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Original Article

Hemodynamic stroke: A rare pitfall in cranio cervical junction surgery

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Abstract

Surgical C1C2-stabilization may be complicated by arterial-arterial embolism or arterial injury. Another potential complication is hemodynamic stroke. The latter might be induced in patients with poor posterior fossa collateralization (risk factor 1) when the vertebral artery (VA) is compressed during reduction (risk factor 2). We report a clinical case where this rare situation occurred: A 72-year old patient was undergoing C1C2-stabilization for subluxation due to rheumatoid arthritis. Preoperative computed tomography angiography (CTA) had shown poor collaterals in the posterior fossa. Furthermore, intraoperative Doppler ultrasound (US) detected unilateral VA occlusion during reduction. It appeared to be a high-risk situation for hemodynamic stroke. Surgical inspection of the VA found osteofibrous compressing elements. Arterial decompression was performed resulting in the normal flow as detected by US. Subsequently, C1C2-stabilization could be realized. The clinical and radiological outcome was very favorable. In C1C2-stabilization precise analysis of preoperative CTA and intraoperative US are important to detect risk factors of hemodynamic stroke. Using these data may prevent this rare, but potentially life-threatening complication.

Key words: Atlanto-axial instability, bow hunter's syndrome, cranio cervical junction, doppler ultrasound, skull base, vertebral artery, vertebro-basilar insufficiency

INTRODUCTION

Surgical C1C2-fusion for atlanto-axial subluxation may be complicated by ischemic stroke in about 1%. Most frequent mechanisms are arterial-arterial embolism or arterial injury. Another mechanism is hemodynamic stroke. The latter may occur through vertebral artery (VA) occlusion during C1C2-repositioning in a patient with a poor posterior fossa collateralization. We report a surgical case where this rare situation was encountered. Intraoperative Doppler ultrasound (US) diagnosed the occlusion. We are convinced that routine use of US prevented stroke in our patient. This rare pitfall in C1C2-fusion is illustrated and thoroughly discussed.

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Clinical case

A 72-year-old female suffering from rheumatoid arthritis presented with progredient neck pain and vertigo depending on head position (especially hyperextension). Clinical exam revealed a painful restriction of head and neck movements. No motor or sensory deficits were present, the reflexes were slightly decreased, no pyramidal signs, no sphincter disturbance was found. Dynamic cervical roentgenograms (flexion/extension) showed atlanto-axial subluxation. Computed tomography angiography (CTA) showed no aberrant VA precluding safe C1C2-fusion. There was no VA stenosis, but arterial supply of

the posterior fossa was poor: On the left there was an atretic p-com and on the other side a fetal uptake of the posterior cerebral artery. The patient was offered a posterior C1C2-fusion.

Surgery

After fiber-optical intubation, the patient's head was fixed in a Mayfield holder. The patient was placed in the prone position with the head slightly flexed under fluoroscopical control. The posterior bony landmarks of C1 and C2 were exposed. The instability could be confirmed intra-operatively by carefully moving C1 with a clamp. The atlanto-axial subluxation could be reduced under fluoroscopic control. In that position, the VA flow was checked with Doppler ultrasonography. Surprisingly, there was a complete flow arrest on the left side (the right being normal). When releasing the atlas, a normal flow on the left returned. When repositioning the atlas to perform arthrodesis, the flow was again interrupted. A careful inspection under the operating microscope (OPMI Pentero, Zeiss, Germany) revealed a small bony spur and a fibrous band over the left VA at the level of the groove just behind the lateral mass of C1 forming an osteo-fibrous canal. While the bony spur could be seen on preoperative CT imaging, the band was not visible [Figure 1]. When reducing the subluxation, the artery was pinched at the canal and the flow as measured by Doppler ultrasonography ceased [Figure 2a and b]. Fearing a stroke resulting from occlusion of one VA and poor p-fossa collateralization, we decided VA decompression before proceeding to fusion. Therefore, the artery was dissected in a subperiostal fashion with a blunt spatula in order to avoid bleeding from the perivertebral venous plexus. Once released, the bony bridge was resected with a Kerrison punch. Remaining fibrous adhesions were sharply released with micro-scissors. Thus, the artery was decompressed from the transverse foramen to its dural penetration [Figure 2c]. This time when atlas and axis were positioned for fusion, the flow in both vertebral arteries remained strong. The rest of the procedure could be performed as planned. A C1C2-fusion by Harms' technique with a rod-screw construct was performed. The postoperative course was uneventful. There were no signs of vertebro-basilar insufficiency, and the CT showed the resection of the bony spur [Figure 3]. At the follow-up, the patient's neck pain and positional vertigo had resolved. Bony fusion was obtained.

