

World Journal of *Clinical Cases*

World J Clin Cases 2024 February 6; 12(4): 671-871



EDITORIAL

- 671 Tenosynovitis of hand: Causes and complications
Muthu S, Annamalai S, Kandasamy V
- 677 Early antiplatelet therapy used for acute ischemic stroke and intracranial hemorrhage
Buddhavarapu V, Kashyap R, Surani S

MINIREVIEWS

- 681 Postoperative accurate pain assessment of children and artificial intelligence: A medical hypothesis and planned study
Yue JM, Wang Q, Liu B, Zhou L
- 688 Application and mechanisms of Sanhua Decoction in the treatment of cerebral ischemia-reperfusion injury
Wang YK, Lin H, Wang SR, Bian RT, Tong Y, Zhang WT, Cui YL

ORIGINAL ARTICLE**Clinical and Translational Research**

- 700 Identification and validation of a new prognostic signature based on cancer-associated fibroblast-driven genes in breast cancer
Wu ZZ, Wei YJ, Li T, Zheng J, Liu YF, Han M

Retrospective Study

- 721 Rehabilitation care for pain in elderly knee replacement patients
Liu L, Guan QZ, Wang LF
- 729 Effect of early stepwise cardiopulmonary rehabilitation on function and quality of life in sepsis patients
Zheng MH, Liu WJ, Yang J
- 737 Influence of initial check, information exchange, final accuracy check, reaction information nursing on the psychology of elderly with lung cancer
Jiang C, Ma J, He W, Zhang HY
- 746 Experience of primary intestinal lymphangiectasia in adults: Twelve case series from a tertiary referral hospital
Na JE, Kim JE, Park S, Kim ER, Hong SN, Kim YH, Chang DK

Observational Study

- 758 Perceived stress among staff in Saudi Arabian dental colleges before and after an accreditation process: A cross-sectional study
Shaiban AS

META-ANALYSIS

- 766 Comprehensive effects of traditional Chinese medicine treatment on heart failure and changes in B-type natriuretic peptide levels: A meta-analysis
Xia LL, Yang SY, Xu JY, Chen HQ, Fang ZY

CASE REPORT

- 777 Mechanical upper bowel obstruction caused by a large trichobezoar in a young woman: A very unusual case report
Scherrer M, Kornprat P, Sucher R, Muehlsteiner J, Wagner D
- 782 Accidental placement of venous return catheter in the superior vena cava during venovenous extracorporeal membrane oxygenation for severe pneumonia: A case report
Song XQ, Jiang YL, Zou XB, Chen SC, Qu AJ, Guo LL
- 787 Gestational diabetes mellitus combined with fulminant type 1 diabetes mellitus, four cases of double diabetes: A case report
Li H, Chai Y, Guo WH, Huang YM, Zhang XN, Feng WL, He Q, Cui J, Liu M
- 795 Clinical experience sharing on gastric microneuroendocrine tumors: A case report
Wang YJ, Fan DM, Xu YS, Zhao Q, Li ZF
- 801 Endoscopic retrograde appendicitis treatment for periappendiceal abscess: A case report
Li QM, Ye B, Liu JW, Yang SW
- 806 Hemichorea in patients with temporal lobe infarcts: Two case reports
Wang XD, Li X, Pan CL
- 814 Monomorphic epitheliotropic intestinal T-cell lymphoma with bone marrow involved: A case report
Zhang FJ, Fang WJ, Zhang CJ
- 820 Inetetamab combined with tegafur as second-line treatment for human epidermal growth factor receptor-2-positive gastric cancer: A case report
Zhou JH, Yi QJ, Li MY, Xu Y, Dong Q, Wang CY, Liu HY
- 828 Pedicled abdominal flap using deep inferior epigastric artery perforators for forearm reconstruction: A case report
Jeon JH, Kim KW, Jeon HB
- 835 Individualized anti-thrombotic therapy for acute myocardial infarction complicated with left ventricular thrombus: A case report
Song Y, Li H, Zhang X, Wang L, Xu HY, Lu ZC, Wang XG, Liu B
- 842 Multiple paradoxical embolisms caused by central venous catheter thrombus passing through a patent foramen ovale: A case report
Li JD, Xu N, Zhao Q, Li B, Li L

