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ORIGINAL ARTICLE

The Relationship Between Hiatal Hernia and Cricopharyngeus Muscle Dysfunction

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Abstract

Although the precise etiology of cricopharyngeus muscle (CPM) dysfunction (CPMD) is uncertain, many have hypothesized that a hypertrophied CPM may develop as a protective compensation against gastroesophageal reflux disease (GERD). The purpose of this investigation was to evaluate the association between CPMD and the presence of hiatal hernia (HH) in an attempt to elucidate the potential etiology of CPMD. The charts of individuals who underwent video fluoroscopic esophagrams between 01/01/14 and 10/30/16 were reviewed from an electronic database. A group of 50 subjects with reported HH were identified and age- and gender-matched to an individual without HH. The prevalence of CPMD was compared between groups. The mean (± SD) age of the HH cohort was 64 (± 13.4) years and 64 (± 12.8) years for the group without HH ($p > 0.05$). Thirty-eight percent was male. The frequency of CPMD in the HH group was 78% versus 58% in the non-HH group ($p < 0.05$). Individuals with a HH were 2.57 times more likely to have evidence of CPMD (95% CI 1.07– 6.15). Although these data suggest an association between GERD and CPMD, further research is required before a causal relationship can be presumed.

Keywords Cricopharyngeus muscle dysfunction · Gastroesophageal reflux disease · Dysphagia · Upper esophageal sphincter · Deglutition · Deglutition disorders · Hiatal hernia · Pharyngoesophageal segment · UES · PES

Introduction

The upper esophageal sphincter (UES) is a 3–4-cm high-pressure zone that forms a barrier between the esophagus and pharynx. The cricopharyngeus (CP) muscle is considered a major functional contributor to the UES high-pressure zone [1, 2]. Cricopharyngeus muscle dysfunction (CPMD) is defined as impaired or uncoordinated cricopharyngeus muscle relaxation or expansion [3]. Sufficient and timely opening of the sphincter is necessary for complete transit of a bolus into the esophagus. CPMD may result in the symptom dysphagia or display evidence of post-swallow residue and aspiration on fluoroscopic swallow study. CPMD is diagnosed by the appearance of a bar or web on radiologic testing [2, 4–6] or increased hypopharyngeal or intrasphincteric intrabolus pressure on pharyngeal–esophageal manometry [2, 7, 8]. Patients with CPMD may report symptoms of either liquid or solid food dysphagia. Over time, obstruction at the UES can result in the development of pharyngeal dilation and weakness [9].

The etiology of CPMD is uncertain. Radiologic findings of CP bars have been reported in 5–19% of patients who undergo a videofluoroscopic swallow study [10] and cricopharyngeal webs are present in 14% of dysphagic patients [11]. In some of these patients, no other structural or neuromuscular etiology for the dysphagia can be identified, and the dysphagia is attributable primarily to the finding of CPMD. Findings of CP bars and webs are significantly more common in the elderly population [5, 12, 13], suggesting that the pathogenesis of CPMD may involve a cumulative process inducing changes in the mucosa and muscle over time. Some investigators have

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hypothesized that a hypertrophied CPM may develop as a protective compensation against gastroesophageal reflux disease (GERD) [14–17]. This has biologic plausibility as the primary function of the CPM is to protect the proximal airway against the regurgitation of gastric and esophageal contents. Previous investigations have demonstrated elevated UES pressures following esophageal acid exposure [17, 18]. Despite these hypotheses and initial findings pointing to a possible involvement of reflux in CP pathology, the association between the two has scarcely been examined.

The purpose of this investigation was to evaluate the association between CPMD and the presence of hiatal hernia (HH) with the goal of clarifying the relationship between GERD and CPMD. Hiatal hernia can result from a lax phrenoesophageal ligament (PEL) which connects the extrinsic diaphragmatic and intrinsic esophageal components contributing to the lower esophageal sphincter high-pressure zone. When the PEL becomes lax, the gastric cardia may herniate above the diaphragm and result in a HH. The altered anatomy associated with hiatal hernia results in LES incompetence and increased risk of GERD [11]. Since HH is a well-known risk factor of severe GERD and its complications [19–21] and can be readily identified on esophagram, it was chosen to serve as surrogate of reflux in our assessment of the relationship with CPMD. Improving the understanding of the pathophysiologic cause of CPMD can help devise more appropriate treatment approaches for alleviating dysphagia in persons with CPMD.

