

Inferior acute myocardial infarction due to acute cardiac herniation after right pneumonectomy

Yasunobu Terauchi · Hiroaki Kitaoka · Katsutoshi Tanioka · Toru Kubo ·
Shunichi Imamura · Yuichi Baba · Yasumasa Kawada · Tatsuya Noguchi ·
Makoto Okawa · Naohito Yamasaki · Toshikazu Yabe · Yoshinori Doi

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Abstract A 69-year-old man underwent right intrapericardial pneumonectomy for lung cancer. After 24 h, he went into shock with inferior acute myocardial infarction. We performed urgent coronary angiography, which revealed total occlusion of the mid-right coronary artery. Intravascular ultrasound showed that the artery seemed to be compressed from the pericardial side. We implanted a coronary stent because the lesion was refractory to balloon dilatation. After the procedure, we performed computed tomography and cardiac herniation was diagnosed. Emergency thoracotomy was performed to return the herniated heart to its normal position. This patient was discharged 38 days after initial surgery.

Keywords Cardiac herniation · Intrapericardial pneumonectomy · Lung cancer

Introduction

Cardiac herniation after surgery was first reported in Bettman et al. [1]. It is very rare but can be fatal because of severe shock or arrhythmia. It can also cause acute myocardial infarction (AMI) due to compression of the coronary artery between the hernial orifice and myocardium. Here we present a case of inferior AMI due to acute cardiac herniation after right pneumonectomy.

Case report

A 69-year-old man underwent a right intrapericardial pneumonectomy for lung cancer (squamous cell carcinoma, T3N1M0 stage IIIA). His coronary risk factor was active cigarette smoking (1 pack/day for 50 years). He had no significant past history or family history.

The surgery was performed in the left lateral position, and a residual 10 × 10-cm defect in the right pericardium remained after surgery. Postoperatively, the patient was hypotensive even after administration of inotropic agents. When the patient was moved to the right lateral position, he experienced a transient decrease in blood pressure to 50/20 mmHg.

After 24 h, the patient went into persistent shock with ST segment elevation in leads II, III, and aVF on electrocardiography, indicative of an inferior AMI. The patient was then referred to the cardiology section.

Blood pressure was 40 mmHg and pulse rate was 140 beats per minute (bpm; regular). No murmur was audible and the jugular vein was dilated. There was no difference in blood pressure between the upper limbs bilaterally.

Electrocardiography showed that the heart rate was 140 bpm (sinus, regular) with ST segment elevation in leads II, III, and aVF and ST segment depression in leads V1, V2, and V3. Chest X-ray revealed deviation of the heart to the right chest cavity. However, because this X-ray was similar to the immediate postoperative X-ray, we did not notice significant changes (Fig. 1). Evaluation of heart condition by transthoracic echocardiography provided no significant information due to poor visualization. We diagnosed the patient as having ST elevated MI (inferior) with hypotension caused by the combination of right ventricular infarction due to right coronary artery (RCA) occlusion and pulmonary hypertension after pneumonectomy.

Y. Terauchi (✉) · H. Kitaoka · K. Tanioka · T. Kubo ·
S. Imamura · Y. Baba · Y. Kawada · T. Noguchi · M. Okawa ·
N. Yamasaki · T. Yabe · Y. Doi
Department of Medicine and Geriatrics, Kochi Medical School,
Kohasu, Oko-cho, Nankoku, Kochi, Japan
e-mail: teya@jewel.ocn.ne.jp

