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Cardiac Herniation Following Pneumonectomy—An Old Complication Revisited

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SUMMARY

Cardiac herniation is a recognised complication of pneumonectomy when a pericardial defect has been made during resection. This complication is very rare and, with the increasing preference for more limited resections, is even less frequently encountered now than it was several decades ago. Uncorrected cardiac herniation is usually lethal, with a high incidence of morbidity and mortality even after correction. We present a case of left-sided cardiac herniation following intrapericardial pneumonectomy to illustrate the difficulty of making this rare diagnosis. Aetiology, pathophysiology, clinical picture and diagnosis of cardiac herniation are reviewed. We also describe the treatment and prevention of this serious complication. Cardiac herniation should be considered in any patient with acute deterioration after pneumonectomy.

Key Words: COMPLICATIONS: acute cardiac herniation. SURGERY: thoracic

Cardiac herniation following pneumonectomy was first described in 1951 by Bettman and Tannenbaum[1]. It is a complication of pneumonectomy that can occur if a pericardial defect has been made during resection[2-9]. This complication is very rare with only about 50 cases reported worldwide since 1951, the majority of which have been published in the surgical literature. With the increasing preference for more limited resections[10] and changes in technique, we believe this complication is even less frequently encountered now than it was several decades ago. Cardiac herniation is lethal with 100% mortality if the complication is unrecognised[3] and a quoted mortality rate of 50% even in those where the complication is managed promptly[3,6,8,9]. We present this case report to illustrate the difficulty of making the diagnosis, particularly as the clinical features may not be sufficiently specific and ECG and chest X-ray findings can be easily misinterpreted[5,6]. We believe that echocardiography in this situation provides a relatively easy and non-invasive bedside test to distinguish cardiac herniation from other causes of collapse or chest pain following pneumonectomy. We also offer this case report as a reminder of the potential for severe harm if the diagnosis remains unrecognised, and that extra vigilance with a high index of suspicion is required in all patients who are left with a pericardial defect following pneumonectomy.

CASE HISTORY

A 54-year-old man was scheduled for left thoracotomy and lung resection for a squamous cell carcinoma of the left upper lobe. He was an ex-smoker of 25 pack-years who presented with a two-month history of moderate haemoptysis. He had no history of cardiovascular disease but was taking pravastatin for hypercholesterolaemia. He had no other medical history of note and had a good exercise tolerance. Physical examination was unremarkable. He had a normal preoperative ECG and preoperative pulmonary function tests were consistent with a mild obstructive defect. Computerized tomography confirmed a mass around the left upper lobe bronchus close to the pulmonary artery. Positron Emission Tomography showed no evidence of metastatic spread.

After full non-invasive monitoring and a peripheral venous cannula had been placed, anaesthesia was induced and maintained with a target controlled infusion of propofol at an initial plasma target level of 6.0 μg/ml. In addition, 100 μg of fentanyl and 50 mg of atracurium were given. The trachea was intubated with a 39 French right-sided double-lumen endo-

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bronchial tube (Rusch UK Ltd) and correct placement was confirmed by auscultation and visualization. A catheter was placed into the right radial artery to invasively monitor blood pressure because of the significant risk of major bleeding in this case. The patient was turned to the right lateral decubitus position and endobronchial tube position was again verified. Anaesthesia was maintained using 40 to 50% oxygen, nitrous oxide and propofol. Neuromuscular blockade was maintained with intermittent boluses of atracurium and 10 mg of morphine was given intravenously for analgesia.

Following a standard left fifth space thoracotomy, a tumour mass occupying the subaortic fossa and not invading the pulmonary artery was found. A left intrapericardial pneumonectomy was performed with the pericardial defect left widely open. A thoracostomy tube was inserted during closure of the chest wall and clamped before connection to an underwater seal. Anaesthesia and one-lung ventilation were uneventful and vital signs remained stable throughout with a systolic blood pressure of 100 to 150 mmHg, a heart rate of 70 to 80 beats per minute and haemoglobin oxygen saturation (SpO_2) maintained at 99%. A left paravertebral catheter for local analgesia was inserted under direct vision at the end of the procedure. The patient was extubated and transferred to recovery uneventfully. In recovery, a paravertebral infusion of lidocaine at 50 mg per hour and a patient-controlled analgesia infusion of morphine were commenced.

