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Coronary air embolism during removal of a central venous catheter

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Abstract

Background. Acute air embolism has been described during central venous cannulation, but it may also occur during catheter removal in a spontaneously breathing patient. We describe an episode of acute coronary ischaemia that occurred during CV catheter removal.

Case report. A 23-year-old male, multiple trauma patient was treated over 27 days in an ITU. He required a tracheostomy, two weeks of mechanical ventilation, and several surgical interventions. On the 27th day, he was scheduled to be transferred to a low-dependency area and his CVC was removed from the left subclavian vein. After five minutes, the pressure pad was released from the site of cannulation; the patient started coughing and became dyspnoeic. He developed tachyarrhythmia with ST depression in the 2nd, 3rd and aVF leads, followed by marked ST elevation, and subsequently, ventricular fibrillation. The patient was placed in the Trendelenburg position and CPR was started. Normal sinus rhythm returned after three defibrillations. Echocardiography revealed the presence of a large amount of air bubbles within the left ventricle, which disappeared spontaneously within one minute. The patient quickly regained consciousness and his condition returned to normal within 12 h, with transient elevation of heart enzymes. Five days later, he was decannulated and transferred to the orthopaedic ward in a satisfactory condition.

Discussion. Air embolism during CV catheter removal is a rare event, but it may occur when a persistent tunnel remains after prolonged cannulation, associated with negative intrathoracic pressure created by a spontaneously breathing or coughing patient. In the case described, acute myocardial ischaemia occurred in the region supplied by the right coronary artery, which is located higher than the left one and is therefore more exposed to air bubbles. We could not demonstrate, however, the presence of a persistent foramen ovale, however some connection had to exist between the right and left sides of the heart in our patient.

Conclusion. Special caution should be exercised during CV catheter removal, and the procedure should be always done with the patient placed in the Trendelenburg position.

Key words: complications, central venous cannulation; air embolism; resuscitation, air embolism

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At present, vascular accesses to central veins are provided in all patients treated in ITUs. Their proper placement and management are the basic practical skills of the entire ITU personnel. The commonest complications related to this procedure are infections and thrombotic lesions [1]. Air embolism resulting from air penetration to central veins is most frequently observed during the access performed in a spontaneously breathing patient and is often accompanied by intravascular hypovolaemia. Unless the gas volume exceeds several to several tens of millilitres, the clinical consequences are usually irrelevant. The air bubbles got stuck in the pulmonary arterioles, from where they are eliminated through the lungs by diffusion. However, in some patients, this complication can lead to serious consequences, severe haemodynamic disturbances, in particular.

The objective of the present report was to describe a case of acute coronary syndrome during air embolism related to central vascular access.

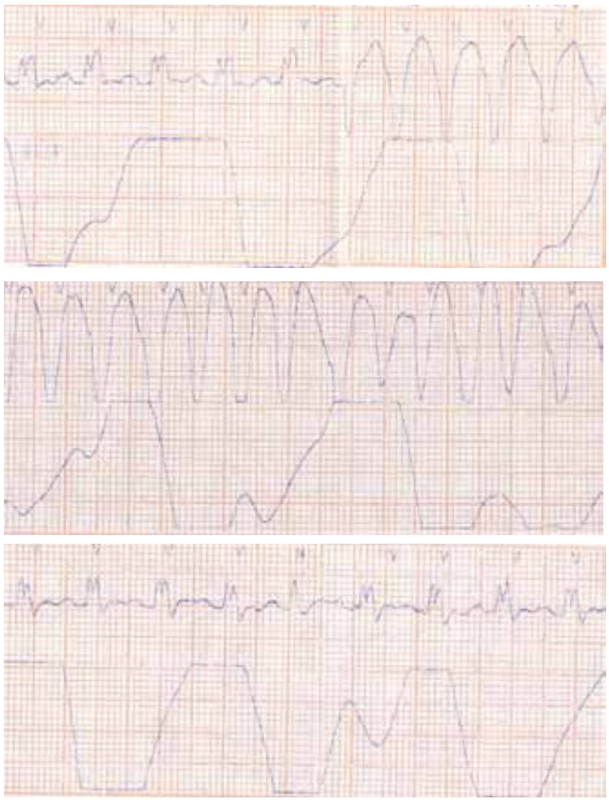

Fig. 1. ECG recording

CASE REPORT

In the evening of the day preceding the ITU admission, a 23-year-old patient, knocked down by a car, sustained multiple organ trauma. The patient suffered the thoracic injury with fractures of ribs IX and X on the left, pneumothorax, contusion of the left lung and abdomen, retroperitoneal haematoma on the right side descending to the right iliac fossa and damage to the right kidney. Moreover, the patient had the transcondylar fracture of the left hip joint, fracture of the pubic bone and the right posterior iliac ala, open fracture of the left shin bones and massive damage to the left forearm soft tissues. Immediately after admission, the injured right kidney was removed; injuries to the left forearm soft tissues were debrided and fractures of the left shin bones externally stabilized.

