CC 101. Glossopharyngeal neuralgia due to lacunar infarction of PICA: an unusual case

María E. Novoa M.[1], María Alfaro-Olivera[1]

1.- Instituto Nacional de Ciencias Neurológicas, Lima-Perú.

Introduction

Glossopharyngeal neuralgia (GPN) is of sudden onset, lasting seconds, usually caused by cold stimuli when chewing, swallowing, talking, coughing, yawning, and sneezing. Painful paroxysms are characteristic, located mostly in the oropharynx and irradiated to the middle ear or vice versa, due to involvement of the pharyngeal branch or auricular branch of the glossopharyngeal nerve (GN). According to the ICHD-3 (International Classification of Headache Disorders-3) classic GPN occurs due to vascular compression. Secondary GPN, due to underlying disease; while idiopathic GPN has no causal evidence. Incidence of 0.7/100,000/year, 9:5 males over females, average age 64 years, left location, 12:9. Infarcts of the posterior inferior cerebellar artery (PICA), an unusual cause of GPN, involving small vessels, associated with risk factors, would explain the ischemic etiology. They represent 40% of cerebellar infarcts, compared to the anterior inferior cerebellar artery (AICA) and the superior cerebellar artery (SUCA). The PICA is divided into 5 segments; anterior medullary (p1), lateral medullary (p2), tonsillomedullary (p3), telovelotonsillar (p4) and cortical (p5). Being the lateral spinal cord segment (p2) the most involved. Represented by Wallenberg syndrome. T2-weighted imaging (T2WI) is more sensitive than fluid-attenuated inversion recovery (FLAIR) in identifying posterior fossa infarcts.

Objective

We present an unusual case of GPN, secondary to a lacunar infarct in the p2-segment of the PICA.

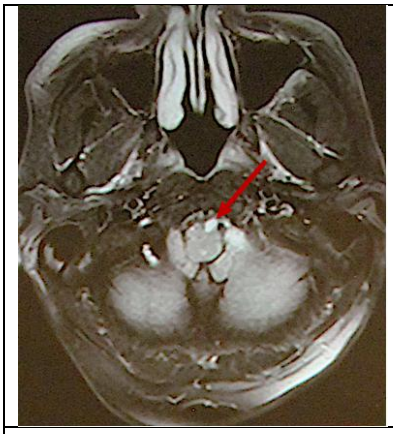
Case description

65-year-old male, with no apparent pathological history. He reports sudden oropharyngeal pain, radiating to the mandibular angle and left ear, as bursts of electric shocks between 7 and 10 seconds, 2 times a day, when chewing, swallowing and speaking. Concomitantly, dizziness and vertigo are limited to the week of onset of pain. Oropharyngeal tactile stimulation causes a burst of pain that paralyzes the patient for a few seconds. The T2 sequence (T2W1) of the MR showed a small hyperintensity in the lateral medullary segment of the left posterior inferior cerebellar artery (PICA). Uncontrolled arterial hypertension, cardiac arrhythmia and hypothyroidism were the risk factors found in the patient. Indicating pregabalin (75 mg/1 time/day) and the management of heart attack risk factors. The case presented did not characterize Wallenberg syndrome. However, it compromised the prominence of the bulbar olive, up to the origin of the rootlets of the GN, where the pharyngeal branch of the left GN could be injured and cause GPN. Meanwhile, the scant surrounding edema in the small ischemia would promptly explain the limited dizziness and vertigo. The theory of

mechanisms of hyperexcitability and ephaptic transmission in central neurons, activating N-methyl-D-aspartic acid receptors in the GN. They would explain the efficacy of antiepileptic drugs in neuralgia. Pregabalin at minimal doses controlled the patient's outbursts of pain.

Conclusion

GPN of small infarct in the PICA territory is an unusual condition. In our case, the lateral anteromedullary segment of the left PICA compromised the ipsilateral GN. The T2W1 of the MR sequence revealed hyperintensity in PICA-p2. Management with pregabalin was effective.



MRI T2W1 sequence showed small hyperintensity in left PICA-p2.