

Immune Checkpoint Inhibitor–Associated Myositis Presenting With Dysphagia: A Case Report

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Introduction

Immune checkpoint inhibitors (ICIs) have improved outcomes in advanced malignancies.

However, immune-related adverse events (irAEs) may develop due to immune system overactivation.

Among irAEs, myositis is uncommon but can be clinically significant, particularly when bulbar muscles are involved, resulting in dysphagia and increased aspiration risk. Early recognition is critical to prevent severe complications.

Notably, ICIs–induced myositis is frequently reported in association with myasthenia gravis (MG), which may further exacerbate bulbar dysfunction. However, cases of isolated myositis without concurrent MG are relatively rare and may pose diagnostic challenges.

Here, we report a case of a 77-year-old man who developed ICI–induced myositis presenting with dysphagia in the absence of myasthenia gravis.

Case presentation

A 77-year-old man with malignant pleural mesothelioma was treated with combination immunotherapy consisting of nivolumab and ipilimumab.

Approximately four weeks after treatment initiation, he developed dysphagia and generalized weakness affecting the neck and proximal limbs.

Neurological examination revealed neck flexor weakness (MRC grade 3) and symmetric proximal limb weakness (MRC grade 4), while sensory function and deep tendon reflexes were preserved.

Serum creatine kinase was markedly elevated at 2,992 IU/L, and myoglobin exceeded 1,200 ng/mL. CK-MB and troponin I were mildly elevated, whereas brain magnetic resonance imaging showed no acute abnormalities (Figure 1).

Clinical Course: CK Level & Treatment Timeline

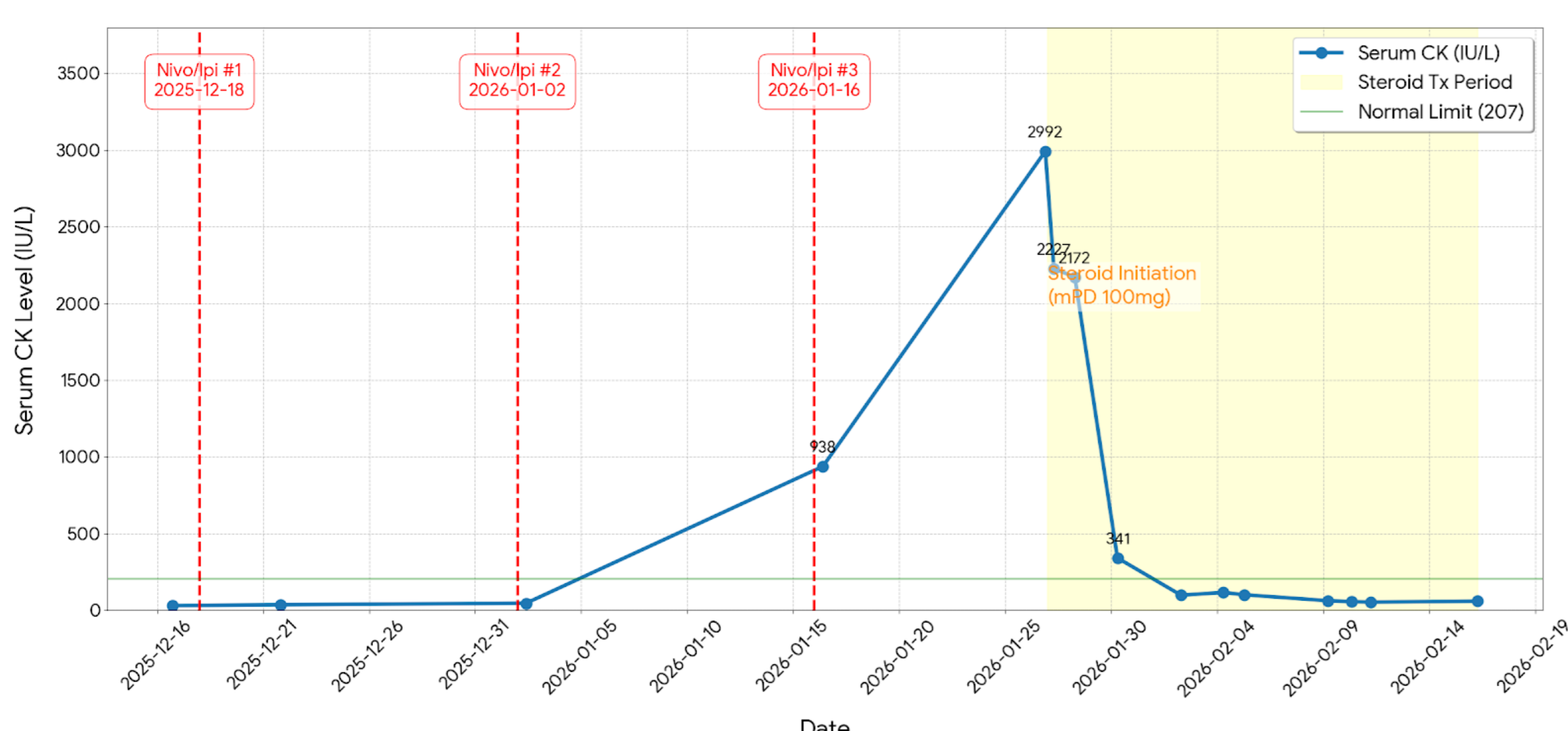


Fig.1 Clinical course of ICI-associated myositis: peak CK level (2,992 IU/L) and rapid response to corticosteroids.

