# Persistent post-stroke dysphagia treated with cricopharyngeal myotomy

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## Abstract

Post-stroke dysphagia is a common problem after stroke. About 8-13% patients have persistent dysphagia and are unable to return to pre-stroke diet even after 6 months of stroke. Use of percutaneous endoscopic gastrostomy (PEG) may be required in these patients, which may be psychologically unacceptable and impair the quality of life. In those with cricopharyngeal dysfunction leading on to refractory post-stroke dysphagia, cricopharyngeal myotomy and injection of botulinum toxin are the treatment options. We present a case of vertebrobasilar stroke who had persistent dysphagia due to cricopharyngeal dysfunction with good recovery of swallowing function following cricopharyngeal myotomy 1.5 years after the stroke.

## Key Words

Cricopharyngealmyotomy, post-stroke dysphagia, videofluoroscopy

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## Introduction

Post-stroke dysphagia is a common problem with incidence ranging widely from 19 to 81% in different studies.[1,2] In majority of the patients, dysphagia is a transient phenomenon, but persistent swallowing difficulty with inability to return to the pre-stroke diet after 6 months of stroke is seen in 8-13% of patients.[3,4] Persistent dysphagia is known to adversely impact the quality of life of stroke patients.[5] Use of percutaneous endoscopic gastrostomy (PEG) tube placement is often required in these patients, which is cumbersome and psychologically unacceptable for the patients. Cricopharyngeal myotomy may be required in these patients with refractory oropharyngeal dysphagia with 75% having complete or partial recovery of swallowing function.[6]

We present a case of vertebrobasilar stroke with persistent dysphagia due to cricopharyngeal dysfunction who had good recovery of swallowing function following cricopharyngeal myotomy 1.5 years after the stroke.

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## Case Report

A 39-year-old male patient with poorly controlled diabetes mellitus presented with acute onset of severe vertigo, ataxia of all limbs, dysarthria, and dysphagia with step-wise deterioration over 9 days. On presentation 12 days after symptom onset, he was noted to have slurring dysarthria, right hemianopia, right Horner's syndrome, right palatal palsy, and bilateral limb and truncal ataxia. In addition, he was tachypneic with evidence of aspiration.

He had multiple diffusion restricted infarcts of different ages in bilateral cerebelli, left pons, left medulla, and left occipital lobe in MRI; and right vertebral artery dissection with occlusion in CT angiography. He was initiated on dual antiplatelets and high dose statin with management of risk factors for his stroke.

As he had pharyngeal pooling of secretions with evidence of aspiration pneumonia at admission, he was electively intubated and subjected to early tracheostomy. He had a second bout of aspiration 3 weeks after weaning from the ventilator. The patient's tracheostomy could be closed 2 months later.

A swallow assessment done at 8 weeks by the speech and language pathologist showed poor swallowing function predominantly due to pharyngeal phase dysfunction. While on conservative management, the patient and family were advised feeding through PEG for which they did not consent initially due to social and personal reasons. But as the patient developed significant malnutrition and weight loss, PEG placement was done after 4 months of the stroke. His nutritional status improved following this and neurological status improved to modified Rankin scale (mRS) 3 with residual ataxia.

The patient's dysphagia however persisted with recurrent spitting and inability to swallow own saliva even after 1.5 years of stroke. The patient had significant depression and impairment of social function related to the swallowing difficulty. On trial swallow test using 8 ml of semisolids and thin liquids, patient demonstrated intact oral preparatory and fairly adequate oral propulsive phases of swallow. Pharyngeal phase of swallow was affected with choking 3-5 s later with effective post swallow cough reflex and expectoration of more than 95% of bolus swallowed despite having adequate hyolaryngeal excursion. Delayed cough reflex was suggestive of post swallow aspiration due to a secondary laryngeal incompetency combined with an inability to swallow own secretions giving the clinical impression of cricopharyngeal dysmotility, which warranted further evaluation.

Videofluroscopic evaluation of swallowing (videofluoroscopy (VFS)) was performed on the patient using bolus of all consistencies to explore the physiology behind altered swallow function and to plan appropriate rehabilitation. Oral preparative and oral propulsive phases of swallowing were adequate. In pharyngeal phase; pharyngeal reflex, hyolaryngeal excursion, and velopharyngeal closure were adequate with fairly efficient laryngeal valving mechanism. Upper esophageal sphincter relaxation was severely affected. Severe cricopharyngeal dysmotility was noted for both semisolids and thin liquids [Figure 1a and b]. Patient had adequate protective and efficient cough reflex and he was able to expectorate out the entire bolus. Secondary laryngeal incompetency was noted once out of five trials; which led to a minimal aspiration for thin liquids. Significant pharyngeal residue at the level of valleculae and pyriform sinus was noted.

He was initially given 40 units of botulinum toxin (20 units on either side) injection into the cricopharyngeus muscle under laryngeal electromyography guidance, which did not improve the dysphagia. Subsequently, he underwent cricopharyngeal myotomy without any postoperative complications.

