Magnetic Resonance Imaging Diagnostics of Vascular Myelopathies: from Basic Sequences to Promising Imaging Protocols

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Abstract

Magnetic resonance imaging (MRI) is the method of choice in diagnostics and differential diagnosis of spinal cord arterial infarction and venous insufficiency. However, imaging of vascular myelopathy is complicated by the lack of clear diagnostic criteria. Basic MRI sequences have low sensitivity at disease onset, and described MR patterns do not sufficiently increase imaging specificity for spinal cord ischemia, so imaging protocols are to be elaborated.

Diffusion-weighted imaging is a key additional sequence that allows establishing the ischemic nature of myelopathy.

Inclusion of spinal MR angiography in comprehensive MR examination allows visualization of aorta abnormalities, its large branches or spinal arteriovenous fistulas, so that they can be treated early.

We presented an optimal MRI protocol for patients with suspected ischemic spinal stroke. Promising high-tech MR sequences for visualization of vascular myelopathies were reviewed.

Keywords: *spinal cord; infarction; vascular myelopathy; diagnostics; magnetic resonance imaging; angiography*

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МРТ-диагностика сосудистых миелопатий: от базовых последовательностей к перспективным протоколам исследования

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Аннотация

Магнитно-резонансная томография (МРТ) является методом выбора в диагностике и дифференциальной диагностике артериального инфаркта спинного мозга и его венозной недостаточности. Однако визуализация сосудистой миелопатии осложнена отсутствием чётких диагностических критериев. При этом базовые последовательности МРТ обладают низкой чувствительностью в дебюте заболевания, а описанные МР-паттерны недостаточно повышают специфичность радиологической картины ишемии спинного мозга, что требует расширения протокола обследования.

Дополнительной последовательностью, позволяющей установить ишемическую природу миелопатии, в первую очередь является диффузионно-взвешенное изображение.

Включение в комплексное МР-обследование спинальной МР-ангиографии позволяет визуализировать патологию аорты, её крупных ветвей или спинальные артериовенозные фистулы, способствуя их ранней коррекции.

Представлен оптимальный технический протокол МР-исследования при подозрении на ишемический спинальный инсульт. Рассмотрена роль перспективных высокотехнологичных МР-последовательностей в визуализации сосудистой миелопатии.

Ключевые слова: *спинной мозг; инфаркт; сосудистая миелопатия; диагностика; магнитно-резонансная томография; ангиография*

Источник финансирования. Авторы заявляют об отсутствии внешних источников финансирования при проведении исследования.

Конфликт интересов. Авторы заявляют об отсутствии явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

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Current challenges in diagnostics of non-compression vascular myelopathies

A search for effective methods for diagnosing myelopathies to differentiate various mechanisms of spinal cord (SC) damage has been ongoing for decades. The imaging methods that were described and introduced in the second half of the 20th century, such as angiography, expanded our understanding of blood supply to the SC and vascular myelopathies [1]. Widespread introduction of magnetic resonance imaging (MRI) into clinical practice and use of various MRI modalities and sequences have revealed many differential diagnostic aspects of central nervous system damage [2–4]. However, most MRI protocols that are successfully used for diagnostics of brain lesions are not used for myelopathies due to the anatomy and physiology of the SC. At the same time, development of neuroimaging methodology and consistent accumulation of knowledge about the pathophysiology of non-compressive vascular myelopathies allows considering the capabilities of MR technologies from new perspective.

Supplementing the definition proposed by the American Heart Association [5], we can say that ischemic spinal stroke (acute SC infarction, subheading G95.1 "Vascular myelopathies" of ICD-10, subheadings 8B43 "Non-compressive vascular myelopathy" of ICD-11[1]) is a severe damage of the SC substance that is manifested by a sudden or rapidly increasing persistent neurological deficit, which is established based on pathological, imaging, or other objective evidence of SC focal ischemic injury in a defined vascular distribution if other intra- or extramedullar abnormalities are excluded. Therefore, the current model for diagnosing vascular myelopathy is based primarily on the rapid exclusion of other acute myelopathies that require immediate surgery (such as compressive myelopathy, SC tumors) or conservative treatment (such as myelitis) [6, 7]. In other words, "vascular myelopathy" is still a diagnosis of exclusion.

