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Case Report
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Air Embolism in a Patient during Hemodialysis

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Abstract

Air embolism is a rare complication of the use of Arteriovenous Fistula (AVF) as vascular access for hemodialysis. We report a patient with AVF who suffered from cough, chest pain, dyspnea and hemodynamic change, because of air leak *via* dialysis tubing that resulted in pulmonary edema. This case illustrates that micro-air-bubbles may leak into the circulation; even the air detector is in operation. The pathophysiologic sequence of pulmonary air-embolism has been discussed.

Keywords: Air embolism; Microbubble; Hemodialysis

Introduction

Arteriovenous Fistula (AVF) is the most frequent used for vascular access for hemodialysis. The blood tubing set is absolutely necessary for hemodialysis. During hemodialysis, the air detector on the dialysis machine protects the patient from inadvertent air embolization. However, use of the tubing outside of hemodialysis may pose a potential source of air entry. We report a case of paradoxical air embolism *via* dialysis tubing from the use of an AVF to draw blood.

Case Presentation

A 63-year-old woman had end-stage renal disease secondary to polycystic kidney disease and was on chronic hemodialysis for 7 years, using an AVF of the cephalic vein and radial artery at the left arm.

The patient had been in good condition on hemodialysis. On December 21st, 2004 the vital signs before hemodialysis were blood pressure of 140/68 mmHg, a pulse rate of 74/min and body temperature of 36.8°C. The attending technician just had completed the total external circulation and starting to dialysis then she left the bed side. About 20 min later, the patient developed nausea, dyspnea, severe cough and chest tightness as initial presentation. Another staff noted the discomfort of the patient, in the meanwhile, many air bubbles were noted in the tubing and the chamber was empty. Only half of the dialyzer was filled with blood. The blood lines were clamped and the blood pump was stopped immediately. The patient was placed in Trendelenburg position on the left side. Blood was withdrawn from the access needle until all the air was removed and connected intravenous line to a cleared needle. Besides, cardiac monitor was placed and oxygen was given by a face mask at high flow rate.

The initial measurement revealed a blood pressure of 163/59 mmHg and a pulse rate of 108/min. Chest examination demonstrated symmetric expansion without crackles or wheezing. The heart sounds were normal without murmur. There was no neurologic impairment including consciousness, speech, vision or muscle power. Immediately, she was transported to Medical Intensive Care Unit (MICU) for further management.

After admission into the ICU, the respiration rate was 25 per minute. The pulse oximeter showed mild oxygen desaturation (a SpO_2 of 89%, using nasal prong cannular with O_2 flow 5 L/min during transportation). Then oxygen with a flow of 15-liter (L)/min *via* non-rebreathing mask with reservoir bag was given and 90 min later Arterial Blood Gas (ABG) analysis showed pH 7.455, partial pressure of O_2 (PO_2) 87.2 mmHg, partial pressure of CO_2 (PCO_2) 41.5 mmHg, HCO_3- 27.9 meq/L, Base Excess (BE) 3.5 meq/L and O_2 saturation 96.9% (calculated $AaDO_2$ 59 mmHg). Forty-three minutes after the event, the electrocardiogram showed normal sinus rhythm with a counterclockwise rotation QRS in precordial leads. Portable Doppler echocardiography showed negative for intracavitary air bubbles or vegetation on the valves. This study revealed a hypertensive

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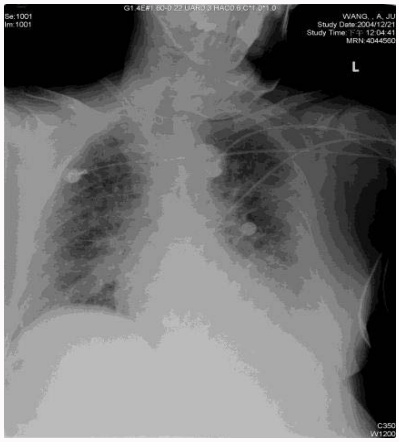
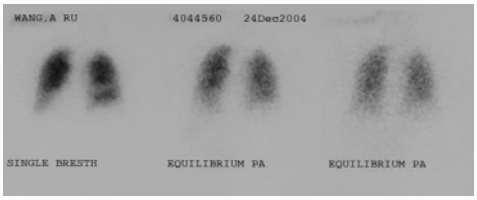
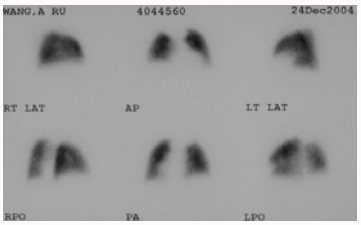


Figure 1: Chest roentgenogram taken 3.5 h after the event revealed LLL congestion, consolidation over bilateral lung field and left side pleural effusion.



