Hiatal hernia as a rare cause of cardiac complications – case based review of the literature

Karol Krawiec¹, Adam Kadej², Marcin Szczasny¹, Andrzej Głowniak¹, Małgorzata Piasecka³, Piotr Blaszczak¹, and Andrzej Głowniak¹

¹ Department of Cardiology, Cardinal Wyszynski Hospital, Lublin, Poland
² Department of General Surgery, Cardinal Wyszynski Hospital, Lublin, Poland
³ Department of Anaesthesiology and Intensive Therapy, Cardinal Wyszynski Hospital, Lublin, Poland

Abstract

Introduction. Hiatal hernia (HH) is a condition which refers to the protrusion of an intraabdominal organ in the thorax cavity through an oesophageal hiatus of the diaphragm. Sliding HH is usually associated with non-specific symptoms, including heartburn, regurgitation or epigastric pain. Importantly, true paraesophageal hernia may lead to cardiac compression. Knowledge of cardiac manifestations of HH is limited.

Objective The main aim of the study is to present the rare case of a patient with gastrothorax due to hiatal hernia which caused cardiac arrest, and to provide a literature-based review of the cardiac aspects of hiatal hernia.

Brief description of the state of knowledge. Patients with paraesophageal hernia may experience arrhythmia, including sinus tachycardia, atrial flutter, atrial fibrillation, supraventricular extrasystole and ventricular tachycardia, as well as left bundle branch block, atrioventricular conduction block and electrocardiographic changes in the ST-segment and T-wave. In echocardiograph, HH may appear as an extracardiac posterior mass encroaching on the left atrial cavity, mimicking the left atrial mass. Rarely, HH may be manifested as tension gastrothorax leading cardiac arrest. In such a case, timely diagnosis and instant adequate treatment of the underlying condition are crucial.

Conclusions. Hiatal hernia should be considered as a possible cause of arrhythmia and changes in ST-T pattern, particularly if symptoms occurred after a meal. Differential diagnosis of the posterior mediastinal mass or intracardiac mass should include hiatal hernia. Gastrothorax is a rare condition associated with hiatal hernia which may lead to cardiac arrest. However, even timely recognition and therapy of gastrothorax does not ensure a positive clinical outcome.

Key words: Gastrothorax, hiatus hernia, cardiac arrest, acute heart failure

INTRODUCTION

Hiatal hernia is a condition which refers to the protrusion of an intraabdominal organ or organs in the thorax cavity through an oesophageal hiatus of the diaphragm [1, 2, 3]. According to the classification based on the location of the gastroesophageal junction in relation to the crural pillar, there are four types of hiatal hernia [1, 4]. Type I is also known as a sliding hernia and is defined as a displacement of the gastroesophageal junction above the diaphragm. This is the most common type of hiatal hernia which accounts for approximately 85–95% of all hiatal hernias. Types II, III and IV are classified as true paraesophageal hernias and represent about 5–15% of cases of hiatal hernia. Type II is characterized by the herniation of the gastric fundus. Type III is the most common type of true paraesophageal hernias, and results from the herniation of gastric fundus and gastroesophageal junction through the diaphragmatic hiatus. The hernia may enlarge, drawing other parts of stomach into the chest [4]. Type IV results from a large defect in the phrenoesophageal membrane leading to the herniation of stomach and other abdominal organs [1–4]. Figure 1 presents classification of hiatal hernia.

There is no single definition of a massive hiatal hernia. It may be defined as a hernia which is bigger than 5 cm, or to the herniation of the entire stomach into the mediastinum [5]. Giant hiatal hernia may also refer to a herniation of more than 30% or – by other authors – more than 50% of the stomach [6]. Some authors indicate that the size of hernia is not sufficient to be the sole criterion of massive hernia [5]. Thus, there are reports of a massive hiatal hernia which encompasses type II, III and IV [5].