[1] Not used in Russia.

Unfortunately, we do not have exact epidemiology data available on prevalence of SC vascular disorders. Rare publications on this topic reported a few studies with small sample size and heterogeneous inclusion criteria. According to them, vascular myelopathies account for 5% to 8% of all acute myelopathies and 1% to 2% of all vascular neurological pathologies [8]. A.I. Qureshi et al. in a population-based study suggested that the incidence may vary from 1.6 to 7.2 per 100,000 person-years [9].

The lack of uniform diagnostic protocols may be a reason for a relatively small number of registered cases of vascular myelopathy. Therefore, we can assume that patients with SC infarction are often misdiagnosed. The lack of diagnostics criteria has also hampered overall progress in spinal angioneurology [10]. However, early diagnostics of SC infarction is crucial for identifying and eliminating potentially manageable causes, as well as early initiation of rehabilitation measures [11].

Both clinical and imaging signs of vascular myelopathy largely depend on the mechanisms and causes of SC hypoperfusion, which are associated with either reduced arterial blood supply or venous dysregulation [12].

Most common causes of arterial spinal cord infarction (SCI) include aortic disease (atherosclerosis, dissection, coarctation), vertebral artery disease, atherosclerosis and mechanical compression of the radicular arteries, and hypotension due to heart failure [13–15]. According to various authors, the proportion of idiopathic SCI varies from 7% to 50% [8, 16]. Venous congestion due to abnormal arteriovenous shunts in the presence of spinal dural or epidural arteriovenous fistulas is a major cause of spinal venous insufficiency [6].

Iatrogenic causes of SC infarction should be mentioned separately. According to N.L. Zalewski et al., aortic aneurysm repair is the most common procedure that is complicated

by SCI (49% of cases) [17]. Other surgical interventions and procedures on the aorta account for 15% of cases; surgical and manual procedures on the spine, endovascular surgery, epidural anesthesia, and blockade of the cervical or lumbar roots of the spinal cord account for 36%.

It should be also noted that the studies mainly included patients after aortic surgery. Therefore, the pathogenesis and natural history of spontaneous or non-iatrogenic SC infarction remain largely unknown [18].

Thus, neuroimaging is a pivotal diagnostics step because it allows narrowing differential search and establishing a specific diagnosis.

Aim. This review aimed at summarizing available information on the sequences and MRI markers that are used in diagnostics of vascular myelopathies.

Materials and methods

A review of publications indexed in PubMed, Scopus, and RSCI databases was carried out using the key words "spinal cord", "ischemia", "infarction", "non-compressive myelopathy", "MRI", "sequences", "DWI", "DTI", "spinal angiography", "vascular malformations".

MRI methods for visualization of ischemic spinal stroke

Due to physical limitations of X-ray diagnostic modalities and spatial resolution of computed tomography (CT), MRI has become the gold standard for visualization of vascular and other lesions of the spinal cord. Crucial for a good-quality spinal examination is the use of magnetic resonance scanners with optimal spatial resolution and signal-to-noise ratio (SNR) [19].

However, available spine and SC MR protocols are less standardized than those for brain imaging. Spinal "visualization barriers" are most challenging for sequence optimization. Artifacts caused by lung excursion and respiratory movements of the chest, CSF dynamics and aortic pulsation, swallowing, can lead to distortion of MR images to a certain extent [17–19].

In addition, 3T MR scanners for SC imaging compared with 1.5T ones are more prone to artifacts caused by field inhomogeneity [20].

However, there are sequences that are used for any spine MRI protocol. These sequences are fast spin echo T2 and spin echo T1; investigations should have sequences in both sagittal and axial planes in the field of view that does not exceed the area of interest and slice thickness of up to 3 mm, which helps identify the exact location of the lesion [21].

