GIANT ZENKER'S DIVERTICULA

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ABSTRACT

Zenker's Diverticula (ZD) is one of the most common diverticula of the gastrointestinal tract and mostly seen in the elderly population. The reported incidence in the general population ranges 0.01-0.11%. This is basically a false and pulsion diverticula having only mucosal and submucosal layers. ZD arises from inherent weak point in the upper posterior oesophageal wall termed as Killian's triangle formed by oblique fibers of inferior pharyngeal constrictor muscle and cricopharyngeus muscle. The presentation of diverticula are mainly progressive dysphagia, regurgitation, halitosis, malnutrition and rarely bleeding. We report a case of large or massive ZD presented with relative less severe symptoms even after having mass effect over the adjacent oesophagus and trachea. This is uncommon finding for such ZD. The presentation was only progressive dysphagia and weight loss and diagnosed by Barium swallow and CT Esophagogram.

KEYWORDS

Zenker's Diverticula, False and Pulsion Diverticula, Dysphagia, Barium and CT Esophagogram.

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INTRODUCTION

ZD is a pulsion diverticula of cervical oesophagus region, also known as pharyngeal pouch, cricopharyngeal, pharyngoesophageal or hypopharyngeal diverticula. It arises from upper posterior oesophageal wall and protruded into superior mediastinum through an anatomically weak point called as Killian's triangle. Killian's triangle formed by oblique fibers of inferior pharyngeal constrictor muscle laterally and cricopharyngeus muscle inferiorly. This occurs predominantly in the elderly population mostly greater than 70 years of age.[1] ZD is rare entity with reported incidence of 0.01-0.11% in general population but the true incidence may be much more due to the number of asymptomatic patients.[2]

The formation of ZD is attributed to chronic increased intraluminal pressure in upper oesophageal area and lower hypopharyngeal area. Abnormal oesophageal motility and upper oesophageal sphincter dysfunction result in discoordination of the swallowing mechanism creating high intraluminal pressure. Since the weakest Killian's point lies posteriorly, this becomes the location of the false diverticulum formation.[3] The main complaints of ZD are dysphagia, regurgitation, hoarseness, halitosis, but the most common presentation is dysphagia in 80-90% of the people. As the diverticula increases in size the symptomatology also worsen. The patients may also have persistent cough in 30-40% population and aspiration with few having aspiration pneumonia.

Uncommon but severe and alarming symptoms are pain in chest and bleeding may signal the development of squamous cell cancer (0.4-1.5%).[4,5] The diagnosis of ZD can be made on a barium esophagogram or CT esophagogram. The diverticulum will be seen filling with barium or ionic contrast at the level of the cricothyroid cartilage posterior to the oesophagus. A lateral view is important so as not to miss the ZD, because of its dorsal position. The definitive treatment of ZD requires excision of the diverticulum and a cricopharyngeal myotomy. We report a case of ZD in an old female patient with the diagnostic approach and review of literature.

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CASE REPORT

A 70 years old female referred to our department due to several months of progressive dysphagia and occasional odynophagia first with solid foods, then with liquid foods but no regurgitation or aspiration. Due to fear to eat the patient had lost weight of approx. 8 kg. For last 15 days she had experienced frequent dry cough and intermittent low-grade fever. Her clinical examination only showed signs of malnutrition and no obvious evidence of respiratory tract infection. Laboratory tests were unremarkable except low level of Hb% and total protein. Local oropharyngeal examination was also normal.

A complete NCCT, CECT and HRCT of thorax was done to rule out any pathology of oesophagus causing dysphagia and any associated or isolated lung parenchymal changes. NCCT and CECT showed an elongated pouch-like lesion occupying most of superior mediastinum posterior to upper oesophagus. The lesion showed mottled density of air and fluid within it without any abnormal enhancement. So on basis of location of lesion and pouch-like appearance clearly it was diagnosed as Zenker's Diverticula. To confirm the diagnosis, CT ionic oral contrast esophagogram was done which clearly depicting a large diverticulum in upper prevertebral space through the posterior pharyngo-oesophageal wall and also the same in barium swallow (Fig. 1a, 1b, 1c and 2b, 2c).

The size of diverticula was 10x5x4 cm, extending from lower cricoid cartilage level to tracheal bifurcation displacing

oesophagus and trachea anteriorly. HRCT was unremarkable. Open surgery was done in the form of diverticulectomy and cricopharyngeal muscle myotomy through a lateral cervical approach. Postoperative events were normal and later on patient was discharged. Histopathology showed large diverticula with acute and chronic inflammatory changes.

Fig. 1 (a) ***Fig. 1 (b)***

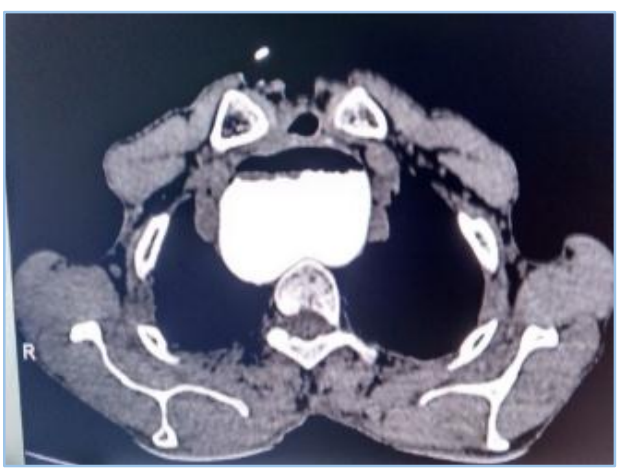

Fig. 1 (c)

Figure 1a (Sagittal), 1b (Coronal) and 1c (Axial) are Computed Tomographic Esophagogram Images of Thorax showing a large Zenker's Diverticulum resting in Lower Cervical and Central Aspect of Superior Mediastinum. Diverticula displacing the Oesophagus and Trachea very anteriorly and contrast remains in the diverticula and Failure of Contrast to pass into the Oesophagus.