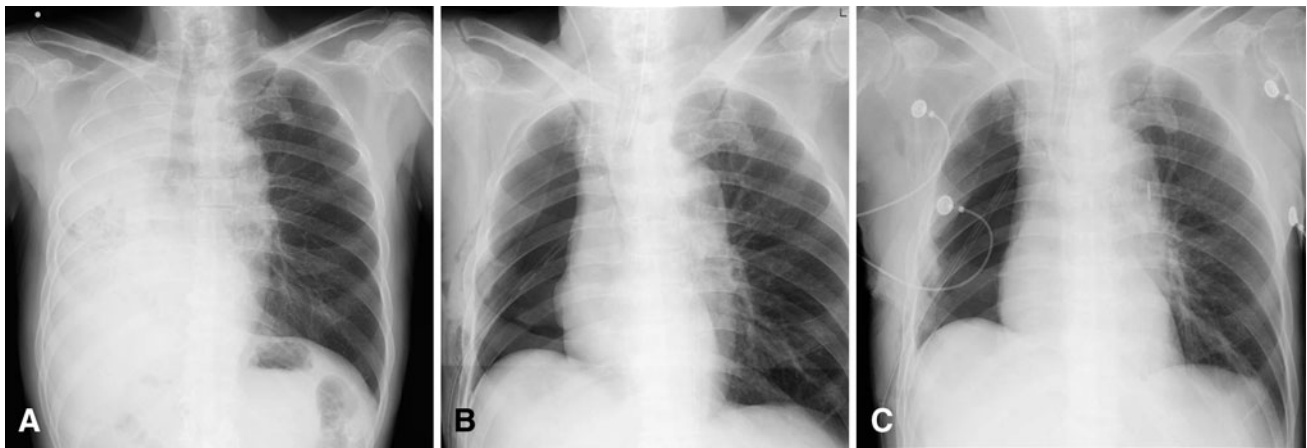


Fig. 1 Chest X-ray. **a** Chest X-ray taken before the operation showing atelectasis of the right lung due to lung cancer. **b** Immediate postoperative chest X-ray showing already deviation of the heart to

the right side of the chest cavity. **c** Although the patient went into persistent shock, no significant change was noted compared with the immediate postoperative X-ray

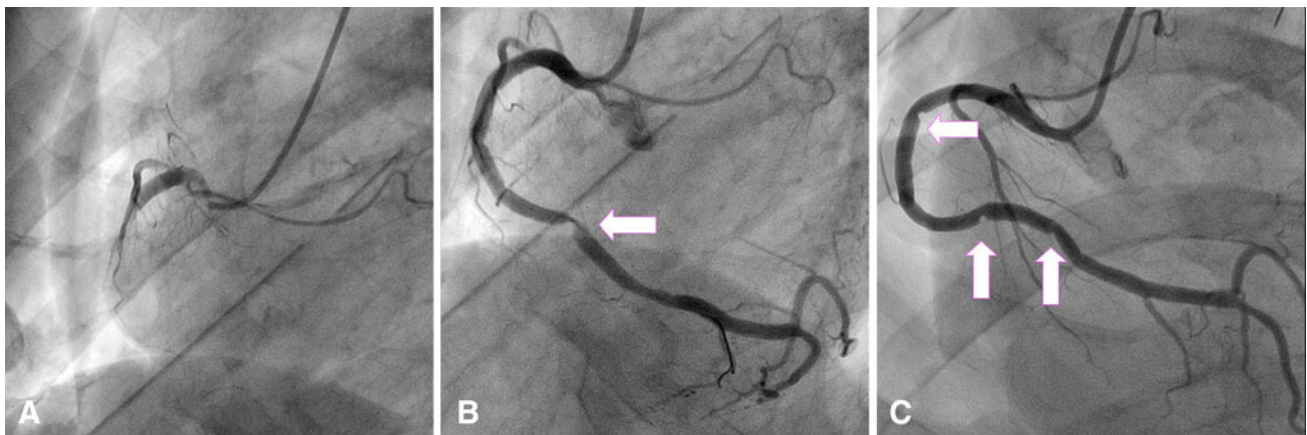


Fig. 2 Coronary angiography (CAG) and percutaneous coronary intervention. **a** CAG demonstrating total occlusion of the mid-right coronary artery (RCA). **b** After guidewire was crossed to RCA, 99% stenosis of RCA segment 3 became apparent (*arrow*). **c** After the stent

was placed at RCA segment 3, new kink lesions appeared on both sides of proximal and distal edge of the stent and RCA segment 2 (*arrows*). We consider that they were caused by accordion phenomenon in the situation of cardiac herniation

