CASE REPORT

AIR EMBOLISM DEVELOPED AS A RESULT OF OPENING THE CENTRAL CATHETER TIP BY THE PATIENT'S RELATIVE

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Central catheterization can be placed in critically ill patients in the intensive care unit (ICU) for some purposes such as dialysis, nutrition, and hemodynamic monitoring. Air embolism is a very rare complication of central catheterization. A 46-year-old male patient with no known comorbidities underwent laparoscopic total colectomy and protective loop ileostomy for colon cancer. He was taken to the general surgery ICU for close hemodynamic follow-up in the postoperative period. Since he was cachectic and could not reach the target of oral nutrition within 1 week, a central catheter was inserted in the right internal jugular vein with ultrasonographic imaging, and total parenteral nutrition (TPN) was started. The patient, who had no additional problems in the follow-up, was transferred to the general surgery ward. Three and half hours after the transfer, the patient became unconscious and had extensor posture. Therefore, emergency cranial computed tomography (CT) was performed and he was taken back to the ICU. There was no finding in favour of bleeding in cranial CT. The patient was intubated to protect the airway, as he had a generalized tonic-clonic seizure during his follow-up. Air bubbles were seen in the main pulmonary artery and right ventricle in the multidetector thorax CT. Cranial CT angiography was taken at the 24th hour, and diffusion cranial MRI was performed for diagnosis of central air embolism. No air was detected to be aspirated in the cerebral arteries in cranial CT angiography. On the 6th day, the patient regained consciousness, extubated, and physical therapy was started. On the 12th day of hospitalization, the patient was discharged with 2/5 loss of motor power in the left upper extremity. When the patient's wife's anamnesis was detailed, it was learned that in order to mobilize the patient, she separated the TPN from the catheter and left the catheter tip open.

Keywords: Central catheterization; Nutrition; Central air embolism

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INTRODUCTION

Central venous catheterization (CVC) is a common medical procedure applied to critically ill patients in the intensive care unit (ICU) for purposes such as dialysis, nutrition, or hemodynamic monitoring. The most common complications of CVC are mechanical complications (haemothorax, pneumothorax, arterial puncture, hematoma), catheter infection, bleeding, and thrombosis during the placement.[1] Air entry into the circulation resulting in air embolism is a very rare but potentially lethal complication of central catheterization. If CVC is no longer necessary in patients discharged from the ICU, it should be removed to avoid those complications.[2] Herein, we report a patient who developed air embolism following discharge from the ICU due to patent CVC in the service.

CASE PRESENTATION

A 46-year-old male patient with no known comorbidities underwent laparoscopic total colectomy and protective loop ileostomy due to colon cancer. No complications or problems were reported during the operation. He was taken to the general surgery intensive care unit (ICU) for a close hemodynamic follow-up in the postoperative period. On arrival, he was conscious, oriented, and cooperative, with a Glasgow coma score (GCS) 15/15, blood pressure (BP):120/60 mmHg, SpO2:95% (in room air), heart rate:89 beats/min, respiratory rate (RR) 20 breaths/min. No abnormality was detected in laboratory findings and arterial blood gas analysis. White blood cell (WBC) was elevated due to recent surgery**.** In the arterial blood gas analysis in room air, the pH was 7.41, PaO_2 103 mmHg, $PaCO_2$ 30.7, lactate 1.7 mmol/L, HCO_3 21.4 mmol/L, base excess -4,3. For venous thromboembolism prophylaxis, enoxaparin 0.6 cc subcutaneous treatment every 24 hours was started in the patient who weighed 75 kg. Prophylactic Cefazolin 1 gram IV every 12 hours started to

prevent surgical site infection. The patient was mobilized 6 hours postoperatively. Since the patient was cachectic and could not reach the target of oral nutrition within 1 week, a central catheter was inserted in the right internal jugular vein with ultrasonographic imaging and total parenteral nutrition (TPN) started. No complications were encountered in the chest X-ray following the insertion of central catheter (Figure-1).