Repetitive nerve stimulation demonstrated no significant decremental response (Figure 2).

Autoimmune serologic testing revealed only low-titer antinuclear antibody positivity (Table 1).

Based on the temporal association with ICI therapy, clinical findings, and laboratory results, ICI-associated myositis was diagnosed.

High-dose intravenous corticosteroid therapy was initiated, leading to normalization of muscle enzyme levels and improvement in limb and neck strength. Nevertheless, dysphagia persisted on follow-up evaluation (Figure3).

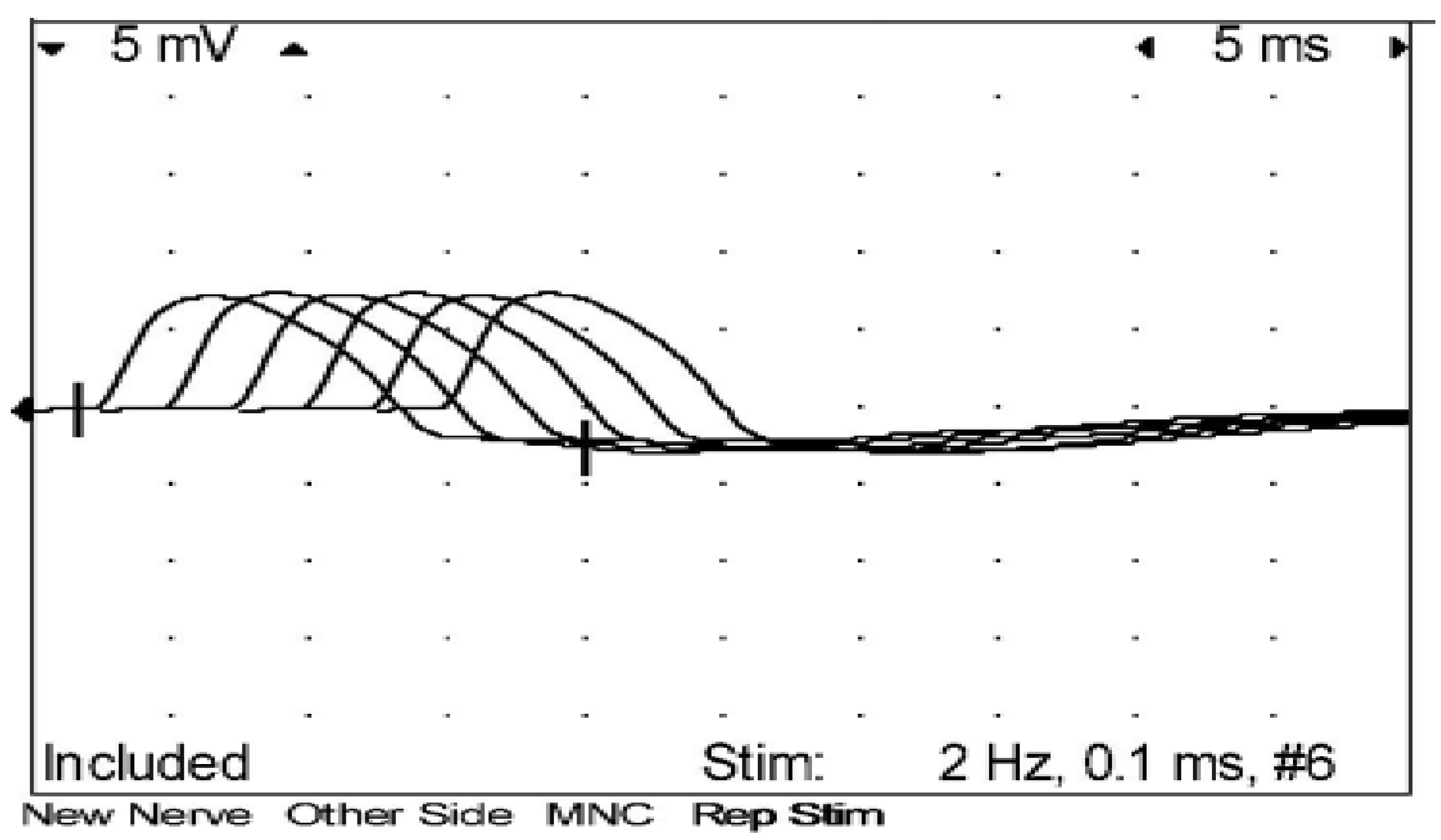


Fig.2 Normal RNS of the spinal accessory nerve

Table 1. Autoantibody results

Test Item	Result
ANA Titer	1:80, Speckled
Anti-AChR Ab	Negative (-)
Anti-MuSK Ab	Negative (<0.02)