- 847** Rupture of a giant jejunal mesenteric cystic lymphangioma misdiagnosed as ovarian torsion: A case report
Xu J, Lv TF
- 853** Adenocarcinoma of sigmoid colon with metastasis to an ovarian mature teratoma: A case report
Wang W, Lin CC, Liang WY, Chang SC, Jiang JK
- 859** Perforated gastric ulcer causing mediastinal emphysema: A case report
Dai ZC, Gui XW, Yang FH, Zhang HY, Zhang WF
- 865** Appendicitis combined with Meckel's diverticulum obstruction, perforation, and inflammation in children: Three case reports
Sun YM, Xin W, Liu YF, Guan ZM, Du HW, Sun NN, Liu YD

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Multiple paradoxical embolisms caused by central venous catheter thrombus passing through a patent foramen ovale: A case report

Jian-Duan Li, Nian Xu, Qiang Zhao, Biao Li, Li Li

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Abstract

BACKGROUND

To date, this is the first case of a paradoxical embolism (PDE) that concurrently manifested in the coronary and lower limb arteries and was secondary to a central venous catheter (CVC) thrombus *via* a patent foramen ovale (PFO).

CASE SUMMARY

Here, we report a case of simultaneous coronary and lower limb artery embolism in a PFO patient carrier of a CVC. The patient presented to the hospital with acute chest pain and lower limb fatigue. Doppler ultrasound showed a large thrombus in the right internal jugular vein, precisely at the tip of the CVC. Transthoracic and transesophageal echocardiography confirmed the existence of a PFO, with inducible right-to-left shunting by the Valsalva maneuver. The patient was administered an extended course of anticoagulation therapy, and then the CVC was successfully removed. Percutaneous PFO closure was not undertaken. There was no recurrence during follow-up.

CONCLUSION

Thus, CVC-associated thrombosis is a potential source for multiple PDE in PFO patients.

Key Words: Paradoxical embolism; Central venous catheter; Patent foramen ovale; Acute myocardial infarction; Case report

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Core Tip: Paradoxical coronary embolism is a rare cause of acute myocardial infarction. Here, we report a case of simultaneous coronary and lower limb artery embolism in a patent foramen ovale (PFO) patient carrier of a central venous catheter (CVC). CVC-associated thrombosis is a potential source for paradoxical embolisms in PFO patients. Meanwhile, transesophageal echocardiography can help us detect PFOs more accurately.

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INTRODUCTION

Paradoxical embolism (PDE) is a clinical condition characterized by a thromboembolism that originates in the right heart or venous vasculature and travels through an intracardiac or pulmonary shunt into the systemic circulation[1]. The clinical presentation is diverse and potentially life-threatening. In this case, we present a patient with a patent foramen ovale (PFO) who suffered from acute myocardial infarction (AMI) and lower limb arterial embolism resulting from a PDE caused by a central venous catheter (CVC)-associated thrombosis.

CASE PRESENTATION

Chief complaints

A 72-year-old female presented to the hospital with acute chest pain and lower limb weakness.

History of present illness

Six days ago, the patient had a CVC implanted for a lumbar disc operation. 6 h before admission, the patient experienced sudden chest pain during the rehabilitation training without shortness of breath or palpitations.

History of past illness

The patient had a medical history of hypertension for 4 years.

Personal and family history

No relevant disorders were identified.

Physical examination

Blood pressure was 145/70 mmHg, heart rate was 65 beats/min, respiratory rate was 20 breaths/min. Lower limb weakness, without pale and pain and the skin temperature was normal. The pulsation of bilateral dorsal foot arteries was weakened.

Laboratory examinations

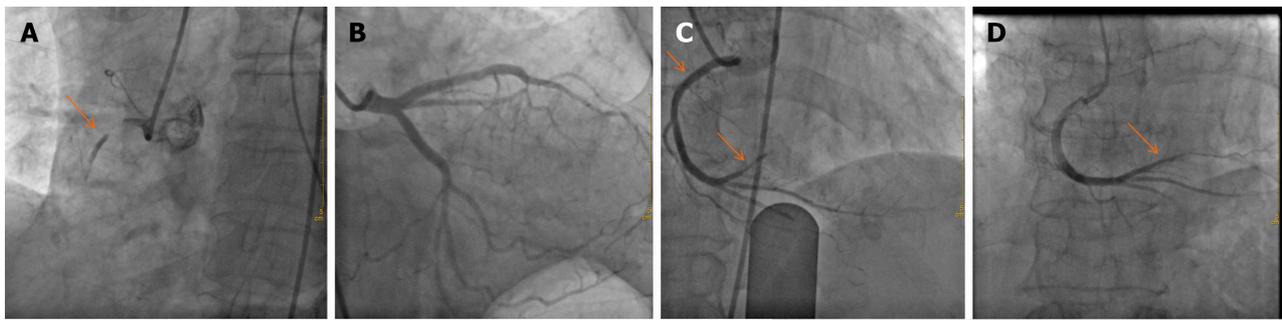
The patient's cardiac biomarkers were elevated, with a high-sensitivity troponin-T level of 0.688 µg/L and a creatine kinase level of 525.3 U/L.