While sliding hernia is usually associated with the gastroesophageal reflux disease, paraesophageal hernias may lead to the obstruction, ischemia, or volvulus of the contents of the hernial sac [3]. It may also cause respiratory distress due to compression of the lung [5]. Large hiatal hernia may also result in the displacement and compression of the heart [7]. However, data about cardiac manifestations of hiatal hernia are scarce and mostly limited to case reports. The aim of this study is to present the case of a patient with a giant hiatal hernia which caused cardiac arrest, and to present a review of the literature of the cardiological aspects of hiatal hernia.
CASE STUDY

A 51-year-old, obese (BMI 30.86 kg/m²) agricultural worker was admitted to the Emergency Department due to severe epigastric pain and vomiting, following the lifting of heavy fertilizer bags.

In physical examination on admission the patient was tachycardiac (150/min) with tachypneic (30/min). Blood pressure was normal 110/60 mmHg although the pulse was hypokinetic and jugular vein distention was present. Chest examination revealed a dull percussion note and absence of respiratory sounds in the left hemithorax. The abdomen was distended, with tenderness in the epigastric area.

Electrocardiogram (ECG) displayed sinus tachycardia, intermediate heart axis, low voltage of QRS complexes, incomplete right bundle branch block, q in leads II, III and aVF.

Computed tomography (CT) of chest and abdomen performed without delay revealed a giant hiatal hernia strangulated in the chest cavity, with hernia’s gate about 7 cm. The stomach was distended, twisted and significantly (> 50%) translocated into the chest resulting in displacement and compression of the heart. The hiatal sac included also a part of the duodenum and its mesentery. The portal venous gas was stated and the left lung significantly compressed (Fig. 2).

The patient was referred for an emergency thoracotomy and laparotomy. Directly after transportation of the patient to the operating theatre, sudden cardiac arrest in the mechanism of pulseless electrical activity (PEA) occurred. Immediate left-sided thoracotomy was performed to release the trapped viscera, and to enable direct cardiac massage which was successfully performed and normal sinus rhythm restored. Afterwards, during laparotomy, hernia content was returned to the abdominal cavity, the hiatus hernia repaired and the patient transferred to the intensive care unit. Regardless of the effective surgical intervention, in the postoperative period the patient experienced significant complications, including cardiac and respiratory failure, post-cardiac arrest brain injury, acute kidney injury and gastrointestinal bleeding. He required mechanical ventilation and continuous infusion of catecholamines, haemodialysis, neuroprotective treatment, and broad-spectrum antibiotics. The patient died on day 22 after surgery due to multiple-organ failure.

DISCUSSION

Tension gastrothorax as hiatal hernia complication.

Tension gastrothorax is a rare and life-threatening condition, which causes contralateral shift and compression of the mediastinal structures by translocation of the stomach into the chest cavity [8, 9]. It is usually associated with congenital diaphragmatic hernia in children, or occurs due to diaphragmatic injury as a result of trauma or complication
of some surgical procedures [10, 11]. Sporadically, tension gastrothorax may be a manifestation of hiatal hernia – as in case of the presented patient [8, 10]. Tension gastrothorax may be difficult to diagnose because its clinical manifestations, including dyspnea, tachycardia and hypotension, may mimic pneumothorax [9, 10]. However, contrary to the pneumothorax, in the gastrothorax, bowel sounds may be auscultated over the lungs fields [9]. Patients with gastrothorax may also present with abdominal pain and vomiting [9].

Tension gastrothorax may lead to cardiopulmonary arrest [9, 10] which ensues due to extrinsic compression of the heart by the strangulated hiatal hernia, which occurred in the presented case.