The patient, who had no hemodynamic and clinical problems in the follow-up, was transferred to the general surgery service on the post-operative 4th day. The patient became unconscious (GCS:5) and had an extensor posture 3.5 hours after the transfer to the ward. On physical examination, there was no nuchal rigidity. Emergency cranial computed tomography (CT) was performed and the patient was taken back to the ICU. On arrival, he was unconscious, eyes were spontaneously open and fixed to the left lateral. Vital findings were blood pressure:193/120 mmHg, heart rate:143 beats/min, SpO2:96% (with 8 L/min oxygen support), RR: 22 breaths/min. Under 10 L/min oxygen therapy, in arterial blood gas analysis pH was 7.41, PaO_2 46.1, $PaCO_2$ 40.8, lactate 5.4 mmol/L, SpO_2 78.4%, HCO_3 20.8 mmol/L, base excess -4,5. The patient was hypoxic, and had high D-dimer levels (2.48 µg/ml), with increased high-sensitive troponin T (Hs trop-T) compared to the basal value of 6.68 ng/L (1 ng/L). There was no finding in favor of bleeding in cranial CT. NT pro-brain natriuretic peptide (NT-proBNP) was measured as 4884 ng/L, higher than the reference range (normal range 0-450 ng/L). On transthoracic echocardiography, left ventricular ejection fraction was 60%, and no pathology was found in the right heart chambers.

The patient was intubated to protect the airway, as he was having generalized tonic-clonic seizures during his follow-up. Lumbar puncture was performed once to exclude central nervous system infection due to fever of 38.5°C, however, no signs of infection were found. In cerebral spinal fluid (CSF), glucose was 74 mg/dL, chlorine was 128 mEq/L, potassium was 2.21 mEq/L, protein was 84.6 mg/dL. No bacteria or leukocytes were seen on the Gram stain of CSF; and in the cell count, no leukocytes or erythrocytes were seen per mm^3. Before echocardiographic assessment patient underwent multidetector thorax CT angiography at the 24th hour of clinical deterioration and air bubbles were detected in the right atrium (Figure-2), and the patient was placed on his upper right lateral side. Air was not observed in transthoracic echocardiography, and transoesophageal echocardiography could not be performed for technical reasons. A diffusion cranial magnetic resonance imaging (MRI) was performed to diagnose and aspirate the central air embolism. Wide diffusion restrictions on the right front parietotemporal and left parietotemporal regions were detected, but no air was detected in the vascular bed (Figure-3). No pathology was detected in bilateral carotid doppler ultrasonography.

Ecopirin 100 mg oral treatment every 12 hours was started, and the prophylactic subcutaneous enoxaparin 0.6 cc treatment was increased to twice a day. The patient regained consciousness on the post-op 6th day, he was extubated, and high flow nasal oxygen therapy was started with FIO_2 100%, due to air embolism to improve absorption. Afterward, the patient was started on physical therapy.

On the post-operative 12th day, the patient was discharged with 2/5 loss of motor power in the left upper extremity. When the patient's wife's anamnesis was deeply investigated, it was learned that in order to mobilize the patient, she separated the TPN from the catheter and left the catheter tip open. Considering the clinical presentation of the patient, it was thought that the air embolism emerged from the central catheter and went to the brain via the arterial route and caused occlusion, resulting in an ischemic cerebrovascular event. But we couldn't detect the air embolism in the cerebral vascular bed because of resorption.

