The objective of this critical narrative review of the literature is to systematize what is known about musicogenic epilepsy in adults, highlighting the epileptogenic mechanisms and the updates in clinical management.

The included articles were selected through the PubMed database with the query "("Musicogenic") AND "Epilepsy"[Mesh])".

The estimated prevalence of ME is 1:1 000 000, with a slight female predominance. The mean age of onset is 28 years old (range: 2–67 years). The role and characteristics of auditory stimuli as triggers for seizures are variable and highly individualized, ranging from simple tones to complex music, and from loud, non-musical sounds to stimuli that are low in volume. The reported cases vary widely regarding the degree of musical training of the patients, occurring in professional musicians as well as in patients with no musical background at all. Seizures' onset zone is mostly located in the temporal lobe, with right-sided predominance. However, some concomitant emotional processing areas are also involved in ME, such as limbic structures, the nucleus accumbens, and the orbitofrontal and prefrontal cortex. The pathophysiology of ME has similarities with comorbid affective disorders, such as depression and anxiety disorders, supporting the involvement of affective networks in the development of ME. Recently there are reports of musicogenic reflex seizures in patients with antiGAD encephalitis. Therefore, all patients with ME should be screened for this pathology, since an earlier diagnosis will result in more effective treatment and a better prognosis. The treatment of musicogenic epilepsy includes some non-pharmacological approaches, and antiepileptic drugs in most cases, rarely culminating in surgery.

Despite being a rare disease, ME should be easily diagnosed and treated to reduce the repercussions in patients' life.

Resumo

A epilepsia musicogénica (EM) é uma forma rara de epilepsia reflexa, cujas crises são desencadeadas pela exposição a estímulos musicais, sobretudo com alto conte-

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údo emocional. Apesar de rara e pouco reconhecida, esta doença poderá ser muito limitante e facilmente subdiagnosticada. Assim, é importante expandir o conhecimento dos clínicos sobre a EM, possibilitando uma melhor avaliação destes doentes. Esta revisão narrativa da literatura tem como objetivo sistematizar aquilo que se sabe sobre epilepsia musicogénica nos adultos, destacando os mecanismos epileptogénicos e as atualizações na abordagem clínica. Os artigos incluídos foram selecionados por meio da base de dados PubMed utilizando a query "("Musicogenic" AND "Epilepsy"[Mesh])". A prevalência estimada de EM é de 1: 1 000 000, com um ligeiro predomínio do sexo feminino. A idade média de início é 28 anos (variação: 2–67 anos). O papel e as características dos estímulos auditivos desencadeadores das convulsões são altamente individualizados, variando desde tons simples a músicas complexas e desde sons de alto volume, não musicais a sons de baixo volume. Os casos relatados variam amplamente quanto ao grau de treino musical, havendo casos relatados tanto em músicos profissionais como em doentes sem formação musical. A zona de início das convulsões localiza-se, maioritariamente, no lobo temporal, com predominância do lado direito. No entanto, algumas áreas concomitantes do processamento emocional também estão envolvidas na EM, nomeadamente estruturas límbicas, núcleo accumbens, córtex orbitofrontal e pré-frontal. A fisiopatologia da EM apresenta semelhanças com algumas perturbações afetivas, nomeadamente depressão e perturbações da ansiedade, suportando a hipótese de um envolvimento das redes afetivas no desenvolvimento da EM. Recentemente, foi relatado o desenvolvimento de crises epiléticas reflexas musicogénicas em doentes com encefalite antiGAD. Deste modo, todos os doentes com EM devem ser rastreados para essa patologia, uma vez que um diagnóstico precoce poderá resultar num tratamento mais eficaz e, consequentemente, num melhor prognóstico. O tratamento da epilepsia musicogénica inclui, na maioria dos casos, abordagens não farmacológicas, associadas a fármacos antiepiléticos, raramente culminando em cirurgia. Apesar de constituir uma doença rara, a EM deverá ser precocemente diagnosticada e tratada, com o intuito de reduzir as repercussões na vida destes doentes.

