



Central Venous Line and Acute Neurological Deficit: A Case Series

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Abstract

Central venous catheter (CVC) insertion is a practical way to assess patients hemodynamic specially in cardiovascular surgery but this relatively simple junior level procedure is not risk free and its common reported complications include; pneumothorax, hydrothorax, hemothorax, local hematoma, cardiac tamponade, vascular injury, thrombosis, embolism, and catheter disruption. Here in this article we are going to present 6 patients with very unusual presentation of CVC complication which was neurological deficit presented by agitation, unconsciousness, disorientation to time and place and hemiparesis. All patients undergone neurologic consult and brain computed tomography. Final diagnosis was brain ischemic damage and finally we kept them on conservative management; fortunately we did not have any permanent damage.

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Introduction

Since 1952, when Aubaniac first described the clinical insertion of a central venous catheter (CVC) in the subclavian vein of battlefield-wounded patients,^{1,2} the CVC has been widely used in various clinical settings.³

The CVC is an essential aid in the management of numerous patients in that it provides reliable venous access for short term and facilitates hemodynamic monitoring, intravenous drug therapy, parenteral nutrition, hemodialysis, and rapid volume resuscitation.^{2,4} Although the placement of a CVC is often considered a relatively safe and junior-level procedure, the installation of such catheters is not risk-free even when it is performed by an experienced operator.^{5,6} Indeed, CVC-related complications such as pneumothorax, hydrothorax,

hemothorax, local hematoma, cardiac tamponade, vascular injury, thrombosis, embolism, and catheter disruption may be lethal and require urgent management.^{5,7}

We here by describe 6 patients, who developed neurological deficit immediately after the removal of the CVC.

Case Reports

Case #1

A 72-year-old diabetic man, who had a history of myocardial infarction (MI) 3 months earlier, was admitted for coronary artery bypass grafting (CABG). He was a former smoker and also had a history of hypertension, hyperlipidemia,

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and stroke. Preoperative evaluations revealed significant obstruction of 2 coronary arteries, an uncomplicated posteriorly located plaque in the left carotid bulb, and a left ventricular ejection fraction (EF) of 35%. Otherwise, he was normal. Prior to surgery, the right internal jugular vein was successfully cannulated with a 0.16-m catheter and threading of the catheter was smooth and uneventful. After an uneventful operation and recovery in the Intensive Care Unit (ICU), the patient was transferred to the Post-ICU Ward, where he remained hemodynamically stable.

Four days after the operation, the CVC was removed. Ten minutes later, the patient became agitated and disoriented to time and place. In addition, he developed severe spasm of the left side extremities, and his eyes were deviated to the left. He was tachycardic (140 beats per minute), and his Glasgow Coma Scale (GCS) was 10/15. After initial management, he was returned to the ICU. Due to agitation, the patient was sedated, intubated, and placed under continuous mechanical ventilation. On brain computed tomography (CT-scan) without contrast, evidence of ischemia in the periventricular and right external capsule was seen with no sign of hemorrhage. Brain magnetic resonance imaging (MRI) revealed old infarctions in the right external capsule and the cerebellar hemisphere. Moreover, an agitated saline contrast test confirmed the diagnosis of a patent foramen ovale (PFO). During the ICU admission, the patient also developed tonic-clonic movements, suggestive of seizure. Three weeks after the operation, the patient was re-admitted to the Post-ICU Ward in good general condition. Three days later, he was discharged from the hospital with a mild left-sided hemiparesis.

Case #2

An 83-year-old diabetic man was referred for CABG. He was a former smoker and was hypertensive and hyperlipidemic, with a history of remote stroke. He was found to have three-vessel disease on coronary angiography. His EF was 55%, and he had posteriorly located plaques in the right carotid bulb. Brain CT-scan revealed an old infarction in the right lentiform nucleus. Otherwise, he was normal. Before surgery, a CVC was placed in the right internal jugular vein successfully. Three hours after the surgery, the patient was re-operated on due to postoperative bleeding. Three days after the second operation, he was transferred to the Post-ICU Ward awake and hemodynamically stable. Two days later, the CVC was removed. Ten minutes later, the patient became unconscious. He had no arrhythmia. After appropriate management, he regained consciousness; however, he was still confused and had lower-limb paresis. Brain CT-scan without contrast reported ischemia of the deep white matter. Ten days after the operation, the patient recovered completely with no residual neurological deficit

and was discharged home from the hospital in good general condition.

Case #3

A 62-year-old hyperlipidemic woman was admitted to the Emergency Department complaining of new-onset angina. Coronary angiography revealed severe stenosis of the left anterior descending coronary artery. Two uncomplicated posteriorly located plaques were found in the bilateral carotid bulbs. Transthoracic echocardiography (TTE) demonstrated an EF of 55% and an abnormal bulging of the interatrial septum; however, the bubble test was not performed. The patient was prepared for CABG, during which a CVC was inserted in the right internal jugular vein successfully. Five days after an uneventful surgery, the CVC was removed. Immediately after CVC removal, the patient became agitated and disoriented and her GCS fell. She was commenced on antipsychotic and anticoagulant therapy. No significant lesion was found on CT-scan. Two days later, her condition improved, and she became awake and alert. No sign of significant stenosis was seen on cervical magnetic resonance angiography (MRA). Twelve days after the operation, the patient was discharged with no neurological symptom.