(A) Ventilation



(B) Perfusion

Figure 2: Ventilation-perfusion scan with Tc-99 was performed on the 4th day. (76 h 30 min after event) and showed low probability of pulmonary embolism but ventilation lesions in the bilateral lower lung field, probably lung parenchymal disease.

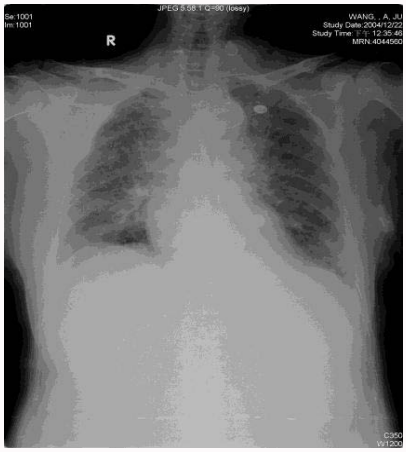


Figure 3: Chest roentgenogram abnormalities of consolidation resolved within one day.

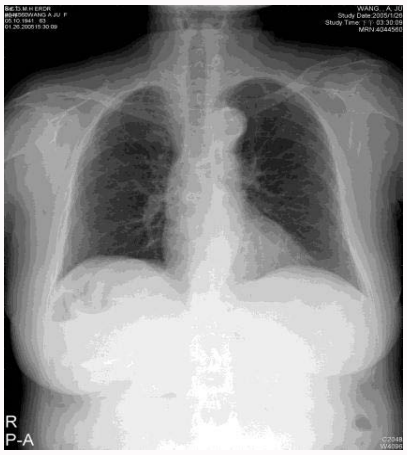


Figure 4: Chest roentgenogram abnormalities of consolidation has been cleared up to complete normal in 35 days follow up film.

cardiovascular disease heart with mitral annular calcification and mild mitral regurgitation, trivial tricuspid regurgitation and aortic regurgitation, decreased biventricular relaxation but adequate left ventricular performance. Chest roentgenogram taken 3.5 h after the event revealed LLL congestion, consolidation over bilateral lung field and left side pleural effusion (Figure 1). Two and half hours later the ABG showed pH 7.455, PCO_2 37.9 mmHg, PO_2 186.5 mmHg, HCO_3 25.5 meq/L, BE 1.6 meq/L and O_2 saturation 99.3% (alveolar-arterial oxygen partial pressure gradient (PA-aO_2) 494.5 mmHg).

Therefore, supportive oxygen supply with venturi mask (FiO_2 50% flow 15 L/min) was administrated till the next day. The ABG follow-up showed pH 7.493, PaO2 63.7 mmHg, $PaCO_2$ 41.8 mmHg, HCO_3- 31.4 meq/L and O_2 saturation 93.9%. The symptoms relieved hours later and only dyspnea on exertion was noted. Continuous pulse oximeter monitor showed SpO_2 in the range of 100% ~ 96%. On the second day, the patient O_2 supplement was changed to nasal cannular with a flow of 2 L/min ~ 4 L/min and 2 h later the ABG showed pH 7.485, PaO_2 67 mmHg, $PaCO_2$ 36.3 mmHg, HCO_3- 29.2 meq/L and O_2 saturation 94.6% (PA-aO_2 137.9 mmHg). Oxygen supplement through nasal cannular was tapered to 2 then to 1 L/min till the patient`s discharge. The dyspnea on exertion persisted for about 48 h after onset of the episode. Ventilation-perfusion scan with Tc-99 was performed on the 4th day (76 h and 30 min after event) and showed low probability of pulmonary embolism but ventilation lesions in the bilateral lower lung field, probably lung parenchymal disease (Figure 2).

