



Misplaced Central Venous Catheter in the Vertebral Artery: Removal with Tract Coiling under Temporary Balloon Occlusion

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Abstract

Inadvertent vertebral artery (VA) cannulation during placement of a central venous catheter (CVC) in a jugular vein is a rare but serious complication. Various endovascular bailout options have been described. We herein illustrate a novel technique for removal of a misplaced CVC in the VA, applying tract coiling to achieve hemostasis under temporary VA balloon occlusion in a 70-year-old woman with sepsis, hyperosmolar coma, and active upper gastro-intestinal bleeding. After the procedure, angiography revealed patent VA without residual extravasation. Follow-up computer tomography (CT) showed no procedure-related complications.

Keywords: vertebral artery, iatrogenic, central venous catheterization, endovascular treatment, tract coiling

Introduction

Inadvertent vertebral arterial (VA) cannulation during jugular placement of a central venous catheter (CVC) at a non-compressible site creates a very high risk situation, as catheter removal may cause bleeding into the pleural cavity, chest wall, mediastinum, or neck. Due to the risks, surgical consultation or careful endovascular treatment planning are required.

We report a case of inadvertent VA cannulation with a 7F CVC in the left neck, with entry point at the V2 segment of VA, passing through the transverse foramen of the 6^{th} cervical vertebra, and ending in the ascending aorta.

Successful removal of the CVC using temporary balloon occlusion and tract coiling was achieved, resulting in complete hemostasis and preserved VA patency. To the best of our knowledge, this is the first reported case of inadvertent VA cannulation treated with this strategy.

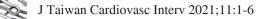
Case history

A 70-year-old woman, with hepatocellular carcinoma, diabetes mellitus, and end-stage renal disease, undergoing regular hemodialysis via Permcath catheter (COVIDIEN AG, Neuhausen am Rheinfall, Switzerland) in the right internal jugular vein, was admitted due to sepsis,

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hyperosmolar coma, and active upper gastrointestinal (UGI) bleeding. Because of unstable hemodynamics and hypercapnic respiratory failure, she was intubated and transferred to the intensive care unit. A 30 cm 7F triple lumen CVC (Arrow International, Cleveland, OH, USA) was inserted into her left internal jugular vein (IJC), percutaneously and without echo guidance. Arterial waveform was noted on pressure tracing. A blood sample yielded typical arterial, rather than venous, blood gas readings. Chest x-ray (Figure 1) suggested erroneous CVC course, as the superior vena cava was clearly outlined by the dialysis catheter. Arterial cannulation was confirmed by computed tomography (CT) angiography (Figure 2), revealing CVC entry into the left VA at the level between the 5th and 6th cervical vertebrae, passing through the 6th transverse foramen, and coursing into the ascending aorta.

Although the patient was neurologically asymptomatic, the anticipated removal of the

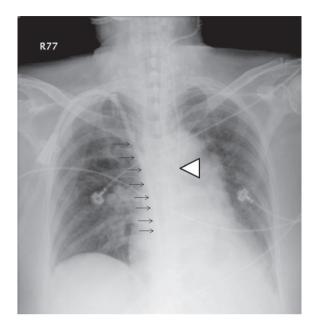


Figure 1. Post-cannulation view of chest, on plain film. Arrow head indicates tip of the central venous catheter. Black arrows indicate Permcath inserted into the right internal jugular vein prior to the insertion of CVC.

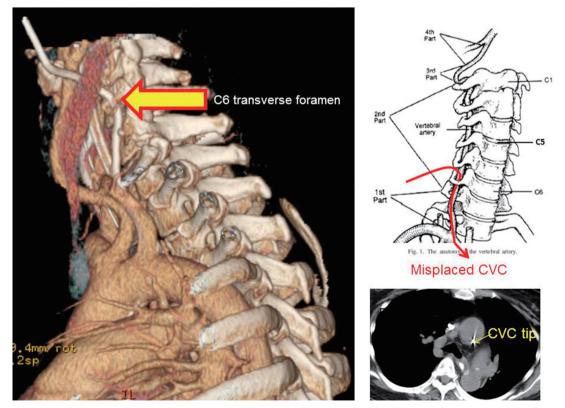


Figure 2. Misplaced CVC entrance into V2 segment of VA, at the level between C5 and C6 transverse process, passing through C6 transverse foramen and ending at the ascending aorta.





misplaced CVC caused significant concern among members of the primary team. In view of the patient's underlying bleeding tendency and the non-compressible anatomy, a simple "remove and observe" strategy was not considered. Surgical repair was rejected in both neurosurgery and vascular surgery consultations, due to relative inaccessibility of the entrance site, large exploration and dissection requirements. There was concern regarding cervical nerve root damage, vital vessel injury, and high anesthetic risk, in this extremely ill patient. The patient was promptly referred for evaluation regarding the possibility of endovascular management.

