Curious Case of a Medullary Lesion following Pontine Cavernoma Resection

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Question

A 36-year-old gentleman, presented with right hemiparesis and internuclear ophthalmoplegia, was operated for left pontine cavernoma [Figure 1]. He followed up in outpatient department with gradual onset of palatal myoclonus 13 months post-surgery post-surgery [Figure 2 and 3]. Patient was evaluated with magnetic resonance imaging (MRI) brain which



Figure 1: Fluid attenuated inversion recovery sequence magnetic resonance imaging brain showing subtotal excision of the left pontine cavernoma



showed the above findings. Can you the guess the sequelae of pontine surgery in this patient? For answer turn to next page.



Figure 2: Axial sections of magnetic resonance imaging brain – (a) (T2W sequence), (b) (T1W post contrast), and (c) fluid-attenuated inversion recovery sequence



Figure 3: T2W coronal sequence with residual cavernoma in the left pons and hyperintensity in the left medulla

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Answer

Left sided hypertrophic olivary degeneration (HOD).

Explanation

The patient was evaluated with MRI brain which showed a hyperintensity in the left inferior olivary nucleus (ION). With a precedent history of brainstem surgery, a rare manifestation of pathology in the Guillain-Mollaret triangle causing HOD was ascertained. Figure 4 depicts the Guillain-Mollaret triangle and its connections.^[1,2] With unilateral hypertrophy of ION, pathology is either in the red nucleus or the central tegmental tract (CTT). In this case, it is more likely the CTT on the left side is affected following the surgery



Figure 4: Illustrative diagram of Guillain-Mollaret triangle

of the left pontine lesion.^[3] There is transsynaptic transneuronal degeneration of the left ION. This degeneration is unique as it causes hypertrophy of the ION rather than atrophy. Focal signal changes seen in the inferior medulla are not pathognomonic for HOD. Non-enhancement on contrast MRI differentiates it from other likely causes such as infarction, demyelinating disease, malignancy, infections, and inflammatory processes could also produce similar signal changes.^[3,4] Symptomatic patients are difficult to treat and rarely resolve, but successful management of symptoms with benzodiazepines and carbamazepine has been reported.

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