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Cervical carotid artery vasospasm during cerebral angiography

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ABSTRACT

Background: Vasospasm occurs commonly in the intracranial arteries as a complication of subarachnoid haemorrhage. On the other hand, extracranial Internal carotid artery (ICA) vasospasm is scarce, and it may occur due to mechanical manipulation during cerebral angiography. We report a case of cervical carotid artery vasospasm during diagnostic cerebral angiography, which caused anterior cerebral artery territory hypoperfusion, to discuss potential risk factors.

Case description: For a 22-year-old female with a ten-year history of epilepsy on multiple drugs, brain magnetic resonance imaging (MRI) showed frontal periventricular developmental venous anomaly. Diagnostic catheter cerebral angiography was used to better identify the vascular abnormality. In the procedure, extra steps were performed, including instruments being sterilized with CIDEX® OPA Solution (phthalaldehyde as the active ingredient), the reuse of the set including the catheters more than twice or triple times, and cold temperature of normal saline that was used in the flushing procedure. Under conscious sedation, the procedure went uneventful until the catheterization of the left carotid artery was performed, where severe vasospasm was noticed in the extracranial ICA, followed by cessation of flow in the ipsilateral ACA. Pulling the catheter to a more proximal location in the extracranial ICA was performed to alleviate the vasospasm. It took twelve minutes for the circulation to be restored, and that was under continuous irrigation and flushing. The patient did not develop any symptoms throughout the procedure or post-procedural course.

Conclusion: Chemical irritation from the sterilizing agent and reuse of the catheters could cause extracranial ICA vasospasm.

INTRODUCTION

Vasospasm in the intracranial arteries is a common complication of subarachnoid hemorrhage, cerebral vasculitis, and reversible cerebral

Keywords
cerebral angiography,
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vasoconstriction syndrome (RCVS) (6,20). Extracranial internal carotid artery (ICA) vasospasm, on the other hand, is rare, and its potential causes may include ergot poisoning or mechanical manipulation (2,16,8). Regarding mechanical irritation, it can attribute either to external manipulation or intraluminal stimulation. Intraluminal stimulation can be seen during catheter cerebral angiography, and it may trigger vasospasm in the ICA (1,10). However, extracranial vessels vasospasm can occur due to various causes depending on the procedural setting and the patient's condition. We report a case of cervical carotid artery severe vasospasm that causes anterior cerebral artery (ACA) territory hypoperfusion during diagnostic cerebral angiography with a discussion of potential factors.

CASE SCENARIO

A 22-year-old female with a ten years history of epilepsy on multiple drugs. Brain magnetic resonance imaging (MRI) showed frontal periventricular developmental venous anomaly (DVA). Diagnostic catheter cerebral angiography to better identify the vascular abnormality. The angiography procedure went with the typical steps used in every case in our institution. It is noted that we are obligated to reuse the instruments because of the cost-related issues and the absence of an insurance system in Iraq. That is why only the additive step that procedural set was reused more than two times at least after the sterilization with CIDEX® OPA Solution (phthalaldehyde as the active ingredient).

Underwent conscious sedation, the procedure went uneventful until the catheterization of the left carotid artery was performed, where severe vasospasm was noticed in the extracranial ICA, followed by cessation of flow in the ipsilateral ACA. We tried to pull the catheter to a more proximal position (2 cm below the skull base) in the ICA along with irrigation with warm normal saline multiple times was done, and it didn't work. So, the subsequent trial was to put the catheter in an even more proximal site at the left common carotid artery, and an irrigation procedure was performed again. It took twelve minutes for the ACA circulation to be restored, and that was under continuous irrigation and flushing (Figure 1). During these events, the patient was assessed clinically because she was

under conscious sedation, and fortunately enough, the patient didn't develop any symptoms throughout the procedure and during the post-procedural course.



Figure 4. Cerebral angiography images. (A) is showing the left ICA (anteroposterior view) showing the normal (pre-vasospasm) ICA with its branches. While (B) is showing ICA (anteroposterior view) showing spasm in the cervical of the ICA with ipsilateral ACA flow absence. (C) shows left ICA (anteroposterior view) showing partial resolution of the vasospasm in the cervical ICA with reestablishment blood flow in the ACA.

DISCUSSION

Intracranial vasospasm is temporary, focal, or diffuse narrowing in the caliber of intracranial arteries that mainly contribute to the arterial wall's smooth muscle contraction (17). It can be classified into intracranial or extracranial, depending on the ICA segments. Intracranial vasculatures are prone to vasospasm compared to extracranial (5,9). However, extracranial vasculatures vasospasm was also reported (2,16). Extracranial ICA vasospasm can be attributed to many factors that may be procedural-associated or patient-related.