Introduction

In reflex epilepsies, seizures are provoked by a specific afferent stimulus or patient's activity.[1] The stimuli precipitating reflex seizures can be visual, auditory, somatosensory or proprioceptive, as well as specific tasks such as reading, writing, eating, hot water immersion, amongst others.[2] Musicogenic epilepsy (ME) is a rare form of reflex epilepsy in which seizures are triggered by exposure to musical stimuli, in particular, those with high emotional content, reflecting the involvement of different auditory and emotional neural networks at seizure onset and propagation. Seizures in ME have a focal onset, mainly in the right temporal lobe, reflecting the nature of brain organization for music processing.[3-7] The pathophysiology is poorly understood and the type of music that can be epileptogenic is yet to be determined since the results of existing studies are not consont. In fact, in most cases, it is not possible to accurately identify which component of the musical stimuli is the epileptogenic trigger.[8] ME can be very disabling and easily misdiagnosed since it is still very unknown to most clinicians. Hereupon, narrowing this knowledge gap will enable a more conscious and informed future approach to ME patients by clinicians.

This work intends to make a critical narrative review of the literature, seeking to systematize what is known about musicogenic epilepsy, highlighting the epileptogenic mechanisms and the updates in the clinical management of these patients.

Methods

This is a narrative literature review based on a qualitative analysis of articles obtained from the electronic database PubMed (http://www.ncbi.nlm.nih.gov/

pubmed/) through the query: "("Musicogenic") AND "Epilepsy"[Mesh])". The research was restricted to the period from 1990 to September 2020 and limited to studies in humans and articles in English. We initially selected 76 articles. After reading the title and abstract, articles that involved children (2) or isolated cases (11) were excluded due to a low level of evidence, as well as those articles that were not fully obtained (15). After reading the remaining selected articles, we excluded 7 which were out of context or not considered relevant and finally included 41 articles in this narrative review (**Fig. 1**).

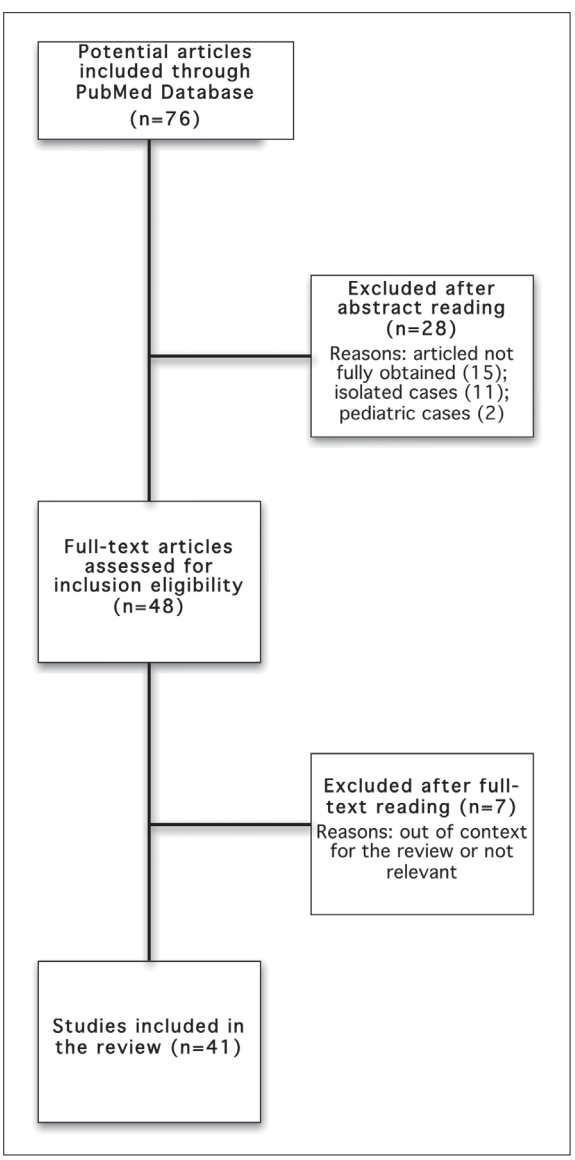

Figure 1. Flow chart of article selection process in the review.