Approximately 30 minutes after arrival in recovery, the patient suddenly lost consciousness. He was immediately mask ventilated with 100% oxygen and a weak pulse was palpated. He remained unconscious for about two minutes but a palpable pulse was maintained throughout. On regaining consciousness he was clammy and complained of central chest tightness. He responded well to intravascular volume replacement with a colloid solution although there was no external evidence of bleeding. Differential diagnoses of haemorrhage, acute myocardial event and cardiac herniation were considered. He was transferred immediately to intensive care where invasive monitoring of arterial blood pressure could be commenced. A 12-lead electrocardiogram showed a sinus tachycardia of 110 and widespread ST depression and T wave inversion in leads III, aVR, aVL and V1 to V5. Chest X-ray at this time was consistent with a post-pneumonectomy picture. With no further evidence of bleeding, the anaesthetic and surgical teams jointly considered a perioperative ischaemic event to be the most likely diagnosis and the patient was given aspirin and commenced on a GTN infusion. In addition a left subclavian central line was inserted and the patient maintained on facemask oxygen.

As per our post-pneumonectomy protocol the chest drain was intermittently unclamped for 30 seconds every hour in order to assess blood loss. The patient initially remained stable but following unclamping of his chest drain on the sixth hour postoperatively, there was a transient loss of consciousness and a fall in systolic blood pressure to 50 mmHg with a concomitant bradycardia of 50 beats per minute. Blood loss from the chest drain during unclamping was 50 ml. Total perioperative blood loss was only 700 ml. His blood pressure did not respond to intravenous fluids and an adrenaline infusion at 2 μg per minute was commenced. A 12-lead ECG showed right axis deviation and marked ST elevation in leads II, III and avF with widespread ST depression in all lateral leads (Figure 1).

A diagnosis of acute inferior myocardial infarction was made by the attending medical team and in addition to supportive treatment, beta blockade was commenced with 25 mg of oral atenolol (following a test dose of intravenous esmolol). Blood was taken to measure cardiac enzymes and urgent coronary angiography was planned for when the patient was more stable. The chest drain was not unclamped again following this episode. Over the next three hours the patient complained of further episodes of chest pain associated with cardiovascular instability, and in view of what appeared to be ongoing ischaemia, his inotrope infusion was changed to dobutamine at 5 μg/kg/min. Overnight his blood pressure remained stable, with no further episode of chest pain or loss of consciousness.

Approximately 18 hours postoperatively he had a further transient episode of loss of consciousness and a severe fall in blood pressure related to being laid flat. A repeat chest X-ray (Figure 2) showed what appeared to be an abnormal cardiac shadow over the left hemithorax. Urgent trans-thoracic echocardiography demonstrated a malrotated and abnormally positioned heart. The diagnosis of cardiac herniation was made and the patient returned urgently to theatre.

Anaesthesia was induced with etomidate 10 mg, fentanyl 100 μg and vecuronium 8 mg. His trachea was re-intubated with a size 9.0 cuffed oral endotracheal tube and he was turned to the right lateral position. The previous incision was reopened and the heart was found to be lying outside the pericardium, rotated to the left. Bruising was noted over the lateral side of the left ventricle, which was con-

