CT angiography of anterior spinal artery in cervical spondylotic myelopathy

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Received: 21 January 2013 / Revised: 1 May 2013 / Accepted: 11 June 2013 / Published online: 18 June 2013
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Abstract

Purpose To identify anterior spinal artery (ASA) infarct or occlusion by CT angiography (CTA) in patients with cervical spondylotic myelopathy (CSM).

Methods Fourteen patients with CSM were performed CTA of ASA after admission. T2-weighted hyperintensity of MR image was compared with image of CTA of ASA.

Results All patients presented spinal canal sagittal diameter compression from 10 to 80 % and different T2-weighted hyperintensity of MR images. No ASA infarct or occlusion was found in CSM patients.

Conclusion ASA infarct or occlusion is not commonly seen in CSM patients with spinal canal sagittal diameter compression less than 80 %. Pathological changes about T2-weighted hyperintensity of MR image in CSM have no close correlation with ASA infarct.

Keywords Cervical spondylotic myelopathy · CT angiography · Anterior spinal artery

Introduction

Cervical spondylosis myelopathy (CSM) may acquire spinal cord physiological changes by cervical compression, which presented as the high signal intensity area of the spinal cord on T2-weighted magnetic resonance imaging (MRI) and the low signal intensity area on T1-weighted

images. It was reported that these intramedullary signal intensity changes are involved in edema, myelomalacia, gliosis, and inflammation, which have been suggested as necrosis and ischemia [1]. Spinal cord ischemia, or even infarct, is much less frequent than cerebral infarction, accounting for only 1 % of all strokes [2]. Most of them have been investigated on vascular surgery, occlusion of the vertebral arteries and vertebral artery dissection [3, 4], and only few of them on CSM. Spinal cord ischemia is usually detected by MRI findings, especially the high signal intensities on DWI and low ADC value findings [5, 6]. However, it is not effective in CSM because of cystic necrosis [7]. The anterior spinal arteries (ASA) supply the anterior horn and the anterior part of the lateral column on the left or right side at each level of spinal cord, which may be theoretically compressed by herniated disc and resulted in spinal cord ischemia in CSM. So we hypothesize that cervical compression may result in ASA infarct or occlusion in CSM. We also hypothesize that T2-weighted hyperintensity of MRI in CSM may have a close correlation with ASA infarct. The objective of this study was to identify ASA infarct or occlusion by CT angiography (CTA) in patients with CSM.

Materials and methods

Patients

Between February 2010 and December 2011, 95 patients of CSM were admitted to investigator group (Z. Z.). Among them, 14 patients were randomly admitted to a doctor in charge (H. W.) and chose to identify ASA infarct or occlusion by CTA. The clinical characteristics of the patients are summarized in Table 1. In these 14 patients

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(11 males and 3 females), the distribution of cervical level was from C3 to C7; the duration from onset until surgery ranged from 1 to 6 months; follow-up ranged from 10 to 16 months (mean, 12.5 months). All patients underwent plain cervical radiographs, CT scans, and 1.5-Tesla MRI. T1-weighted and T2-weighted MRIs were performed to define cord compression, spinal canal sagittal diameter compression proportion, and the extent of intramedullary signal intensity change. In all patients anterior decompression and fusion surgery were performed. Preoperative and postoperative neurological statuses were evaluated using the American Spinal Injury Association (ASIA) impairment classification and the motor score [8].

CT angiography of ASA

With the institutional review board approval of the research protocol, CTA of ASA was performed on the patients. One or two days after performed MRI, patients were scanned with 64-slice MDCT scanners (LightSpeed 64, GE Medical System, Milwaukee, WI, USA). All CT scans were obtained with 0.5 s rotation, 0.625 mm nominal detector widths, pitch 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 with a dose of 120–150 ml (350 mgI/ml) at a rate of 5 ml/s. The scan delay was determined by a preliminary 20-ml test injection at the level of basilar artery. All observations were made retrospectively by two neuroradiologists. To examine the ASA

transverse sections, multiplanar reformation (MPR), curved planar reformation (CPR) and thin-slab (2–4 mm) maximum intensity projection (MIP) 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. The ASA was identified by maintaining the MIP-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. Inter-rater reliability, in the form of consensual identification of ASA by two radiologists, was required.