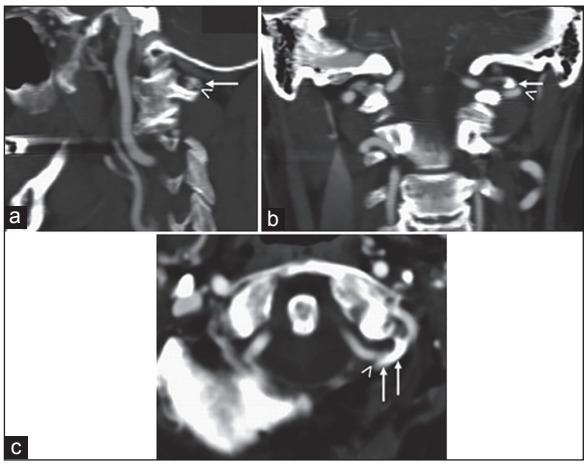

Figure 1: Computed tomography angiography with sagittal and coronal reconstructions, preoperatively (a-c). There is a bony spur (arrow) above the left vertebral artery; the fibrous band may not be seen (the position such as identified intra-operatively is indicated by an arrow-head)

DISCUSSION

Clinical case

The presented patient had an atlanto-axial subluxation due to rheumatoid arthritis. During the surgery, the subluxation could be reduced. However, routine US revealed an occlusion of the left VA in that position. The arterial compression occurred at the level of the C1 lateral mass. There were anatomical findings typically seen in bow hunter's syndrome (BHS). In fact, there were bony and fibrous elements forming a fibro-osseous canal impinging the VA [Figure 4a]. Due to a poor collateralization of the posterior fossa on CTA [Figure 4b], which is the second characteristic of BHS, we decided to decompress the artery before proceeding to C1C2-fusion. The patient's collateral circulation of the posterior fossa was underdeveloped due to a rare anatomic configuration: There was a fetal p-com up-take on the right and an aplastic p-com on the left side. We are convinced that the patient would have suffered a stroke, if the VA had not been decompressed before definitive C1C2-fusion.

Atypical bow hunter's syndrome

In general, BHS is characterized by symptomatic rotational occlusion of the VA at C1C2. Most often osteo-fibrous bands may be found. BHS may be surgically treated by arterial decompression and/or C1C2 fusion.[1,2]

Our patient's condition may be classified as an atypical form of BHS. Typical features were: Vertigo depending on head position,

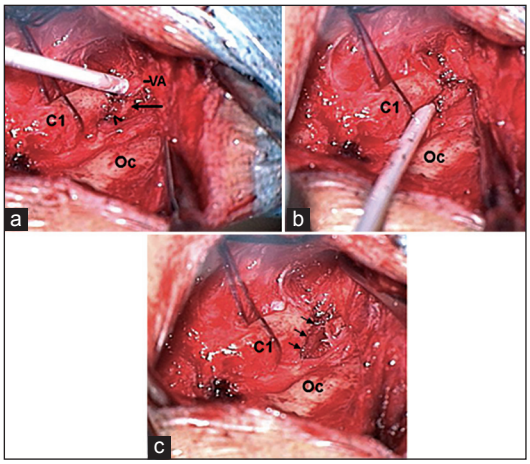

Figure 2: View through the operating microscope. There is an osteo-fibrous canal formed by a bony spur (arrow) and a fibrous band (arrow-head) around the left vertebral artery ("C-canal"). The Doppler probe measured neither flow at the entrance (a) nor at the exit (b) of this canal when reducing atlanto-axial subluxation. After decompression of the vertebral artery between the lateral mass of C1 and the dura (small arrows) normal flow was restored (c)