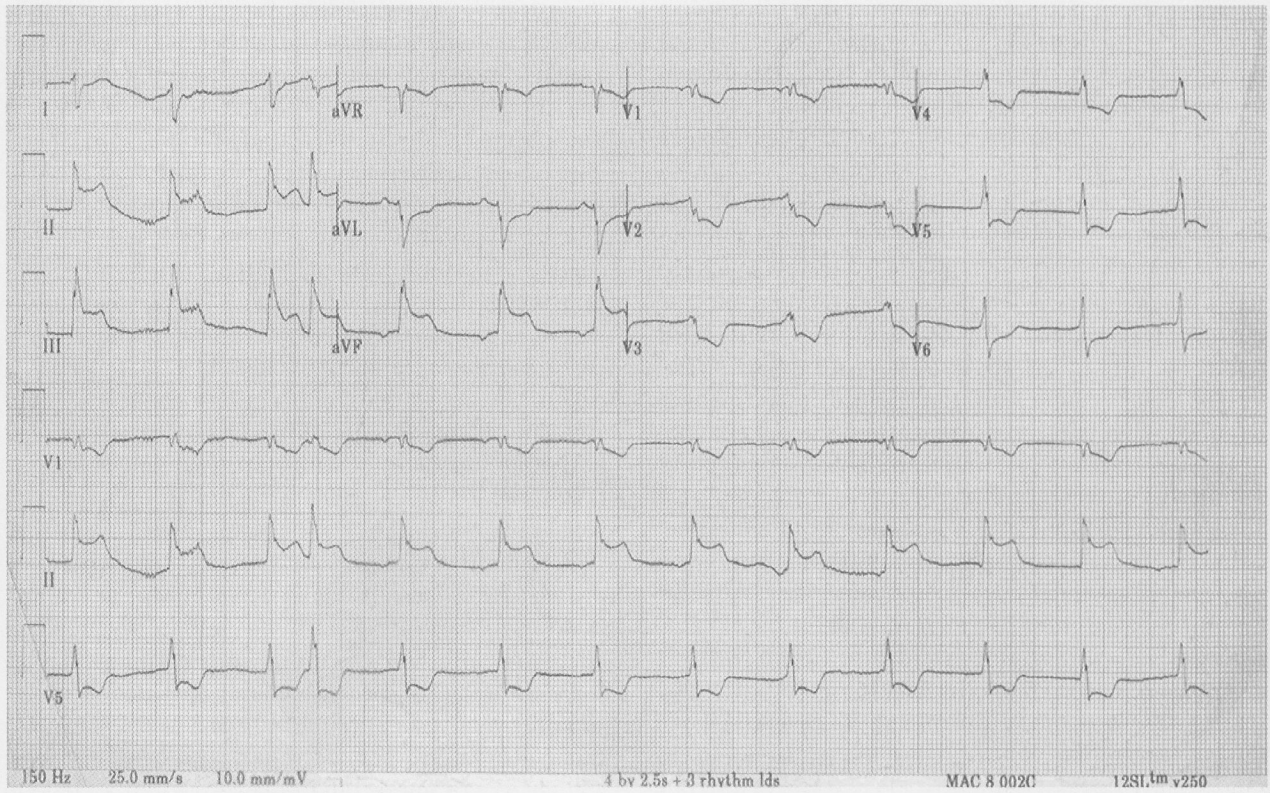

FIGURE 1: ECG of this patient.

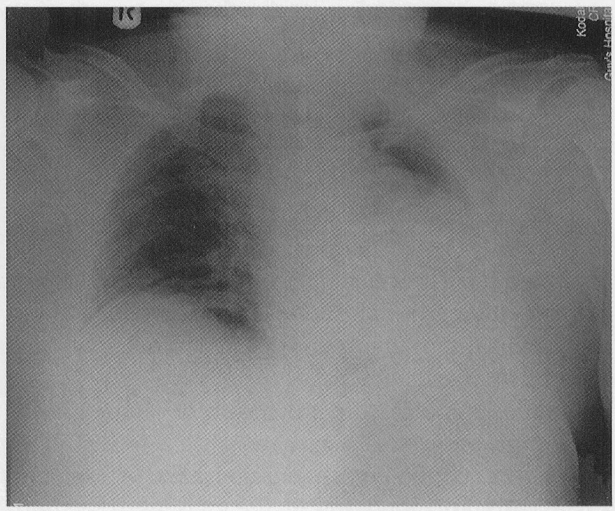

FIGURE 2: AP chest X-ray showing abnormal (?) cardiac shadow over left hemithorax.

