Short communication

Bilateral acute anterior uveitis and anticonvulsant hypersensitivity syndrome

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ABSTRACT

Clinical case: A case is presented of a patient who developed skin lesions on the legs and a bilateral anterior uveitis after taking Oxcarbazepine (Trileptal).

Discussion: The presence of uveitis in the context of an allergic reaction to an anticonvulsant is a very uncommon and can affect the internal organs. For this reason, it should be taken into account when carrying out a full assessment of a patient with a possible hypersensitivity reaction to a particular drug.

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Keywords:
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- Bilateral
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Uveítis anterior bilateral aguda e hipersensibilidad a anticonvulsivantes

RESUMEN

Caso clínico: Se presenta el caso de una paciente que tras la toma de oxcarbacepina (trileptal) desarrolló lesiones cutáneas en miembros inferiores y una uveítis anterior bilateral.

Discusión: La presencia de uveítis en el contexto de una reacción alérgica a un anticonvulsivante es un hecho poco frecuente que transduce una afectación de órganos internos, por lo que debe ser tenida en cuenta en la valoración completa de un paciente con posible reacción de hipersensibilidad a un determinado fármaco.

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Introduction

The clinical presentation of hypersensitivity to an anti-epileptic drug is highly variable. The typical triad comprises fever, skin rash and systemic involvement. In the latter, uveal involvement is infrequent.

Clinical case

A patient, aged 34, visited the emergency ward of our hospital referring pain and blurred vision in both eyes with one week of evolution.

The ophthalmological exploration revealed a visual acuity (VA) of one unit in both eyes, the presence of cellular tyndall of 2+ and fine keratic precipitates in both eyes. The ocular fundus assessment was normal.

The patient was being assessed by the Neurology service for epileptic attacks that require treatment with oxcarbacepine (Trileptal® Novartis). One month before the bilateral anterior uveitis episode and matching an increase in the oxcarbacepine dosage, the patient developed skin lesions in the lower limbs. Said lesions were biopsied and reported as leucocytoclastic vasculitis with moderate eosinophilia, probably associated to the administration of carbamacepine. The patient did not exhibit any other sign or symptom of systemic involvement. The analytics gave normal results.

Oxcarbacepine was substituted by topiramate (Topamax®, Ortho-McNeil Neurologics). The patient was treated with topical dermic steroids, exhibiting a rapid regression of the skin lesions. In addition, the ocular topical treatment with steroids and cyclopegic achieved a complete resolution of the anterior ocular inflammation in both eyes.

Discussion

The epileptic hypersensitivity syndrome is a reaction induced by drugs. It courses with fever, skin rash, eosinophilia and systemic findings that include lymphadenopathy, liver function alterations and kidney alterations, among others. In 1988 the term “anticonvulsive hypersensitivity syndrome” was coined to describe the large range of symptoms related to aromatic anti-convulsive drugs (carbamacepine, phenytoine, phenobarbitral). Recently this clinical entity has been renamed as “drug-induced hypersensitivity syndrome”. However, no universally accepted term is utilized to designate said condition. The exact prevalence of this syndrome is not known due to the variability of its presentation and lack of strict diagnostic criteria.

Considering the criteria applied by the WHO (the Naranjo algorithm) to assess the cause of adverse reactions to a drug, our patient exhibited a score of 7. This includes her in the probable allergic reaction category. The anticonvulsive hypersensitivity syndrome lacks clearly defined diagnostic criteria, but the literature seems to agree in that it is characterized by the typical triad of fever, exanthema and involvement of internal organs. Within the ocular expressions, the literature describes dyopia, conjunctivitis, visual alterations and a single case of unilateral uveitis. Our patient did not exhibit fever throughout the episode; instead, the expression was in the form of characteristic skin lesions in lower limbs, confirmed with said biopsy.

The pathogenic mechanisms of idiosyncratic reactions to drugs are not fully defined. Sullivan and Shear proposed a multifactorial model, according to which the development of said syndrome would be determined by the combination of a susceptible patient and exposure to a drug in sufficient dosage and duration to cause said reactions.

Another alternative for the pathogenic mechanism is related to an active viral infection concomitant with the utilization of antiepileptic drugs: lymphocytes CD4 and CD8, recruited for the immune response against the virus, would exhibit a crossed reaction with the antiepileptic drugs. This theory is supported by descriptions of reactivation of infections due to CMV, VHS and VEB associated to an anticonvulsive hypersensitivity syndrome. Finally, another proposed pathogenic mechanism describes the appearance of reactive intermediate compounds during the metabolization of aromatic anticonvulsives through the pathway of cytochrome p450 (arene oxides) that the body is not able to eliminate due to structural or functional damage of the appropriate enzyme (epoxide hydroxylase), which give rise to direct cell death, also regulated by hypersensitivity type IV. Most cases of adverse reaction to anti-convulsive drugs have been described in association with phenytoine, carbamacepine and phenobarbital, with very few cases published in the literature referring oxcarbacepine.

The presence of uveitis in the context of an allergic reaction to an anti-convulsive drug is an infrequent occurrence which involves internal organs. Therefore, it must be taken into account in the full assessment of a patient with possible hypersensitivity reaction to a given drug.

Conflict of interest

The authors declare they have no conflict of interest.

References