After oxygen therapy, hemodialysis and supportive treatment for 5 days the patient recovered from the distress and discharged to home directly from MICU. Following up visits revealed that the patient was fine in daily activity without any symptoms and vital signs had kept stable in the later hemodialysis procedures. The chest roentgenogram abnormalities of consolidation resolved rapidly within one day (Figure 3) and has been cleared up to complete normal in 35 days follow up film (Figure 4).

Discussion

Air embolism is a rare but potentially life-threatening event that occurs as a consequence of the entry of air into the vasculature. A venous or pulmonary air embolism occurs when air enters the systemic venous circulation and travels to the right ventricle and/or pulmonary circulation [1-3]. Air in the pulmonary arteries can interfere gas exchange, induce changes in pulmonary pressure,

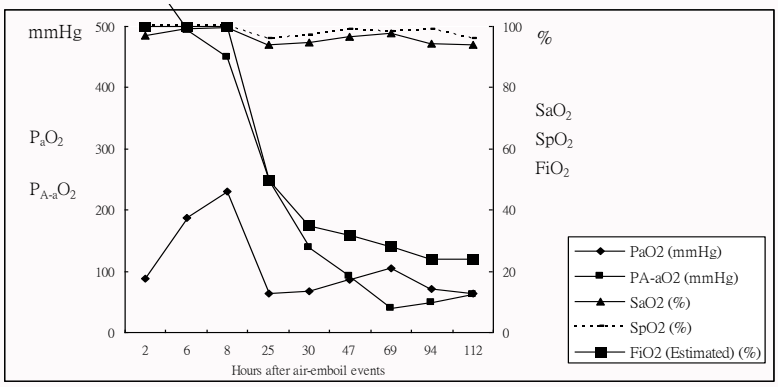


Figure 5: The progressive changes of oxygenation data. The PA-aO_2 showed rapid decreasing while the SaO_2 and SpO_2 remained stable. The PaO_2 has been kept high for fear of tissue hypoxia in the first day.

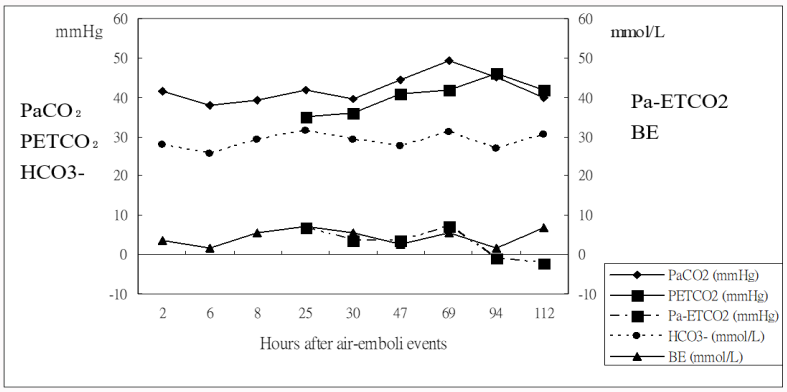


Figure 6: The progressive change of acid-base and CO_2. The mostly ABG showed neutral in pH but metabolic alkalosis has been noticed in occasion. Pa-$ETCO_2$ shows slightly fluctuated.

cardiac arrhythmias, cardiac failure, and even death.

AVF is commonly used for permanent vascular access for hemodialysis. During dialysis, air detectors may warn the personnel and then prevent air from being pumped into the venous system. Air can potentially leak during puncture of AVF, as well as the hemodialysis tubing set is capped improperly or inadvertently disconnected from a line. Air embolism in such situations is rarely clinically significant because in the absence of a blood pump or pressurized infusion only little amount of air is likely to enter the patients' veins, and is usually confined to the venous circulation.