Results

Epidemiology

The estimated prevalence of ME is 1:1 000 000 and there seems to be a slight female predominance (females/males = 54/46%).[4,9-11] However, since acoustic stimuli are not usually applied during routine electroencephalogram (EEG) in the assessment of epilepsy, the possibility of prevalence being higher is very plausible.[4,5,9,12] The mean age of onset is at 28 years old (range: 2–67 years).[3,4,9,10,12]

Music as a trigger for epilepsy

Music is an organization of sounds with aesthetic, artistic or playful intentions, variable according to the author, geographical area, historical epoque, among other features. It can be categorized as instrumental, vocal, or as a subtype of vocal music which includes songs combining instrumental music and the human voice.[9] The characteristics of the auditory stimuli triggering seizures are variable and highly individualized, ranging from simple to complex tones, as well as from loud to low in volume. In addition, physical qualities of the stimuli, including tone and bandwidth, may also be important.[4,9,10,13-15] The ME auditory triggers can be divided into different categories, such as music, sounds/tones and a combination of both, with a higher prevalence in the music category (78%), followed by the combination of music and sound (14%) and, lastly, by the isolated sound/tone category (4%).[4] There is no characteristic pattern of the type of music reported as a trigger by these patients. It varies according to several aspects, namely the instruments of the music (highlighting the organ and piano); content of music; types or categories of music (classical, popular, religious, lyric, jazz, instrumental); languages; composers; familiarity of the sound.[4,9,10] Curiously, some reports suggest that seizures may be triggered not only by the act of listening to music, but also in situations where patients are playing their instrument, or even just thinking about a certain song or sound.[2,4,12,16]

Musical skill and its association with ME

It is known that musical training has an influence on brain anatomy, and its impact is related to the age of initiation and the intensity of practice. Imaging data show a larger corpus callosum and primary motor cortices, predominantly on the right side in people with musical training.[5] During musical stimuli, neural networks res-

ponsible for musical processing are activated. Therefore, it is logical that if there is a sensitization of these hyperexcitable cortical regions, there may be a greater propensity for the development of epilepsy, namely ME.[5,15] However, reported cases vary widely in the degree of musical training of the patients, namely from professional musicians to patients with no background in music at all.[8,10,17,18]

Understanding epileptogenesis in ME

Several studies have used EEG and advanced functional imaging in the search for an understanding of how the brain generates seizures in patients with ME. Most of these exams are not usually applied in clinical practice, but they provided data that allowed to identify areas of the brain related to epileptogenesis in these patients. We describe the most important findings of these studies:

- **EEG**: There is evidence that epileptogenic sounds induce bursts of theta activity over the right posterior temporal region and high-voltage spiking bursts in both frontal areas, with a slight predominance over the right hemisphere.[19] The ictal and interictal EEG findings are in tune with some single-photon emission computerized tomography (SPECT) and functional MRI (fMRI) studies, indicating hemisphere lateralization, with a predominance of the epileptic activity on the right side.[8,20]
- **SPECT:** Ictal SPECT findings demonstrate the dominant role of the right temporal lobe in ME, including mesial temporal lobe structures, with earlier changes in the metabolism in the temporal lobe, ipsilateral to the expected focus. There is a right neocortical posterior temporal hypoperfusion and a right temporal antero-polar hyperperfusion at the peak of seizure, including deep temporal lobe structures, and involvement of fronto-orbitary and insular regions as well.[2,4,8,11,20-25] The contribution of mesial temporal structures is in line with the clinical presentation of most patients, with episodes of *déjà-vu* and automatisms like lip smacks and swallowing.[8] The immediate postictal scan presents moderate hypoperfusion of the right temporal lobe, supporting the observations during the ictal period.[8]
- Interictal Fluorodeoxyglucose positron emission tomography (**FDG-PET**): Most studies identified right temporal hypometabolism in ME patients, indicating interictal dysfunction in these brain areas.[3,10,26-28,29]
- **fMRI**: The fMRI studies revealed a cascade of recruitment, with frontal lobe involvement as propagation from a temporal lobe origin.[3] Performing simultaneously EEG and fMRI during a musical trigger, it was possible to identify in the ictal fMRI of these patients an increase in BOLD signal, with significant signal change within bilateral frontal lobes (with a predominance of the right side), including the insula, middle cingulate cortex, orbitofrontal cortex, accumbens, and frontal pole, and in the right temporal lobe.[2,3,19,30,21] In the initial period of the seizure, it appears that the signal in the frontal lobe is unilateral, progressing to the front and finally becoming bilateral. The change in cerebellar signal is contemporary, with bilateral expansion as well. These findings are consistent with the patient's behavioral changes when the seizure progresses and with the ictal activity recorded on the EEG.[3] In the final 30 seconds of imaging, fMRI localized the activity in bilateral frontal lobes, bilateral cerebellum, and the right temporal lobe. This moment corresponds to the aura.[3,21]
- Magnetoencephalography (**MEG**): The studies identified a right temporal onset with spread to the right frontal region.[3,31]

From these findings, we can say that ME has consistent lateralization to the right hemisphere, with early changes in the posterior temporal lobe and propagation through the limbic system and anterior frontal region, highlighting the intrinsic relationship between areas belonging to limbic circuits and emotional processing and control.[3,21,23,31-33] Thus, the hypothesis that ME is a limbic epilepsy should be considered, since temporal and frontal limbic regions are involved: on the one hand, the gyrus rectus is probably associated with the response to the trigger music and, on the other hand, the orbitofrontal and anterior temporal lobes are involved in the occurrence of auras.[3,30]

The role of emotions and affective disorders in ME

It is known that the primary auditory cortex is not the usual generator of the seizures in ME and that the affective experience of music has a relevant role in the development of these reflex seizures. This knowledge led to speculation that the pathogenesis of ME may be

more related to the intermediary emotions produced by music than to its pure auditory content.[3,20] Therefore, besides the cortical areas related to audition, there may be other areas involved in triggering seizures in ME, in particular those who contribute to cortical processing of acoustic stimuli and emotion.[21] The majority of seizures in ME manifest as focal dyscognitive seizures with automatisms. However, given the epileptic sensitivity of the limbic system to sensory input through the neocortex, subsequent processing at this location may produce the seizures. The exposure to music is usually a pleasurable and emotional situation, so the trigger music is often a familiar and known song to the patient (or parts from these songs), regardless of the specific technical characteristics of that music (such as tone, timbre or the melody itself), further reinforcing the hypothesis previously mentioned.[3,4,9,10,14] Functional tests are also in line with this hypothesis: for example, the observed pattern of BOLD activity in fMRI shows this dominance of emotion-related areas in the period immediately preceding seizure onset.[21] This emotion resulting from different possible combinations of sound characteristics, namely the melody/lyrics combination, or even the attempt to confine that same emotion, may be at the basis of the epileptogenesis, highlighting the role of areas related to the control of emotions.[21] In fact, if we take into account the cases described so far, we can conclude that the songs with the highest emotional load are the major trigger of seizures in ME.[7]

The pathophysiology of ME has similarities with comorbid affective disorders, such as depression and anxiety disorder. In fact, in the three entities, there is a deficit of serotonergic and noradrenergic function. This relationship is another factor in favor of the importance of the processing of affective networks in the development of ME.[1,12,34] It is important to highlight this point, given the high prevalence of cases of ME closely related to the emotions provoked by sound stimuli. This will therefore have implications in treatment.[34] However, it is necessary to safeguard that specific mechanisms responsible for the seizures in ME still remain very much unknown.[7,19,21,27,34]