We performed urgent coronary angiography using an intra-aortic balloon pump (IABP), which revealed total occlusion of the mid-RCA (Fig. 2a). After crossing the guidewire to RCA, coronary flow improved to thrombolysis in myocardial infarction (TIMI) grade 3, and 99% stenosis of RCA segment 3 became apparent (Fig. 2b). Because the patient was still in the early postoperative period, we tried to treat this coronary lesion by balloon dilatation only; however, recoil occurred immediately. We checked the lesion using intravascular ultrasound (IVUS). No significant plaque was found at that, and it seemed to be compressed by something from pericardial side (Fig. 3). When we removed the guidewire, coronary flow quickly decreased to TIMI grade 2, and diastolic blood pressure augmented by IABP fell from 80 to 50 mmHg. We decided to place a bare metal stent and finally could keep the good coronary flow. After placing the stent, new kink lesions

appeared on both sides of proximal and distal edge of the stent and RCA segment 2 (Fig. 2c). Because coronary blood flow was good and diastolic blood pressure could be maintained at 80 mmHg, we finished the procedure.

After percutaneous coronary intervention (PCI), we performed a computed tomography (CT) scan to clarify the cause of the compression. Intravenous contrast-enhanced CT scan revealed cardiac herniation; the right atrium was incarcerated within the right pericardial defect and the stent site of RCA was sandwiched between the hernial orifice and cardiac muscle (Fig. 4).

Emergency thoracotomy was performed to return the heart to its normal position and to close the pericardial defect with a polytetrafluoroethylene sheet. Blood pressure stabilized immediately after the repositioning of the heart.

After 24 h from the second operation, bleeding in the thoracic cavity occurred, and a third thoracotomy was

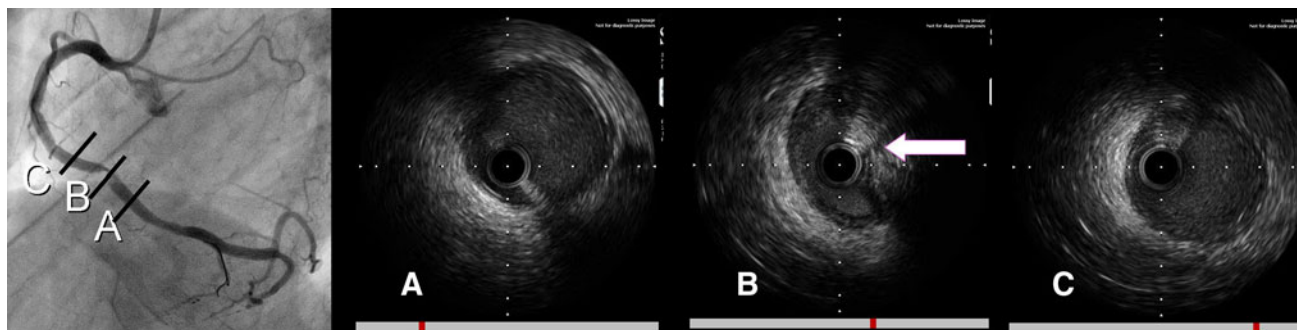


Fig. 3 Intravascular ultrasound (IVUS) findings. IVUS showed no significant plaque around the lesion of the right coronary artery (RCA). **a** Distal site of the lesion. **b** RCA was compressed by something from pericardial side (*arrow*). **c** Proximal site of the lesion