In the ITU, artificial ventilation of the lungs was implemented, initially at F_IO_2 0.5 followed by 0.4, and the cardiovascular parameters were maintained with the infusion of dobutamine. Furthermore, the patient was administered the suitable antibiotic therapy, enteral feeding, antithrombotic prophylaxis and analgosedation. Thanks to the treatment applied, his general condition stabilized. Percutaneous tracheotomy was performed on hospitalization day 10 and artificial ventilation was continued until day 19. The ITU management was however complicated with massive infection soft tissues. Due to the shin compartment syndrome symptoms, the patient underwent fasciotomy of the left shin and repeated surgical interventions to remove the infected and necrotic tissues. Thanks to this management, his general condition improved.

On day 27, due to no further indications for central vascular access and intention to transfer the patient to the department of orthopaedics, the decision was made to remove the vascular catheter. The patient was conscious, responsive, and breathed spontaneously; oxygen therapy was carried out through the tracheostomy tube. The circulatory parameters were stable. Having placed the patient horizontally with the headrest elevated by 15°, the 7F triple-lumen catheter was removed from the left subclavicular vein. Immediately after the removal, the subclavicular region was pressed. After about 5 min, the pressure was relieved to place the dressing. A massive cough fit occurred followed by forced inspiration, with retraction of the intercostal spaces and supraclavicular region. The ECG recording disclosed additional supraventricular contractions and a decrease in ST-T segment, followed by its elevation and rapid evolution to the Pardee`s wave. The site of catheter removal was immediately pressed and the patient was placed in the Trendelenburg position. Shortly, tachycardia with a wide QRS complex (Fig. 1) was observed, which subsequently resulted in ventricular fibrillation. The chest compression and artificial lung ventilation at F_IO_2 1.0 were immediately initiated. Triple defibrillation was performed; ischaemic changes gradually subsided. After about 1 min, the haemodynamically efficient heart rhythm was restored. Five minutes after the episode, echocardiography was carried out, which demonstrated the presence of numerous air bubbles within the left ventricle (Fig. 2). During the further several tens of seconds, their number gradually decreased to subside completely. The echogram of the heart was normal, except for gradually regressing contractility disturbances and the slightly enlarged right ventricle in diastole (2.6 cm).

The patient's general condition improved quickly. Artificial ventilation (F_IO_2 1.0) was carried out for 2 h. The patient regained consciousness, no symptoms of focal CNS lesions were observed. After the next 4 h, the concentration of creatinine kinase was 22.0 U L^{-1} and of troponin I – 2.9 ng mL^{-1}. The follow-up after 12 h revealed normalisation of these parameters.

On hospitalization day 33, the tracheostomy tube was removed and 24 h later the patient in good general condition was transferred to the department of orthopaedics for further treatment.

DISCUSSION

Air penetrates the vascular system only when the pressure difference is favourable for this phenomenon. In the horizontal body position, the pressure in the superior vena cava is usually higher than the atmospheric pressure. However, this changes radically at coexisting hypovolaemia, particularly during the inspiratory phase, in a spontaneously breathing patient. Therefore, the majority of physicians (91%) provide the central vascular access with patients placed in the Trendelenburg position.

Only 26% of physicians believe that the risk of air embolism occurs also during the removal of a central venous catheter.

