Sudden ventricular fibrillation due to absence of pericardium in left upper lobectomy: a case report

Guo-Cao Wang, M.D.[1,2,†]; Xi-Rong Li, M.D.[2,†]; Ning Huang, M.D.[2]; Hai-Tao Tian, M.D.[1,*]

[1] School of Clinical Medicine, Jining Medical University, Jining, Shandong Province, China

[2] Department of Anesthesiology, Affiliated Hospital of Jining Medical University, Jining, Shandong Province, China

[†]Guo-Cao Wang and Xi-Rong Li contributed equally to this work.

Running title: Pericardium absence and cardiac arrest

Corresponding author

* Hai-Tao Tian, M.D.

School of Clinical Medicine, Jining Medical University, Hehua Road 133#, Jining, Shangdong, China

Tel: (+86) 13639435218, E-mail: mzxwgc@163.com

Previous presentation in conferences: Not applicable.

Conflicts of interest:

No potential conflict of interest relevant to this article was reported.

Funding: Not applicable

Acknowledgments: Not applicable.

IRB number: Not applicable

Clinical trial registration number: Not applicable.

Sudden ventricular fibrillation due to absence of pericardium in left upper lobectomy: a case report

Running title: Pericardium absence and cardiac arrest

Abstract

Background: Congenital absence of the pericardium (CAP) is a rare cardiac abnormality. As pericardial defects are usually asymptomatic, most cases are diagnosed during surgery or on autopsy. The patient in this case was found to have CAP during thoracoscope.

Case: We present the unusual case of a 69-year-old patient with CAP who experienced sudden ventricular arrhythmia and developed ventricular fibrillation during left upper lobectomy. Surgical operations, the lateral decubitus position, and other external stimuli may be important risk factors for ventricular fibrillation. The patient regained sinus rhythm soon after intrathoracic cardiac compression and pharmacological treatment, including lidocaine spray (2%, 10 ml) administered to the heart surface. The surgery was then completed without any additional instances of ventricular arrhythmia.

Conclusion: Patients with CAP are more susceptible to cardiac-related adverse events during thoracotomy or thoracoscopy. Treatment of ventricular arrhythmias that occur during lung resection in patients with CAP should be emphasized.

Keywords: Amiodarone; Congenital absence of the pericardium; Intrathoracic cardiac compression; Lidocaine; Lobectomies; Ventricular fibrillation.

Congenital absence of the pericardium (CAP) is a rare cardiac abnormality with an estimated incidence ranging from 0.007% to 0.015% on autopsy and 0.044% in a surgical case series [1]. CAP can be classified into entire left- or right-side absence, partial left- or right-side absence, and complete absence of the pericardium, the latter of which is the rarest [2]. Because it is usually asymptomatic and imaging findings are atypical, clinicians may have difficulty diagnosing it [2]. Anatomically, the "bare heart" (absence of pericardium) is susceptible to external stimuli from the lung, pleura, and chest wall, especially when in the lateral decubitus position, during mechanical ventilation, and during thoracoscopic surgery. Many case reports [2,3] have shown a correlation between the absence of the pericardium and adverse cardiac events. A heart without the pericardium is particularly susceptible to external physical stimulation. Here, we present the case of a patient with CAP that experienced frequent premature ventricular contractions and sudden ventricular fibrillation during a left upper lobectomy.

Case report

A 69-year-old male patient (172 cm, 73.5 kg) with a medical history of hypertension and coronary artery disease was scheduled to undergo a left upper lobectomy for space-occupying lesions in the lung. A preoperative echocardiography demonstrated trivial tricuspid valve regurgitation and normal ejection fraction (62%). The chest computed tomography (CT) revealed a space-occupying lesion in the left upper lung lobe, chronic inflammation in the right upper lung lobe, bronchitis, and emphysema (Fig. 1A). The coronary CT showed right coronary dominance and a severe lesion in the proximal left anterior descending coronary artery with 70% luminal narrowing. Electrocardiography (ECG) results showed sinus bradycardia and an incomplete bundle branch block. However, the patient

did not report any significant discomfort and no significant laboratory abnormalities were noted. The patient was given an American Society of Anesthesiologists (ASA) grade of III. Perioperative anesthesia was maintained using a nerve block combined with general anesthesia.