There are only few published case reports of tension gastrothorax complicated by cardiac arrest [8–15]. The majority were caused by trauma [9, 11–14], while by hiatal hernia in only two cases by [8, 10]. Solé et al. reported the case of a 75-year-old man with chest pain, dyspnea and nausea. Minor increase of troponin I with ST-segment depression in ECG suggested acute coronary syndrome. The patient experienced cardiopulmonary arrest, recovering after resuscitation, but with persistent hypotension. Echocardiogram did not show any significant abnormalities. Chest CT revealed a giant hiatal hernia causing cardiac displacement, and compression of the left lobar bronchus [8]. Shoij et al. presented the case of a 60-year-old man with suspected myocardial infarction as an underlying condition of cardiopulmonary arrest. However, in echocardiogram there were no abnormalities, and further imaging studies revealed tension gastrothorax due to a hiatal hernia as the actual cause of cardiac arrest [10]. Thus, it appears that tension gastrothorax may mimic acute coronary syndrome and should be considered as a potential reason of cardiac arrest.

Diagnosis of tension gastrothorax should be based on clinical signs and symptoms, although timely imaging studies are essential for the appropriate diagnosis. Radiological signs suggesting gastrothorax include the presence of abdominal organs in the thorax, elevated diaphragm, compressive atelectasis, mediastinal displacement, visible fluid levels, and/or the presence of gas bubbles in the chest [12, 16]. In the current case, a CT scan of the abdomen and chest revealed the presence of massive hiatal hernia which compressed the heart to the anterior thoracic wall, and constricted the left lung.

The emergency initial management of gastrothorax included decompression of the stomach by the placement of a nasogastric or orogastric tube [10]; laparotomy or thoracotomy was the treatment of choice. In the presented case, the patient required both. Similar management was needed in a patient reported by Shoij et al. [10]. However, according to the literature, some patients with gastrothorax complicated by cardiac arrest underwent solely laparotomy [13, 14] or thoracotomy [9, 11]. During the surgery, the anatomical position of the organs is restored and the hiatus hernia repaired [10]. It should be highlighted that laparoscopy can increase the pressure in the abdominal cavity, thus it should be performed with caution [10].

**Hiatal hernia and changes in electrocardiogram.** Changes in the electrocardiogram pattern and rhythm have been observed in patients with hiatal hernias [17–35]. However, the linkage between these conditions has not been fully elucidated. Table I presents a summary of selected case reports of patients with abnormal findings in electrocardiogram, presumably triggered by the hiatal hernia reported in the literature.