Results

ASA infarct

The clinical data are summarized in Table 1. The ASA was visualized in all 14 patients. The course of the ASA extended from C1 to C7 in sagittal, coronal and transverse view. The ASA runs along the entire length of the anterior surface of the spinal cord. Anterior radiculomedullary arteries (ARAs), ASA branch which supply the cord throughout its length by anastomoses branching upward and downward, were not visualized for the angiography, because ARAs are very little or even no flow in two directions. The Adamkiewicz artery (AKA) was also visualized in most cases. No ASA infarct or occlusion was found in 14 CSM patients, even with some cases of 80 % spinal canal sagittal diameter compression and T2-weighted hyperintensity of MR image (Table 1).

Table 1 Clinical summary of 14 patients with CSM

Case no.	Sex/age	Level of compression	T2 cord hyperintensity	Location of T2 cord hyperintensity	Spinal canal sagittal diameter compression (%)	ASA infarct	Type of operation	ASIA on admission/at last follow-up/months
1	M/65	C4/5 C5/6	C4/5	Anterior horn	80	No	ACCF	D/D/10
2	M/44	C3/4 C4/5	None	None	80	No	ACCF	D/E/13
3	M/44	C4/5 C5/6	C4–C6	Central cord	70	No	ACCF	D/E/16
4	M/75	C4/5 C5/6	None	None	70	No	ACCF	D/E/10
5	F/60	C3/4 C4/5 C5/6	C3/4	Central cord	70	No	ACCF	D/E/13
6	M/81	C3/4 C4/5	None	None	50	No	ACCF	D/E/13
7	M/47	C5/6 C6/7	C5/6	Central cord	40	No	ACCF	D/E/16
8	M/71	C4/5 C5/6	None	None	30	No	ACDF	D/E/15
9	M/62	C4/5	C4/5	Central cord	30	No	ACDF	D/E/10
10	M/43	C4/5 C5/6	None	None	30	No	ACCF	D/E/10
11	M/41	C4/5 C5/6	None	None	30	No	ACDF	D/E/10
12	M/42	C3/4	None	None	10	No	ACDF	D/E/14
13	F/42	C5/6	None	None	10	No	ACDF	E/E/10
14	F/47	C4/5	None	None	10	No	ACDF	E/E/10

M male, *F* female, *C* cervical, *CCM* cervical compression myelopathy, *ACDF/ACCF* anterior cervical disctomy/corpectomy fusion

Neurological status

Preoperative and postoperative neurological statuses of the patients are given in Table 1. All CSM patients had performed anterior cervical disctomy/corpectomy fusion (ACDF/ACCF). Two patients without paraplegia did not encounter neurological complication. For 12 ASIA D patients, only one 65-year-old patient's neurological status presented no improvement for 10-month follow-up, the other eleven patients improved to ASIA E after over 10 months follow-up.

Illustrative cases (case 5) (Fig. 1)

A 60-year-old female was admitted with limbs weakness and numbness for 3 months. Neurological examination revealed that strength of limbs was 4/5 (ASIA D), Deep tendon reflexes were increased in all limbs, the Babinski sign was positive, sensations were almost normal. T2-weighted MR images demonstrated that the cervical spinal cord was compressed at the C3/4 (for almost 70 %), C4/5 and C5/6 intervertebral levels. Spinal cord hyperintensity was showing in the central area at C3–C4. The patient was diagnosed as CSM. CTA presented normal ASA at C3–C6. The patient was performed ACCF and neurological status was recovered to ASIA E after 13 months following up.