After entering the operating room, vital signs including ECG, SpO2, heart rate, respiratory rate, and invasive blood pressure were continuously monitored. The ECG showed sinus rhythm. Anesthesia was induced with etomidate (0.2 mg/kg), rocuronium (0.6 mg/kg), and sufentanil (0.3 mg/kg). After tracheal surface anesthesia, a double-lumen tracheal tube was inserted using a visual laryngoscope. Correct tracheal positioning of the catheter was confirmed using a fiberoptic bronchoscope. The patient was then placed in the right lateral decubitus position. A thoracic paravertebral block (TPVB) was performed under ultrasound guidance at the T7 level. The patient was mechanically ventilated using volume-controlled ventilation with a breath volume of 6 ml/kg using a mixture of gases in proportion to 50% oxygen and 50% air. Anesthesia was maintained with sevoflurane (2–2.5%) and rocuronium was administered as needed. An arterial blood gas analysis was performed before surgery, with normal results. The circulatory parameters were also stable.

For the procedure, the seventh intercostal space of the axillary midline was used for the endoscope and the fourth intercostal space of the axillary front line was used for the operation. At this time, the ECG showed occasional premature ventricular beats. As the lens entered the pleural cavity, we discovered a complete absence of the pericardium in the heart (Fig. 1B). The frequency of premature ventricular contractions gradually increased as the surgery continued. The blood pressure became unstable and began to decrease. Ephedrine (6 mg) was administered to improve the blood pressure. The adhesions were separated, freeing the lung lobes. At this time, his heart rate suddenly increased

to 110 mmHg and blood pressure decreased to 90/56 mmHg. ECG revealed paroxysmal ventricular tachycardia. Lidocaine (1 mg/kg) was administered immediately. Continuous lidocaine and norepinephrine were then administered using the micropump, at 80 mg/h and 0.06 μg/kg/min, respectively. The blood pressure increased to 121/78 mmHg. As the patient was in the right lateral decubitus position, his heart experienced significant stimulation during the operation. To obtain a more satisfactory surgical field and complete the operation as soon as possible, the surgeon decided to perform an open thoracotomy. Premature ventricular contractions continued to occur frequently after opening the thoracic cavity. ECG showed polymorphic ventricular tachycardia, accompanied by profound hypotension, which was immediately followed by ventricular fibrillation. The patient's blood pressure decreased to 45/31 mmHg. Intrathoracic cardiac compression was immediately performed: a single hand was extended straight into the thoracic cavity and the heart was intermittently compressed. A continuous intravenous infusion of amiodarone (30 mg/h) and epinephrine (1 mg) was initiated immediately. Fortunately, the patient quickly regained sinus rhythm. Lidocaine spray (2%, 10 ml) was then administered onto the surface of the heart. No further incidences of ventricular arrhythmia occurred for the remainder of the surgery. The patient was extubated in the postanesthesia care unit and was maintained on continuous monitoring. . The patient was then returned saftely to the ward and discharged on postoperative day 7. No further cardiac-related adverse events were observed at the 1-month follow-up.

Discussion

The pericardium, which comprises two sacs [4], an outer fibrous membranous sac and an inner serous sac that covers the heart and great blood vessels, serves several physiological and protective functions.

