Research Article

Is the anterior spinal artery occluded in severe cervical compressive myelopathy?

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Purpose: The goal of this study was to identify whether the anterior spinal artery (ASA) is occluded in severe cervical compressive myelopathy (CCM) according to computed tomography angiography (CTA); occlusion was defined as spinal canal sagittal diameter compression of more than 80%.

Study design: A retrospective study.

Setting: Xinqiao Hospital, Chongqing, China.

Participants: The sample comprised 11 patients with spinal canal sagittal diameter compression of more than 80%.

Interventions: The patient underwent CTA of the ASA after admission. Covisualization of the ASA and the artery of Adamkiewicz was used to identify the ASA.

Outcome Measures: Spinal cord compression and decompression were determined with 1.5-Tesla magnetic resonance imaging. The neurologic status was evaluated according to the classification of the American Spinal Injury Association (ASIA).

Results: No ASA occlusion was found in all 11 severe CCM patients who presented with chronic or acute onset. All patients demonstrated improved neurological status after the anterior cervical decompression and fusion procedure.

Conclusion: ASA occlusion was not observed in CCM patients with spinal canal sagittal diameter compression of more than 80%.

Keywords: Cervical compressive myelopathy, Computed tomography angiography, Anterior spinal artery, Occlusion

Introduction

Cervical compressive myelopathy (CCM) may result in spinal cord physiological changes and may present notably as pyramidal tract syndrome with cervical compression, such as cervical disc herniation and spinal canal stenosis. The anterior spinal arteries (ASAs) (diameter, 0.2–0.8 mm) run along the entire length of the anterior surface of the spinal cord and distribute blood to the anterior two-thirds of the spinal cord.¹ Thus, the ASA may be theoretically compressed by a herniated disc, resulting in spinal cord ischemia or infarct, which presents as a high signal intensity area in the spinal cord with T2-

weighted magnetic resonance imaging (MRI) and as a low signal intensity area with T1-weighted imaging. It is easily hypothesized that cervical compression may result in ASA infarction and anterior spinal artery syndrome in CCM. However, in our previous computed tomography angiography (CTA) study of the ASA, ASA occlusion was not found in CCM patients with spinal canal sagittal diameter compression ranging from 10% to 80%. Pathological changes in the hyperintensity of T2-weighted MR images in CCM were not closely correlated with ASA infarction.² Nevertheless, there is still certainly a theoretical risk of ASA occlusion in case of severe CSM with spinal canal sagittal diameter compression of more than 80%. The objective of this study was therefore to identify ASA occlusion by CTA in severe CCM, which defined as spinal canal sagittal diameter compression of more than 80%.

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Materials and methods

Patients

Between May 2011 and May 2014, 122 patients with CSM were included in the investigator group (Z. Z.). Among them, 11 patients (9%) presented with spinal canal sagittal diameter compression of more than 80% and chose to undergo the identification of ASA occlusion by CTA. The clinical characteristics of the 11 patients are summarized in Table 1. In these 11 patients (10 males and 1 female), the distribution of cervical level was from C3/4 to C5/6; hyperintensity on T2-weighted MR images was demonstrated in 6 patients; the duration from onset until surgery ranged from 1 day to 6 months (2 presented with acute onset and 9 with chronic onset); and the follow-up for each patient was more than 1 year after surgery. All patients underwent plain cervical radiographs, CT scans, and 1.5-Tesla MRI. T1-weighted and T2-weighted MRI was performed to define cord compression, the proportion of spinal canal sagittal diameter compression, and the extent of intramedullary signal intensity changes. All patients underwent anterior decompression and fusion surgery, including anterior cervical disctomy fusion or anterior cervical corpectomy fusion. The preoperative and postoperative neurological status was evaluated using the American Spinal Injury Association (ASIA) impairment classification and the motor score.³

CTA of the ASA

CTA allows visualization of both spinal cord and bone anatomy, which is beneficial for the identification of the ASA in CCM. With institutional review board approval of the research protocol, CTA of the ASA was performed in the patients. CTA of the ASA and identification of the ASA was followed the protocol described previously.^{2,4,5} Briefly, patients underwent imaging with 64-slice MDCT scanners (LightSpeed

64, GE Medical System, Milwaukee, WI, USA). All CT scan images were obtained with a rotation of 0.5 s, nominal detector widths of 0.625 mm, and pitches of 0.984, 120 kV and 480 mA. Transverse sections were reconstructed with 50% overlap relative to the effective section thickness of 0.6 mm. Iohexol (Amersham Health Princeton, NJ, USA) was administered through an antecubital vein at a dose of 120–150 ml (350 mgI ml⁻¹) and a rate of 5 ml s⁻¹. The scan delay was determined by a preliminary 20 ml test injection at the level of the basilar artery. To examine the ASA transverse sections, multiplanar reformations, curved planar reformation and thin-slab (2–4 mm) maximum intensity projections were generated and displayed on a workstation (Advantage Workstation 4.3, GE Medical Systems) with window and level settings selected to maximize arterial to background discrimination.

