PS-15

Isolated acute dysphagia as a rare presentation of Guillain Barré Syndrome showing complete recovery: A Case Report

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Case description

A 76-year-old woman was hospitalized with sudden-onset dysphagia during meals, her second episode of dysphagia. Three years ago, she experienced transient dysphagia which spontaneously resolved after a week. She had a history of left PCA infarction 9 years ago, but there were no neurological sequelae. She denied any recent history of vaccination, infective illness prior to the onset of symptoms. She experienced coughing with sputum and lost 9 kg in 2 weeks. Apart from bilateral loss of the gag reflex, other cranial nerve functions appeared intact. There was no weakness in the neck or limb muscles, and sensory examination was normal. Deep tendon reflexes were normal in the upper limbs and mildly decreased in the lower limbs, without signs of pathologic reflexes. Results from brain imaging, electrophysiologic studies, and laboratory tests collectively indicated a low probability of other conditions causing dysphagia. Based on the acute monophasic progression of disease course and CSF analysis result showing albuminocytological dissociation, we consequently diagnosed the patient with an atypical variant of Guillain-Barré syndrome (GBS), specifically involving cranial nerve (CN) X, and initiated intravenous immunoglobulin (IVIg) treatment for 5 days, from day 21 to day 25 of the illness. The patient showed a rapid response to IVIg treatment, with symptoms beginning to improve from the first day. Complete recovery of dysphagia was confirmed on VFSS performed on day 48 of onset to safely consume any food without restriction.