The association of ME and autoimmune encephalitis

Anti-GAD antibodies are associated with different neurological pathologies, including limbic encephalitis (LE), temporal lobe epilepsy (TLE), stiff-person syndrome (SPS), cerebellar ataxia and eye movement disorders.[28,35] Epilepsies related to the elevation of this antibody are mainly unexplained focal epilepsies, beginning in adulthood, with predominant involvement of the temporal lobe (LT), manifesting as drug-resistant seizures. There seems to be a greater likelihood of anti-GAD encephalitis manifesting as chronic, slow-developing focal epilepsies that have become drug-resistant, instead of acute epilepsies usually associated with limbic encephalitis. There are several reports of patients with ME with positivity for anti-GAD antibody. However, its correlation with this antibody is not yet fully understood. There may be a possible link between epilepsy in anti-GAD encephalitis and ME since they present similar anatomical and clinical characteristics. In addition, interestingly but not surprisingly, there seems to be an association between the positivity of the anti-GAD antibody and the simultaneous presence of ME and SPS.[36] Extrapolating to clinical practice, this association could be the key to a possible early diagnosis of patients with SPS and other autoimmune diseases associated with anti-GAD--antibody, namely, anti-GAD autoimmune encephalitis, considering the presence of ME as a clue to the diagnosis of these pathologies.

Clinical features

In people with ME, both spontaneous and reflex seizures may occur, with two-thirds having spontaneous seizures preceding musicogenic seizures. There are commonly autonomic and automotor manifestations (25% oroalimentary automatisms), according to the main area of origin of the seizures in the right temporal lobe.[3-7] As in other temporal lobe epilepsies, patients often report an epigastric aura, with abdominal discomfort, nausea, tightness and an unpleasant ascending epigastric sensation, dysautonomic features, hand paresthesia, flushing, tachycardia, oroalimentary automatisms, *déjà-vu* sensations, with loss of awareness during longer seizures. Rarely there is a secondary generalization.[3,6,25] Seizures are initiated by an auditory trigger.[37] The time lag between the stimulus and the seizure ranges from an almost instantaneous seizure to a seizure that develops a few minutes after the stimuli.[5,7,25,38] There are some reports of agitation, tachycardia and shortness of breath during this delay.[5]

Diagnostic evaluation

For a complete diagnosis, in addition to the detailed clinical history of seizures and their triggering factors, an EEG with acoustic stimuli and a brain MRI are essential. When the diagnosis of ME is plausible and brain MRI does not show a structural lesion, it is also recommended to search for anti-GAD antibodies, given that sometimes anti-Gad autoimmune encephalitis may present with temporal lobe epilepsy with reflex musicogenic seizures.[26] An earlier diagnosis will result in more effective treatment and a better prognosis for these patients.

Treatment options

Despite being a rare condition, it is easy to understand that musicogenic epilepsy can have serious consequences in the daily lives of patients. Usually, they can identify their own music or sound trigger, leading to its avoidance. However, seizures may be so impactful that these patients end up developing an aversion to all types of music, leading to musicophobia. This clinical situation can culminate in other psychological and/or psychiatric conditions since these patients will have a greater tendency to isolate themselves socially and develop depression. This medical condition can be particularly limiting in certain populations such as professional musicians. Since some studies (but not all) point out the fact that high musical expertise may lead to a greater predisposition to ME, it is very important to find solutions for these patients, so that they can have a relatively normal day-to-day life with no need to be deprived in their professional or leisure activities.[12,39]