A condition for the entry of air into the venous system is sub atmospheric pressure. Causes include leaks around tubing connections and accidental removal of the arterial needle. Air may also enter from empty intravenous-fluid containers (glass bottles) connected to the blood circuit before the blood pump. In this case, the most possible leaking site is the tubing connections or the T-line before the blood pump and the major cause is improperly capping or clumping. Acute pulmonary air embolism occurs when air enters the venous system, from which it is transported into the lung where it causes serious physiological derangements. About 10% of people with pulmonary embolism suffer some pulmonary infarction. The body may break up small air emboli quickly, keeping damage to a minimum. Larger emboli frequently cause sharp pleuritic chest pain especially when the person inhales. The symptoms of pulmonary embolism usually develop abruptly, whereas the symptoms of pulmonary infarction develop in several hours. Symptoms of infarction often last several days but usually become milder day by day. The quantity of air that is associated with symptoms depends on the rate of air entry, and the vessel size. Microbubbles are tolerated to a greater extent than a macroscopic bubble; a slow infusion is tolerated better than a large bolus. It has been reported that as little as 12 ml ~ 15 ml of rapidly injected air may result in death. However, animal studies have shown that at least 3 ml to 8 ml air/Kg body weight is necessary to produce death from air embolism [4,5].

The surfaces of air bubbles are covered by a network of fibrin, platelets, and fat globules. Those may induce neutrophil-mediated microvascular damage, activate the intrinsic coagulation cascade and obstruct capillaries. This will cause an increased physiological dead space [6,7], ventilation-perfusion mismatching [8] and reduced cardiac output as a result of right ventricular outflow obstruction, leading to decreased end-tidal Carbon Dioxide tension ($P_{ET}CO_2$), arterial oxygen concentration, and systolic arterial pressure, together with an increased end-tidal arterial Carbon Dioxide gradient (Pa-$_{ET}CO_2$). High flow oxygen should be applied immediately. Gas from the air enters the blood with about 79% nitrogen [9]. The oxygen in the bubble quickly equilibrates with the blood, but the nitrogen equilibration takes longer. Administration of high inspired percentage of oxygen can drop the partial pressure of nitrogen in blood and increases the net nitrogen gradient from air to blood. Embolic bubbles size decreased as nitrogen dissolved in blood [10]. Air bubbles can be present in the intravascular space for more than 48 h [11]. Therefore, high inspired percentage of oxygen or hyperbaric

therapy should be considered. In our patient, her ABG initially revealed high oxygen alveolar-arterial oxygen partial pressure gradient (PA-aO_2), decreased PaO_2 and $P_{ET}CO_2$ and increased Pa-$_{ET}CO_2$. There was a rapid improvement after ventilation with oxygen 100% (Figure 5, 6). The fluctuation of parameters of oxygenation may be caused by rapidly decreased FiO_2.

The microbubble travels in the blood stream and lodged in the microcirculation. During the course, the bubble is compressed against the endothelial capillary wall, causing changed the functional structure of the endothelial cells, and created the gaps between endothelial cells [12]. It results in intravascular fluids leak into the surrounding tissue. Pfitzner, et al. [13], have shown that air embolism produces hypoxemia prior to the development of pulmonary edema [13]. It mimics that seen with larger boluses of intravenous air.

The clinical manifestations of air embolism are dependent on the patients` body position at the time of embolization. If the patient is upright, a rushing sensation to the head may soon be followed by neurological dysfunction. When patient is lying supine may complain of chest pain or shortness of breath. Those are lying in the Trendelenburg position at the time of embolization, air occludes the venous vasculature of the lower extremities with the findings of patchy cyanosis. If the patient is recumbent, foam is formed and results in the occlusion of the pulmonary vasculature.

Air detectors are essential components of the dialysis blood circuit for the prevention of air embolism. The ideal air detector will respond to foam as well as to air and not to saline; if there is an alarm it stops the blood pump and activates a tubing clamp immediately downstream of the air chamber. Many different physical principles have been employed: Probably the most reliable air detectors are ultrasonic devices. Ultrasonic waves pass through the bubble chamber and recorded at the opposite side. In the machine this patient was using, the air detector can detect the bubble larger than 0.05 ml. The microbubble less than 0.05 ml may pass through the air detector without an alarm and accumulate in the blood circuit. The most possible leaking site is the tubing connections. Having benefited from an improvement in technology, the sensitivity of air detector has increased from 0.05 ml/bubble to 0.01 ml/bubble, and now down to 0.003 ml/bubble. Although the new model was released in the year 2003, the old one is still commonly used in many centers. Therefore, a latent risk of air leak remains but the accidental air leak can be easier detected and the dialysis may be safer.