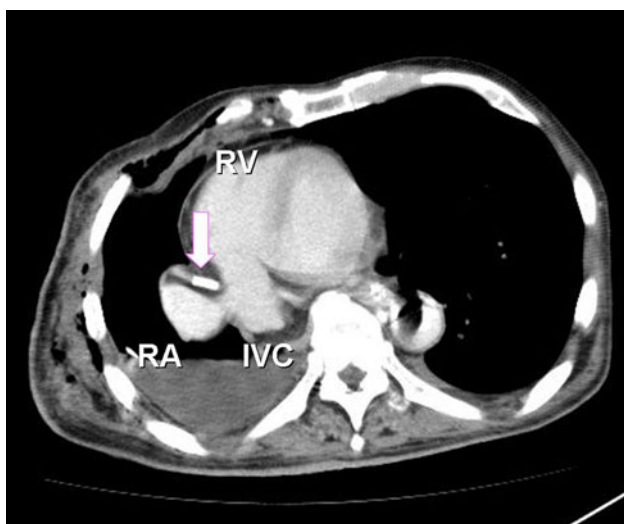


Fig. 4 Intravenous contrast-enhanced computed tomography scan after percutaneous coronary intervention. **a** Right atrium herniated to the right chest cavity due to a pericardial defect. *Arrow* the stent in the right coronary artery (RCA). The stent site was compressed between the hernial orifice and the myocardium. *IVC* inferior vena cava, *RA* right atrium, *RV* right ventricle

performed, after which the patient's clinical course was uneventful (peak creatine kinase 2200 IU/L, peak creatine kinase MB 377 IU/L). Anti-platelet agents were administered continuously and subacute stent thrombosis did not occur.

After 30 days from the first thoracotomy we evaluated the patient's cardiac condition by a cardiac CT scan; no kink lesions were detected in RCA. Patency of the stent site was confirmed (Fig. 5). No cardiac herniation was observed. The patient was discharged on foot 38 days after the first thoracotomy.

Discussion

Cardiac herniation occurred due to the protrusion of the heart through the pericardial defect. This is rarely seen as a

complication after intrapericardial pneumonectomy [2], but causes potentially life-threatening hemodynamic instability.

Kimura et al. [3] reviewed 68 cases of cardiac herniation after lung surgery, and reported that it occurred more frequently on the right side (46 cases) than on the left side (22 cases). In those patients, 12 right-sided and 9 left-sided cases were fatal. Veronesi et al. [4] reported that mortality due to cardiac herniation is 100% in unrecognized cases and 50% in recognized and surgically corrected cases.

Cardiac herniation into the right chest cavity causes incarceration of the superior vena cava, inferior vena cava, and/or right atrium, resulting in decreased filling of the right ventricle and subsequent shock. On the other hand, according to the literature, cardiac herniation into the left chest cavity results in myocardial ischemia [5]. However, in some cases of right side herniation, myocardial ischemia may also occur, similar to our case [3].

Myocardial ischemia is caused by compression of the coronary artery between the hernial orifice and cardiac muscle. Myocardial ischemia can also be due to distortion of the coronary artery caused by deviation of the heart. In our case the major cause of myocardial ischemia was compression of the coronary artery. The CT scan taken just after PCI showed that the stent site in RCA was located between the hernial orifice and the cardiac muscle (Fig. 4). Otherwise, we did not check the new kink lesions observed in RCA after placing the stent with IVUS, but cardiac CT scan taken after repositioning of the heart showed no detectable kink lesions in RCA (Fig. 5). Hence, we consider that they were caused by accordion phenomenon in the situation of cardiac herniation.

Previously reported triggers of cardiac herniation after pneumonectomy are suction of the chest tube, coughing, positive pressure ventilation, and repositioning of the patient with the operated side downwards [6].

