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Massive pulmonary air embolism during the implantation of pacemaker, case reports and literature analysis

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Abstract. – OBJECTIVES: Pacemaker implantation has developed into a mature technology, meanwhile, implantable cardioverter-defibrillators (ICD) and cardiac resynchronization therapy (CRT), as extended pace making technology, are both carried out in rising frequency. Massive pulmonary air embolism is a rare but fatal complication accompanying with such pace making process. The objective of this study was to investigate the epidemiology, pathophysiological mechanism, occurrence and treatment for this kind of complication.

PATIENTS AND METHODS: Two cases of complicated massive pulmonary gas embolism were presented: one in CRT and the other in pacemaker implantation, both of which were captured rapidly and treated successfully by inhalation of high flow oxygen, closure of gas inflow tract, position change, and vasoactive drugs. Moreover, published literatures about air embolism in the process of pacemaker implantation or CRT/ICD were summarized and analyzed.

RESULTS: Complicated massive pulmonary air embolisms could be successfully resolved with satisfied short-term prognosis. Literature analysis showed that massive pulmonary air embolism is very rare in the course of pacemaker implantation, and coughing or deep breathing, advanced age, preoperative sedation, sheath with large cavity, improperly operating the hemostasis valve and diminished compliance of pulmonary circulation might be risk factors for air embolism.

CONCLUSIONS: Massive pulmonary air embolism during pace making which is very rare in the course of pacemaker implantation is one kind of life-threatening complication. Rapid judgment and timely treatment can avoid a catastrophic event, which could prevent adverse impact on the short-term prognosis, while further observation is required to explore the long-term prognosis.

Key Words:
Pacemaker, Pulmonary air embolism, Literature analysis.

Introduction

Pulmonary air embolism is a kind of complication related to iatrogenic reason or patients' own cause, which means gas enters into right ventricle through venous entrance, and then into pulmonary artery, resulting in varying degrees of pathophysiological damages. In the implantation of pacemaker, such as implantable cardioverter-defibrillators (ICD) or cardiac resynchronization therapy (CRT), there may be several complications, including bleeding, infection, pneumothorax, and cardiac tamponade[1], but the massive pulmonary air embolism is rarely encountered. After entering into the circulation, the extent of air embolism's damage depends on the moving speed and the volume of gas[2]. Studies on animals have shown that a 1.5 cm^3 air embolism per-second per-kilogram of body weight can kill a dog[3]. Ericsson et al[4] reviewed 93 cases of venous air embolism, in which the unhandled patients' mortality rate was is 93%, while the handled of is 33%. Pathophysiological damages of massive pulmonary air embolism are mainly as follows, plugging right ventricular outflow tract, blocking right ventricular ejection, acute dilatation of right ventricular or even heart failure. What's more, because of increased right atrial pressure, the air embolism is easy to cross the foramen ovale or the atrial septal defect (ASD), then into left atrium, left ventricle, and eventually arriving at the brain, resulting in disastrous consequences such as acute cerebral embolism.

Particularly, when the gas, which is not pure oxygen but atmosphere containing less oxygen, enters into the pulmonary circulation, it will decline blood flow and blood oxygen content, leading to acute disorder of pulmonary ventilation/perfusion ratio, anatomy streaming and functional dead space, which will in turn worsen the circulatory state. At the same time, acute expansion of right ventricular will squeeze ventricular sep-

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tal to the left, affecting left ventricular diastole. Finally, failed pulmonary circulation will significantly reduce the blood supply to left ventricular.

Here we presented two cases of large pulmonary air embolism in order to explore the epidemiology, pathophysiological mechanisms, the occurrence and treatment of this complication.