The pericardium stabilizes the position of the heart inside the thorax by sternopericardial ligament, conferring cardioprotection from mechanical trauma, and acts as a lubricant. CAP is a rare cardiac malformation. Most patients with CAP are asymptomatic. However, some patients may exhibit atypical symptoms such as chest tightness, chest pain, and palpitations [3, 4]. The imaging findings are also atypical, making clinical diagnosis of CAP difficult. As with this case, CAP was not diagnosed preoperatively based on clinical manifestations, laboratory findings, or imaging features. However, we did reanalyze the preoperative examinations retrospectively with the help of radiologists. The chest CT had shown evidence of an absence of the pericardium (Fig. 1a); however, CAP remains difficult to diagnose without surgical outcomes. Additionally, the patient had an incomplete bundle branch block and coronary stenosis, both of which may be associated with CAP. Similarly, some studies have reported [1-5] that patients with CAP may also have sinus bradycardia, ventricular tachycardia, incomplete right bundle branch block, and myocardial infarction. Three cases have been reported [1] with patients who presented with sudden death due to cardiac strangulation across a partial left-sided pericardial defect. Additionally, chest radiography in patients with CAP often shows levoposition of the heart. A tendency toward cardiac levorotation may increase the risk of cardiac torsion in left-sided lobectomies or pneumonectomies [6]. Anesthesiologists should be vigilant about various adverse cardiac events associated with the lack of a pericardium.

Ventricular fibrillation associated with CAP has been reported previously [7]. In that case, the presumptive diagnosis was anterior infarction. However, angiography revealed normal coronary arteries. The authors [7] hypothesized that acute torsion of the great vessels secondary to cardiac hypermobility could have led to ventricular fibrillation. That patient received five shocks to restore

the pulse. In our case, the coronary CT revealed a severe lesion in the proximal left anterior descending coronary artery. This may have led to insufficient myocardial blood supply and cardiac dysfunction. Additionally, the “bare heart” was compressed due to the surgical operation, consisting of a thoracotomy or thoracoscopic approach. Indeed, any surgery can affect the cardiac conduction system. Surgical stimuli continued throughout the procedure, which corresponded to frequent ventricular arrhythmias. Ventricular arrhythmia may occur during left upper lobectomies in patients with a normal heart due to hypotension, hypovolemia, hypoxemia, and acidosis. However, the blood gas and circulatory parameters were normal before ventricular arrhythmia occurred. Thus, one could hypothesize that surgical stimuli may trigger ventricular arrhythmias.

Early in the treatment course, we focused only on intravenous drugs to correct the ventricular arrhythmias. Amiodarone is an effective antiarrhythmic medication frequently used in the treatment of ventricular and atrial arrhythmias that can block potassium channels, which increases the duration of the cardiac action potential. Lidocaine is a Class IB antiarrhythmic agent that exerts its action by blocking sodium channels. It can reduce arrhythmogenic transient depolarization and twitch tension by decreasing the inward sodium current [8]. However, even though amiodarone and lidocaine were administered in this case, ventricular arrhythmias continued to occur frequently before ventricular fibrillation. We concluded that ventricular arrhythmias would continue as long as the stimuli persisted. After ventricular fibrillation, intrathoracic cardiac compression and pharmacological treatments were administered.

Fortunately, the patient quickly regained sinus rhythm. Spraying lidocaine on the heart surface is not commonly performed;. Lidocaine is an antiarrhythmic drug and effective local anesthetic. Spraying

lidocaine on the larynx and bronchi is well known to inhibit the stress response during endotracheal intubation. We believed that spraying lidocaine on the heart surface could enhance the ability of the heart to respond to external stimuli. Lidocaine reduces the frequency of sodium channel opening and decreases the autorhythmicity of the heart by acting directly on the Purkinje cells and ventricular myocytes. It can also block inward potassium rectifier channels in cardiomyocytes and improve the threshold for ventricular fibrillation. The heart becomes less sensitive to external stimuli after lidocaine spraying. We believe that attenuation of stimulation may contribute to the termination of ventricular fibrillation, as no ventricular fibrillation occurred postoperatively. One could hypothesize that the absence of surgical stimuli, which may have initially triggered ventricular fibrillation, contributed to its termination. In addition, spraying lidocaine on the heart surface has a prompt onset of action. In this case, this administration method did not result in heart block or any other toxic side effects. However, no relevant studies have reported evidence regarding administering lidocaine spray on the surface of the heart, and thus the concentration, dosage, and mechanisms underlying this treatment need to be investigated.