Quick diagnosis is necessary in cases of cardiac herniation to avoid sudden death. Diagnosis is sometimes difficult; however, AMI and pulmonary embolism are possible differential diagnoses. Deviation of the heart shown in chest X-ray

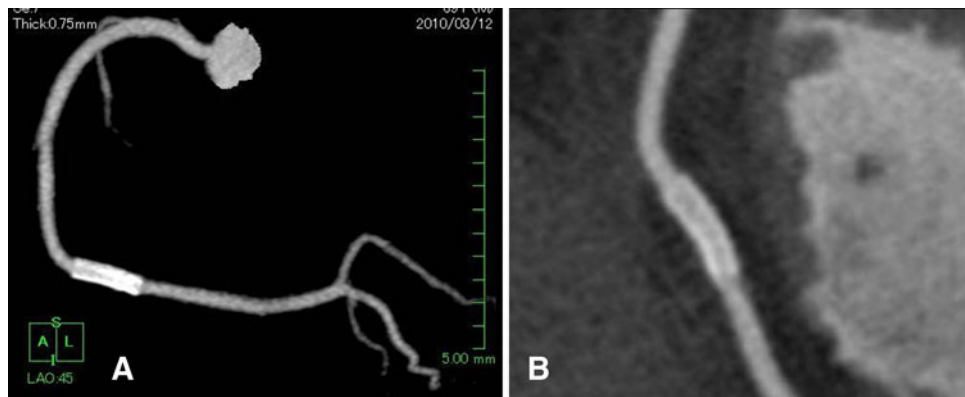


Fig. 5 Cardiac computed tomography (CT) scan before discharge. Cardiac CT was performed using a 64-slice multi-detector CT (Somatom Definition; Siemens). Parameters: 64×0.6 mm collimation, 120 kV, 380 mAs/rotation, 0.33-s rotation time, pitch value 0.2. Retrospective ECG gating was used, and reconstruction was done with a 0.75-mm slice thickness and a 0.4-mm interval. Retrospective reconstruction of the image data was performed for acquisition of phase images starting from late systole (20% of the R–R interval) and

ending at late diastole (80% of the R–R interval) using 10% increments. In checking these eight phase images, we detected no kink lesions in the right coronary artery (RCA) and confirmed the patency of the stent site. **a** Angiographic view of RCA in the end-diastolic phase showing no detectable kink lesions. **b** Curved planar reconstruction of RCA in the end-diastolic phase showing the patency of the stent site

may be important for the diagnosis. In the case of cardiac herniation occurring after pneumonectomy, however, it is possible for the heart to deviate from its normal position just after the operation, as in our case. Rodgers et al. [7] reported that thoracoscopy or CT is useful for diagnosis. Otherwise, Kimura et al. [3] recommended that exploratory thoracotomy should be done when hemodynamic instability is observed after intrapericardial pneumonectomy.

Of course, the prevention of cardiac herniation is most important. Closure of pericardial defects by suturing directly or using an artificial patch is necessary after intrapericardial procedures in order to prevent cardiac herniation. However, it has been seen even in cases where the pericardial defect was sutured directly [3]. Pericardial defects are often sutured loosely to avoid postoperative cardiac tamponade. Suturing too tightly can cause an increase in the intrapericardial pressure. Both situations may instigate cardiac herniation.

On the other hand, in cases undergoing artificial patch closure, the closure is very close to the edge of the bronchial stump, increasing the risk of infection. However, considering the bad prognosis associated with cardiac herniation, Junzo et al. [8] asserted that it is essential to avoid direct suturing whenever possible, and to prevent cardiac herniation by patch closure.

Conclusion

We report a case of inferior AMI due to acute cardiac herniation after right pneumonectomy. Because we could not diagnose it immediately, we performed urgent coronary

angiography. During this procedure we observed compression of RCA by IVUS and diagnosed cardiac herniation by CT scan and managed to save this patient.

We have to recognize cardiac herniation as a fatal complication after pneumonectomy with intrapericardial procedure.

Conflict of interest There is no conflict of interest associated with the authors in connection with this manuscript. This is an original manuscript that has not been previously published elsewhere.

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