On follow-up at 3 months after the procedure, he could swallow with the adoption of the chin-tuck maneuver. His VFS showed only minimal pharyngeal phase dysphagia with normal upper esophageal sphincter opening [Figure 1c and d]. His PEG tube was subsequently removed and on follow-up after 10 months, he continued to maintain good nutrition and was able to swallow without any specific techniques. The improvement in dysphagia made a marked change in his quality of life.

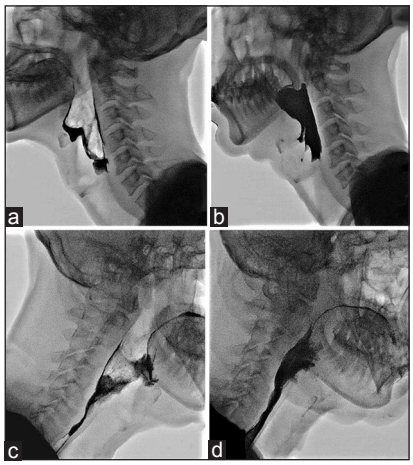


**Figure 1: Videofluoroscopic images of barium swallow showing (a) Cricopharyngeal spasm with thin barium and (b) Cricopharyngeal spasm and secondary penetration with thick barium preoperatively. Post cricopharyngeal myotomy videofluoroscopic images showing (c) Free flow of thin barium and (d) Free flow of thick barium**

## Discussion

Our case is an extremely rare case of improvement of post-stroke dysphagia following cricopharyngeal myotomy after 18 months of stroke. The persistence of dysphagia in our patient was related to the abnormal relaxation of the upper esophageal sphincter. The dysphagia had severe impact on the general condition of the patient as well as his quality of life. This patient had severe weight loss and depression; as a result of which he was unable to pursue even activities of daily living by self, in spite of good improvement of his motor function. The improvement of dysphagia following cricopharyngeal myotomy illustrates that even those patients treated late in the course of the illness can have a favorable outcome. It is important to suspect cricopharyngeal dysfunction in patients with persisting dysphagia and treat them appropriately as 75% of them make a good recovery of their swallowing function.

Recovery from dysphagia following acute stroke occurs spontaneously in 87-90% of patients by 6 months;[3] majority of them within the first 2 weeks. Studies from patients with brainstem stroke indicate a higher incidence of dysphagia and aspiration in them, ranging between 40 and 80%.[7] The involvement of the medullary swallowing center and the lower cranial nerve nuclei result in prominent swallowing dysfunction and slower recovery in these patients.[8,9]

Cricopharyngeal dysfunction in stroke results from the incoordination between the pharyngeal contraction and upper esophageal sphincter relaxation. This condition is relatively rare and is reported in up to 5.7% in patients with neurogenic dysphagia[10] and 5.2% in patients with acute stroke using videofluoroscopic studies.[11] Post-stroke dysphagia due to cricopharyngeal dysfunction is frequently noted in lateral medullary syndrome in contrast to hemispheric strokes where it is uncommon.[12]

VFS using radiopaque substances of different liquid and solid textures is the gold standard for the detection of

cricopharyngeal spasm and other stroke related swallowing disorders. This is widely available and has the advantage of being able to assess all the stages of swallowing. However, it has the disadvantages of radiation exposure and need for trained personnel for interpretation. The alternative methods are fiberoptic endoscopic evaluation of swallowing (FEES), esophageal manometry and electromyography of the esophageal sphincter which are more cumbersome and are not as widely available as VFS.

Cricopharyngeal spasm is eminently treatable. Conservative strategies like compensatory chin-tuck and head rotation maneuvers, strap muscle strengthening exercises, and changing consistency of fluids should be tried initially. In patients who are unresponsive, disruption of the upper esophageal sphincter using botulinum toxin and cricopharyngeal myotomy are the best techniques. These should be considered only after at least 6 months of acute stroke, as improvement in swallowing can occur spontaneously in this period.

Botulinum toxin injection to the cricopharyngeus muscle under electromyographic guidance has been reported in persistent post-stroke dysphagia in many case reports, but the effect is frequently transient and is associated with the risk of dysphagia due to the infiltration of toxin to the surrounding musculature.[13,14] Failure to respond to botulinum toxin injection can occur in long-standing muscular dysfunction resulting in fibrosis and due to improper needle placement of the injection.

Cricopharyngeal myotomy is the best established treatment for cricopharyngeal spasm with about 50-70% of the patients undergoing surgery for neurogenic dysphagia reporting satisfactory results.[6] The improvement in patients with neurogenic dysphagia is less robust than those with muscular dysphagia with some of the symptoms persisting after treatment.[15] Cricopharyngeal myotomy is usually accompanied by minor and self-limiting perioperative complications occurring in upto 22% cases. The reported complications include pharyngeal leaks, recurrent laryngeal nerve injury, and pneumonia.[16]

Dysphagia produces an immense negative impact on the quality of life with more than one-third of sufferers reporting poor self-esteem, impaired socialization, and inability to enjoy life.[5] Thus, in addition to the malnutrition and recurrent aspirations, persisting dysphagia can result in poor functional recovery after stroke.

Our case illustrates that cricopharyngeal spasm can be one of the causes for a persistent dysphagia after stroke for which the patient should be evaluated. Cricopharyngeal myotomy is a safe and relatively simple technique with considerable benefit in these patients.

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