Fig. 2a & 2b

Figure 2a (Antero-Posterior) and 2b (Lateral) are Barium Esophagogram views showing a large Zenker's Diverticulum where contrast remains in the Diverticulum without evidence of contrast in Oesophagus and Bronchus.

DISCUSSION

Oesophageal diverticula are as such rare and constitute of 0.08-4.0% according to endoscopic and radiologic series.[6] and ZD is the most common upper gastrointestinal diverticula representing 75% of them.[7] ZD is usually seen in older population in late 70 and 80 decades of life and rare in adult life.[8] Recent study shows the mean age of presentation was 73±12.3 years and higher frequency in male population.[7] It is false and pulsion diverticula arises dorsally at pharyngoesophageal junctional wall through Killian's triangle a region of relative anatomical weakness, formed by oblique fibers of inferior pharyngeal constrictor and transverse fibers of cricopharyngeus muscle.[9]

Small ZD is mostly asymptomatic and in symptomatic patient most common complaint is dysphagia (80-90%).[8] Other symptoms include odynophagia, halitosis, food stuck in throat, regurgitation of food, chronic cough. The symptoms may vary from weeks to several years depending upon the size of lesion. With enlargement of lesion, the symptoms may become more worsened; 30-40% of patient may also present with chronic cough, hoarseness, aspiration pneumonia. Sudden increase in symptoms like severe dysphagia, local pain and/or haemoptysis/haematemesis are alarming and raised the suspicion of ulceration or malignant tumoural change within the pouch.[8,10]

The pathophysiology of ZD is altered or decreased compliance of upper oesophageal sphincter (Cricopharyngeus muscle) and failure to open completely the sphincter for bolus clearance results into an increase in intrahypopharyngeal pressure.

Greater intrahypopharyngeal pressures have been noted in patients with ZD as compared with an age matched healthy population.[11] Since this is a disease of elderly people, degenerative muscular weakness and dysfunction of cricopharyngeus muscle are commonly attributed as aetiology.[8,6] ZD lined with stratified squamous epithelium and very rarely cancerous change in pouch reported (0.3-7% of cases), secondary to chronic irritation and inflammation over many years. Carcinoma in situ or small carcinomas are difficult to diagnose radiologically and even miss on endoscopic examination.[6,10]

Oesophageal barium swallow is the most important diagnostic tool. Complete anatomical description is supplemented by contrast CT esophagogram. Atypical outpouching noted on the dorsal surface of the oesophagus at the level of sternoclavicular joint in superior mediastinum. Depending upon the size (Craniocaudal), ZD is classified into: small (up to 2 cm), intermediate (2-4 cm) and large (4-6 cm).[10,12] Dynamic continuous fluoroscopy allows monitoring of the swallowing mechanism and may detect small diverticula, which mostly remain as symptomatic. Overflow and aspiration can be also accessed. Hiatus hernia or reflux esophagitis are other abnormalities noted sometime with these patients on endoscopic evaluation. A progressive filling defect or loss of the smooth contour of interior of the pouch raises the possibility of malignant change.[8,10]

Many different open surgical and intraoral endoscopic approaches are available for treatment, but still debate continues about which approach is superior for treatment. The choice of approach should be a careful discussion between the surgeon and the patient in order to obtain the optimal outcome with minimal complication.

Principle open surgical approach is diverticulectomy and cricopharyngeal myotomy and it was common practice prior to the use of the flexible endoscope. Intraoral endoscopic surgery is also effective and safe process, but not suitable for small size ZD (<3 cm). However, endoscopic method is preferred for high risk cases, where general anaesthesia can complicate the condition and has the advantage of faster recovery and shorter hospital stay.[3] and thus can be applied as OPD procedure.

For better result, a careful selection of approach must be considered by meticulous discussion between the operating surgeon and the patient. However, open surgical method is preferred choice if patient is asymptomatic while endoscopic approach is for symptomatic and large diverticula.

CONCLUSION

Zenker's diverticulum is one of the most common diverticula of upper gastrointestinal tract, typically presents in elderly population. The basic pathophysiology of ZD formation is altered and/or decreased mobility of muscles around the upper oesophageal sphincter causing increased intraluminal or intrabolus pressure with resultant protrusion of mucosa and submucosa through weak Killian's point dorsal to oesophagus. A small ZD can be asymptomatic, but large ZD always presents with severe symptoms especially increasing to almost complete obstructing acute dysphagia. Large diverticula can cause regurgitation and often complicated by aspiration and recurrent pneumonia. But with our patient with such large or massive ZD, no severe symptoms noted except experience of gradual increasing dysphagia for a long time without aspiration, aspiration pneumonia or bleeding, which is uncommon in general.

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