Methods

This study was approved by the institutional review board of the University of California, Davis School of Medicine. The charts of individuals who underwent fluoroscopic video-esophagrams between 01/01/14 and 10/30/16 were reviewed from an electronic database. A total of 162 charts were reviewed. All patients were referred for video-esophagrams due to complaints of dysphagia. Patients with a medical history of head and neck cancer or neurological disorder were excluded. Patients with a history of CP dilation, myotomy, or repair of Zenker's diverticulum prior to the date of the esophagram were also excluded from the investigation. A group of 50 subjects with HH were identified and each was individually matched to the next chronologically adjacent individual without HH findings who was matched by gender and age (± 5 years). Data collected from the chart included presence/absence of HH, CP bar/CP web, impairment of esophageal motility, and objective pharyngoesophageal segment (PES) measurements made from the lateral fluoroscopic view during a 20 ml bolus swallow.

The esophagography was completed in accordance with our standard protocol: 1 mL and then 20 mL liquid contrast (EZ-PAQUE barium sulfate suspension, 60% w/v, 41% w/w; E-Z-EM, Inc, Westbury, New York) was offered in the lateral seated position. Next, after effervescent crystals were given, 20 mL liquid contrast and a 13-mm barium tablet were administered in the AP standing position. Patients were then instructed to drink a 20 mL single swallow followed by consecutive swallows of approximately 100 mL from a cup in the right anterior oblique position. Finally, a water siphon test was performed in the supine position. The anatomic structures and function of the PES and esophagus were evaluated.

CPMD was defined as the presence of a CP bar, CP web or Zenker's diverticulum, based on the clinical findings noted in the esophagram report. The clinical interpretation of esophagrams in our center identifies CP bars as an indentation at the PES apparent on the posterior hypopharyngeal wall and they are stratified by degree of obstruction, as described previously [11]. CP webs are identified by observing an indentation of the web on the posterior arch of the cricoid which corresponds to a similar indentation on the adjacent posterior hypopharyngeal wall [11] (Fig. 1). Cricopharyngeal webs have been implicated as an extrinsic cause of CPMD [22].

A HH was defined as an extension of the esophagogastric junction (EGJ) above the diaphragmatic hiatus visualized on fluoroscopy (Fig. 2). For the purposes of this investigation, an extension of at least 2 cm was required to ascertain the presence of HH. This 2 cm demarcation was utilized to account for normal deglutitive and respiratory variations in EGJ movement and exclude insignificant fluctuations [23, 24]. Based on this definition, true paraesophageal hernias, where the EGJ remains at the level of the diaphragm, were not included in the investigation.

To evaluate a potential correlation between HH size and PES opening, the HH size was measured using the universal desktop ruler (AVPSoft, Pittsburgh, PA). PES opening diameter was measured from a 20 mL bolus swallow in the lateral view, using Swallowtail software (Belldev Medical Arlington Heights, IL) (Figs. 2, 3). Measurements made with both software were calibrated to an opaque ring of known diameter that had been taped to the patient's neck during the fluoroscopic exam.

All data were recorded and coded into SPSS 19.0 for Macintosh (SPSS Inc., Chicago, IL). The frequency of