Patients and Methods

Basic Information of Cases

Case 1:

A male patient, with a hypertension history for 10 years, was installed a temporary pacemaker in local hospital, then transferred to our hospital for further treatment. Elecrocardiotgraphy (ECG) showed a third degree atrioventricular block and complete left bundle branch block, with a broadened quantum resonance spectrometer (QRS) complex 210 ms. His left-ventricular end-diastolic diameter (LVEDD) was 81.5 mm, with an ejection fraction (EF) of 36%. Coronary angiography report was negative. The patient was diagnosed as dilated cardiomyopathy, third degree atrioventricular block and complete left bundle branch block. According to New York Heart Association (NYHA) functional classification, the patient is in the third grade with the indications for CRT. In CRT process, the left ventricular electrode was successfully implanted into lateral vein, at that moment, the patient felt very hot, and took a deep breath, then followed by continuous discomfort, such as sudden chest tightness, difficulty in breathing, pale, sweating, blood oxygen saturation fell off from 98% to 78%, blood pressure suddenly dropped down to 70/40 mmHg, and heart rate reduced to 46 bpm. From the cine, a massive air embolism was seen lingering around the pulmonary artery.

Case 2:

An 81 year-old male, with a history of hypertension, diabetes mellitus, and surgery for esophageal cancer, complained of recurrent syncope for three years. Holter monitoring showed a significant sinus bradycardia and long intermittent of 5.2 seconds. ECG showed a LVEDD of 69 mm, with an EF of 58%. This patient was diagnosed as sick sinus syndrome, and received dual-chamber pacemaker implantation. We selected the pulse generator of Medtronic Company (Santa Rosa, CA, USA), and a Medtronic 5076-58 cm electrode in right ventricular, a Medtronic 5076–52 cm in right atrium. When the venous sheath (inner diameter, 8 F) was placed through subclavian vein approach, no blood spilling was found from the sheath, which suggested very low pressure in central venous. With the aid of sheath and guide-wire, ventricular electrode successfully reached the apical of heart. When right atrial electrode was implanted, the patient coughed, which was followed by a deep breath, immediately a massive air embolism was seen rising from the right ventricular outflow, across the pulmonary valve, gathered and extended to the pulmonary trunk. The patient's blood pressure and oxygen saturation was were normal, and heart rate was 110 bpm. He did not feel uncomfortable.

Literature Review and Analysis

We searched the Chinese domestic literature from 1979 to 2011 using the keywords "pacemaker" and "air embolism" or "complication", and 4 cases of massive pulmonary air embolism were retrieved. In the database of MEDLINE, PubMed, Embase, OVID and so onothers, we found 6 articles with 7 cases of pulmonary air embolism. Through the Web, we also tracked the pacemaker-related monographs and meeting reports, one case was caught. Coupled with our own case, there are all 14 cases of massive pulmonary air embolism in the process of pacemaker implantation or ICD/CRT, whose demographic data, surgical data, processing results and outcome were recorded.

Results

The Treatment and Results of the Two Patients in Our Center

Case 1:

The patient was inhaled with high flow oxygen via face mask, given rapid intravenous infusion to increase blood volume, removed the pillow and taken the Trendelenburg' s position to avoid the air embolism entering the pulmonary artery. Five minutes later, his symptoms mitigated, meantime, the air embolism disappeared from pulmonary valve with each electrode still in normal location (Figures 1 and 2).

Case 2:

We removed the vein sheath, closed the entry path of the gas, and then gave the patient high flow oxygen via face mask, placed him in Trendelenburg's position. From the X-ray, we could

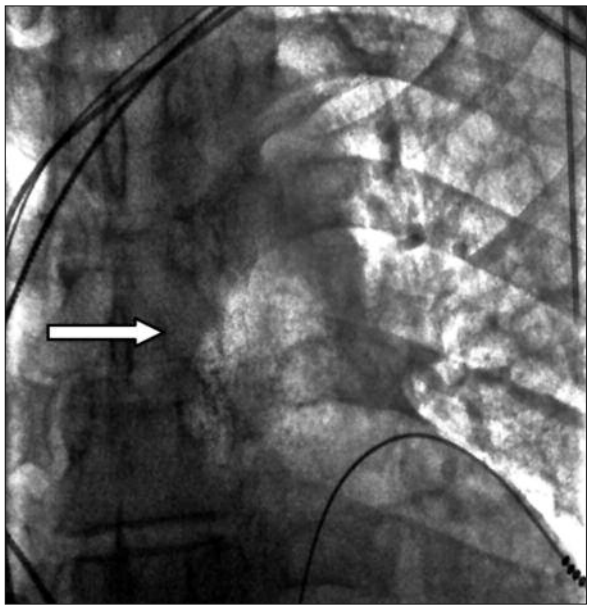

Figure 1. Air embolism blocking pulmonary valve.

