Acute Dysphagia and Dysphonia Associated with Anti-GM3 Ig Antibodies: A Case Report

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INTRODUCTION

Gangliosides are glycosphingolipids that contain one or more sialic acid residues linked to the oligosaccharide core. They are highly abundant in the nervous system, especially in neuronal membranes, where they participate in cell signaling and cell-to-cell communication. Anti-ganglioside antibodies can be associated with Acute Inflammatory Demyelinating Polyneuropathy (AIDP) or Acute Motor Axonal Neuropathy (AMAN) or their variants and Chronic Inflammatory Demyelinating Polyneuropathy (CIDP).

We describe a case of acute clinical onset of dysphagia and dysphonia worsening in six months, with detection of GM3 antibodies in the serum.

MATERIALS AND METHODS

- 74-year-old male patient
- Sudden dysphagia and hypophonia
- EN: hypomobility of the soft palate on the left and left facial asymmetry with diminished nasolabial fold prominence
- Previous PEG at another centre

- Auto-Antibodies
- Onconeural Antibodies
- Tumor markers

- ORL assessment whit fibrolaryngoscopy
- Neurophysiological assessment: ENG, EMG and F waves
- swallowing reflex

- Full body CT scan with contrast
- Spine MRI with contrast
- Body fluorodeoxyglucose PET

RESULTS

Full body CT and spine MRI were unremarkable. FDG-PET identified a small hypermetabolic focus at the right vocal cord. Fibrolaryngoscopy revealed left hemilaryngeal paralysis in abduction, reduced palatal mobility, with an ineffective cough. Neurophysiological assessment demonstrated chronic neurogenic damage of the IX and X cranial nerves (left > right), mainly affecting the pharyngeal phase of swallowing. Bilateral sural SAPs were absent with a slight reduction of SAPs amplitude in the upper limbs. F waves were normal. Swallowing reflex was preserved. Serum analysis revealed antibody anti-titin and anti-GM3 Ig positivity.

During hospitalization the patient was treated with intravenous immunoglobulins (0,4 g/kg/die for 5 days), which led to partial improvement in dysphagia and dysphonia.

Nerve	Segment	SCV	SAP (mcV)	Sensory distal latency (ms)	MCV	CMAP (mV)	Motor distal latency (ms)
Median (R)	Elbow - Wrist	53	27	3.8	51	14	3.9
Ulnar (R)	Elbow - Wrist	55	25	2.8	53	13	3
Common peroneal (R)	Ankle - Calf				40	7	5.2
Posterior tibial (R)	Ankle - Calf				43	11	4.8
Sural (R)	Calf - Lateral Malleolus	//	NO	SAP			
Sural (L)	Calf - Lateral Malleolus	//	NO	SAP			

DISCUSSION AND CONCLUSIONS

This case underscores a rare clinical phenotype of anti-GM3 Ig-associated neuropathy manifesting as acute onset with slowly progressive bulbar and facial nerve dysfunction associated with a mild sensory neuropathy. The presence of anti-GM3 antibodies in this context may represent a distinct autoimmune neuropathy variant, expanding the spectrum of disimmune neuropathy presentations. Awareness of this potential association is critical for early recognition and timely initiation of immunomodulatory therapy in similar atypical cases.

REFERENCES

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