Discussion

The anterior spinal artery (ASA; diameter, 0.2–0.8 mm) runs along the entire length of the anterior surface of the spinal cord and distributes blood to the anterior two-thirds of the cord tissue by central and pial branches [9]. Numerous anterior radiculomedullary arteries (ARAs) supply the cord throughout its length by anastomoses, branching upward and downward. The posterior spinal arteries are usually paired, course on the posterolateral surface of the spinal cord along its entire length, and may occasionally be discontinuous. ARAs and posterior spinal arteries are less than 0.5 mm in diameter and even no flow in either direction [10], and at present, can only occasionally be depicted in vivo by catheter angiography. Thus, visualization of ASA is able to depict spinal cord blood supply in normal spinal cord and to some extent spinal cord pathological ischemia.

Catheter, CT and MR angiography are the ultimate imaging techniques for diagnosing, localizing, and classifying spinal vascular lesions. Despite its superior spatial resolution and image quality, catheter angiography has, however, several major drawbacks including invasive technique, ionizing radiation exposure, risk for major complications, and only be performed by experts [11, 12]. MR angiography benefits from strong background suppression

techniques are available, which allow the depiction of vessels smaller than the voxel size at acquisition [13]. However, it can not allow to visualize the cord and bone anatomy which is very important for spine and spinal cord in addition to spinal vessels injury compared. That is the reason why we chose CTA for visualization ASA and identification cord ischemia, although its disadvantages include inherent exposure to ionizing radiation and administration of a potentially nephrotoxic iodine contrast agent. In our series, the ASA was visualized in all 14 patients.

Myelopathy in cervical spondylosis includes edema, myelomalacia, gliosis, and inflammation, which have suggested as necrosis and ischemia [1]. ASA infarct was considered by some authors as one of very suggestive causes of traumatic spinal cord injury [14, 15], CSM [16, 17] and anterior spinal cord syndrome (ASCS) concomitant cervical spondylosis [18, 19]. The diagnosis of spinal cord infarction in these papers was confirmed on the basis of the history, clinical symptoms and MRI findings. However, no ASA infarct image was reported in the patient in CSM.

In our series, no ASA infarct was found in 14 CSM patients, even with cases of 80 % spinal canal sagittal diameter compression and T2-weighted hyperintensity of MR image. It indicates the spinal cord is easier compression compared with ASA in its anterior surface. It also indicates ASA infarct or occlusion maybe occur in CSM patients with over 80 % spinal canal sagittal diameter compressions. These negative findings are similar to our report that ASA rupture is not commonly seen in acute blunt cervical spinal cord injury [20], however, more patient population are need to identify. Although ischemia is a key factor and key contributor to the secondary pathogenesis for CSM [16], ARAs pathological injury including occluded and compressed, is possible reason for cord ischemia but not ASA occlusion. For visualization of ARAs, we failed through catheter, CT and MR angiography (data not show). Advancing imaging technique will definitely further improve the possibility to non-invasively visualize intradural spinal cord arteries and veins for spinal cord.

Although no ASA infarct or occlusion was found in CSM, the limitations of this preliminary study include visualization of ARAs, quantification of spinal cord CTA image, blinded CT perfusion measurements, patient population, and CTA and MRI taken 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.

Conclusion

Visualization of ASA to CSM is crucial for the ongoing discussion about spinal cord pathological ischemia which

Fig. 1 Case 5 a 60-year-old female with CSM at C3/4, C4/5 and C5/6. **a** Sagittal T2-weighted MR images showing spinal cord compression at the C3/4, C4/5 and C5/6 intervertebral levels. Spinal cord hyperintensity is showing in the central area at C3–C4. **b–d** Transverse T2-weighted MRI showing spinal cord compression at the C3/4 (**b**), C4/5 (**c**), C5/6 (**d**) and cord hyperintensity at C3/4. **e, f** Sagittal view (**e**) and coronal view (**f**) of CTA showing normal ASA at C3–C6. **g** Lateral radiograph view of ACDF of 3 days postoperation

influences the functional deficits and the necessity of decompression. ASA infarct or occlusion in CSM can be identified by CT angiography of ASA, and is not commonly seen in CSM patients with spinal canal sagittal diameter compression less than 80 %. Pathological changes about T2-weighted hyperintensity of MR image in patients with CSM have no close correlation with ASA infarct, but, maybe ARAs infarct.

Conflict of interest The authors declare no conflict of interest.

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