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Dysphagia due to Liver Cirrhosis

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BACKGROUND

Acquired hepatocerebral degeneration (AHD) is a chronic neurological syndrome occurring in individuals with advanced hepatobiliary diseases. Diagnosis relies on neurological manifestations, laboratory findings, and the presence of liver cirrhosis (LC). Clinical features encompass movement disorders, predominantly parkinsonism, and cognitive impairment, with dysphagia being a rare occurrence. Herein, we present a case of dysphagia secondary to acquired hepatocerebral degeneration, notably exhibiting improvement following dopamine agent therapy.

CASE REPORT

An 82-year-old male with chronic liver cirrhosis presented to the dysphagia clinic due to recent onset, non-progressive dysphagia. Additionally, he had experienced transient myoclonus, which had resolved without any lasting effects. During physical examination, mild dysarthria and symptoms of aspiration during regular feeding were noted. The initial videofluoroscopic study (VFSS) revealed cricopharyngeal dysfunction and aspiration, as depicted in Figure 1. Consequently, based on the VFSS findings, the patient's oral diet was discontinued and substituted with tube feeding. Further assessment via brain MRI displayed bilateral T1 high signal intensities in the basal ganglia, as illustrated in Figure 2. Considering the amalgamation of MRI results, liver cirrhosis, transient myoclonus, and dysphagia, the patient received a diagnosis of dysphagia attributable to acquired hepatocerebral degeneration. Subsequently, he was initiated on Perkin (levodopa with carbidopa) 100mg b.i.d., resulting in amelioration of his dysphagia symptoms. Follow-up VFSS showcased marked improvement, facilitating the reintroduction of oral feeding, as depicted in Figure 3.

Figure 2. Brain magnetic resonance imaging depicting high signal intensities in both basal ganglia on T1-weighted image, suggestive of acquired hepatocerebral degeneration (arrows)