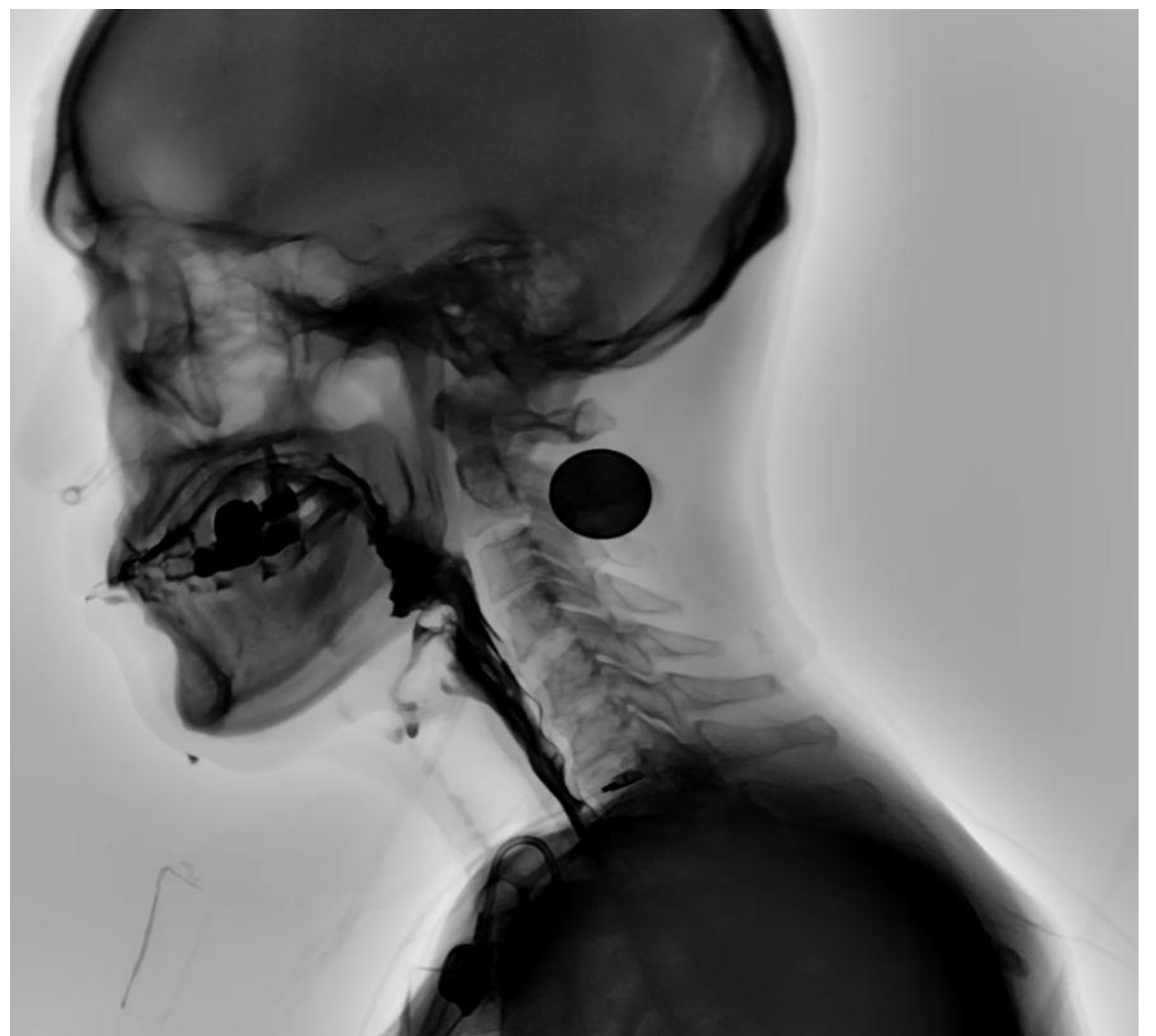


Figure 3. Decreased pharyngeal contractility& reduced epiglottic inversion with aspiration during swallowing.

Conclusions

ICI-associated myositis should be considered in patients presenting with acute muscle weakness and dysphagia within weeks of initiating immunotherapy. Although corticosteroid therapy can improve biochemical abnormalities and motor weakness, bulbar dysfunction may persist.