Identification of the ASA

The ASA was identified by maintaining the maximum intensity projection slab parallel to the anterior surface of the spinal cord at each vertebral level assessed from C1 to C7. An enhanced artery on the midline ventral surface of the spinal cord was interpreted as the ASA. At the same time, an artery originating from the aorta and coursing through the intervertebral foramen to join the ASA in a hairpin configuration was interpreted as the Adamkiewicz artery. Covisualization of the ASA and the Adamkiewicz artery was used to identify the ASA (Fig. 1); the covisualization of veins was not a criterion for identifying the ASA. Another criterion was that the ASA originated from the vertebral artery in the skull base. Interrater reliability, in the form of the consensual identification of the ASA by two radiologists, was required.

Table 1 Clinical summary of patients.

Case no.	Sex / Age	Level of compression	T2 cord hyperintensity	Onset	ASA occlusion	Spinal canal sagittal compression (%)	Type of operation	ASIA on admission/1W/1 year
1	M/44	C4/5 C5/6	C4-C6	Chronic	No	90	ACCF	D/D/E
2	M/39	C3/4	C3-C4	Chronic	No	90	ACCF	C/C/D
3	F/60	C3/4 C4/5 C5/6	C3/4	Chronic	No	90	ACCF	D/D/E
4	M/44	C3/4 C4/5	None	Acute	No	90	ACDF	D/E/E
5	M/66	C4/5 C5/6	C4/5	Chronic	No	90	ACCF	D/D/D
6	M/47	C4/5	C4/5	Chronic	No	90	ACCF	D/D/E
7	M/42	C4/5	C4/5	Acute	No	80	ACDF	D/D/E
8	M/61	C4/5	None	Chronic	No	80	ACDF	D/D/E
9	M/40	C4/5 C5/6	None	Chronic	No	80	ACDF	D/D/E
10	M/78	C3/4 C4/5	None	Chronic	No	80	ACDF	D/D/E
11	M/74	C4/5	None	Chronic	No	80	ACDF	D/D/E

M, male; F, female; C, cervical; CCM, cervical compression myelopathy; ASCS, anterior spinal artery syndrome; ACDF, anterior cervical disctomy fusion; ACCF, anterior cervical corpectomy fusion.

Figure 1 Identification of the ASA. Coronal (A), sagittal (B) and oblique-sagittal (C, D) CTA views showing the ASA and the adamkiewicz artery (arrows) as the suppliers of the ASA.

Results

ASA occlusion

The clinical data are summarized in Table 1. The ASA was visualized in all 11 CCM patients. The course of the ASA extended from C1 to C7 in sagittal, coronal and transverse views. The ASA runs along the entire length of the anterior surface of the spinal cord. Anterior radiculomedullary arteries (ARAs), the ASA branches that supply the cord throughout its length by anastomoses branching upward and downward, were not visualized with angiography, because ARAs have very little or even no flow in two directions. For all 11 CCM patients with more than 80% spinal canal sagittal diameter compression, no ASA occlusion was found (Table 1), including in 6 patients with hyperintensity on T2-weighted MR images.

Neurological status

The preoperative and postoperative neurological statuses of the patients are provided in Table 1. None of the 11 CCM patients experienced complications after anterior cervical discotomy fusion or anterior cervical corpectomy fusion. With the exception of the neurological status of sixty-six-year-old patient' who demonstrated no improvement after one year of follow-up (case 5), the other 10 patients demonstrated improved neurological status of one level on the ASIA scale after over 12 months of follow-up. Compared with the chronic onset patients, the 2 patients with acute onset (cases 4 and 7) demonstrated faster (within 2 weeks) and more complete neurological improvement after emergency surgery.

Illustrative cases (Case 7) (Fig. 2)

A 42-year-old male was admitted with limb weakness and numbness for 2 days. Neurological examination revealed that the strength of the limbs was 4/5 (ASIA D). Deep tendon reflexes were slightly increased in all limbs, the Babinski sign was negative, and sensations were almost normal. T2-weighted MR and CT images demonstrated that the cervical spinal cord was compressed at the C4/5 (over 80%) intervertebral levels (Fig. 1A, B, C). Spinal cord hyperintensity was observed in the central area at C4/5 (Fig. 1A, B). The patient was diagnosed with CCM. CTA demonstrated a normal ASA at C3-C6 (Fig. 1D, E, F). The patient underwent anterior cervical discotomy fusion, and the neurological status recovered to ASIA E after 12 months of follow up.

