**Paroxysmal Postprandial Atrial Fibrillation Suppressed by Laparoscopic Repair of a Giant Paraesophageal Hernia Compressing the Left Atrium**

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**INTRODUCTION**

The current anatomical classification of hiatal hernia (HH) includes four types. Type I are sliding HHs with the gastroesophageal junction (GEJ) migrated above the diaphragm while the stomach is in its normal anatomical position. Type II are pure paraesophageal hernias (PEHs) with the GEJ in its normal anatomical position while the fundus is herniated through the hiatus. Type III hernias are a combination of type I and II and type IV are defined by the herniation of other structures than the stomach. Types II – IV hernias as a group are referred to as PEHs and represent 5-15% of the total HHs, with more than 90% of the PEHs being Type III [1].

Two potential mechanisms could explain the etiology of a giant HH: gastroesophageal reflux disease (GERD) that leads to esophageal scarring and shortening, and chronic positive pressure on the diaphragmatic hiatus with a predisposition to herniation [2].

Atrial fibrillation (AF), GERD and HHs are often seen in clinical practice and it has been suggested that GERD and HH represent risk factors for AF [3, 4]. Hiatal hernia appears to be associated with increased frequency of AF both in men and women of all age groups [5]. Hiatal hernia can directly impinge on the left atrium in a similar way as atrial masses. Since atrial masses are associated with various arrhythmias due to cardiac compression, HHs could potentially result in a similar effect [6, 7]. The link between the upper gastrointestinal tract and the AF is suggested also by the reported reduced number of recurrences of AF with the use of PPI or Nissen fundoplication [8].

**CASE REPORT**

We report the case of a 77-year-old female who presented at the Cardiology Department complaining of minimal effort dyspnea. The patient also mentioned daily acid regurgitation and postprandial palpitations. Her medical history included chronic heart failure, arterial hypertension, anxiety disorder, chronic obstructive pulmonary disease (COPD) stage III GOLD and a recent left humeral neck fracture, surgically treated.

Her heart rate was of 110 bpm and the blood pressure measured 130/80 mmHg with no antihypertensive medication.
The 12-lead electrocardiogram indicated AF. The 24 hours Holter monitoring demonstrated AF to be paroxysmal and associated with eating. Hormone levels, the clinical and echographic assessments of the thyroid function and structure showed no abnormalities.

Chest radiography showed widening of the mediastinum and a large abnormal shadow overlapping the heart (Fig. 1). The transthoracic echocardiography demonstrated a mass compressing the left atrium, a mild mitral regurgitation and no signs of hypertensive cardiopathy (Fig. 2). Left ventricular size and the contractile function were preserved and there was no significant valvular disease. Upper endoscopy was performed as well and revealed reflux esophagitis (grade A) and a giant PEH with 2/3 of the superior part of the stomach being situated intrathoracally without any mucosal abnormalities (Fig. 3). Spirometry proved mixed ventilator dysfunction with a FVC of 75%, FEV1 of 55%, Tiffeneau index of 62% and a positive bronchial challenge test.

The barium swallow revealed important reflux, a giant HH (13.5 cm) with the GEJ located intrathoracally and containing almost the whole stomach, except a small portion of the pyloric antrum that was very thin (Fig. 4a).

The documented correlation between symptoms and HH and the large size of the hernia led to the indication for surgery [1]. Pre-operatively she was treated with cardioselective beta-blockers, low molecular weight heparin and high doses of PPI. We performed a complete reduction of the herniated stomach, excision of the sac and crural repair through laparoscopic

![Fig. 1. Chest radiography: a) preoperative PA view: mixed hydroaeric image located in the inferior mediastinum; b) postoperative PA view: ascended left hemidiaphragm and normal mediastinum; c) preoperative lateral view: mixed hidroaeric retrocardiac image; d) postoperative lateral view: normal mediastinum.](image1)

![Fig. 2. Transthoracic echocardiogram: a) preoperative apical window: giant hiatal hernia compressing the left atrium; b) postoperative apical window: left atrium without any compression. LV – left ventricle; LA – left atrium; HH – hiatal hernia; X – space previously occupied by the giant hiatal hernia.](image2)
approach. Due to the stomach laxity we achieved a complete (360°) Nissen fundoplication.

Postoperative barium swallow and chest radiography showed a subdiaphragmatic stomach and GEJ (Figs. 1, 4). Transthoracic echocardiography showed that the compression on the left atrium was relieved and the mild mitral regurgitation ceased (Fig. 2). Postoperatively, the AF was suppressed and converted to normal sinus rhythm as per the electrocardiogram and the Holter monitoring. The patient was discharged 5 days after surgery in sinus rhythm.