tracting sluggishly. The heart was returned to the pericardium and the defect closed with a sheet of pleura raised from the postero-lateral chest wall and a new thoracostomy tube was inserted. His blood pressure remained stable throughout the procedure whilst continuing on a dobutamine infusion of 5 μg/kg/min. He was extubated at the end of the procedure and returned to intensive care on facemask oxygen (60%).

Repeat electrocardiograms showed no ST abnormalities and creatine kinase MB fraction was not elevated. He remained in intensive care for a further 48 hours during which time the dobutamine infusion was slowly discontinued. He returned to the ward on the third postoperative day where he continued to make an uneventful recovery. He was discharged home on the seventh postoperative day.

DISCUSSION

This case report illustrates the difficulty that cardiac herniation can present to the physician in the immediate postoperative period. Pneumonectomy has among the highest morbidity and mortality of any thoracic procedure with a perioperative death rate ranging from 7 to 11%[10]. Pneumonectomy was formerly the standard curative resection for lung cancer but currently is indicated only when lesser resections are not possible[10,11]. Intrapericardial pneumonectomy is reserved for resection requiring intrapericardial ligation of the great vessels[11]. Cardiac herniation is a

recognised complication of this procedure. In cardiac herniation the heart herniates through a defect in the pericardium into the evacuated hemithorax, usually leading to profound cardiovascular collapse. Herniation can occur into the right or left chest spaces depending on the side of the resection.

Cardiac herniation most commonly occurs in the immediate postoperative period with 75% occurring at the end of surgery[12] and nearly all occurring within the first 24 hours[8,12]. Therefore this complication is most likely to be encountered when the patient is still under the care of the anaesthetist in recovery[5] or the intensive care physician in an intensive care or high dependency unit. It is worth noting our patient had his first episode of cardiovascular collapse within 30 minutes of returning to the recovery unit. Late onset is rare, due to the rapid development of adhesions between the heart and pericardium that are thought to develop within three days of operation[6,13]. There have been several case reports of delayed onset, with two case reports documenting cardiovascular collapse occurring at least 24 hours following surgery[6,7]. In these cases, herniation was thought to have occurred initially without any cardiovascular consequences. One case report documents herniation occurring at 14 weeks following lobectomy with partial pericardectomy[13].

Cardiac herniation can occur spontaneously due to congenital pericardial defects or following traumatic injury[8,13], but most commonly occurs following pulmonary surgery[13]. It is particularly associated with intrapericardial pneumonectomy where the defect in the pericardium has deliberately been left open[8]. It can occur with defects of any size[7] and has even been recorded following closure of the pericardial defect at the initial operation[7,14,15]. It is likely to occur less often on the left than the right side as a wide excision of the pericardium on the left should logically prevent strangulation[2]. There are believed to be several possible mechanical events that may precipitate cardiac herniation into the evacuated chest space. Any rise in negative pressure in the intrathoracic space may precipitate herniation such as suction on the thoracostomy tube[3,5,6,8,16]. In addition, coughing on extubation and positive pressure ventilation have also been described as precipitating factors[3,5,6]. Changes in patient position have also been associated with precipitating herniation, particularly when the operative side becomes dependent and gravity facilitates dislocation of the heart through any defect[5]. In our patient there was no immediate precipitating event preceding the first collapse in recovery. The patient had been smoothly extubated whilst breathing spontaneously at the end of surgery. Although a thoracostomy tube was inserted, it remained clamped initially in recovery and no external suction was ever applied. Subsequent decompensation in our patient was associated with unclamping of the thoracostomy tube, the unclamping possibly leading to a slight negative pressure that in retrospect may have precipitated herniation[5]. Also the final episode of collapse was associated with a change in position from the sitting to the supine position, which presumably facilitated further herniation.