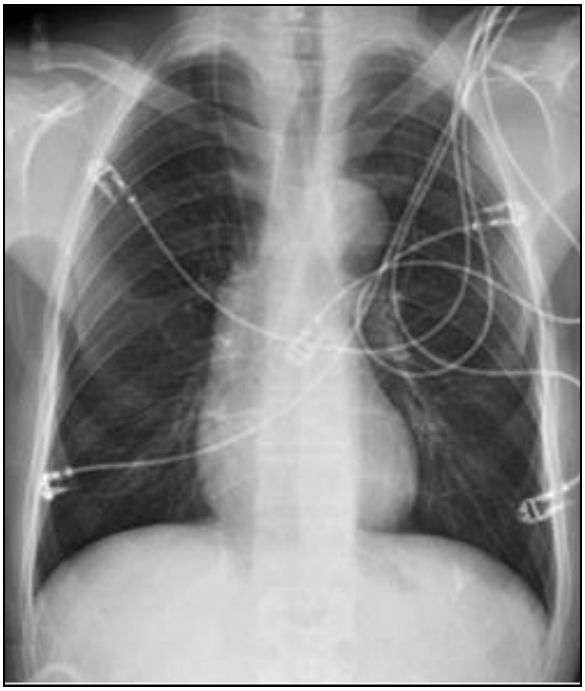

Figure-1: The location of the right internal jugular catheter is seen in the upper part of the right hilum in the postero-anterior chest radiography

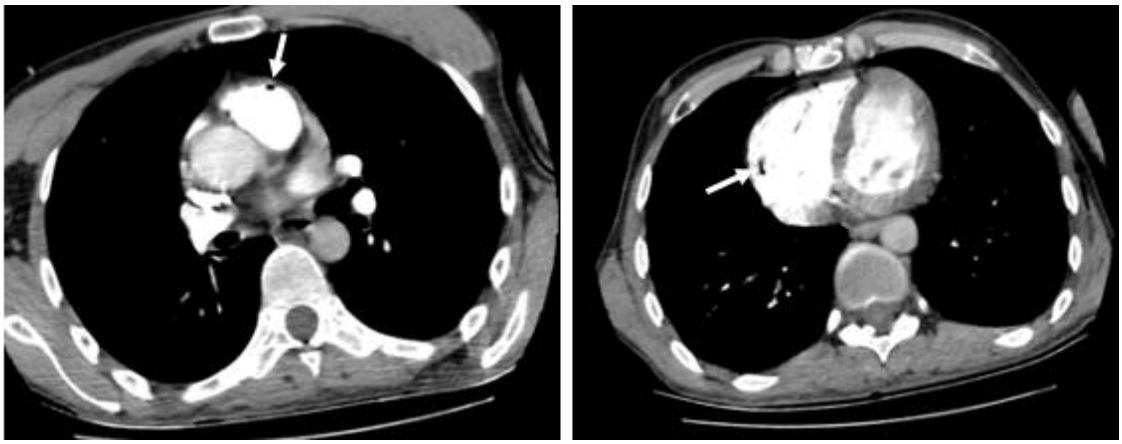

Figure-2: The main pulmonary artery and air in the right ventricle are seen in multi-detector lung computed tomography