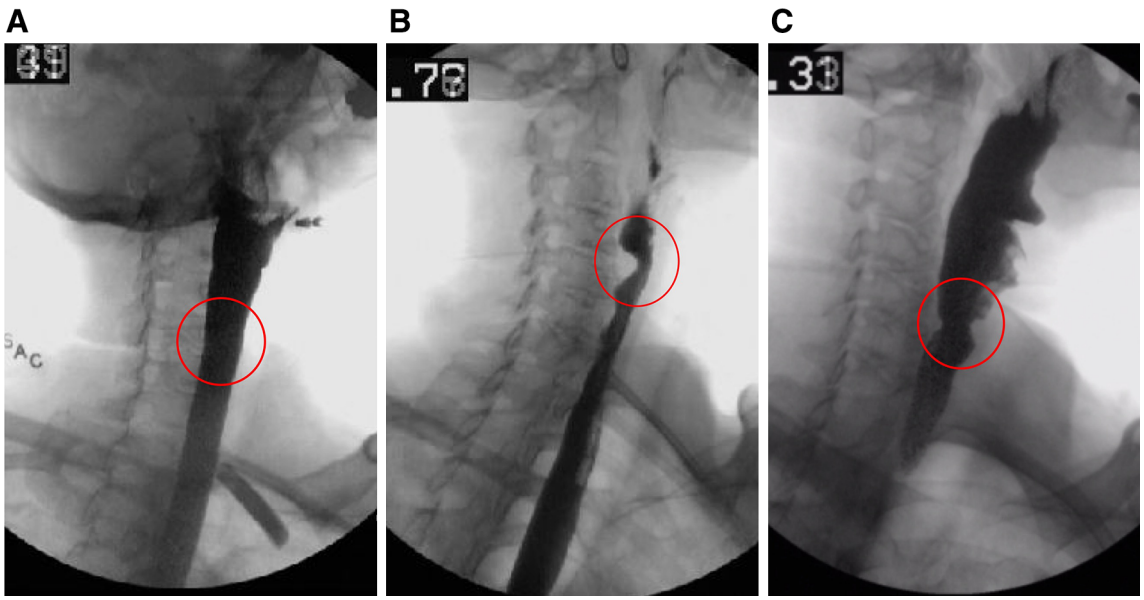

Fig. 1 Right anterior oblique fluoroscopic views displaying normal appearance of the pharyngoesophageal segment during bolus flow (**a**), cricopharyngeus bar (**b**) and cricopharyngeus web (**c**)

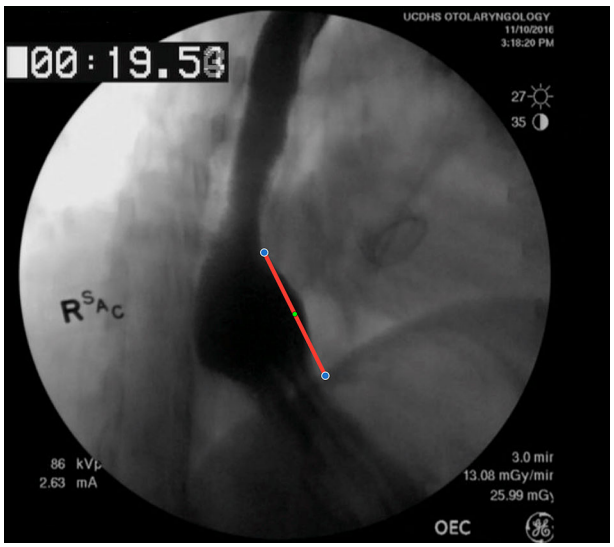

Fig. 2 Measurement of a large hiatal hernia (HH) in a 70-year-old male from a fluoroscopic image in the right anterior oblique position. Measurements of HH were taken from the gastroesophageal junction to the diaphragm using Universal Desktop Ruler (AVPSoft, Pittsburgh, PA)

CPMD findings was calculated for both the HH positive and HH negative group. An association between HH and CPMD was evaluated by calculating the odds ratio with associated 95% confidence interval. The difference between UES opening diameter was assessed with an independent sample T test. The correlation between HH size and UES opening diameter within the HH group was assessed with the Pearson correlation coefficient.

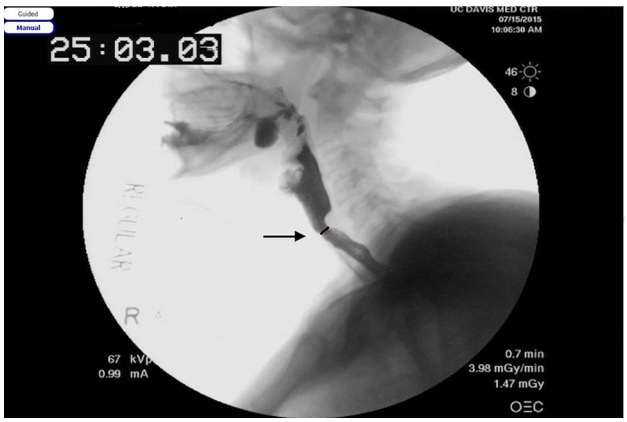

Fig. 3 Pharyngoesophageal segment opening diameter of a 20 mL bolus swallow in a 75-year-old female with findings of a cricopharyngeus bar and hiatal hernia displayed in the fluoroscopic lateral view. Measurement was made using Swallowtail software (Belldev Medical Arlington Heights, IL)

Results

Subject ages ranged from 32 to 88 years in the HH group and 35 to 93 years in the control (no HH) group. The mean age (± SD) of the HH cohort was 64 (± 13.4) years and 64 (± 12.8) years of the group without HH ($p > 0.05$). Thirty-eight percent of were male. Esophageal motility was noted to be impaired in 22 (44%) of the HH cohort and in 24 (48%) of the control group ($p > 0.05$).