The pathophysiological process and the clinical picture are dependent on which side herniation occurs[16], although this complication can occur without any clinical signs or symptoms at all[17]. In right-sided herniation the heart dextrorotates[2], leading to torsion of the great vessels and obstruction of the superior vena cava and inferior vena cava (IVC)[6,8,13]. This in turn leads to symptoms of superior vena cava syndrome (dusky face, distended veins in neck and upper torso) which may be confused with a diagnosis of acute thrombosis or stenosis of the superior vena cava[5]. In addition, because of the obstruction to the superior vena cava and inferior vena cavas, there is a rapid decrease in venous return and cardiac filling, leading to a dramatic fall in cardiac output and blood pressure[6,16,19].

Left-sided herniation leads to strangulation of the ventricles by the edges of the pericardial defect. There is obstruction to coronary artery flow causing the myocardium to become increasingly oedematous, ischaemic and permanently damaged if untreated[6,8,13,18]. Left-sided herniation therefore presents with signs of dysrhythmia, myocardial ischaemia or infarction accompanied by ECG changes consistent with ischaemia or infarct[6,8]. In addition, strangulation of the ventricles leads to outflow obstruction and a profound fall in cardiac output[18]. Common to herniation on either side is a sudden loss of cardiac output with subsequent hypotension, dyspnoea and displacement of the apex beat[7].

In hindsight, our patient probably developed a left-sided herniation in recovery, which led to his fall in cardiac output and loss of consciousness. Obstruction to the coronary blood flow by the margins of the pericardial defect caused the ischaemic ECG changes we witnessed. Unclamping the drain or changes in the position of the patient exacerbated or initiated further episodes of herniation. In between these episodes the heart probably returned to a position where its mechanical function was less obstructed, although the continued presence of ischaemic changes on ECG suggests there still remained obstruction to the coronary vessels.