Cerebral angiography confirmed the arterial entry site of the CVC at the V2 segment of the left VA. The diameter of the left VA was 3 mm, and cephalad flow into the basilar artery (BA) was diminished at Thrombolysis in Myocardial Infarction (TIMI) score of 2 (Figure 3). Left common, external, and internal carotid arteries were not involved. No arterio-venous fistula or aneurysm was found. The right VA was patent, but not connected to the BA directly; only through collaterals from the posterior inferior cerebellar artery (PICA) (Figure 4). No significant collateral perfusion was found from the carotid system to the basilar system.



Figure 3. Selective left subclavian artery angiogram revealing preservation of antegrade flow in the left vertebral artery.

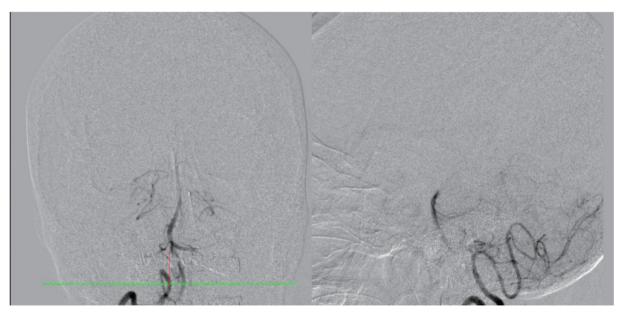
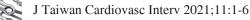


Figure 4. Right vertebral artery ends in the PICA, with collaterals to the basilar artery.



Procedure and technique

The nature of the condition, possible outcomes and complications, and potential treatment options were thoroughly reviewed and discussed. The primary goal was to remove the CVC, achieve good hemostasis and maintain flow within the left VA. Since the right VA was connected to the basilar artery only through collaterals, left VA occlusion by detachable balloon or coil was excluded because of the risk of basilar insufficiency. Similarly, a stent graft was rejected, due to the high entry point of the CVC and surrounding bony fixation points. The stress during neck rotation, or flexion, would likely lead to mechanical fatigue and disruption of the stent struts, resulting in stent crushing/deformity, migration, distal embolism, and/or endoleak with late extravasation. To achieve CVC removal with hemostasis, and preserve VA patency without stent graft, we devised the plan of coiling the tract after CVC withdrawal, under temporary balloon occlusion of the VA.

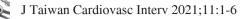
A 6F JR4 guiding catheter (Launcher, Medtronic, Minneapolis, MN, USA) was placed into the left subclavian artery via a right femoral puncture. Heparin was given to maintain activated clotting time (ACT) at 250 seconds. A 0.014" guidewire (Fielder FC, Asahi Intecc, Aichi, Japan) was advanced through the JR4, into the left VA passing distally into the V3 segment. A 3.0 x 20 mm balloon (Sapphire II, OrbusNeich, Hong Kong) was loaded onto the wire, delivered distal to the CVC entry site, and inflated to 6 atm for 5 minutes to confirm patient tolerance. After confirming a stable neurological condition, the balloon was deflated and kept there on stand-by status. Another 0.014" guidewire (Sion, Asahi Intecc) was inserted through the CVC via its connection hub, and advanced into the ascending aorta retrogradely. The CVC was withdrawn from the VA and the balloon on stand-by was then pulled back quickly to cover the CVC entry site. The balloon was inflated immediately to 6 atm, in order to control bleeding as well as to trap, and anchor, the Sion wire. A 2.6F microcatheter (Finecross, Terumo, Tokyo, Japan) was advanced over the Sion wire into the tissue tract created by the CVC, with its tip just outside of the VA entry site. The Sion wire was then withdrawn, and a small amount of contrast was injected via the Finecross micro-catheter to confirm its tip position. The hemostatic microcoil (VortX, Boston Scientific, Marlborough, MA, USA) was loaded into and pushed through the micro-catheter, and deployed in the tissue space just outside of the VA. The balloon was deflated temporarily, 5 minutes after deployment of four 2.5 x 3 mm coils. Only minimal extravasation and skin puncture site bleeding were observed. Complete hemostasis was achieved after 5 more minutes of balloon occlusion, and VA patency was confirmed by final angiogram (Figure 5).

Post-procedure course

The patient was transported back to the intensive care unit in stable clinical and hemodynamic condition. The left neck was examined and no bleeding, hematoma, or oozing was detected. Neurological examination revealed no evidence of posterior circulation insufficiency, and follow-up CT one month later showed patent left VA without aneurysm formation (Figure 6).