Air leak from the blood circuit is rarely reported. Minor cases of air embolism occur frequently and are minimally symptomatic. Severe cases are characterized by acute vascular insufficiency of specific organs such as the brain or spinal cord and/or hemodynamic collapse. Differentiation from pulmonary air emboli, acute myocardial infarction, or cerebrovascular accident may be difficult [1,2,14,15]. Air embolism should be considered in the differential diagnosis of any patient who has the sudden onset of cardiopulmonary or neurologic decompensation in a clinical setting that puts the patient at risk for air embolism [16]. Confirming the diagnosis of air embolism is difficult, and is complicated by the fact that air may be rapidly absorbed from the circulation while diagnostic tests are being arranged. Cardiac Doppler ultrasonography, end-tidal CO_2 monitoring, pulmonary artery catheterization, ventilation-perfusion scan and pulmonary angiography may be useful in supporting the clinical diagnosis of air embolism. In this case, cardiac Doppler ultrasonography 5 h later was performed, but there was not any positive finding. May be due to the timing of examination and the rapidity of the absorption of air. Doppler ultrasonography has been used to document the presence of air in the right ventricle and may show evidence of acute right ventricular dilation and pulmonary artery hypertension consistent with air embolism [3,17]. Esophageal ultrasonic Doppler emits acoustic signal in addition to the optical display on the monitor screen and is a useful alternative to the precordial ultrasonic Doppler [18]. It is often used during neurosurgical procedures, procedures with the patients in the sitting position, and other procedures with a high risk of air embolism. Ventilation-perfusion scan abnormalities which mimic those seen in pulmonary thromboembolism may be seen in the setting of massive air embolism. However, the perfusion defects due to air embolism resolve more rapidly, frequently within 24 h [19]. In this patient, the ventilation-perfusion scan was performed on the 4th day and the air may be rapidly absorbed from the circulation. This may explain the examination reports as "low probability of pulmonary embolism". Moderate heterogeneity of radioactivity distribution in the bilateral lung fields may be caused by the hypoxemic vasoconstriction due to recent emboli. This suspicion is difficult to exclude. Follow up lung scan may give us a clue.

Air embolism causes increased capillary permeability in the lung and adult respiratory distress syndrome has been shown experimentally in a sheep due to the production of superoxide anion and release by activated leukocytes [20]. It has been reported that the increased plasma levels of endothelin-1, big endothelin-1 and thromboxane were noted during acute pulmonary air embolism in pigs. The overexpression and production of these potent mediators can explain the concomitant deterioration of gas exchange and pulmonary hypertension during the course of this life-threatening disease [21]. In this case, the roentgenogram presented increased infiltration, congested vasculatures over bilateral lung field and consolidation of LLL with pleural effusion in left side. The presentation may be resulted from the increased permeability of pulmonary capillary and causing transient lung edema which may be explained by the adequate left ventricular performance shown in Doppler and echocardiography, and rapid clearance of the congestion and consolidation after supportive therapies but without antibiotics.

The aims of treatment of air embolization are identification of the source of air entry and prevention of further air embolization, removal of embolized gas, and restoration of the circulation. Supportive care is the cornerstone of management but active measures may also be helpful. After the air leak noted, immediately the blood lines are clamped and blood pump is stopped. The patient is placed in Trendelenburg position and lying on the left side and oxygen is given. No specific treatment is available, but administration of 100% oxygen would seem prudent. But, in animal study the repositioning provided no benefit in hemodynamic performance. The combination of acute increase in right ventricular afterload and arterial hypotension with subsequent right ventricular ischemia are the major contributing factors to cardiac dysfunction after air embolism [22].

Conclusion

Just like every kind of monitor air detector is not a perfect device. The size of micro bubble less than the designed size of air detector can make the air pass through the air detector without an alarm. Here we report a patient of air embolization during hemodialysis and either the connection of puncture needle and tubing or the T-line is the most possible site of air leak. For the purpose of airproof, these sites should be connected tightly or clamped correctly. The accident is fortunately

detected in the first seconds by the attending staff. Therefore, the staff is very important during dialysis for observation of any abnormal condition to compensate the defect of the machine. Besides, the staff should obey the guidelines of hemodialysis to avoid making mistakes and recheck the potential leaking site correctly.