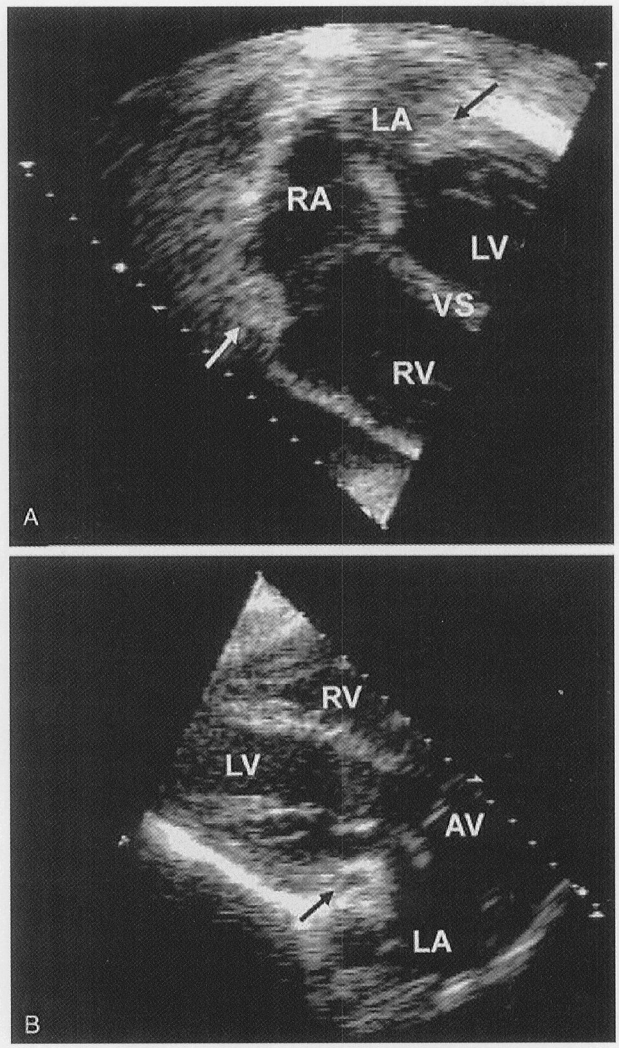

FIGURE 3: Cardiac herniation: Reproduced with permission from Journal of the American Society of Echocardiography[25]. A: From the paraapical transducer position, the atria appear elongated and compressed. In the atrioventricular grooves, there is a mass of encircling pericardial tissues (arrows). The ventricles appear bulbous relative to the atrial configuration. B: Transducer in the parasternal position obtaining a four-chamber view. The retracted pericardium produces a mass effect in the left atrium (arrow). The left atrium is small and elongated whereas the ventricles are bulbous, hypermobile, and distorted. AV, Aortic valve; LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle; VS, ventricular septum.

Diagnosis rests on an awareness of the potential problem and the correct interpretation of data from clinical signs, ECG and chest X-ray. The chest X-ray may not always confirm a diagnosis particularly if the herniation is on the left, as often the X-ray picture is not striking[3,5,6,16]. X-ray signs on the right are often more striking with displacement of the cardiac apex into the right chest cavity. There may also be associated displacement of any thoracostomy tube, a sharp kink in any central venous line or a clockwise rotation of any pulmonary artery catheter[19]. In either right or left cardiac herniation, the heart may appear laterally displaced (obvious for herniations into the right chest cavity) with the apex at right angles to the mediastinum[3]. The heart may appear to be more spherical in shape and a notch may appear between the cardiac mass and great vessels[3]. ECG analysis may show axis deviation, rhythm disturbances (which are common after pneumonectomy) and ST segment changes. The appearance of ECG changes may be the first signs of cardiac herniation[6]. These signs, symptoms and simple tests are often not sufficiently specific to allow an early diagnosis[6]. Additional investigations have been used to confirm the diagnosis of cardiac herniation such as bedside angiocardiography[2] and thoracoscopy under local anaesthesia[20]. However we believe that echocardiography offers a relatively easy and non-invasive additional technique for confirming or supporting the diagnosis (Figure 3).

The differential diagnosis of postoperative hypotensive collapse following pneumonectomy (Table 1) includes cardiovascular events such as hypovolaemia from haemorrhage or third space losses, acute mediastinal shift, acute myocardial ischaemia or infarction, cardiac tamponade, vagal responses, pulmonary oedema, pulmonary embolus, cardiac herniation and arrhythmia[3,6]. Other differential diagnoses for early postoperative deterioration include respiratory events such as aspiration or acute lung injury, neurological events such as cerebrovascular accidents, or seizures, metabolic derangements or immunological events such as adverse drug reactions or anaphylaxis. Management should involve concurrent resuscitation and investigation of the underlying cause. The conscious patient may be able to provide a history of events including pain, palpitations or dyspnoea. General examination should include assessment of neurological status, fluid balance and evidence of drug adverse reaction. Examination of the cardiorespiratory system should seek evidence of bleeding, mediastinal shift, pneumothorax, embolus, cardiac failure or herniation. Particular attention should be paid to pallor, cardiovascular indices, chest drain losses, tracheal displacement, position of the apex beat and precordial impulses, heart sounds and chest examination. Much of this may be quite difficult to interpret in this setting and further investigation may be required.

These should include an ECG for evidence of arrythmia, ischaemia or embolus; a chest X-ray for evidence of mediastinal displacement, cardiac herniation, air or fluid in the chest cavity, acute lung

TABLE 1
Some causes of hypotension post-pneumonectomy with key diagnostic features and initial treatment