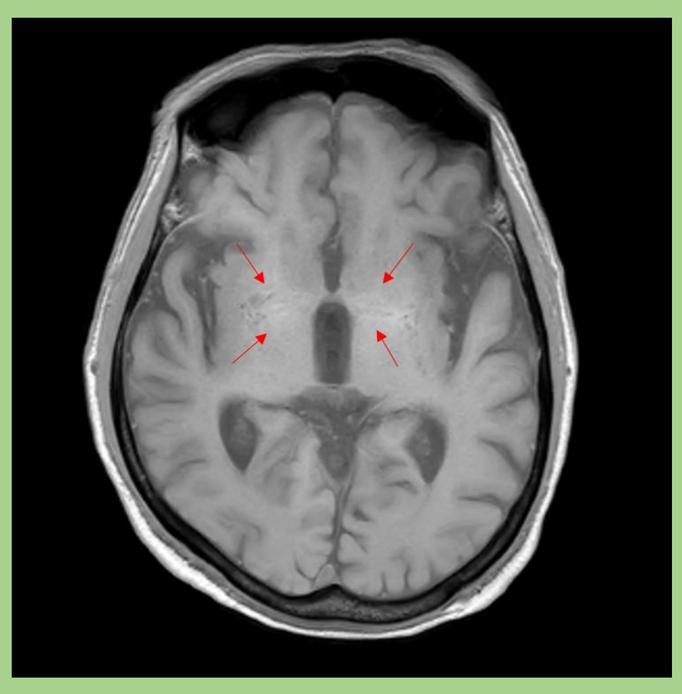
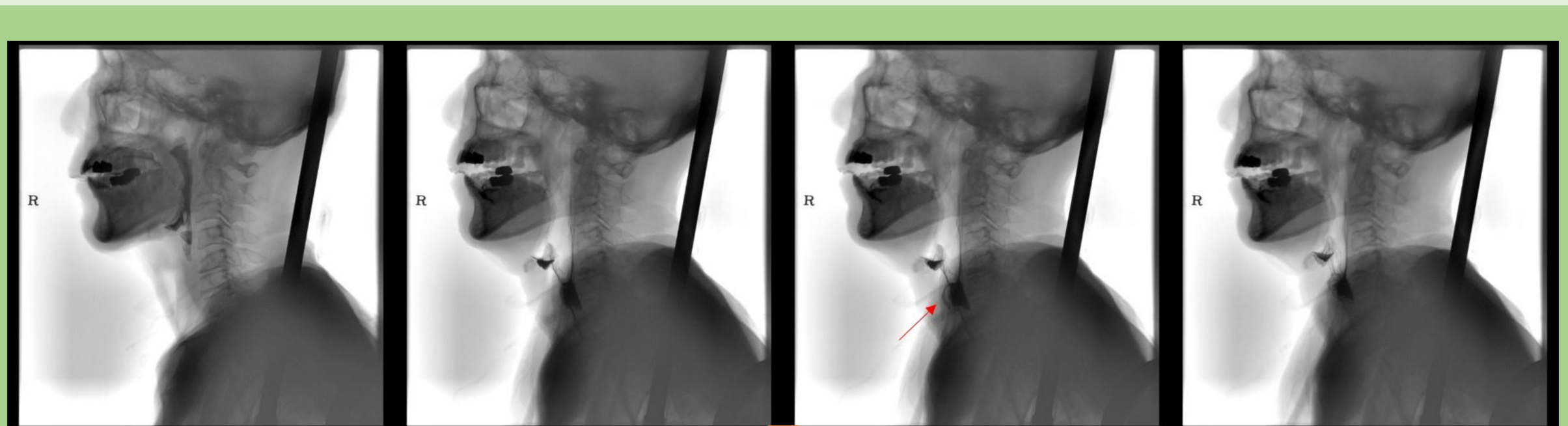
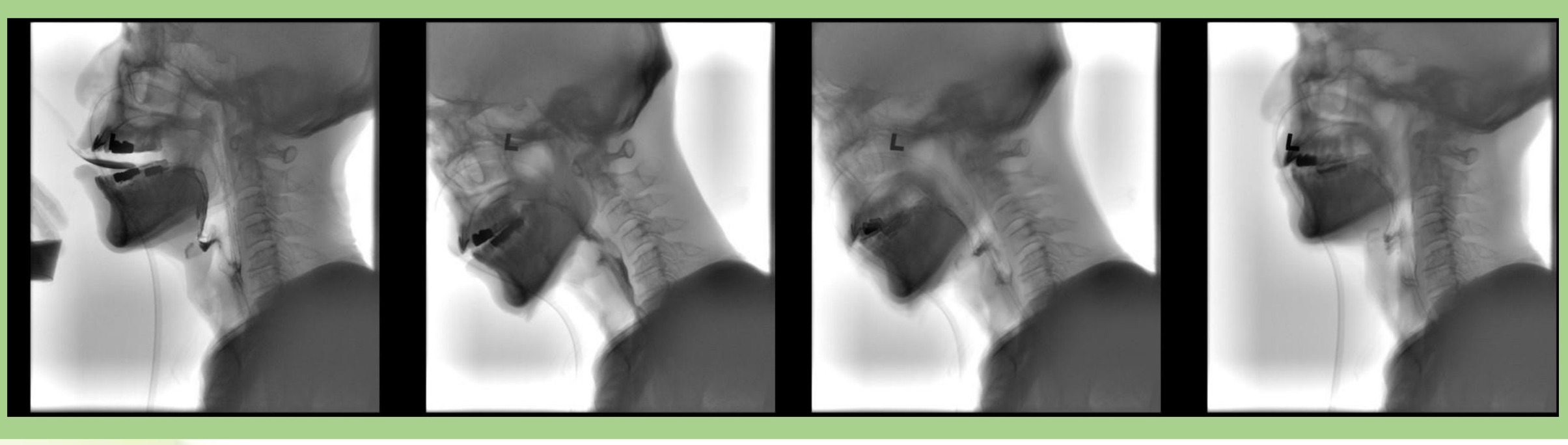


Figure 1. Initial videofluoroscopic studies using barium highlight cricopharyngeal dysfunction, indicated by arrows denoting post-swallow aspirations.



Perkin (levodopa with carbidopa) 100mg b.i.d.

Figure 3. Follow-up videofluoroscopic studies using liquid demonstrate the absence of penetration or aspiration, indicating significant improvement in cricopharyngeal muscle relaxation.



CONCLUSION

Dysphagia is often presented in patients with liver cirrhosis. This case underscores the significance of considering acquired hepatocerebral degeneration as a potential cause of dysphagia in cirrhotic patients. Importantly, our findings highlight the efficacy and ease of improving dysphagia through the prescription of dopamine agents, offering valuable insights for clinicians managing such cases.