Reumatol Clin. 2010;**6(1)**:71

Reumatología Clínica

www.reumatologiaclinica.org

Letter to the Editor

Ageusia associated to treatment with pregabalin

Ageusia asociada a tratamiento con pregabalina

Mr. Editor:

Pregabalin (PGB) is an analogue of the neurotransmitter gamma amino butyric acid (GABA) that has not shown any direct GABA-mimetic effects, but that increases neuron concentration of GABA.[1] It has analgesic, anti-epileptic and anxiolytic effects,[2] and was approved in 2004 by the European Drug Agency for the treatment of peripheral neuropathic pain (diabetic polyneuropathy and post-herpetic neuralgia), for partial epileptic crisis with or without secondary generalization and for generalized anxiety disorders in adults.[2-5]

We present the case of a rare adverse event of PGB: ageusia or loss of taste associated to treatment with PGB in a patient with neuropathic pain due to spinal cord compression.

The patient is a 49 year old male teacher with no history of importance or toxic habits, who presented lumbar back pain and sciatica since two months prior. He had not had any similar events in the past and had not carried out important physical activity. His family physician started treatment of muscle relaxants and non-steroidal anti-inflammatory drugs, without any improvement, and a magnetic resonance imaging (MRI) study was carried out which demonstrated a disc hernia at L5-S1 with cord compression. With this diagnosis, surgical treatment was proposed, something that was rejected by the patient, starting PGB (75mg at night, increasing to 75 mg/12 h after 7 days), in addition to paracetamol (1 g every 12 h). After 15 days, the patient presents complete loss of taste, not being able to distinguish salty from sweet tastes, and treatment with PGB is stopped. A central nervous system MRI is obtained showing no alterations. Four months after suspending PGB, the patient starts to recover the sense of taste, with complete recovery at 10 months.

According to the products drug insert,[5] clinical trials in all of the combined populations (neuropathic pain, postherpetic neuralgia, epilepsy and generalized anxiety disorders) 14% of patients treated with PGB and 7% of patients treated with placebo abandoned the study prematurely due to adverse events. In the PGB group, the most frequent adverse events were dizziness (4%) and somnolence (3%), with a lesser frequency (1%) of other effects that also motivated the discontinuation of treatment and that also appeared in the group of patients under treatment with PGB: ataxia, confusion, fatigue, difficulty to concentrate, lack of coordination, transitory loss of visual acuity and peripheral edema. The insert also documents, among the rare side effects, loss of taste,[5] which was present in one patient out of 100 and 1,000 treatments.

Alterations in the sense of taste can be due to conditions that interfere with access to the substance to be tasted to receptors on taste cells or in the neuronal pathways involved n transmitting the nervous stimulus to the cerebral cortex. In clinical practice, the most common cause for loss of taste is drug treatment. Drugs that are more commonly associated to these alterations are those containing sulphydril groups (penicyllamine, captopril), but are also produced by products without the sulphydril geoups (enalapril, metronidazole and certain anti-neoplastic agents).[4]

In the case described, the dose of PGB was low and the time since drug start was short, leading to the conclusion that it was an idiosyncratic reaction.

Because of the satisfactory resolution of the problem after suspension of the drug, with complete recovery of the sense of taste, no complementary testing was performed, such as tests of the perception of the quality and intensity of tastes or neuronal conduction tests, which would allow for the precise determination of the taste deficit in the different quadrants of the tongue.

PGB is a very commonly used drug in clinical practice, which must be considered in spite of this very rare side effect.[5]

References

1. Tremon-Lukaats IW, Megeff C, Backonja MM. Anticonvulsionants for neuropathic pain syndromes: Mechanism of action and place in therapy. Drugs. 2000;60: 1029-52.
2. López-Trigo J, Sancho J. Pregabalina: un nuevo tratamiento para el dolor neuropático. Neurología. 2006;21:96-103.
3. Tassone DM, Óbice E, Guyer J, Nuzum D. Pregabalin: A novel gamma-aminobutyric acid analogue in the treatment of neuropathic pain, partial onset seizures, and anxiety disorders. Clin Ther. 2007;29:26-48.
4. Henkin RI. Drug induced taste and smell disorders. Drug safety. 1994;11:318-77.
5. Lyrica®. Ficha técnica. Available at: www.emea.europa.eu/humandocs/PDFs/EPAR/lyrica(H-546-PI-es.pdf

Victor González Vallés, Javier Calvo Catalá,*
Cristina Campos Fernández, and Amalia Rueda Cid

Unidad de Reumatología y Metabolismo Óseo,
Consorcio Hospital General Universitario, Valencia, Spain

* Corresponding author.
E-mail address: calvo_jav@gva.es (J. Calvo Catalá).