Imaging examinations

ECG showed sinus rhythm and an ST-segment elevation of 0.1-0.2 mm in the inferior (II, III, and aVF) leads. Emergent coronary angiography (CAG) revealed an acute total occlusion at the proximal segment of right coronary artery (RCA) (Figure 1A), with normal blood flow of the left coronary artery (Figure 1B). After opening the RCA with balloon angioplasty, the repeat angiography revealed a smooth vascular wall without evidence of atherosclerotic plaque (Figure 1C) but the thrombus shattered and moved into the distal of the RCA (Figure 1C). After reperfusion, the patient was administered cardiovascular secondary prevention medication and low-molecular-weight heparin. Doppler ultrasound revealed thrombotic occlusions in the left anterior tibial artery and the right popliteal artery without any evidence of venous thrombosis.

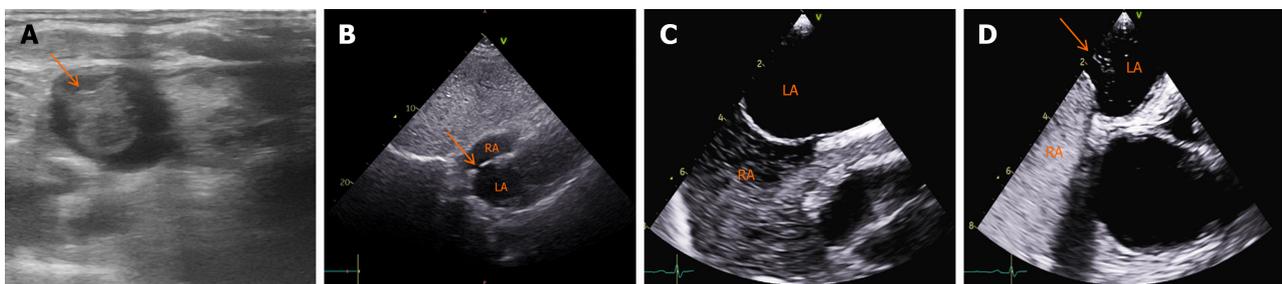
FURTHER DIAGNOSTIC WORK-UP

A large thrombus (18 mm × 15 mm × 13 mm) at the tip of the CVC was detected by ultrasound (Figure 2A). A transthoracic echocardiogram initially showed suspicion for a PFO accompanied by mild pulmonary hypertension (Figure 2B). This preliminary diagnosis was subsequently corroborated through an agitated saline contrast examination conducted *via*



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Figure 1 Coronary angiography. A: Emergent coronary angiography (CAG) revealed a total occlusion in the proximal segment of the right coronary artery (RCA); B: CAG revealed no significant atherosclerotic lesions in the left main, left anterior descending and left circumflex; C: After percutaneous coronary intervention (PCI), repeat angiography revealed the result of thrombolysis in myocardial infarction (TIMI) 3 blood flow in RCA. Thrombus were observed in the first posterior descending artery and posterior lateral artery; D: Two months after PCI, CAG showed TIMI 3 blood flow in the distal region of the RCA without signs of thrombus.



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Figure 2 Imaging examination of central venous catheter associated thrombosis and patent foramen ovale. A: Doppler ultrasound of the right internal jugular vein showing a large thrombus (18 mm × 15 mm × 13 mm) at the tip of the central venous catheter; B: Transthoracic echocardiogram showing a suspicious patent foramen ovale (PFO); C: In resting conditions, transesophageal echocardiography (TEE) with saline contrast showing no evidence of a right-to-left shunt; D: TEE following saline contrast clearly demonstrates the presence of bubbles in the left atrium, with Valsalva indicating the presence of a moderate right-to-left shunt at the atrial level through a PFO. RA: Right atrium; LA: Left atrium.

transesophageal echocardiography (TEE). In resting conditions, the TEE with saline contrast revealed no evidence of a right-to-left shunt (Figure 2C). Furthermore, the induction of a right-to-left shunt across the intra-atrial septum (grade 2) was successfully achieved by applying a Valsalva maneuver (Figure 2D).