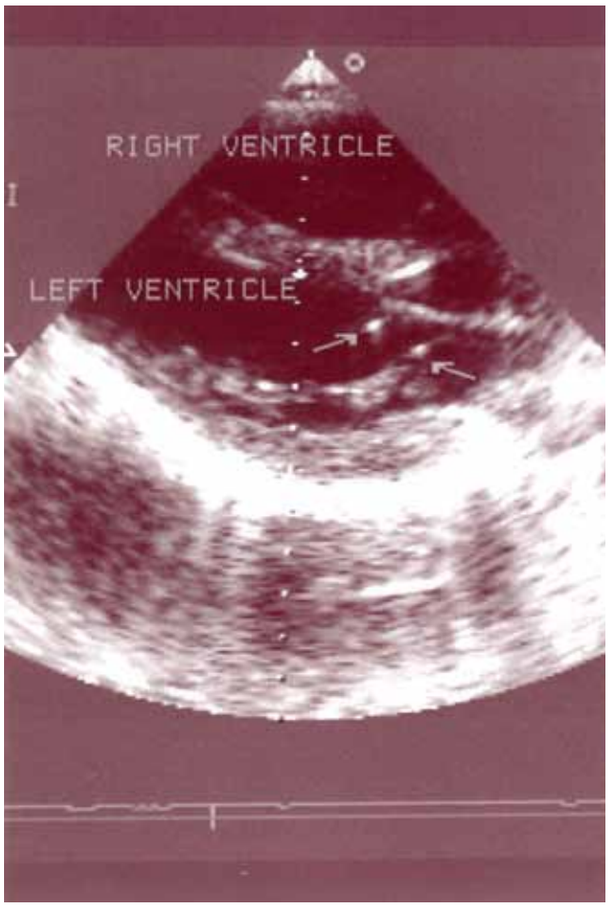

Fig. 2. Echocardiogram. Gas bubbles in the left ventricle (arrows)

Interestingly, 13.9% claim that they remove the catheter when the patient is placed in a reclining position [2].

The incidence of central vascular access-related air embolism is unknown as in the majority of cases the air volume permeating the circulatory system is so low that it does not cause any clinical symptoms. It is believed that the key role in its development, besides the total gas volume, is the rate with which the air penetrates the blood. The rate of 20 mL sec^{-1} will definitely induce various clinical symptoms whereas the 75-100 mL sec^{-1} will contribute to death in the majority of cases. The studies in dogs demonstrated that fast injection of air to vena cava resulted in death once the volume of 7 mL kg^{-1} was exceeded whereas air administration over several hours generated severe haemodynamic abnormalities after injection of 1400 mL [3]. It is worth remembering that a 14 G catheter enables the gas flow at the rate of about 100 mL s^{-1}, at the pressure difference of only 5 cm H_2O (0.49 kPa) [4].

During the first 24 h after introduction of central venous catheter, the inflammatory reaction enhances due to contact of the catheter with the adjacent tissues and the tissue tunnel is formed, which with the time passing should increasingly tend to maintain the lumen open. This can intensify the infection at the catheter site. The size of this canal tunnel is also directly dependent on the catheter diameter. Moreover, another independent risk factor is the choice of a central vessel for catheterization; some believe that subclavicular access induces higher risk than the jugular or femoral one [5].

In the case presented, a variety of phenomena developed, which supplementing one another, significantly increased the pressure gradient between the atmosphere and chest veins. During the catheter removal, the patient was not lying flat. The triple-lumen catheter was kept in the subclavicular vein for 10 days; immediately after its removal, the patient had a massive cough fit accompanied by forced inspiration through the 8.5 mm tracheostomy tube.

According to the available case reports and reviews, in the majority of cases the course of a given complication depends on the volume and speed of gas penetration to the circulatory system [3]. At large volumes, increased pressure in the pulmonary bed, right ventricle and right atrium reverses the interatrial gradient of pressures. In cases of persistent foramen ovale, this leads to the symptoms of paradoxical embolism. The alternative route enabling the passage of air to the systemic circulation is via the pulmonary capillaries.

At moderately severe embolism, larger gas bubbles are eliminated thanks to excellent filtrating properties of the lungs [6]. However, if the volume of gas is bigger or increases quickly, some of air bubbles are transported with blood to the systemic circulation bed leading to the final localisation-dependent clinical symptoms. In our case, the coronary vessels were the place of destination. The primary ECG changes in leads II, III and aVF indicate the abnormal flow within the right coronary artery, which is understandable as in the patient's recumbent position the right coronary artery is situated above the left one, which facilitates the transfer of gas bubbles from the blood. Similar localisations of air emboli were also described by other authors [7, 8]. In our case, the diagnostic procedure did not demonstrate the presence of persistent foremen ovale, which can be observed in 25-27% of paradoxical embolism cases [9, 10]. The explicit assessment in this case was however quite difficult. The final imaging examination, clinical symptoms and results of enzymatic tests were indicative of past acute coronary syndrome.

The risk of transfer of gas bubbles to arteries supplying the brain in paradoxical embolism cases is high. Clinical symptoms may significantly vary and their severity may be changeable [11, 12]. In our patient, however, no features of focal brain injury were observed. Among the implemented methods of management, which were found so beneficial, artificial ventilation at F_IO_2 1.0 is worth stressing; thanks to it, partial pressure of nitrogen in the embolic material was reduced and thus absorption of gas bubbles and their elimination through the lungs were accelerated. It is thought that an air bubble, 4 mm in diameter, is completely absorbed within 560 min of air ventilation and within 56 min during 100% oxygen ventilation [4].

The case described is not the first or the only one available in literature. Yet it is another piece of evidence that not a single medical case is simple. This is proved by our case, in which the catheter was removed by an experienced specialist under meticulous supervision of two other highly qualified specialists and a well-trained intensive therapy nurse.

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