Serial VFSS study of the patient

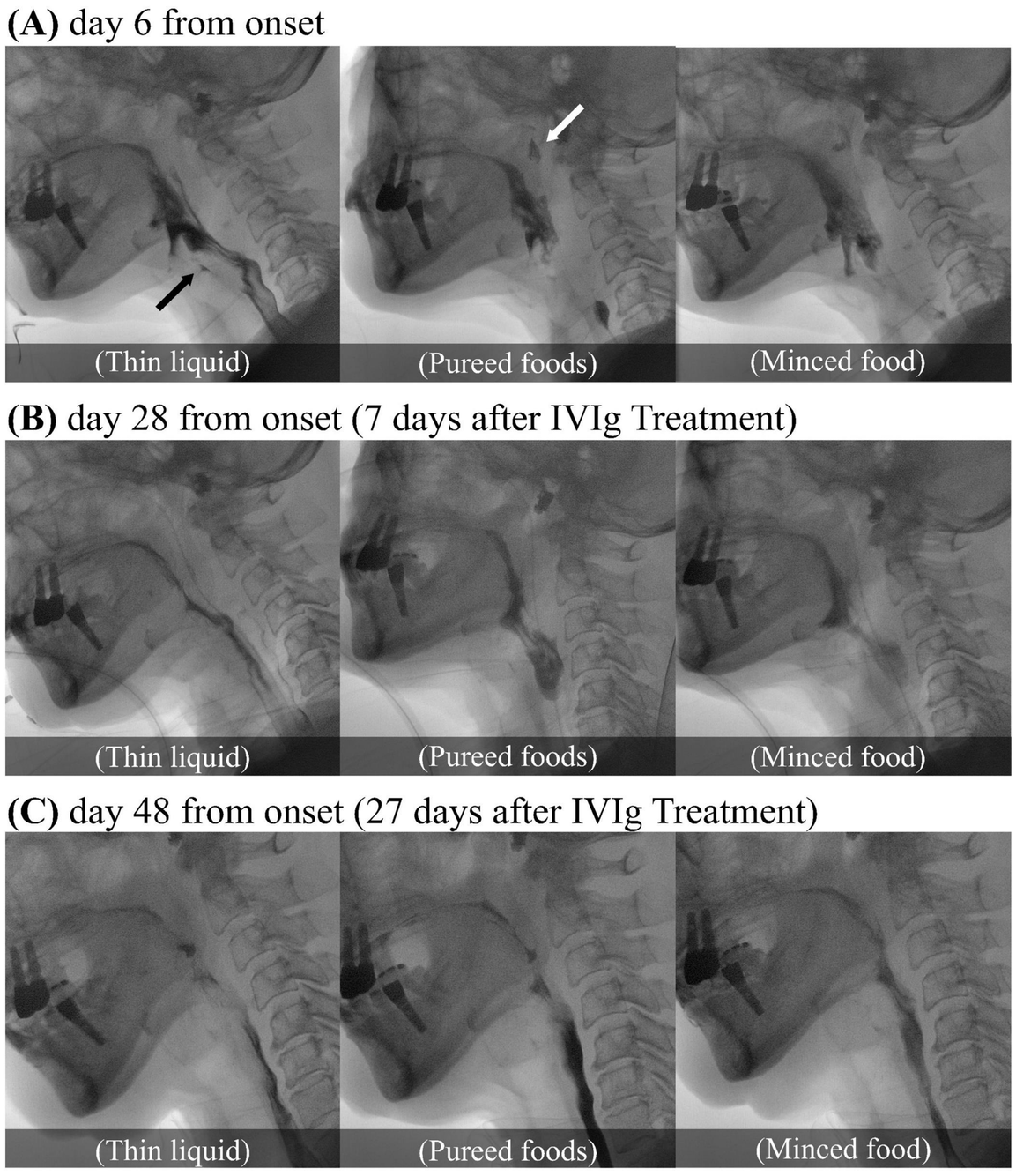

Figure 1.

(A-C) Serial Video Fluoroscopic Swallowing Study (VFSS) of the patient on day 6, day 28, and day 48 of the illness, using thin liquid (International Dysphagia Diet Standardization Initiative (IDDSI) level 0), pureed food (IDDSI level 4), and minced food (IDDSI level 5).

Differential diagnosis of the patient with oropharyngeal dysphagia

Oropharyngeal dysphagia

Structural	Neurogenic	Myogenic
Zenker's diverticulum, Head & Neck cancer, Esophageal web, Pharyngeal infection, Osteophyte, Prior surgery, Prior radiotherapy, Thyromegaly	Stroke, Brainstem tumor, Parkinson's disease, ALS and other MND, Multiple sclerosis, NMOSD, Guillain-Barre syndrome, Spinal muscular atrophy	Myasthenia gravis, Inflammatory myopathies, Paraneoplastic syndrome, Oculopharyngeal muscular dystrophy, Myotonic dystrophy

Laryngoscopy, Neck CT	Brain MRI	NCS, EMG, RNS	Lab
Unremarkable findings			

Anti-GM1 Ab IgM/IgG	-/-
Anti-GD1b Ab IgM/IgG	-/-
Anti-GQ1b Ab IgM/IgG	-/-

Tumor markers	WNL
Paraneoplastic antibodies	All negative
Rheumatoid markers	WNL
Myasthenia gravis antibodies	All negative
Antiganglioside antibodies	All negative
CSF study	**Albuminocytologic dissociation**

GBS spectrum diseases

GBS spectrum core feature	GBS spectrum supportive feature
▪ Mostly symmetric limb and/or motor cranial n. weakness ▪ Monophasic course, onset - nadir interval: 12hrs~28days ▪ Other possible mimic diseases are excluded	▪ Hx. of antecedent illness (up to 4 wks before onset) ▪ CSF analysis: albuminocytological dissociation ▪ presence of IgG against neural antigens (gangliosides)

Classic GBS	Pharyngeal-cervical-brachial weakness	Acute bulbar palsy (APB)	Miller-Fisher syndrome
Limb/Bulbar weakness Respiratory weakness Sensory deficit	Cervical, arm weakness Areflexia in upper limb Bulbar weakness	Prominent bulbar palsy without neck/limb weakness Plus feature (ABPp)	Opthalmoplegia Ataxia Areflexia

Conclusion

Dysphagia in the elderly constitutes a critical condition that can substantially increase patient morbidity and mortality, even over a short duration. With a thorough evaluation of systems impacting the swallowing mechanism and awareness of uncommon causative factors, such as neuro-autoimmunity, clinicians can implement effective disease-modifying therapies, potentially leading to the resolution of dysphagic symptoms..