Abstract:
Cricopharyngeal myotomy improves dysphagia of neurogenic origin to a satisfactory to excellent subjective results in up to 75 percent of patients. The literature on cricopharyngeal myotomy for neurogenic dysphagia is few. In this background, we report a 44 year female presented to our department who has been fed through feeding gastrostomy for duration of 18 years, which she had undergone for neurogenic dysphagia as a result of a stroke. She was further evaluated in our department to find out exact cause of dysphagia in February 2011 including a complete neurological assessment by neurologists. We excluded myogenic and organic causes for dysphagia by performing esophageal manometry, upper GI Endoscopic examination, Laryngoscopic examination, MRI of brain, Barium swallow. A Cricopharyngeal myotomy was planned and was performed on 1-3-2011. Patient had an uneventful post operative recovery and has been able to swallow solid and liquid foods till now.

Keyword: Neurogenic dysphagia, cricopharyngeal myotomy, upper esophageal sphincter-UES

Neurogenic dysphagia is due to disruption of highly coordinated swallowing mechanisms in patients with central nervous system disease or cranial nerve involvement. Stroke is the most frequent neurogenic cause of this condition and other causes are Parkinson’s disease, amyotrophic lateral sclerosis, motor neuron disease, bulbar poliomyelitis. The mechanism of dysphagia is due to incoordination between pharyngeal contraction and cricopharyngeal sphincter (Upper esophageal sphincter-UES) relaxation as a result of stroke. This results in misdirection of swallowed bolus of food leading to laryngotraacheal aspiration and pharyngonasal regurgitation.
Complete absence of relaxation of UES is also seen resulting in functional obstruction at the level of UES leading to dysphagia. Cricopharyngeal myotomy aims to mitigate the functional obstruction seen in this condition but do not correct the basic problem of incoordination between pharyngeal contraction and cricopharyngeal sphincter (Upper esophageal sphincter-UES) relaxation. In other words myotomy decreases the resistance to pharyngoesophageal transit. This results in good palliation of dysphagia but still patients can have episodes of aspiration though improved in significant amounts as reported in literature. In 1951, Kaplan reported the first successful cricopharyngeal myotomy to treat a patient with significant dysphagia from bulbar poliomyelitis. Patients presenting with oropharyngeal dysphagia needs a systematic assessment regardless of the cause. The clinical assessment that is the subjective assessment of symptoms is the most important. This helps to localize the dysphagia. Patients need to have a well preserved voluntary deglutition act, because cricopharyngeal myotomy does not correct the oral phase of dysphagia. The other recommended clinical criteria are adequate tongue movement, intact laryngeal function and phonation and a minimum period of 6 months of conservative management after stroke. Our patient satisfied almost all clinical criteria. A minimum waiting period of 6 months is recommended because about 80% of patients with stroke have spontaneous recovery of symptoms. Endoscopic assessment of entire esophagus has to be done to rule out organic cause. Laryngoscopic assessment of larynx, pharynx and esophageal inlet also is mandatory. Manometric evaluation of entire esophagus is done to rule out myogenic cause. Assessment of UES relaxation and coordination with pharyngeal contraction is difficult due to the strategic location and dynamic nature of UES. Videofluoroscopic evaluation helps us to detect and analyze the functional impairment of pharyngoesophageal junction.

Our patient presented with history of dysphagia to liquids and solids for 18 years. She underwent feeding gastrostomy 18 years ago for dysphagia since she had a stroke. Patient was nutritionally depleted on clinical examination. Her phonation and tongue movements were normal. She was further evaluated by Barium swallow which showed abrupt cut off at cricopharyngeal level as shown in the figure.

**Barium swallow which showed abrupt cut off at cricopharyngeal level**

Upper GI Endoscopy revealed cricopharyngeal spasm, but scope was passed beyond into esophagus and stomach which were normal and hence an organic cause for dysphagia was ruled out. Laryngoscopy showed normal vocal cords. MRI of brain showed cerebellar atrophy. Esophageal manometry was performed. Since upper esophageal sphincter manometric assessment is very difficult to perform due to the above mentioned reason, we were not able to perform UES manometric assessment. Manometry of rest of esophagus was found to be normal. A Cricopharyngeal
Cricopharyngeal myotomy was performed on 1/3/2011. The steps of surgery are shown in the figure below.

completed myotomy:
Patient had an uneventful recovery and has been able to swallow solid and liquids from 3rd POD till now and gastrostomy tube was removed latter. Our patient satisfied almost all clinical criteria for myotomy which are considered to be good prognostic factors for the results of surgery. We conclude that majority of patients with dysphagia due to neurogenic cause recover spontaneously. In the remaining patients, feeding gastrostomy (Percutaneous endoscopic gastrostomy/surgery) is an established treatment for nutritional purpose. Cricopharyngeal myotomy would be considered as a viable option in selected cases of those patients as they provide a better quality of life.

References:


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