In most cases, electrocardiographic alternation disappeared after initial stomach decompression or surgical correction of the hiatal hernia [18, 20, 21, 30–35], or after successful conservative management with dietary control and proton pump inhibitor [29]. Resolution of abnormal findings in ECG after management of hiatal hernia may imply a causal relationship between the hiatal hernia and changes in the electrocardiogram pattern and rhythm. However, ECG repolarization disorders persistent up to three months have been observed after successful surgery, possibly due to pericardial irritation [26]. Roy et al. carried out a retrospective analysis of patients with hiatal hernia for the presence of atrial fibrillation. The authors demonstrated that 7.1% of all patients with hiatal hernia experienced atrial fibrillation. Interestingly, the prevalence of atrial fibrillation was 17.5-fold and 19-fold higher in men and women younger than 55 years with hiatal hernia, compared to the general population [36]. Although the exact mechanism of changes in electrocardiogram related to the hiatal hernia is not well understood, some hypotheses have attempted to explain these findings. Schilling et al. suggested that persistent compression of the left atrium by the hiatal hernia may result in an area of relative ischemia and conduction block, causing reentry [31]. Another explanation of changes in electrocardiogram may be the stimulation of the vagal nerve by pressure from the hiatal hernia [31, 37]. Increased vagal tone may precipitate the onset of tachycardia in the mechanism similar to that in bradycardia-tachycardia syndrome [29]. Kounis et al. presumed that the increase in direct or indirect pressure exerted on the global surface of the heart appears to be the cause of the electrical alternation observed in electrocardiography of patients with increased intrathoracic pressure, including those with hiatal hernia [38]. Maruyama et al. implied that mechanical contact and irritation on the left atrium or pulmonary veins by the hiatal hernia may contribute to the ectopic firing leading to the atrial fibrillation [39]. Roy et al. suggested that since hiatal hernia is related to reflux oesophagitis, the inflammation may extend to the surrounding organs, including the left atrium, and lead to tachyarrhythmia due to mechanical or chemical/ neural impact mediated through the vagal or sympathetic nervous system [36]. Basir et al. hypothesized that ST elevation may be related to torsion or compression of the epicardial artery from direct pressure from the hiatal hernia [21]. In patients without hiatal hernia but with extreme abdominal distension and hemi-diaphragm elevation, it has been also suggested that elevation of ST in the electrocardiograph may result from the mechanical compression or transient vasospasm of one or more coronary arteries from the diaphragm impinging on the myocardium from severe abdominal distention [40]. Patients with a large hiatal hernia may experience arrhythmias, including sinus tachycardia, atrial flutter, atrial fibrillation, supraventricular extrasystole and ventricular tachycardia, as well as left bundle branch block, atrioventricular conduction block and electrocardiographic changes in the ST-segment and T-wave. There is a need to consider hiatal hernia as a reason for the abnormal electrocardiogram pattern and rhythm, particularly in patients with clinical symptoms that occurred after meals or in the supine position.
<table>
<thead>
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<th>Case</th>
<th>Gender</th>
<th>Age</th>
<th>Symptoms</th>
<th>ECG</th>
<th>ECHO</th>
<th>X-ray</th>
<th>CT</th>
<th>Surgical method</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schmer W. 2017 [17]</td>
<td>M</td>
<td>73</td>
<td>Dyspnea, chest pain</td>
<td>Left bundle branch block</td>
<td>No information</td>
<td>Cardiomegaly, large mass behind the heart</td>
<td>Large HH</td>
<td>Not described</td>
</tr>
<tr>
<td>Hokama J. et al. 2005 [18]</td>
<td>F</td>
<td>79</td>
<td>Chest pain, vomiting</td>
<td>ST-T changes</td>
<td>Mass compressing LV, LV, IVC</td>
<td>Large shadow overlapping cardiac silhouette</td>
<td>Type III giant HH compressing the heart and IVC</td>
<td>Nissen fundoplication, Hill’s method</td>
</tr>
<tr>
<td>Kakarala K. et al. 2015 [24]</td>
<td>M</td>
<td>41</td>
<td>Chest pain, dyspnea</td>
<td>ST-T changes</td>
<td>No information</td>
<td>HH, pneumopericardium, pleural thickening or fluid in the left base</td>
<td>Type I HH, pneumopericardium, pericardial effusion</td>
<td>Thoracotomy, laparotomy</td>
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<tr>
<td>Rubini-Gimenez M. et al. 2019 [26]</td>
<td>F</td>
<td>61</td>
<td>Dyspnea, chest pain</td>
<td>ST-T changes</td>
<td>Diastolic dysfunction, high PAP</td>
<td>No information</td>
<td>Giant HH</td>
<td>Laparoscopy</td>
</tr>
<tr>
<td>Avind A. et al. 2019 [27]</td>
<td>F</td>
<td>87</td>
<td>Chest pain, nausea</td>
<td>ST-T changes</td>
<td>Mass compressing LA and LV causing tamponade physiology</td>
<td>No information</td>
<td>Large HH compressing the heart</td>
<td>Toupet fundoplication</td>
</tr>
<tr>
<td>Gürgün C. et al. 2002 [28]</td>
<td>F</td>
<td>76</td>
<td>Dyspnea</td>
<td>Atrial fibrillation</td>
<td>Normal</td>
<td>Cardiomegaly, a dome shaped air level overlapping cardiac silhouette</td>
<td>No information</td>
<td>Laparoscopy</td>
</tr>
<tr>
<td>Duygu H. et al. 2008 [29]</td>
<td>F</td>
<td>79</td>
<td>Chest pain</td>
<td>Atrial fibrillation</td>
<td>TR, MR, high PAP</td>
<td>No information</td>
<td>HH</td>
<td>Non-operative treatment</td>
</tr>
<tr>
<td>Cristian D.A. et al. 2015 [30]</td>
<td>F</td>
<td>77</td>
<td>Dyspnea</td>
<td>Atrial fibrillation</td>
<td>Mass compressing LA, MR</td>
<td>Widening of the mediastinum, large shadow overlapping the heart</td>
<td>No information</td>
<td>Nissen fundoplication</td>
</tr>
<tr>
<td>Schilling R.J. et al. 1998 [31]</td>
<td>M</td>
<td>72</td>
<td>Heart palpitations</td>
<td>Atrial flutter with 2:1 AV block</td>
<td>Normal</td>
<td>Large mediastinal mass</td>
<td>No information</td>
<td>Not described</td>
</tr>
<tr>
<td>Patel A. et al. 2014 [32]</td>
<td>F</td>
<td>80</td>
<td>Failure to thrive and weakness</td>
<td>Atrial flutter</td>
<td>High PAP</td>
<td>Cardiomegaly, a large lucency involving the mid and lower hemi-thoraces</td>
<td>Large HH</td>
<td>Gastropexy</td>
</tr>
<tr>
<td>Tursi A. et al. 2001 [33]</td>
<td>F</td>
<td>75</td>
<td>Weakness, dysphagia, heartburn</td>
<td>Supraventricular extrasystole, atypical right bundle branch block, and inferior axis</td>
<td>No information</td>
<td>Giant gastric HH compressing LA</td>
<td>No information</td>
<td>Nissen-Rossetti fundoplication</td>
</tr>
<tr>
<td>Gnanenthiran S.R. et al. [34]</td>
<td>M</td>
<td>78</td>
<td>Syncope</td>
<td>Ventricular tachycardia</td>
<td>Mass compressing LA, dyskinesis of LV</td>
<td>No information</td>
<td>No information</td>
<td>Laparoscopy</td>
</tr>
<tr>
<td>Gleadle J. et al. 1989 [35]</td>
<td>F</td>
<td>65</td>
<td>Vomiting</td>
<td>Sinus tachycardia, ST-T changes</td>
<td>HH compressing and displacing the heart</td>
<td>Very large HH</td>
<td>No information</td>
<td>Not described</td>
</tr>
<tr>
<td>Present report</td>
<td>M</td>
<td>51</td>
<td>Abdominal pain</td>
<td>Sinus tachycardia, low voltage of QRS complexes, incomplete right bundle branch block</td>
<td>No information</td>
<td>No information</td>
<td>Giant HH compressing and displacing the heart</td>
<td>Thoracotomy, laparotomy</td>
</tr>
</tbody>
</table>