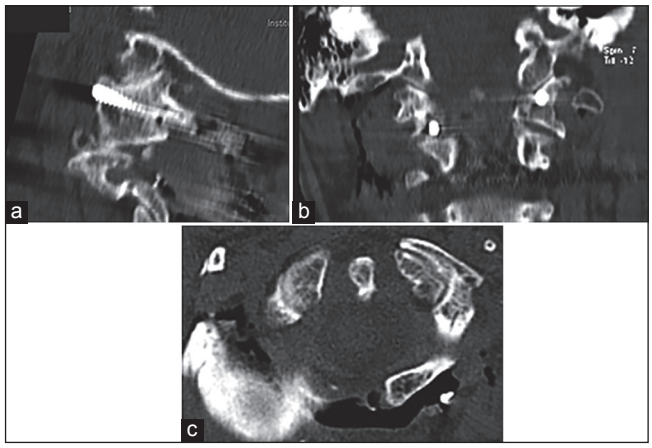

Figure 3: Computed tomography, bone windows, postoperatively (a-c). Note the resection of the bony spur and the screw-rod system

poor arterial supply of the p-fossa and osteo-fibrous elements compressing the VA when moving C1 and C2 relatively to each-other. What was somewhat atypical is that almost no rotation was necessary to occlude the VA. The fact of reducing the chronic subluxation by hyperextension was enough.

Neurovascular complications during C1C2-fusion and prevention strategies

The incidence of neurologic complications after posterior C1C2 fusions is about 1%.[3] The most frequent patho-mechanisms are thromboembolic strokes and arterial injuries. We believe that extrinsic VA compression and concomitant poor posterior fossa collateralization creating a BHS-like situation as detailed in the presented case may be another etiology.[1,2,4,5] Although this pitfall is probably very rare, it may be avoided by routine use of intraoperative Doppler echography during C1C2-fusions.[6] Furthermore, a subtle analysis of preoperative CTA or magnetic resonance angiography to assess posterior fossa collateralization should be done in every case.

CONCLUSION

The most typical neurovascular complications in C1C2-fusion are thromboembolic strokes or arterial injuries. Another rare etiology may be a positional extrinsic VA compression additionally to a poorly collateralized posterior fossa. This condition may be recognized by subtle analysis of preoperative CTA and intraoperative Doppler echography. Awareness and recognition of this pitfall is important to prevent stroke.

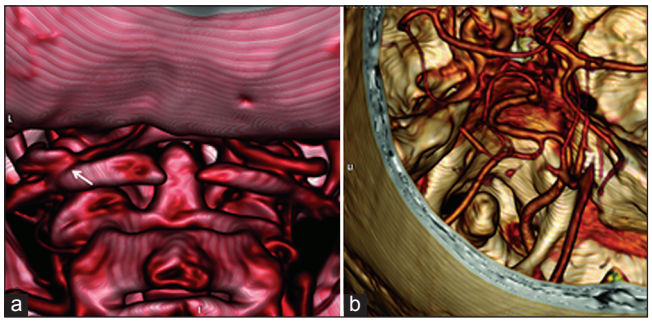

Figure 4: Three-dimensional reconstructed computed tomography. (a) View from behind: The left vertebral artery was pinched at the level of a fibro-osseous canal behind the lateral mass of atlas (arrow). Note the regular course of the right vertebral artery in its arterial groove. A nonunion of the atlas may also be seen. (b) Circulus arteriosus Wilisii seen from a left oblique view. Note the fetal configuration on the right side and an aplastic p-com on the left side

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