FINAL DIAGNOSIS

Combined with the patient’s medical history, the final diagnosis was Paradoxical coronary and lower limb arteries embolism caused by CVC-associated thrombosis passing through a PFO.

TREATMENT

On the basis of the guideline-based medical therapy for STEMI, the patient was administered an extended course of anticoagulation therapy, specifically low-molecular-weight heparin for seven days, followed by a regimen of apixaban, dosed at 15 mg daily. Before discharge, the CVC was successfully removed.

OUTCOME AND FOLLOW-UP

A follow-up examination conducted two months postdischarge, which included CAG and Doppler ultrasound, indicated successful resolution of the thrombus in the distal region of the RCA (Figure 1D), bilateral limb arteries, and superior vena cava.

DISCUSSION

The PFO has been identified as a potential source of PDE since the late 19th century and is now recognized as the most common intracardiac shunt in PDE cases[1,2]. The most common sites of PDE are the extremities, accounting for 49% of cases, followed by the cerebrum at 37%. The coronary, renal, or splenic arteries are less frequently affected[3,4]. Instances of PDE resulting in multiple arterial embolisms are rare. Simultaneously, myocardial and renal infarction resulting from a PDE through a PFO was reported[5]. Islam *et al*[6] reported a case of PDE through a PFO involving the left upper extremity, brain, and coronary artery. However, there have been no reported cases of simultaneous myocardial infarction and lower limb thrombi due to a PDE caused by a CVC-associated thrombosis. This highlights the complexity and variability of PDE and underscores the need for continued research and understanding.

Acute myocardial infarction (AMI) is often precipitated by coronary atherosclerosis. Despite presenting several risk factors for atherosclerosis, the patient in this case demonstrated a different pathogenesis of AMI. Emergency CAG revealed a thrombotic occlusion in the RCA. After opening the RCA with balloon angioplasty, a repeat angiography of the RCA revealed a smooth vascular wall without evidence of atherosclerotic plaque. Her intact left coronary artery also excluded the presence of coronary atherosclerosis. Hence, we considered that the total occlusion of the RCA is caused by acute thromboembolism rather than chronic due to plaque. Evidence of acute thrombosis was also found in the Doppler ultrasound of the lower limb arteries, where the thrombi had completely disappeared after anticoagulation therapy.

In most cases, the elevated right atrial pressure is secondary to an acute process, such as the Valsalva maneuver, coughing or acute pulmonary embolism with cor-pulmonale or right-sided strain. This permits paradoxical emboli to easily cross the PFO from the right to the left atrium and enter the arterial circulation. Paradoxical emboli have the potential to traverse any branch of the coronary artery *via* antegrade blood flow, thereby precipitating an AMI. Furthermore, these emboli can disseminate to various organs, including peripheral arteries, resulting in widespread arterial embolisms. In this case, the presence of a small PFO and right-to-left shunting was confirmed through transesophageal echocardiography (TEE) and an agitated saline contrast examination. As reported, TEE is the gold standard because it can offer superior sensitivity and precision in detecting atrial anomalies and intracardiac shunts compared to transthoracic echocardiogram[7].

CVC-associated thrombosis constitutes 10% of all deep venous thrombosis (DVTs) in adults and 50%-80% of DVTs among children[8]. In this case, Doppler ultrasound showed the presence of a large thrombus at the tip of the CVC. Brain infarction caused by a CVC-originated paradoxical thrombus was previously reported[9,10]. CVC-associated thrombosis risk factors include catheter material and size, duration of placement, hereditary disorders or hypercoagulable states, blood flow stagnation, and potential endothelial damage from corrosive drugs[8,11]. The most likely risk factor for this case of CVC-associated thrombosis was the extended duration of the CVC placement and potential infection. As suggested by Citla *et al*[8], it is necessary to continue anticoagulant therapy for at least three months after catheter removal. The EAPCI advises that patients over 65 years of age should be evaluated for PFO closure on an individual basis [12]. In this instance, percutaneous PFO closure was not undertaken due to the PFO's relatively small size and the potential for eliminating and preventing CVC-associated thrombosis, which was considered the source of the PDE. Considering the patient's personal preferences, a pharmacological treatment approach was selected.

CONCLUSION

CVC-associated thrombosis is a potential source for PDE in PFO patients. Examinations for PFO and CVC in unexplained thrombotic events are important to look for possible sources of embolism. Furthermore, TEE can help us detect PFOs more accurately.

FOOTNOTES

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