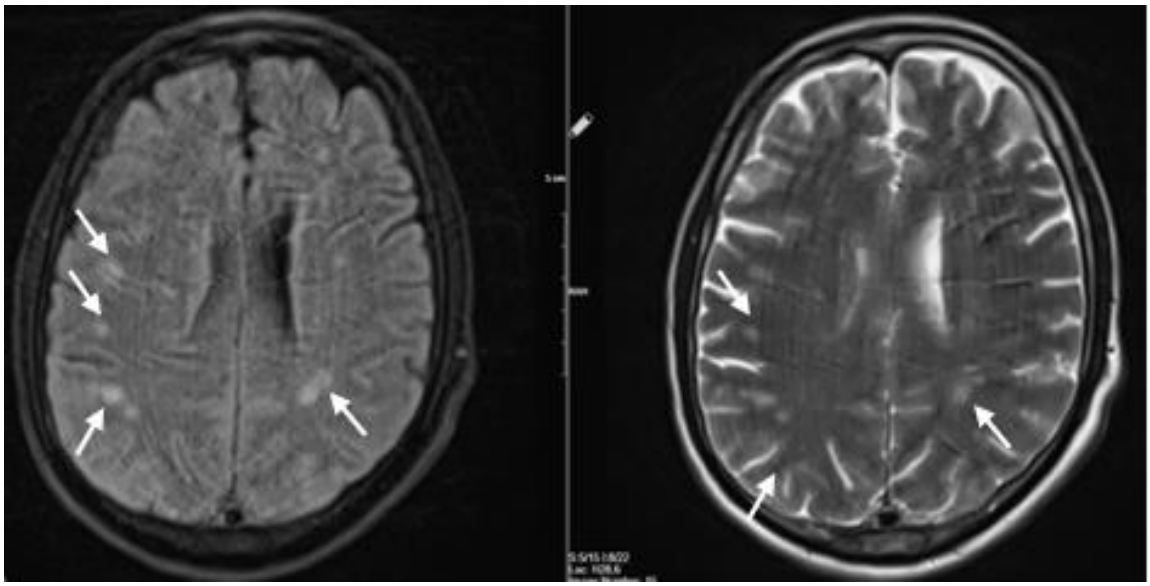

Figure-3: Right frontoparietotemporal and left parietotemporal wide diffusion restrictions are present in diffusion MRI images.

Table-1: Laboratory findings at the patient's first and second admission to the intensive care unit

Parameters	Laboratory values at the first admission to Intensive Care	Laboratory values at the second admission to Intensive Care	Reference value
Glucose	100 mg/dl	80 mg/dl	74- 106 mg/dl
Urea	18 mg/dl	12 mg/dl	19- 49 mg/dl
creatinine	0,87 mg/dl	0,5 mg/dl	0.7- 1,2 mg/dl
Total Bilirubin	0,99 mg/dl	0,34 mg/dl	0-1,2 mg/dl
Direct Bilirubin	0,29 mg/dl	0,17 mg/dl	0-0,3
AST (Aspartate-amino transferase)	27 U/L	60 U/L	0-40 U/L
ALT (alanine-amino transferase)	11 U/L	25 U/L	0-41 U/L
Calcium	8,35 mg/dl	8,1 mg/dl	8,6- 10,2 mg/dl
Creatine Kinase	- U/L	191 U/L	0-190 U/L
CK- MB	- U/L	14 U/L	0-25 U/L
Na	139 mEq/L	137 mEq/L	136- 145 mEq/L
K	4,88 mEq/L	3,83 mEq/L	3,5- 5,1 mEq/L
Cl	108 mEq/L	102 mEq/L	98- 107 mEq/L
White blood cell	25,540 x 10^3/µL	11,54 x 10^3/µL	3,57- 11,01x 10^3/µL
Hgb	12,9 g/dl	11,5 g/dl	13,2- 17,3 g/dl
Platelet	384 x 10^3/µL	386 x 10^3/µL	150- 372 x 10^3/µL
C-reactive protein	6 mg/L	45,69 mg/L	<5 mg/L
D-dimer	0,2 µG/mL	2,48 µG/mL	0-0,05 µG/mL
Hs-Troponin T	1 ng/L	6,68 ng/L	0-14 ng/L

DISCUSSION

Air embolism is a preventable, relatively rare but catastrophic hospital-acquired complication of CVC. Air can be introduced to the vascular system iatrogenically at the time of catheter insertion, removal, or during accidental or unawaredisconnection of the catheter.[3] In our patient, a central catheter was not removed for the continuation of TPN treatment due to cachexia during the postoperative period.