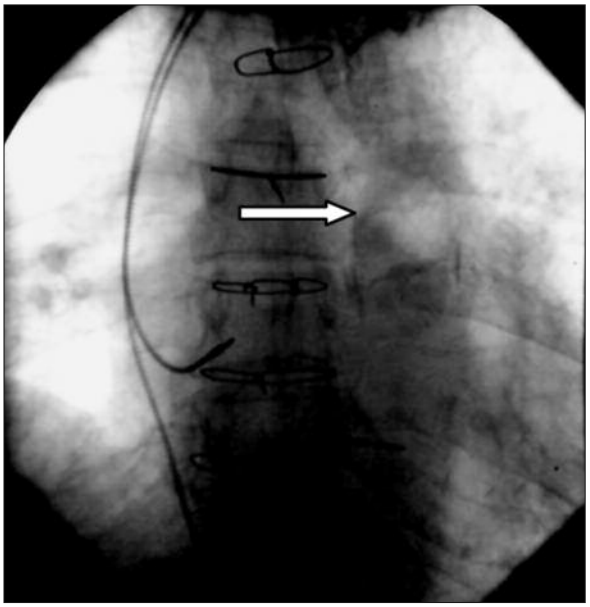

Figure 3. Air embolism blocking pulmonary artery.

see the embolism gradually moving upward, becoming smaller and smaller until dissipation. The patient did not present with any hemodynamic changes, and was free of the administration of vasoactive drugs (Figures 3 and 4).

Follow-up:

After six months follow-up, the two patients were both in good condition, and their cardiac function was improved. The related indexes of their heart function are presented in following table (Table I).

Summary and Analysis of all the Cases About Pulmonary Air Embolism

Coupled with the cases in our center, we found a total of 14 cases complicating pulmonary air embolism[5-15], 9 males (64.3%) and 4

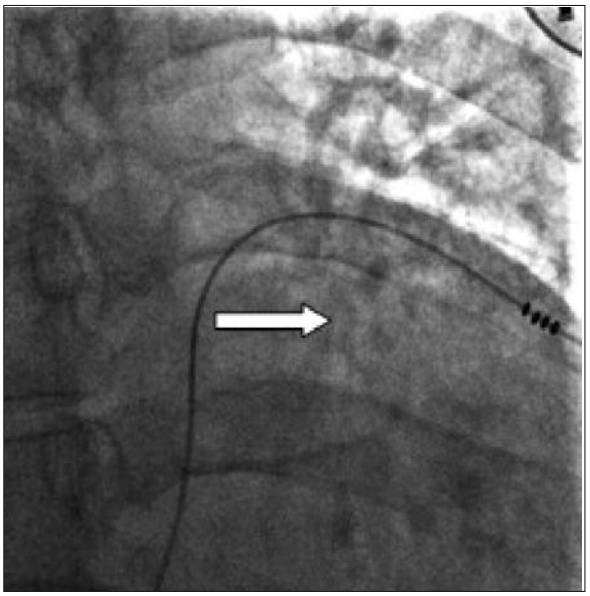

Figure 2. Air embolism invisible.