Case #4

A 58-year-old male smoker, who was also diabetic and hyperlipidemic, was referred for CABG. Preoperative assessments demonstrated significant stenosis of two coronary arteries, an EF of 55%, and normal bilateral carotid arteries. Prior to surgery, his right internal jugular vein was cannulated with a 0.16-m catheter uneventfully. Three days postoperatively and immediately after the removal of the CVC, the patient transiently became unconscious, aphasic, and left-sided hemiparetic for 30 minutes. Stroke management was performed for the patient. Normal imaging (CT scan and MRI) confirmed the diagnosis of transient ischemic attack (TIA). Postoperative TTE demonstrated an aneurismal interatrial septum; nevertheless, a post saline-injection bubble passage was seen neither at rest nor after the Valsalva maneuver. Four days later, the patient was discharged from the hospital in good general condition.

Case #5

A 65-year-old man, complaining of new-onset chest pain, was admitted with a diagnosis of acute MI. Except for a positive family history of coronary artery disease, he had no other cardiac risk factors. Further evaluations revealed three-vessel disease, an EF of 50-55%, and posteriorly

located plaques in the bilateral carotid bulbs. Preoperative electrocardiogram (ECG) confirmed the diagnosis of posterior Q-wave MI. The patient was prepared for CABG, during which a CVC was placed in his right internal jugular vein successfully. Three days after an uneventful surgery, the CVC was removed. Two minutes after CVC removal, the patient's oxygen saturation dropped abruptly, and he developed left-sided hemiparesis. His heart had a regular normal rhythm. The neurologist made the clinical diagnosis of embolic stroke and commenced him on anticoagulant, Dexamethasone, and Citicoline. The patient's brain CT scan was normal. One week later, he was discharged with residual neurological deficit in his left leg.

Case #6

A 52-year-old diabetic man was admitted for CABG.

Preoperative evaluations confirmed the diagnosis of three-vessel coronary artery disease and an EF of 50%; the patient was, otherwise, normal. Prior to surgery, a 0.16-m CVC was inserted in his right internal jugular vein. Six days after the operation, the CVC was removed. Minutes later, the patient seized and became right-sided hemiparetic; he was, accordingly, returned to the ICU. Hours after conservative management in the ICU, his hemiplegia recovered completely. Brain CT scan without contrast did not show any abnormal lesion. In postoperative TTE, bubbles passed through the interatrial septum (IAS) during the saline-agitated test, in favor the diagnosis of a PFO. Nine days after the surgical operation, the patient was discharged from the hospital in good general condition.

Table 1 depicts the patients' history in brief.

Table 1. Patients characteristic

| | Case 1 | Case 2 | Case 3 | Case 4 | Case 5 | Case 6 |
|----------------------------------|---|--|---|--|-------------------------------------|--|
| Age (y) | 72 | 83 | 62 | 58 | 65 | 52 |
| Gender | Male | Male | Female | Male | Male | Male |
| CCs FC | 2 | 0 | 0 | 2 | 0 | 0 |
| MI history | 3 months earlier | - | - | - | New MI | - |
| CVA history | + | + | - | - | - | - |
| Risk factors | | | | | | |
| DM | + | + | - | + | - | + |
| HTN | + | + | - | - | - | - |
| HLP | + | + | + | + | - | - |
| FH | - | - | - | + | + | - |
| Smoking history | Former | Former | - | Current | - | - |
| Number of involved vessels | 2VD | 3VD | SVD | 2VD | 3VD | 3VD |
| EF (%) | 35 | 55 | 55 | 55 | 50-55 | 50 |
| Carotid Doppler | Carotid LB plaque | Carotid RB plaque | Carotid LB & RB plaques | No plaque | Carotid LB & RB plaques | No plaque |
| Acute neurologic signs | Left-sided spasm, Eyes deviation to left | Unconsciousness, Confusion, Lower-limb paresis | Disorientation, Agitation | Unconsciousness, Aphasic, Left-sided hemiparesis | Left-sided hemiparesis | Seizure, Right-sided hemiparesis |
| CT-Scan result | Old infarction in periventricular and right external capsules | Deep white matter ischemia | Normal | Normal | Normal | Normal |
| TTE result | PFO | - | IAS bulging | Aneurismal IAS | - | PFO |
| Other neurological complications | Tonic-clonic seizure | - | - | - | - | - |
| Discharge status | Mild left-sided hemiparesis, otherwise healthy | Good general condition with no symptom free | Good general condition with no symptom free | Good general condition with no symptom free | Left leg paresis, Otherwise healthy | Good general condition with no symptom |
| Status 1 month after discharge | Alive, Mild left-sided hemiparesis | Alive, Symptom | Alive, Symptom | Alive, Symptom | Alive, Symptom | Alive, Symptom |