The follow-up at 10 days after surgery revealed that the shortness of breath improved significantly, the pulse rate was 80 bpm and the electrocardiogram showed no AF.

**DISCUSSION**

The anatomical proximity of the left atrium to the HH makes the atria liable to mechanical irritation, neural connections and inflammation that may increase the risk for AF [5]. The relationship between gastrointestinal symptoms and arrhythmias was first described by Ludwig Roemheld, under the name of “Roemheld gastrocardiac syndrome”, in which an esophageo-gastric stimulus was able to induce arrhythmia-related symptoms [9, 10].

Large PEH predominantly occurs in the elderly population. Besides causing regurgitation and heartburn due to accompanying GERD, it may present with a wide spectrum of manifestations mimicking those of cardiovascular pathology such as postprandial syncope, angina-like chest pain, recurrent dyspnea and acute heart failure due to HH induced cardiac compression [11-13]. Further, HH may simulate the appearance of an intra-atrial mass on transthoracic echocardiography [13, 14].

Giant HH and GERD are two separate diseases, which frequently coexist, although either can occur without the other one, both being able to act as risk factors for each other and also for AF [2, 4]. Our patient was diagnosed with both GERD and giant HH. There have been numerous case reports and large retrospective studies that have suggested an association between AF and GERD due to the locally released cytokines from esophageal injury or due to the vagal overstimulation that create an environment that can induce and perpetuate AF [4, 8, 15-18]. On the other hand, the hypothesis that the presence of HH contributes to the development of AF is supported by previous reports. Roy et al. demonstrated epidemiologic association between HH and AF in young patients (age < 55) due to the cardiac compression and subsequent atrial irritation [5]. Gillinov et al. reported the case of a patient who had a surgical correction of a HH and had resolution of AF; however, this patient also had catheter ablation to treat the AF [19]. Schilling et al. reported that the repair of a large PEH resulted in the termination of AF [15]. Patel et al. also reported a case of a patient with atrial flutter caused directly by the mechanical effect of a large HH that occupied more than 50% of her thorax without reflux or heartburn that was resolved after surgery [20]. Duygu et al. reported a case of paroxysmal atrial flutter that was refractory to electrical cardioversion and calcium channel blockers in a patient with severe acid reflux and daily heartburn symptoms secondary to a large HH [21].

Patients with large HHs have also significant dyspnea and it has been suggested that left atrial compression has a causal role in the pathogenesis of HH-associated dyspnea [22]. Moreover, in our case the dyspnea was also determined by the rapid ventricular rate accompanying AF. Our patient’s shortness of breath improved significantly after the HH repair.
It is not stated yet if AF is caused by the direct compression of the HH on the left atrium or by the association of GERD, which accompanies most cases of giant HH.

Besides the direct compression of the HH and the associated GERD, there are also other possible risk factors that can induce AF such as ischemic heart disease, chronic heart failure and thyroid dysfunction. For a 77-year old patient, it is difficult to completely rule out the presence of ischemic heart disease without performing coronaryarography. However, besides the age, the patient had no other cardiovascular risk factors (diabetes, obesity, dyslipidemia) and was a non-smoker. The arterial hypertension mentioned in her medical history seemed to have been more linked to the previous episodes of anxiety, as at the time of evaluation the blood pressure values were normal without any antihypertensive medication. The cardiac echography showed no sign of hypertensive cardiopathy either. Furthermore, the good medication. The cardiac echography showed no sign of pressure values were normal without any antihypertensive episode of anxiety, as at the time of evaluation the blood history seemed to have been more linked to the previous smoker. The arterial hypertension mentioned in her medical history seemed to have been more linked to the previous episodes of anxiety, as at the time of evaluation the blood pressure values were normal without any antihypertensive medication. The cardiac echography showed no sign of hypertensive cardiopathy either. Furthermore, the good tolerance of the patient, without any chest pain, at the onset of fast rhythm AF represents a strong argument against myocardial ischemia.

The “chronic” heart failure mentioned in her medical history was in fact related to the symptomatology induced by the non-permanent AF which in its turn was caused by the cardiac compression of the HH. The normal volume of the left atrium on echocardiography examination contradicts the “chronic” evolution of the heart failure. In addition, the mild mitral regurgitation ceased after the reversion of the cardiac compression as a result of the HH surgical treatment.

CONCLUSION

To the best of our knowledge there are no case series and no case reports in the literature of giant HH associated with paroxysmal AF which was suppressed after laparoscopic surgical repair. This could indicate that direct cardiac compression represents the main mechanism for inducing AF. In order to determine the full relationship between HH and AF, future prospective investigation of patients with both AF and HH would be required.

Conflicts of interest: None to declare.

REFERENCES