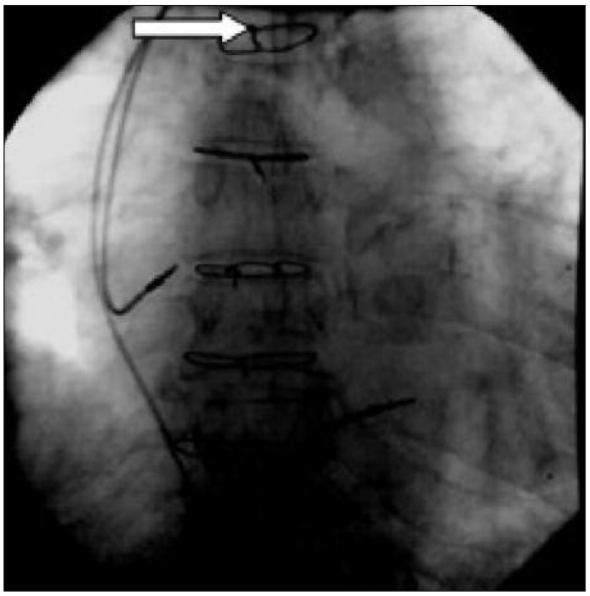

Figure 4. Air embolism gradually disappeared.

Table I. Preoperative and postoperative heart function indexes.

	Pre-operation	Post-operation
Case 1		
EF	36%	44%
LVEDD	81.5 mm	78 mm
QRS duration	210 ms	145 ms
NYHA grade	IV	II
Case 2		
EF	58%	60%
LVEDD	69 mm	69 mm
QRS duration	163 ms	168 ms
NYHA grade	I	I

EF: ejection fraction; LVEDD: left-ventricular end-diastolic diameter; QRS: quantum resonance spectrometer; NYHA: New York Heart Association.

females (28.6%), one case without report of gender and age. The average age of the 13 patients was 69.91 ± 9.87 years old. In 10 cases (71.4%), the sheath with large lumen diameter was used, the finest was 8F, and the largest was 11F. Among all the reported predisposing factors for air embolism, there were "cough and deep breathing" in nine cases (54.3%)[5-8,10,13,14], "relatively large gap between electrode and sheath" in one case (7.1%)[12], "improper operation of the hemostatic valve" in one case (7.1%)[9], and "preoperative sedative drugs" in three cases (21.4%)[6,8,13].

After occurrence of massive air embolism, 2 (14.3%) patients presented with severe cardiac arrest[5,6], six patients with hypotension (42.9%)[5-8,12], four patients with severe bradycardia (28.6%)[5,6], and five cases were associated with dropped oxygen saturation (36.7%)[5-7].

When it comes to the treatment, there were 13 cases of patients were successfully rescued by non-interventional therapy, such as postural change, oxygen inhalation, vasoactive drugs and immediate cardiopulmonary resuscitation (92.9%), whereas interventional suction of air embolism through catheter (7.1%) was required in one patient[5]. All patients were successfully rescued (100%) with a satisfied result during hospitalization, while the postoperative long-term follow-up data were absent. We summarized the demographic data of the 14 patients, the occurrence and development of air embolism, intra-operative treatment for air embolism, as well as the results and prognosis record (Table II).

Table II. Summary of the clinical reports about massive pulmonary air embolism*.

Number	1	2	3[5]	4[6]	5[7]	6[8]	7[9]	8[10]	9[11]	10[12]	11[13]	12[14]	13[14]	14[15]
Sex	M	M	F	M	M	F	M	M	F	M	M	F	?	M
Age(years)	60	60	65	69	58	73	74	61	80	68	83	88	?	75
Disease	DCM	SSS	SSS	AVB	ICM	SSS	DCM	SSS	SSS	DCM	AVB	SSS	?	sss
Operation	CRT	PM	PM	PM	ICD	PM	CRT	PM	PM	CRT	PM	PM	PM	PM
Sheath	8F	8F	9F	9F	10F	9F	9F	9F	8F	9F	None	None	None	11F
Chest tightness	Yes	No	Yes	?	?	?	Yes	No	No	Yes	No	No	?	no
Hypotension	Yes	No	Yes	Yes	Yes	Yes	?	No	No	Yes	No	No	?	No
SPO2%	78	95	Lower	0	Lower	?	?	?	Normal	90	Normal	Normal	?	Normal
Cardiac arrest	No	No	Yes	Yes	No	No	No	No	No	No	No	No	No	No
Bradycardia	Yes	No	Yes	Yes	No	Yes	No	No	No	No	No	No	?	No

*DCM: dilated cardiomyopathy; SSS: sick sinus syndrome; AVB: atrioventricular block; ICD: implantable cardioverter defibrillator; PM: pacemaker implantation; ICM: ischemic cardiomyopathy; ? : The original article does not provide related information. Number 1, 2 are from our center.