In cerebral angiography, advancing the catheter and wires in the ICA may induce mechanical vasospasm. Ishihara H et al, examined 147 patients who underwent therapeutic neuro-endovascular procedures; severe vasospasm was noted in up to 40% of the sample (8). Individuals with significant anxiety, having a history of vasospasm in diagnostic angiography, and increase vascular tortuosity with a degree $\geq 30^\circ$ vessel bends were all potential risk factors for severe vasospasm (12). Patients with high anxiety have a high probability of developing vasospasm in both general anesthesia and conscious sedation because of the increase in the sympathetic response in the body during the procedure (8,12). The history of diagnostic angiography and the increase in vascular tortuosity may cause vasospasm because of mechanical irritation of the vascular lumen.

Based on the literature review, the intracranial location of the catheter within the ICA is a well-known cause of irritation in the form of pain or vasospasm (2,8,9). However, extracranial ICA vasospasm is either not well reported or usually doesn't affect the distal flow (9). During the procedure, the wire may induce vasospasm if it reaches the skull base. However, in our case and our practice, we localize the wire in the proximal portion of the cervical carotid. Here, we describe a case with apparent distal flow affection in the form of ipsilateral ACA flow cessation due to cervical carotid vasospasm.

Vasospasm due to mechanical irritation caused by sterilizing material is not described in the literature. In our case, after exclusion of the above risk factors, we propose the following points as potential causes for severe vasospasm, 1) chemical irritation with the use of CIDEX® OPA Solution, which has phthalaldehyde as the active ingredient, the primary tool for the sterilization of the procedure tools 2) re-sterilization of the equipment in the procedure and the set may be used twice or triple times 3) cold temperature of normal saline that was used in the flushing during the procedure. In the procedure, the reuse of the catheters has obvious advantages from a cost perspective and has several disadvantages because catheters are made for single-use (15). The practice of reusing catheters in cerebral angiography is common, and in Iraq, the re-usage can occur particularly when there is a limitation of resources.

The treatment of vasospasm in the setting of subarachnoid hemorrhage is well established by using calcium channel blockers (4,7). Mechanical irritation-related-vasospasm treatment needs further studies because it is clinically significant, and it is difficult to pinpoint the cause. There are a few reports on the efficacy of intra-arterial injections of calcium channel blockers, papaverine, and lidocaine in the mechanical irritation-related-vasospasm. Still, no comprehensive studies have been conducted on this topic (3,14). Reports listed that deep anesthesia or muscle relaxants may effectively reduce vasospasm and others suggested warm compression, especially while using the radial access (11). We propose different steps in the management of vasospasm in the setting of neuro-endovascular procedure, 1) dragging the catheter in a more proximal portion within the affected artery (in our case, the cervical carotid), 2) copious irrigation with

normal saline, 3) administration of vasodilators as discussed above, 4) the use of angioplasty. The use of one of these steps or a combination may result in the resolution of the vasospasm. In our case, continuous irrigation with warm normal saline was sufficient to resolve the vasospasm and restore the distal circulation.

As for prevention, vasospasm is expected to be relieved by prophylactic treatments in patients with risk factors such as sedative drugs and warm compresses (3,12). Also, using the optimum sterilization technique is needed as well as utilizing the tools once only is the preferable setting. While in limited resources setting where re-usage represents the only viable option, sufficient irrigation to ensure clearance of any sterilizing agent is mandatory. Flushing and the irrigating fluid' temperature can be monitored to use the fluid with body temperature as much as possible.

Clinical assessment of the patient is quite ready if the patient is under conscious sedation, and this fits well for diagnostic cerebral angiography. However, in certain cases, general anesthesia can be applied. Sriganesh K et al, proposed that the Bispectral index BIS might be utilized to detect and monitor substantial changes in regional cerebral blood flow following neuro-endovascular procedures with general anesthesia (19). In our case, the patient was on conscious sedation, the assessment of vasospasm was imminent, and it was carried out radiologically and clinically during the procedure.

In summary, vasospasm may occur in diagnostic cerebral angiography due to mechanical manipulation, chemical irritation, equipment usage, and temperature fluctuations in normal saline. The treatment options include the administration of intra-arterial calcium channel blockers or papaverine, or lidocaine. Prevention can be achieved by targeting the risk factors. Vasospasm as a complication during endovascular management should be highlighted and kept in mind during the explanation of the procedure to the patient, and it should be disclosed as there is a plurality of causes of this condition; we haven't scratched its surfaces yet.

CONCLUSION

Cervical carotid artery severe vasospasm is a rare complication during diagnostic cerebral angiography. Chemical irritation from the sterilizing

agent and re-usage of the catheters should be kept in mind as potential causes of vasospasm.

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