Vascular air embolism (VAE) can occur during surgery, traumatic events such as blunt or penetrating traumas, endovascular or other interventional procedures such as central catheterization, peripheral catheters, or during intravenous infusions when residual air remains in the closed system or might be a complication of mechanical ventilation.[4] The volume, rate and rapidity of the air or the medical gas entry into the patient's venous or arterial circulation and the position of the patient determine the degree of physiologic impairment VAE, ranging from being trivial to cause catastrophic outcomes. Common symptoms of VAE include cardiovascular collapse, neurological deficits, and pulmonary complications.[1] The pathophysiology of VAE requires both a negative pressure gradient and a communication channel between the vasculature and the atmosphere.[4] Air accumulation in the left ventricle impedes diastolic filling, and during systole air is pumped into the coronary and systemic arteries, disrupting coronary perfusion and resulting in acute hypoxemia and hypercapnia. This may result in elevated pulmonary artery and right ventricular pressures and decreased cardiac output, which can be followed by right heart failure, systemic circulatory collapse, and even death. If air emboli were to reach the cerebral circulation, neurological deficits may be observed ranging from confusion to seizures and cerebrovascular events.[5] The acute phase mortality was reported in 1/5 of patients.[6] The air emboli not only cause a reduction in perfusion distal to the obstruction in pulmonary arteries, but the air bubbles also initiate an inflammatory response, which can result in pulmonary oedema, bronchospasm, and increased airway resistance.[1]

The prevalence of VAE due to CVC is underestimated due to the transient and nonspecific clinical presentation and difficulties encountered in confirming the diagnosis. The frequency of VAE due to CVC is estimated to differ from 1 in 3000 cases to 1 in 47 cases in different studies, with a corresponding mortality rate ranging from 23–50%.[7] Malinoski *et al*.[8] published a prospective study including 594 patients over 2000 catheter days[8]. In this study performed in two trauma centers, a total of 354 central catheter-related complications were encountered, and 68% of the patients were male, as in our case. Pulmonary embolism was detected in 7 patients (0.5%) within 7 days of the removal of the central catheter. However, the frequency of air embolism was not mentioned in this study. Bell *et al*.[9] investigated the complication rates according to the opening sites of central catheters. In this study a total of 801 patients were examined, 32% of the patients had multiple catheters and 73 events of complication were detected. In the study, in which the complication rate was 5.9%, no difference was found in the mechanical complication rates in terms of catheter locations. They recommended the use of USG, as applied in our case, to prevent mechanical complications. Deep vein thrombosis/pulmonary thromboembolism was detected in a total of 26 patients (2.2%), and no event of air embolism was reported.[9]

The “gold standard” for the detection of venous air embolism is the trans-oesophageal echocardiography (TEE) due to its ability to detect as little as 0.02 mL/kg of air; however, the cost, expertise, and resource requirements limit its utility as a standard screening tool.[10] In our patient, TEE could not be performed due to technical reasons, and air could not be seen in TEE.

Treatment for VAE is mostly supportive. Using the Durant manoeuvre, the patient is placed on the left lateral decubitus position with the head lying down. This positioning helps to keep the trapped air within the heart away from the right ventricle outflow tract and may consequently reduce or dislodge the blockage caused by air bubbles within the vasculature. It is also recommended that patients receive high flow oxygenation. Hyperbaric oxygen is the definitive treatment, which facilitates gas reabsorption, improve tissue oxygenation and reduce ischemic injury by decreasing the size of air emboli.[11,12] In our case cerebral angiography taken at the 24th hour, it was observed that there was no air large enough to be aspirated in the vascular structures and there was no air in the control echocardiography, therefore, we did not plan hyperbaric oxygen therapy.

In patients with central air embolism the use of high-flow oxygen (15 L/min or more) can achieve high inspired fractions of delivered oxygen, but pulmonary toxicity should be considered while administering such high concentrations for extended periods. High concentrations of oxygen (approaching 100%) can generally be tolerated for up to 12 h, however if longer periods are necessary, room air brakes may be interspersed.[13] While 100% oxygen was given during mechanical ventilation in our

patient, high flow oxygen was given with 100% FIO_2 after extubation.

Air embolism associated with catheter removal either iatrogenically or accidentally can be a serious and frustrating medical complication that occurs at the time the patient's condition improves. Careful attention of physicians and healthcare workers is required to prevent air embolism related to CVC. Leading from this case, we suggest that the patients who were discharged from the ICU should be re-evaluated regarding the removal of their invasive instruments, and if they seem necessary, the relatives of the patients who took care of them should be informed about the necessary precautions.

Declaration of patient consent:

Written informed consent was obtained from the patient for the publication of this case presentation.

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