Incidence of Massive Air Embolism During Pacemaking in History

In the last decade, from 2002 to the end of 2011, our Center implanted a total of 1450 cases of pacemaker (including CRT/ ICD), if this number was regarded as denominator, the prevalence of pulmonary massive air embolism during the implantation in our center is was 0.14%. China's domestic earliest literature about massive air embolism was in 1993, since then, six cases had been reported, but there was no literature summing up the incidence of massive air embolism. Since the pulmonary air embolism was firstly discovered in 1967 worldwide, a total of seven cases were confirmed, however, there was still no statistical data about that, only some studies about small gas embolism in central venous catheter inserting was reported, whose prevalence was 5-19%[16-18].

Discussions

Massive air embolism is a rare complication in pacemaker implantation, and till now, there was is no accurate definition for it till now. Since Rotem et al[13] first reported pulmonary air embolism in 1967, however, all the described "massive pulmonary air embolism" confirmed that the embolism's size was is closed to the inner diameter of pulmonary trunk, which appearsed as a massive translucent area from fluoroscopy.

As for risk factors of air embolism previous researches had have told us as follows. First of all: coughing and deep breathing. According to all the records in past literature, "coughing" or "deep breathing" might be the most direct factor, which could quickly lower the pressure of central venous, and then the rapid decompression would promptly drive the gas into central venous. In 9 retrieved articles above, operators observed with certainty that the massive air embolism was is triggered by coughing and deep breathing. Secondly: advanced age. In the above literatures, patients suffering from air embolism were are all older than 60 (the average age was is 69.55 ± 9.79 years old). Due to elderly patients' poor tolerance and weak activity, they were are more prone to coughing, and because they liked high-pillow lying position, which kept keeps the pulmonary artery at a high level, once the air entered enters into circulation, it was is opt to linger around pulmonary artery[19]. The third: sheath with large cavity. According to the principle of Poiseuille equation, when flow fixedfixes, the pressure gradient is inversely proportional to the four power of the sheath's radius. That is to say, when the sheath diameter increases, pressure within the sheath will decline significantly, which directly facilitate the gas invasion of the gas. The fourth: sedative drugs were used to calm patients before surgery. One of the drugs' shortcomings was is that, after sedation, the patient would have involuntary breathing during operation, which would also increase the risk of air embolism. The fifth: compliance of pulmonary circulation and right heart system. In Ninio-DANIEL's report[7], he believed diminished compliance of pulmonary circulation would make patient prone to encounter air embolism. The last, CRT or ICD has its particularity in operation, such as, long operative time, repeatedly flushing the hemostasis valve and tee, earlier removing the hemostasis valve[9,12], as well as inappropriate operation of the bridge flap, which would also increase the risk of embolism.

Prevention and treatment for massive air embolism isare an interesting topics, timely detecting and removing risk factors is are of most importance. If risk factors are not eliminated, gas will continue to enter into the circulation, which will bring fatal damage to the patient. Besides above risk factors, we should also pay more attention to the central venous pressure. Our Center has a novel and simple way to estimate the central venous pressure (as shown in Figures 5 and 6). When removing the inner core of sheath,