Patients with CAP are more susceptible to ventricular arrhythmia and fibrillation during thoracotomy or thoracoscopy. Intraoperative ventricular arrhythmia in a patient with CAP is described in this case to increase awareness and provide information on its effective management.

References

[1] Lopez D, Asher CR. Congenital absence of the pericardium. Prog Cardiovasc Dis 2017; 59: 398-406.

[2] Shah, Ankit B, and Itzhak Kronzon. Congenital defects of the pericardium: a review. Eur Heart J Cardiovasc Imaging. 2015;16:821-7.

[3] Xu B, Betancor J, Asher C, Rosario A, Klein A. Congenital absence of the pericardium: a systematic approach to diagnosis and management. Cardiology 2017; 136: 270-8.

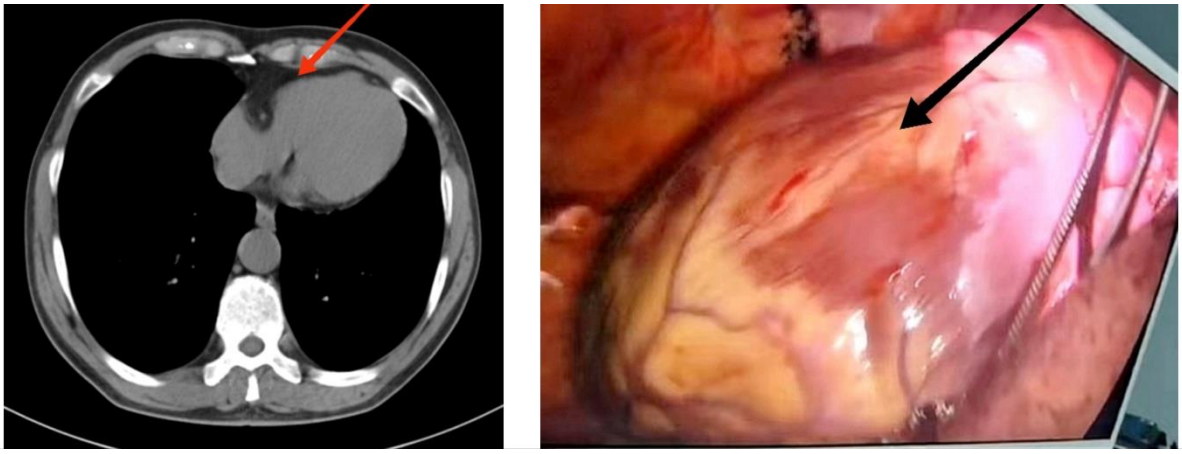
[4] Rayamajhi S, Shrestha R, Shahi K, Adhikari B, Mahaseth A. Congenital pericardial agenesis presenting as non-specific chest pain: a case report. Radiol Case Rep 2022; 17: 4462-5.

[5] Brunet-Garcia L, Carretero Bellon JM, Pérez-Casares A, Prada Martínez FH, Sánchez-de-Toledo J. Partial absence of the pericardium: only an incidental finding. An Pediatr (Engl Ed) 2022; 96: 540-2.

[6] Nicholas J, Barber E, Grondin S, Haber J. Congenital absence of the pericardium encountered during thoracoscopy. Can J Anaesth 2022; 69: 898-9.

[7] Steinberg C, Pelletier MJ, Perron J, Kumar A, Champagne J. Sudden cardiac arrest due to subtotal absence of left-sided pericardium--case report and review of the literature. Congenit Heart Dis 2013; 8: E92-8.

[8] Güler S, Könemann H, Wolfes J, Güner F, Ellermann C, Rath B, et al. Lidocaine as an anti-arrhythmic drug: are there any indications left? Clin Transl Sci 2023. Advance Access published on Oct 2, 2023. doi:10.1111/cts.13650.



A B

Fig. 1. The evidence of CAP in CT and thoracoscope

A) Chest CT showing absence of the pericardium (red arrow).

B) Image during intraoperative observation showing a “bare heart” without a pericardium (black arrow).