Possible Cause	Diagnosis	Treatment
Hypovolaemia	Review fluids/blood loss, CVP reading, swing on arterial pressure trace, unclamp drain to check for continued bleeding	• Oxygen by mask • IV crystalloid, clear colloid or blood • X-match further blood • Return to theatre with continued bleeding
Primary arrhythmia	ECG	• Oxygen by mask • Check potassium and correct • Check magnesium and correct • Consider DC shock • Adenosine 6-12 mg for SVT • Amiodarone 150-300 mg IV for fast AF
Myocardial ischaemia/infarction	Serial ECG, cardiac troponin	• Oxygen by mask • Oral aspirin 75 mg • Sublingual GTN • GTN infusion • IV β-blocker • Consider urgent angiography
Mediastinal shift	CXR	• Oxygen by mask • Unclamp chest drain • Clamp drain when intrapleural pressure is – 5 cm H_2O • If no drain, measure intrapleural pressure and add/remove air to produce – 5 cm H_2O
Anaphylaxis	Clinical, urinary tryptase	• Oxygen by mask • Stop giving initiator • Epinephrine • IV fluids
Contralateral pneumothorax	CXR	• Needle decompression • Chest drain insertion
Cardiac herniation	Clinical, CXR, echocardiography	• Oxygen • Lateral position (good side down) • Keep chest drain clamped • Return to theatre

injury/fluid overload and central venous pressure catheter tip position. Blood should be taken for arterial blood gas analysis as well as haemoglobin, electrolytes and glucose, clotting studies and cardiac enzymes. An echocardiogram may show evidence of ventricular failure, wall motion abnormalities, cardiac herniation or rotation, or pulmonary embolus.

This case illustrates the difficulty, particularly with left-sided herniation, of diagnosing this phenomenon and how it can mimic the signs and symptoms of other complications of pneumonectomy. In our case, we made a provisional diagnosis of collapse secondary to a primary ischaemic event. The intermittent nature of our patient's symptoms and the initial lack of any precipitating cause made diagnosis of cardiac herniation relatively difficult. The ECG findings were consistent with an ischaemic event and the initial chest X-ray signs lacked any striking abnormality. In addition, the surgical team felt that the deliberately large hole in the left pericardium from the primary operation made strangulation of the heart unlikely. These problems led to a potentially critical delay in the diagnosis and it was only through echocardiography that we were clearly able to reach a definitive diagnosis.

Immediate treatment of cardiac herniation requires turning of the patient so that the non-operative side is dependent[5]. Gravity reduces the herniation and the heart may return into the pericardial sac. This will often lead to an improvement in cardiac output. Increases in inspiratory pressure should be avoided and the injection of air into the evacuated hemithorax may be considered[4,5]. Definitive treatment requires open surgery to reduce the hernia and repair the pericardial defect by primary closure, prosthetic patch or autologous graft[4,7]. Anaesthesia should be induced whilst maintaining the lateral position with the non-operative side dependent[17].

There have been a number of surgical approaches

to preventing cardiac herniation but none can definitively avert this complication. Classically, as in our case, defects (particularly those on the left side) are widened to prevent incarceration of the heart[2,7,24]. Increasingly, some authors have recommended routine closure of all pericardial defects by a variety of means. This can be by primary closure[5], patch closure with artificial materials[7], patch closure using pleural flap[7,22,23] or patch closure using fascia lata from the lateral thigh[21]. Closure of the pericardium increases the risk of tamponade and artificial materials provide a risk of infection[7,21].

In summary cardiac herniation is a documented but very rare complication of intrapericardial pneumonectomy. It may occur with a defect of any size and even after the pericardium has been closed. It is likely to become increasingly rare as more limited resections are now undertaken for the treatment of lung cancer. Cardiac herniation remains a complication which, even after treatment, has a high mortality. Diagnosis can be difficult, particularly when herniation occurs on the left side. We recommend that any patient who has undergone intrapericardial pneumonectomy should be closely monitored and cardiac herniation should be considered as one of the primary differential diagnoses of collapse. We believe echocardiography is a useful non-invasive tool to confirm the diagnosis. Cardiac herniation is most likely to occur when the patient is still under the care of the anaesthetist or intensive care physician and therefore we should remain vigilant to the onset of this potentially lethal complication.

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