The frequency of CPMD in the HH group was 78% versus 58% in the non-HH group ($p < 0.05$). Zenker's diverticulum was noted in three patients in the HH group and one patient in the non-HH group. Individuals with a

HH were significantly more likely to have evidence of CPMD ($p = 0.02$), odds ratio (OR) 2.57, 95% CI 1.07–6.15. PES opening diameter was significantly smaller in the HH group compared to the non-HH group ($p = 0.016$) (see Table 1). Within the HH group, no correlation was found between HH size and UES opening diameter (Pearson's $r = 0.005$, $p = 0.975$). A summary of the measurements is presented in Table 1.

Discussion

Cricopharyngeus muscle dysfunction can lead to impaired UES opening, resulting in significant oropharyngeal dysphagia. Deglutitive UES dysfunction may arise from a non-relaxing upper esophageal sphincter resulting from neurological disorders such as medullary lesions and Parkinson's disease [25]. Impairments in the opening of the sphincter are also common in some muscular disorders [26, 27] and following chemoradiation therapy for head and neck cancer patients due to stricture formation [28–31]. Many patients, however, have no known etiology for their CP muscle dysfunction. Histopathologic studies of the CP muscles of patients with CPMD and Zenker's diverticulum show myopathic features including muscle fiber necrosis, phagocytosis, and increased fibroadipose tissue [4, 32]. These changes in the CP muscle can potentially influence the elastic properties of the muscle and its ability to open sufficiently during swallow [4, 33].

Many clinicians and investigators have hypothesized that CPMD, indicated by the radiologic appearance of a cricopharyngeal web or cricopharyngeus muscle bar, may result from direct exposure to excessive refluxate or from reflexive hyperfunction secondary to chronic gastroesophageal reflux, intended to prevent extraesophageal reflux. Previous studies have reported an increase in UES pressure following experimental acid infusion into the esophagus [17, 18]. Similarly, Chernichenko et al. [34] identified a spike in electromyographic (EMG) activity of the pig CPM in response to acid exposure, which was not evoked in response to bile perfusion. This finding was interpreted by authors as a response to prevent GERD from entering the pharynx. Brady et al. [35] found that premature contraction of the CP muscle was a positive predictor of the diagnosis of reflux. Also, Hunt et al. [16] reported high CP muscle resting pressure in some patients with reflux esophagitis that normalized in most patients following the successful repair of HH. Individuals with HH in our investigation were 2.57 times more likely to display evidence of CPMD ($p < 0.05$) and PES opening diameter was significantly smaller in this group ($p < 0.05$). These results support a relationship between CMPD and chronic reflux disease.

No correlation was observed in this investigation between HH size and the extent of PES opening within the HH group. Although the presence of HH as a risk factor for GERD has been well established, findings regarding the correlation of hernia size with the severity of reflux are equivocal [21, 36, 37]. Due to this uncertainty, use of HH as a surrogate for reflux may have presented a limitation for assessing the nature of the GERD-CPMD relationship. Future investigations that include measurements such as ambulatory pH and impedance monitoring could help further elucidate this relationship.

The prevalence of CPMD reported in this study is greater than those reported in previous videofluoroscopic studies [10, 38]. A possible explanation for this observation is that individuals who report solid food dysphagia are more likely to be referred for esophagrams and as such our cohort may inherently contain more cases of CPMD in comparison to all-comers referred for standard videofluoroscopic swallow evaluations. Furthermore, technical differences in videofluoroscopic evaluation procedures may also partially explain the differences in rates of CPMD findings. In our center, we utilize the anterior oblique (RAO) view which enables an unobstructed view of PES anatomy and uses a 60% weight/volume ratio of barium sulfate, allowing for improved visualization of CP webs and bars. It should be noted that this investigation did not include an assessment of the degree of dysphagia caused by the CPMD, but rather focused on the radiographic findings of CPMD, in an attempt to illuminate the relationship with HH.

Conclusion

These results suggest an association between the prevalence of hiatal hernia and cricopharyngeus muscle dysfunction. Individuals with HH were 2.57 times more likely to display evidence of CPMD ($p < 0.05$) and upper esophageal sphincter opening diameter was significantly

Table 1 Statistical summary of Hiatal Hernia and UES measurements

	UES diameter in lateral view (cm)	Hiatal Hernia size (cm)
Non-Hiatal Hernia Group	0.93 (± 0.25)	
Hiatal Hernia Group	0.81 (± 0.24)	3.73 (± 1.76)

Values are mean ± SD

smaller in this group ($p < 0.05$). These results support the role of gastroesophageal reflux in the development of CPMD, although further investigation is required to establish a causal relationship.

Compliance with Ethical Standards

Conflicts of interest The authors have no funding, financial relationships, or conflicts of interest to disclose.

Informed Consent Informed consent was obtained from all participants in the research study.

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