References

1. Dudney TM, Elliott CG. Pulmonary embolism from amniotic fluid, fat, and air. Prog Cardiovasc Dis. 1994;36(6):447.
2. King MB, Harmon KR. Unusual forms of pulmonary embolism. Clin Chest Med. 1994;15(3):561.
3. Muth CM, Shank ES. Gas embolism. N Engl J Med. 2000;342(7):476.
4. Durant TM, Long J, Oppenheimer MJ. Pulmonary (venous) air embolism. Am Heart J. 1947;33(3):269-81.
5. Holt EP, Webb WR, Cook WA, Unal MO. Air embolism: Hemodynamics and therapy. Ann Thorac Surg. 1966;2(4):551-60.
6. Presson RG, Kirk KR, Hasselby KA, Linehan HJ, Zaleski S, Wagner WW. Fate of air emboli in the pulmonary circulation. J Appl Physiol. 1989;67(5):1898-902.
7. Albertine KH, Wiener-Kronish JP, Koike K, Staub NC. Quantification of damage by air emboli to lung microvessels in anesthetized dogs. J Appl Physiol. 1984;54:1360-8.
8. Orebaugh SL. Venous air embolism: Clinical and experimental considerations. Crit Care Med. 1992;20(8):1169-77.
9. Dexter F, Hindman BJ. Recommendations for hyperbaric oxygen therapy of cerebral air embolism based on a mathematical model of bubble absorption. Anesth Analg. 1997;84(6):1203-7.
10. King MB, Harmon KR. Unusual forms of pulmonary embolism. Clin Chest Med. 1994;15(3):561-80.
11. Tovar EA, Delcamp C, Borsari A, Webb RP, Dell JR, Weinstein PB. Postoperative management of cerebral air embolism: Gas physiology for surgeons. Am Thorac Surg. 1995;60(4):1138-42.
12. Barak M, Katz Y. Microbubbles: Pathophysiology and clinical implications. Chest. 2005;128(4):2918-32.
13. Pfitzner J, Petito SP, McLean AG. Hypoxaemia following sustained low-volume venous air embolism in sheep. Anaesth Intensive Care. 1988;16(2):338-40.
14. Heckmann JG, Lang CJ, Kindler K, Huk W, Erbguth FJ, Neundörfer B. Neurologic manifestations of cerebral air embolism as a complication of central venous catheterization. Crit Care Med. 2000;28(5):1621.
15. Orebaugh SL. Venous air emboli: Clinical and experimental considerations. Crit Care Med. 1992;20(8):1169.
16. Tellides G, Ugurlu BS, Kim RW, Hammond GL. Pathogenesis of systemic air embolism during bronchoscopic Nd:YAG laser operations. Ann Thorac Surg. 1998;65(4):930.
17. Marcus RH, Weinert L, Neumann A, Borow KM, Lang RM. Venous air embolism: Diagnosis by spontaneous right-sided contrast echocardiography. Chest. 1991;99(3):784.
18. Henze D, Menzel M, Lindner J, Roth S, Soukup J, Hennig C, et al. Validating of a transoesophageal ultrasonic-Doppler-probe to the detection of air in the venous blood. Anaesthesist. 1998;47(5):406-8.
19. Sessler CN, Kiser PE, Raval V. Transient pulmonary perfusion scintigraphic abnormalities in pulmonary air embolism. Chest. 1989;95(4):910.
20. Flick MR, Peril A, Staub NC. Leukocytes are required for increased lung microvascular permeability after microembolization in sheep. Circ Res. 1981;48(3):344-51.
21. Simon M, Battistini B, Joo Kim Y, Tsang J. Plasma levels of endothelin-1, big endothelin-1 and thromboxane following acute pulmonary air embolism. Respir Physiol Neurobiol. 2003;138(1):97-106.
22. Geissler HJ, Allen SJ, Mehlhorn U, Davis KL, Morris WP, Butler BD. Effect of body repositioning after venous air embolism. An echocardiographic study. Anesthesiology. 1997;86(3):710-7.