F – female; HH – Hiatal hernia; IVC – inferior vena cava; LA – left atrium; LV – left ventricle; M – male; MR – Mitral regurgitation; PAP – pulmonary arterial pressure; TR – Tricuspid regurgitation
Hiatal hernia and echocardiography. Hiatal hernia may be visualized in the transthoracic echocardiogram while it is encroaching on the posterior part of the left atrium and left atrioventricular junction [41]. Hiatal hernia is usually seen as an extracardiac posterior mass encroaching on the left atrial cavity, mimicking a left atrial mass on transthoracic echocardiography [25, 42–54]. However, this may not be obviously apparent since its visualization relates to the imaging plane and respiratory fluctuation [41]. It has been observed that hiatal hernia is seen in its maximal dimension while the left atrium is imaged in a posterior plane. On the other hand, while the left atrium is imaged in an anterior plane, the hiatal hernia appears to be progressively smaller or even absent on transthoracic echocardiography [55].

D’Cruz et al. described features of hiatal hernia commonly seen on the echocardiograp, including:

- a large ill-defined amorphous solid mass apparently filling all or most of the left atrial chamber in the apical 4-chamber view (imaging the posterior atrial plane), which sometimes may extends across the atrial septum into the adjacent right atrial space;
- a large convex poorly-demarcated mass impinging on the posterior left atrial wall, atrioventricular junction, or even occasionally on the postero-basal left ventricular wall in the parasternal or apical long-axis views;
- paradoxical motion of the left ventricular wall if there is its encroachment;
- respiratory fluctuation in degree of encroachment of the hiatal hernia mass on the left atrium;
- normal sonolucency of the descending thoracic aorta in the apical 4-chamber and long-axis views partly or completely obscured by the large echorgenic hiatal hernia [55].