CCs FC, Canadian Cardiovascular Society Angina Function Class; MI, Myocardial infarction; DM, Diabetes mellitus; HTN, Hypertension; HLP, Hyperlipidemia; FH, Family history; CVA, Cerebrovascular accident; VD, Vessel disease; EF, Ejection fraction; LB, Left bulb; RB, Right bulb; TTE, Transthoracic echocardiography; PFO, Patent foramen ovale; IAS, Interatrial septum



Discussion

Neurological deficit is a relatively rare and poorly explained but critical complication of the CVC removal. Neurological deficit may be caused by a number of different mechanisms, the most common of which is believed to be paradoxical embolism after the removal of the CVC.

Paradoxical embolism may occur following the insertion, presence, or removal of the CVC and may lead to lower-limb ischemia, coronary artery occlusion, stroke, or a combination of these conditions.^{3, 8-10} The PFO or any other atrial septal defect predisposes patients to paradoxical embolism. The PFO is a common asymptomatic condition that may be found in up to 35% of people.^{11, 12} It is a well-recognized cause of stroke in cases of venous thrombotic events or air embolism.³ Be that as it may, paradoxical embolism may occur in the absence of intracardiac defects.¹³

Thrombotic Embolism

Previous studies have demonstrated that clots or fibrin commonly accumulate in or around venous catheters. Large-vessel thrombosis may occur in 8%-63% of patients with the CVC,^{3, 14, 15} and clot formation has been found in 30% of samples taken from heparinized catheters.^{3, 16} Therefore, either these clots or other debris within the CVC may be dislodged at the time of catheter removal³ and enter the arterial circulation through an intracardiac defect. The most common intracardiac defect associated with paradoxical embolism is the PFO, which may be undiagnosed or may open temporarily.¹⁷ Paradoxical thrombotic embolism is expected to manifest as pulmonary embolism, followed by an ischemic stroke; nonetheless, there have been a few reports of paradoxical embolism in the systemic circulation coexistent with pulmonary embolism. Hence, ischemic stroke has been described as the most important clinical manifestation of paradoxical embolism.¹⁸

Cerebral air embolism occurs under various conditions associated with the CVC, including CVC insertion and removal¹⁹⁻²⁴ or accidental disconnection of a CVC.^{19, 25, 26} A number of factors such as deep inspiration during the CVC insertion or removal, hypovolemia, and upright position of the patient reduce the central venous pressure and increase the risk of catheter-related air embolism.^{19, 26, 27}

Air entrainment in the venous system due to negative pressure following the CVC removal is a well-known but uncommon complication of routine postoperative care.²⁸ Even in patients without intracardiac defects, cerebral air embolism may occur following systemic venous air entrapment.¹³ Nevertheless, the exact mechanism whereby such systemic venous air reaches the cerebral vasculature in the absence of intracardiac defects is unclear.¹⁷ One possible mechanism for air embolism in patients without intracardiac

defects is that the pulmonary venous microcirculation is overwhelmed and eventually traversed because air emboli can easily deform.^{17, 27} Another explanation could be an undiagnosed PFO that opens temporarily when pulmonary arterial pressure rises.^{17, 29}

As was mentioned earlier, in the cases presented herein, neurological deficit appeared immediately after the CVC removal. The close association between the CVC removal and neurological impairment is in favor of paradoxical embolism with either thrombotic or air origin. The diagnosis of paradoxical embolism is more probable in patients who had or were suspected of having a PFO or other intracardiac defects.

Although only 4 patients in this study had evidence of intracardiac defects, paradoxical embolism in the other cases could have been caused by an undiagnosed temporarily-opened PFO or through pulmonary vasculature.

Thrombotic embolism may be caused by the dislodgement of clots or debris following the CVC removal, and air embolism can be caused by air entrainment during or immediately after the CVC removal.

Considering the high prevalence of the PFO, it may be expedient to perform pre CVC-insertion agitated saline contrast test to detect patients with this defect and, accordingly, commence those with a PFO on heparin prior to the CVC removal. This modality, however, does not seem to be cost-effective.³

Conclusion

It is important to detect any intracardiac defect in patients who need the CVC and to monitor neurological status after the CVC removal to detect the early symptoms of neurological impairment. We should consider the probability of both air and thrombotic paradoxical embolism in patients who develop neurological deficit after the CVC removal even in the absence of a PFO or other intracardiac defects. Prompt diagnosis and adequate treatment can minimize further cerebral damage, and protect the patient from developing lethal complications. Furthermore, it is highly recommended that the nursing staff follow the CVC removal protocols so as to drastically reduce costs and risk of air embolism.

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