Cerebrovasc Dis 2013;36:153–154
DOI: [10.1159/000352063](https://doi.org/10.1159/000352063)**Hypertrophic Olivary Degeneration after Pontine Hemorrhage: A Cause of Delayed Neurological Deterioration**Inês Menezes Cordeiro^{a,c}, Joana B. Tavares^b, Sofia Reimão^b, Ruth Geraldes^a, José M. Ferro^a^aStroke Unit, Department of Neurosciences, Santa Maria Hospital, University of Lisbon, and ^bNeuroimaging Department, Santa Maria Hospital, Lisbon, and ^cNeurology Department, Faro Hospital, Faro, Portugal**Background**

In patients who suffer acute stroke, the improvement of neurological deficits varies. Late-onset neurological worsening, after an initial clinical improvement, may be caused by prolonged focal motor seizures [1], cognitive decline and dementia [2], depression and apathy [3], emotional disturbances [4] and fatigue [5].

Hypertrophic olivary degeneration is associated with late-onset neurological worsening after brainstem injury, but this has seldom been reported after stroke [6–9].

Case Report

A 71-year-old female suffered a sudden onset of diplopia, dysarthria and paresthesias of the left hemiface and the upper left extremities. She had hypertension and dyslipidemia.

Upon admission, the patient showed a right IV cranial nerve palsy, skew deviation, right internuclear ophthalmoplegia, dysarthria, left central facial palsy, left hemihypoesthesia, right appendicular ataxia and gait ataxia.

Brain CT showed two acute intraparenchymal hemorrhages, a larger one in the pontine tegmentum adjacent to the right superior cerebellar peduncle, and a second one in the homolateral basis ponti. Brain MRI (on day 3) confirmed these two subacute hemorrhages, which involved the central tegmental tract (fig. 1a, d).

The patient was discharged 11 days later with a modified Rankin Scale (mRS) score of 4. During the following weeks, the patient improved and she could walk with unilateral assistance (mRS score 3).

Fig. 1. Brain MRI – imagiological evolution of hypertrophic olivary degeneration shows progressive hyperintensity signal on T2-weighted MRI and hypertrophy of the bulbar olive. Axial T2-weighted images: day 3 (a), month 11 (b) and month 18 (c). Coronal gradient-echo T2-weighted images: day 3 (d), month 11 (e) and month 18 (f).

Starting 3 months after stroke, a progressive neurological deterioration with worsening of the dysarthria and ataxia was noticed, conferring increased disability at 6 months (mRS score 4). Brain MRI at 11 months disclosed a hyperintensity on T2-weighted sequence and mild hypertrophy of the right anterolateral part of the medulla, corresponding to the inferior olivary nucleus, consistent with hypertrophic olivary degeneration (fig. 1b, e).

Eighteen months after the initial injury, MR showed an increase in the size of the olivary hypertrophy (fig. 1c, f).

At the last follow-up examination, 2 years after the stroke, the patient had also developed palatal myoclonus.

Discussion

Hypertrophic olivary degeneration is a rare condition, caused by the interruption of the neuronal connections of the dentatorubral olivary pathway, often referred to as the 'Guillain Mollaret triangle' [6].

It is considered a form of transsynaptic degeneration, following the disconnection of afferent axons to the inferior olivary nucleus [7].

Hypertrophic olivary degeneration occurs after a focal lesion in the brainstem and is characterized by a hypertrophic response of the inferior olivary nucleus to deafferentation [8].

The current view is that the enlargement of the olivary nucleus occurs as a consequence of the lack of inhibition after its disconnection from the dentate nucleus, whose projections to the olivary nucleus are mainly inhibitory GABAergic neurons [10].

Clinical findings include palatal myoclonus, dentatorubral tremor, ataxia, dysarthria and diplopia, appearing months after the initial insult [9, 10].

There are few reported cases in the literature, most after pontine hemorrhage but also in association with tumors and neurosurgical intervention [6, 7, 10].

There is scarce information regarding the prognosis of these patients due to underreporting of long-term follow-up.

This case report underlies the importance of recognizing this condition and its association with delayed neurological deterioration after stroke.

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