Case report

Spontaneous tension hydropneumopericardium complicating serofibrinous pericarditis

Ming Wu a, Xueming He a, Guangzhao Yang b,*

a Department of Thoracic and Cardiovascular Surgery, 2nd Affiliated Hospital, School of Medicine, Zhejiang University, Hangzhou 310009, PR China
b Department of Radiology, 2nd Affiliated Hospital, School of Medicine, Zhejiang University, Hangzhou 310009, PR China

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Abstract

A hydropneumopericardium is a rare but critical condition, which is mostly ascribed to primary infiltrative lesions from adjacent organs, pericardial infections, or trauma. Although there have been reports about pyopneumopericardium, no case of spontaneous non-purulent hydropneumopericardium has been documented in literature. We report a case of a previously healthy man of spontaneously occurred tension hydropneumopericardium complicating serofibrinous pericarditis. A surgical exploration followed by partial pericardiectomy was performed to stop cardiac tamponade and possible later constriction. Remarkably, no definite pathogenesis was identified despite surgical and laboratory investigations. The spontaneity and management of hydropneumopericardium are discussed.

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1. Introduction

Most cases of hydropneumopericardium are secondary to diverse primary lesions or trauma and are exceptionally lethal. As an extremely rare entity, we report a case of spontaneous tension hydropneumopericardium complicated with non-purulent pericarditis that obtained a favorable result after a partial pericardiectomy.

2. Case description

A previously healthy 49-year-old male was admitted with worsening precordial pain, dyspnea, and palpitation on exertion for 7 days. He was a hardworking peasant and had a smoking history of 25 years. He denied history of trauma or abdominal discomfort. On admission, his temperature, respiratory rate, pulse, and blood pressure were 36.7°C, 20 beats/min, 74 beats/min (without pulsus paradoxus), and 101/73 mmHg, respectively. Cardiac auscultation revealed distant heart sounds without pericardial rub. No pedal edema, subcutaneous emphysema, or pneumothorax was identified except mild distention of the bilateral jugular vein. All routine variables were within the normal range except increased serum C reactive protein (19.5 mg/L) and hypoalbuminemia (3.11 g/dL). The results of serum anti-tuberculous antibody and HIV antibody tests were negative. The ECG revealed reduced voltages in all leads and diffuse ST-segment abnormalities. Chest roentgenogram, chest CT, and cardiac MRI demonstrated hydropneumopericardium and mild pericardial thickening (Fig. 1A–C). Transesophageal echocardiography revealed intra-pericardial fluid and normal heart ejection function. Bronchoscopy and gastrografin swallow excluded any involvement of trachea or upper gastrointestinal tract. A further gastrofiberscopy was performed without finding any peptic ulcer or healing lesion. As the hemodynamic conditions were stable and symptoms improved with oxygen inhaling, the patient was intensively monitored without emergent pericardiocentesis. On day 3, he complained a deteriorating dyspnea with pulse rate increment (110 bpm) and blood pressure reduction (90/65 mmHg). A cardiac tamponade was confirmed as the central venous pressure rose to 20 cmH2O measured through an internal jugular vein catheterization. A median sternotomy was immediately performed and exposed an enlarged and remarkably stressed pericardial sac. When the thickened pericardium was cut, an audible gush of air was released, along with about 200 ml serofibrinous exudates. The epicardium was covered by fibrinoid adhesions (Fig. 2A). Partial pericardiectomy and intra-pericardial exploration were performed without discovering any communication between pericardium and contiguous organs including lung, diagram, and esophagus. The postoperative period was uneventful and the patient was discharged 10 days after
operation. He was kept disease-free during a 12-month follow-up (Fig. 1D).

The epicardial fibrinoid adhesions, intra-pericardial fluid, and blood were cultured with negative findings for microorganisms or parasite. The histological analysis demonstrated a serofibrinous pericarditis (Fig. 2B). Diagnostic examinations including ANA, dsDNA, ENA, SM, RNP, anti-SS-A, anti-SS-B, anti-SCL-70, anti-JO-1, anti-mitochondria antibody, CEA, CA199, CA125, CA153, PSA, NSE, TPA, IgA, IgM, and IgG were done without revealing positive results.