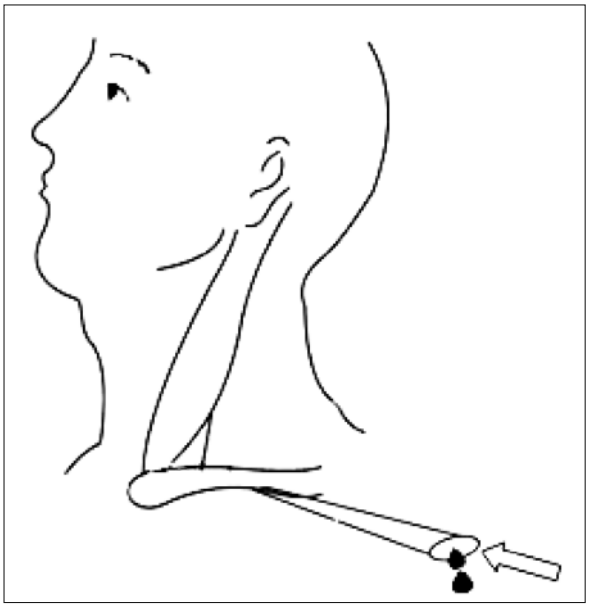

Figure 5. Blood spilling, higher pressure.

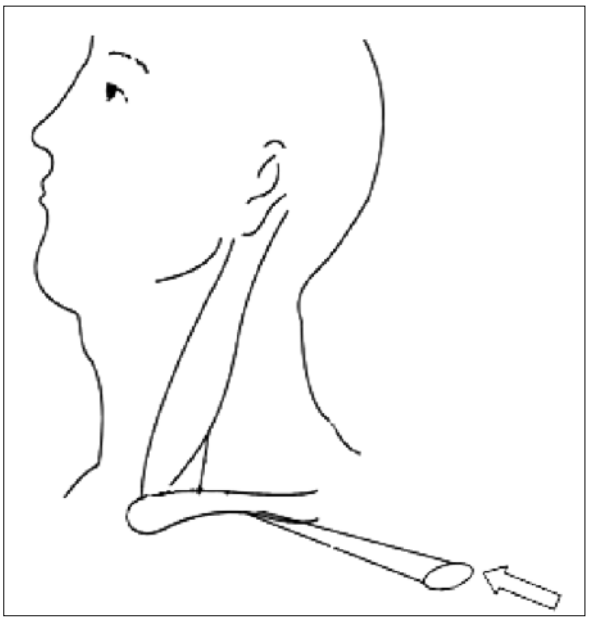

Figure 6. No blood spilling, lower pressure.

if there is no blood spilling, it can be confirmed that the central venous pressure is lower than atmospheric pressure, which suggests the high risk of air embolism. In this case, the sheath entrance should be blocked by operator's thumb as quick as possible, once pacing electrode reaches the superior vena cava, we should tear the sheath to prevent the entry of gas. Our another experience is that, when a thin electrode is in a thick sheath resulting in an apparent lacuna around the electrode, which also predicts a high-risk of air embolism, thereby venous sheath should be torn as quick as possible.

We found that, timely fluoroscopy and cine was a convenient way for capturing air embolism, which played a crucial role in first bedside aid. When air embolism occurred, the first thing we should do is to block entrance of gas, and then give the patient high-flow oxygen supply to increase concentration of oxygen in the blood. What's more, position change, such as left lateral position and Trendelenburg posture, could keep right ventricular apex at a high position, and delay the gas gathering into pulmonary artery.

Once respiratory and cardiac arrest happensed, rapid cardiopulmonary resuscitation was is commonly used, not only because it is a basic life supporting method but also because external cardiac massage could increase intrathoracic pressure, helping drive away the gas. When the above methods does not work, and the gas still gathers and continues to clog pulmonary artery, we could suction gas should be performed through a catheter as Ostovan and Asiani[5] had told usdescribed, which was is an effective intervention treatment. In addition, hyperbaric oxygen is also a certain positive way for the rehabilitation later by treating tiny infarction and extenuating hypoxia.

The final topic is prognosis of massive pulmonary gas embolism. Most researchers believed that the pathophysiological effect of the embolism is transient, and the damage is reversible if the patients receive proper treatment timely. Oppenheimer et al found report that the vast majority of air embolism disappeared disappears from the X-ray in 15 minutes[20]. Pathology study also confirmed that, after intravenous injection of air embolism, venous or and pulmonary artery endothelia both had have a only short-term hyperplasia, which could be cured[21]. Till now, however, there were are no reports about long-term prognosis of pulmonary air embolism till now.

Conflict of Interest

The Authors declare that they have no conflict of interests.

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