Discussion

The incidence of inadvertent arterial puncture during internal jugular vein (IJV) catheterization has been reported with a frequency of 0.5-11.4%, mostly into the carotid artery.¹ The actual incidence of inadvertent VA puncture during IJV catheterization is not clear, but should be infrequent, and dictated by individual anatomy. Punctures at the V1 portion have been more frequently observed, followed by punctures at V2.¹ A misplaced CVC indwelling in the VA may cause arterial occlusion and thrombosis, leading to posterior ischemia or distal embolization. The VA may suffer a puncture and the removal of the CVC may also cause dissection, hemorrhage, and pseudo-aneurysm formation. An enlarging neck



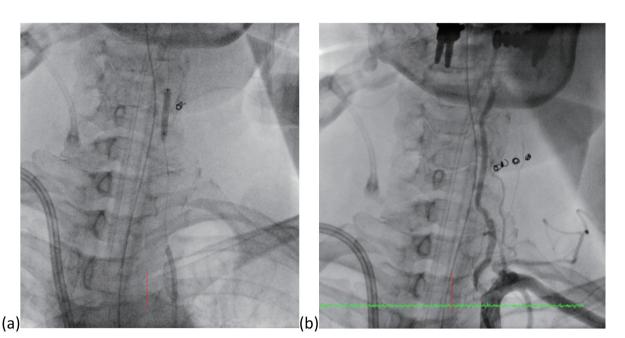


Figure 5. (a) Anchored balloon inflation for bleeding control and tract coiling. (b) Preservation of antegrade flow in the left vertebral artery after tract coiled.



Figure 6. Computed tomography angiography reveals patency of the left VA (arrow head). The white arrow indicates subcutaneous coil.

hematoma may lead to airway obstruction and cranial nerve injury.

Several management strategies have been proposed for inadvertent arterial cannulation by catheter. The simplest approach is direct removal with manual compression. But non-compressible arterial sites, such as the V2 segment in the present case, have only previously been managed through surgical intervention. Chung et al. reported a case of VA cannulation into the origin of the left VA with a dialysis catheter, which was resolved with surgical removal and suture repair of the artery under general anesthesia.³ However, there is a concern over potential procedural complications, such as Horner syndrome, injury to the phrenic or lower cranial nerves, and hemorrhage, especially in a critically ill patient with bleeding tendency.

Various endovascular techniques to achieve hemostasis after removal of misplaced CVC at non-compressible sites have been reported.⁴ Detachable balloon occlusion and coil embolization of the VA proximal to the entry site may be applicable in certain cases. However, as the right VA was not directly connected to the basilar artery in the present case, we felt that the left VA should not be sacrificed.

Covered stent deployment may be a good choice for preservation of lumen patency while achieving hemostasis. Akkan et al. reported a case with a misplaced CVC with entrance at the proximal VA, terminating in the right mediastinum, in which hemostasis was achieved



with a covered stent after catheter removal.⁴ However, the entry site in the present case was the V2 segment, where the artery was under constant mechanical stress due to neck movement. Therefore, the risk of stent crushing, deformation, fracture, migration, or endoleak had to be given serious consideration. Another issue in this patient was the active UGI bleeding with bleeding diathesis, prohibiting antiplatelet use following stent deployment.

Percutaneous arteriotomy closure devices have been applied in inadvertent arterial cannulation. Nicholson et al. summarized 4 cases with misplaced subclavian catheter, in which successful arterial closure with a collagen plugbased device (Angioseal, St. Jude Medical, Saint Paul, MN, USA) was achieved.⁵ However, in the present case, Angioseal (St. Jude Medical, Saint Paul, MN, USA) was contraindicated because of the small vessels (< 4 mm in diameter). Fraizer et al. reported the use of a percutaneous vascular suture device (Perclose, Abbott Vascular Devices, Abbott Park, IL, USA) for closure of an inadvertent subclavian artery puncture.⁵ However, the long and tortuous tissue tract in the present case, involving the C5 and C6 transverse processes precluded adequate sliding and tightening of the suture knot.

Given the unfavorable entry and long tissue tract, and the need to preserve VA patency, coil embolization with temporary balloon occlusion for bleeding control was deemed to be the safest strategy for our patient. Some essential technical points bear mentioning in this procedure. First, we advanced the Finecross micro-catheter with its tip as close to the VA as possible in order to avoid potential secular pseudoaneurysm formation later in the course. The risk of coil deployment into the VA with this proximity was mitigated by simultaneous balloon occlusion inside the VA. Second, intermittent release of the VA occlusion balloon was necessary to avoid thrombosis and ischemia in the basilar system. Complete thrombosis and hemostasis of the tract may not have occurred within 5 minutes, but the residual bleeding would flush the coils outward against the tip of the microcatheter. Therefore, the risk of inadvertent coil migration into the VA during balloon release was minimal.

Conclusion

We report a novel technique in managing inadvertent CVC cannulation of the VA in a patient at high risk for bleeding. The catheter was removed under temporary VA balloon occlusion, and the tissue tract was coiled to achieve final hemostasis. The patency of the VA was maintained and there was no need for anticoagulation/antiplatelet use after the procedure.

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