The first-line treatment of ME, just like in any other type of epilepsy, are antiepileptic drugs (AED).[12,34] However, since it is mainly a reflex epilepsy, if the precipitating music can be identified, it may be possible to implement therapeutic measures that go beyond medication,[4,12,39] and may even prevent the AED from being taken continuously. In terms of non-pharmacological approaches, we can summarize them in the following proposals:

1) Strictly avoiding the stimuli: if the epilepsy is strictly musicogenic, only provoked by a single identified musical stimulus, it may be controlled by avoidance of the trigger or by diverting the attention from it. However, the success of this measure will depend on the trigger sound, since there are sounds that can hardly be avoided.[40]

2) Modifying the stimuli: since triggers can vary from high to low in volume, if the patient takes this into account, he may be able to control, at least partially, the development of seizures by controlling the sound volume. The same applies if the main problem is the length of exposure time. However, once again, the success of this measure will depend on the stimuli.[40]

3) Desensitization: this technique can be done in different ways: through the repetitive presentation of the changed stimuli, namely, in its intensity; through stimulation in the postictal refractory period, thus altering the trigger threshold; by inhibiting surveillance; or still, due to conditioning of avoidance. Constant reinforcement and training are necessary, which may become incompatible with its applicability.[40]

4) Inhibition: Training to acquire the capacity to recognize and prevent the development of the seizures may involve relaxation techniques, clue--controlled arousal, the imagination of the aura followed by the application of the stimulus and biofeedback of the cortical direct current (DC) potential.[40]

Knowing that in many ME there is a time lag (seconds to several minutes) between the stimulation and the development of ictal activity, these non-pharmacological techniques may benefit from this factor and act in this pre-ictal period.

When patients that only have reflex seizures can predict when they will be exposed to the stimulus, it is possible to prescribe an AED a little time before the expected exposition. In these cases, the most suitable drugs are benzodiazepines.

Based on the previously mentioned hypothesis that the pathogenesis of seizures is, in large part, associated with the affective and emotional content of the trigger, and given the considerable prevalence of affective disorders (namely depression and anxiety) in these patients, it may be adequate to complement the therapy of these patients with antidepressants. Selective serotonin receptor inhibitors (SSRI) seem to be the ideal drugs.[34] Citalopram and fluoxetine are two of the drugs already used and which seem to have good results in controlling these pathologies, with a reduction in the frequency of seizures.[34] In cases of anti-GAD related epilepsies, immunosuppressant drugs must be used, besides AED.

Surgical treatment should be considered in the rare cases of ME refractory to medical treatment, especially those with intractable seizures with unilateral focal onset, as well as in cases of patients with a brain lesion causing epilepsy. That is the case of brain tumors that progressed from low-grade to high-grade malignancy.[12,24] However, in situations of independent bilateral foci or without structural lesion, vagal nerve stimulation (VNS) treatment may be considered.[24] A complication of this type of surgery that should not be overlooked, is the effect on patient's musicality. This impact may translate into the impaired capacity to recognize songs or altered musical perception for pitch, timbre, tone and rhythm.[12] This compromised musicality can have very significant consequences if we are, for example, facing a case of a professional musician, whose profession will be highly compromised.

Conclusion

Musicogenic epilepsy is a probably underdiagnosed reflex epilepsy evoked triggered by a musical stimulus, especially with high emotional content. Some patients may also have unprovoked seizures. Clinicians should be aware of the possible occurrence of musicogenic reflex seizures in patients with autoimmune encephalitis with anti-GAD antibodies. Therefore, all patients with ME must carry out the determination of anti-GAD antibodies, even those with normal structural MRI. This correlation may allow an earlier diagnosis and treatment of autoimmune diseases associated with anti-GAD antibodies. There is a high prevalence of affective disorders in ME so the role of SSRI should not be underestimated. Although the primary treatment of ME involves antiepileptic drugs, non-pharmacological approaches may also be implemented, namely stimulus avoidance, stimulus modification, desensitization and inhibition. These measures may help the patient to have a life as normal as possible. ■

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