Oral ingestion of a carbonated beverage may lead to visualization of swirling echodensities in the mass or distinct echo-free space replacing a substantial portion of the original ‘mass’, facilitating the differential diagnosis between a true left atrial mass and a hiatal hernia on transthoracic echocardiogram [42, 50, 55, 56]. The use of intravenous echocardiographic contrast may also be helpful in establishing the diagnosis of hiatal hernia [56]. Smelly et al. suggested that the combination of an oral ingestion of carbonated beverage mixed with echocardiographic contrast media should better delineate extracardiac structures [56].

Hiatal hernia may be difficult to visualize in the transesophageal echocardiography because of the inability to obtain adequate images [57]. However, Frans et al. suggested that on the transesophageal echocardiography, hiatal hernia may be seen as a posterior, mass-like lesion with microbubbles, and thick inner lining resembling the stomach mucosa [58].

There have been several papers reporting left atrial compression with haemodynamic collapse and heart failure due to hiatal hernia [59–62]. However, only Ishibashi et al. have presented a hiatal hernia which protruded into the left ventricle and caused paradoxical movement in the posterior wall [60]. Hiatal hernia should be taken into consideration if a posterior mediastinal mass impinging on the left atrium is visualized in the echocardiogram [56]. However, differentiation of hiatal hernia with other posterior mediastinal mass lesions or intracardiac masses may be challenging for echocardiographists. Thus, it requires additional imaging, including computed tomography or magnetic resonance imaging [63].

Hiatal hernia mimicking cardiac tamponade. Hiatal hernia has been reported as a rare cause of cardiac compression mimicking cardiac tamponade due to a distended stomach after coronary artery bypass and post-type A aortic dissection repair surgery [64–66]. It has been suggested that patients with hiatal hernia may benefit from a prophylactic nasogastric tube placement prior cardiac surgery, limiting the risk of gastric stasis, vomiting, aspiration pneumonia and haemodynamic compromise [64, 65]. Prompt decompression of the distended stomach results in the improvement of the haemodynamic status of patient and resolution of the symptoms [65, 66].

Extra-pericardial pathologies including hiatal hernia should be considered in patients’ post-cardiac surgeries with symptoms suggesting cardiac tamponade without its objective evidence on echocardiography [64, 65].

Hiatal hernia and syncope. In the literature there are several reports of a syncope due to a huge hiatal hernia. Saito et al. reviewed nine cases of syncope related to hiatal hernia from the literature between January 1995 – August 2016 [67]. Most of the reported episodes of syncope occurred after eating an unusually large meal. It has been suggested that compression of the enlarged stomach on the left atrium may be the cause of syncope [68]. Haemodynamic instability due to the cardiac compression may result in a decreased preload and cardiac output which lead to syncope [69]. Differential diagnosis of left atrium compression should also include tumours and thrombosis; therefore, further imaging studies may be necessary for final recognition [70]. Maekawa et al. reproduced a syncopal attack in their patient using a water pouring test through a nasogastric tube [71]. Hiatal hernia should therefore be considered in the differential diagnosis of postprandial syncope.

CONCLUSIONS

In conclusion, hiatal hernia should be considered as a reason of arrhythmia and changes in the ST-T pattern, particularly if clinical symptoms occurred after a meal. Differential diagnosis of posterior mediastinal mass lesions or intracardiac masses should include hiatal hernia. Gastrothorax is a rare condition associated with hiatal hernia which may lead to cardiac arrest. Early diagnosis based on imaging studies enables appropriate management. However, even timely recognition and therapy of gastrothorax does not ensure a positive clinical outcome.

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