Fig. 1. Chest roentgenogram demonstrated free intra-pericardial air and fluid as well as pericardial thickening and bilateral pleural effusion (A). Neither pericardial defect nor fistula connection between neighboring organs was identified by chest CT (B) or MRI (C). Chest radiography revealed a normalized cardiac size and silhouette 12 months after the operation (D).

Fig. 2. Operative schema showed the thickened pericardium and fibrinous depositions (arrow) on epicardium. (A) Histological section (hematoxylin and eosin stain, original ×100) diagnosed a serofibrinous pericarditis by demonstrating fibrous tissue proliferation, angiogenesis and little lymphocyte infiltration. Fibrinous depositions were proved fibrinous lamina accompanied by little lymphocyte infiltration (B).
3. Discussion

In pneumopericardium, air was known to access the pericardial sac through: (1) concomitant posttraumatic pneumothorax and pericardial rupture; (2) pre-existed pleural-pericardial connections, such as congenital pericardial defect; (3) notably, leaked air may travel along peribronchial and perivascular sheaths to the lung hilum and enter the pericardial sac through the discontinuation at the reflection of parietal onto visceral pleura near the ostia of the pulmonary veins [1]. With additional presence of intra-pericardial free fluid, hydropneumopericardium occurs extremely rare and was mostly reported due to intra-pericardial gas-forming bacterial infection and primary infiltrative diseases of adjacent air-containing organs. And as evidence, a variety of secondary fistulas have been reported including bronchopericardial, esophagopericardial, and gastropericardial fistulas [2–4]. Interestingly, etiology of this case is far from categorical. There was no trauma, peptic ulcer, fistulous tract, or pulmonary infection. A gastrofiberscopy excluded any evidence about ‘spontaneous quick healing’ of penetrating gastric lesions [5]. Furthermore, serum examinations about infectious, rheumatic, neoplastic, and autoimmune disease were done without positive findings. Therefore, we have to define it as an idiopathic case.

In an attempt to explain the origin of gas, fluid, and pericarditis, we once assumed that the pathogenesis might start from an unapparent, self-limited pericarditis caused by gas-forming bacteria. However, this additional presumption was supported by neither negative clinical findings nor the fact that intra-pericardial bacteria infection is life-threatening and barely asymptomatic or self-limited [6]. Since pathologists defined the pericardial disease as a non-purulent, serofibrinous pericarditis, we hypothesize that the air and fluid emerge first: alveoli impairment possibly occurred in this hardworking peasants and air may appear through a congenital direct small tracheobronchial–pericardial communication that enabled pneumopericardium [1]. Subsequently, intra-pericardial exudates may initiate pericarditis. With the developing of pericarditis and intra-pericardial fibrinous deposition, the gas-inlet tract was gradually blocked and tension physiology finally occurred as a result of effusion increment. To our knowledge, this represents the first reported instance of spontaneous hydropneumopericardium complicated with tension and non-purulent pericarditis [7].

Tension was present in about 37.7% patients with pneumopericardium and is most often secondary to infants receiving mechanical ventilatory support (74%), pyopneumopericardium (14%), and posttraumatic pneumopericardium (11%) [6,8]. Once tension or tamponade is recognized, immediate pericardiocentesis is suggested. In this case, a median sternotomy was performed because we supposed that an exploration was needed for the reason why the pericardial thickened and where the air and liquid originated. The patient received partial pericardectomy and enjoyed a favorable result, which support our belief that pericardectomy is needed to avoid recurrence or later pericardial constriction in hydropneumopericardium [9].

This case suggests that a possibility of idiopathic origination should be kept in mind when encountering a hydropneumopericardium, which usually occurs secondary to trauma or severe primary diseases.

References