Discussion

In our series, no ASA occlusion was found in 11 CCM patients with spinal canal sagittal diameter compression more than 80% and with hyperintensity on T2-weighted MR images. This finding indicates that the spinal cord more easily experiences compression than the ASA in its anterior surface. These negative findings are similar

to our report that ASA rupture is not commonly observed in acute blunt cervical spinal cord injury,⁴ in CCM patients with spinal canal sagittal diameter compression less than 80%,² and in cervical spondylotic amyotrophy and CCM patients with the “snake-eye” MRI sign.⁵ These results are helpful for understanding the large difference in incidence between CCM and anterior spinal artery syndrome (ASAS), which is caused by occlusion or hypoperfusion of the ASA and results in spinal cord infarction.

A few reports have described ASAS with ASA occlusion in cervical spondylosis according to clinical presentation and/or MR imaging studies.^{6–13} However, only one case with ASA visual images has been reported in patients.¹¹ The typical presentation of ASAS is acute and painful myelopathy. The usual presentation of cervical spondylosis is chronic compressive myelopathy, which is different from ASAS. In our series, even in the patients with acute onset, no patients had both acute transient neck pain and spinal cord compression, which does not support the diagnosis of acute ASAS. It is interesting that reported ASAS cases caused by compression due to disc herniation and ASA occlusion

Figure 2 A 42-year-old man with CCM at C4/5. (A) Sagittal T2-weighted MRI showing spinal cord compression with severe C4/5 disc herniation (arrow). (B, C) Transverse T2-weighted MRI and CT scan showing spinal cord compression at C4/5 (arrows). (D, E, F) Right oblique-sagittal view (D), left oblique-sagittal view (E) and coronal view (F) of CTA showing the ASA at C4/5 (arrows).

demonstrate small herniated disc sizes in MR images.^{6–13} This finding indicates that ASA occlusion has no absolute correlation with the severity of compression.

Spinal cord infarction demonstrates an intramedullary high signal intensity area in the spinal cord on T2-weighted MRI; this characteristic is also associated with other myelopathies such as edema, myelomalacia, gliosis, and inflammation.¹⁴ In other words, although ischemia is a key factor of and key contributor to the secondary pathogenesis for CCM,¹⁵ pathological changes in T2-weighted hyperintensity in CCM can be considered as only one very suggestive causes of spinal cord infarction. However, no cases of ASA occlusion were found in the 11 severe CCM patients. ARAs may be occluded and compressed in severe CCM patients, which may be a possible reason for cord ischemia but not ASA occlusion. For the visualization of ARAs, we failed with the use of a catheter and with CT and MR angiography (data not shown). Advancing imaging techniques will definitely further improve the possibility of non-invasively visualizing intradural spinal cord arteries and veins in the spinal cord.

The treatment for CCM and reported ASAS with cervical spondylosis consists of early surgical decompression and fusion. The long-term prognosis of ASAS is poorly described from many causes.¹⁶ In reports of ASAS with cervical spondylosis, neurological recovery is demonstrated in most but not all cases after decompression surgery.^{6–13} In contrast, all patients in this series demonstrated neurological improvements of 1 or 2 ASIA scales after 1 year following up, especially in patients with acute onset after emergency surgery (Table 1). This finding indicates that spinal cord infarction caused by ASA occlusion (e.g. ASAS with cervical spondylosis) is a worse factor compared with spinal cord compression (e.g. severe CCM) in terms of long-term prognosis.

Although this is the first study to show ASA visualization in severe CCM, the limitations of this preliminary study include the patient population, visualization of ARAs, quantification of spinal cord CTA images, blinded CT perfusion measurements, and the lack of CTA and MRI performed during follow-up. Current and future advances in different imaging techniques will be (and already are being) combined to exponentially increase the knowledge gained from these types of studies.

Conclusions

Visualization of the ASA in CCM is crucial for the ongoing discussion of spinal cord pathological ischemia, which influences the functional deficits of and the necessity for decompression. ASA occlusion was not

observed in CCM patients with spinal canal sagittal diameter compression of more than 80%. Pathological changes in hyperintensity on T2-weighted MR images in patients with CSM did not have an absolute correlation with ASA occlusion, but, ARA infarction may occur.

Disclaimer statements

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Declaration of interest None.

Conflicts of interest Authors have no conflicts of interest to declare.

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