Widely used in spinal neurology, T2-weighted imaging (WI) with the spin-echo inversion-recovery method (Short-Tau Inversion Recovery, STIR) is manifested by SC hyperintensity, since it enhances abnormal processes due to the short time inversion value, which suppresses the signal from fat. However, T2-STIR sequence has a lower signal-to-noise ratio and greater susceptibility to the spinal "imaging barriers" mentioned above. Therefore, this sequence has high sensitivity but low specificity for spinal lesions [19, 20].

As with cerebral infarction, T2-weighted images are sensitive to the total volume of tissue fluid in the SC. Therefore, signal intensity change appears only once significant vasogenic edema of the infarcted tissue has developed. Therefore, the SC ischemia lesion is not seen on T2-weighted MR images at the onset of clinical symptoms [6, 22]. In a study by K. Nedeltchev et al., only 45% of patients with acute SC ischemia had signal intensity changes on T2-weighed MR images performed on day 1 of the onset of clinical symptoms [8].

M.M. Thurnher et al. assessed MRI findings in 23 patients with SCI: MR signals on T2-weighted MR images were not visualized in 3–4 hours but were seen in 8 hours after the onset of clinical symptoms [23]. According to S. Weidauer et al., slight signal change on T2-weighted MR images can be seen as early as in 3 hours but significant signal change is seen only in 12–24 hours after the onset of clinical symptoms [24]. Based on these observations, the authors suggested that MRI should be performed not earlier than 12 hours from the onset of clinical signs of myelopathy or later, since the infarction lesion is best visible in the subacute stage of its development [11].

Thus, basic MRI sequences have low sensitivity at the onset of SCI, when the accuracy of the differential diagnosis of myelopathy is especially important and critical. On the other hand, low sensitivity of T2-weighted images at the onset of SCI (i. e. no signal enhancement from the lesion and the development of edema) can be itself a useful differential diagnostic sign [16, 24].

However, T2 signal hyperintensity and SC edema are non-specific findings that can be also seen in patients with myelitis of various origin. In this context, diffusion MRI sequences are of key relevance [6, 23, 24].

Diffusion-weighted imaging (DWI) sequence, which has a high sensitivity to cerebral cytotoxic edema, is widely used in angioneurology to determine the most acute stage of cerebral infarction [2, 25]. High sensitivity of DWI for acute ischemic processes in the brain has been demonstrated in multiple studies. Diffusion and perfusion MRI is an important investigational tool in the acute phase of ischemic stroke, as it may differentiate reversible brain tissue damage from irreversible [25].

Experience with DWI in patients with SCI is limited. This is mainly related to technical difficulty with DWI in the spinal

canal. Pulse sequences specifically designed for the spine and spinal cord are not commonly available and require optimization [23].

For DWI of the SC, single echoplanar imaging is used with a maximum b-factor of 600–1000 sec/mm^2 and a slice thickness of 3 mm. In SCI patients, apparent diffusion coefficient varied from 0.23 to 0.9 × 10^3 mm^2/s (Figure 1) [23].

None of the studies established the exact time frames for the manifestation of diffusion changes in the SC substance. According to different authors, the average time between the onset of clinical symptoms and signal change on DWI is 3 to 4 hours [26]. In a study by M.M. Thurnher et al. in 23 SCI patients, persistent hyperintensity of the SC ischemia lesion on DWI was reported when MR images were obtained between 2 and 9 days [23]. In a study in 11 patients by N. Yadav et al., diffusion limitation was not seen during imaging on day 17 to 21 from the onset of clinical symptoms [27]. Therefore, larger studies are needed to establish the temporal threshold for diffusion changes on MRI in patients with SCI.

There is no doubt that DWI-MRI can help in identifying SCI at early stage. However, despite the use of techniques to reduce spatial distortion and improve the quality of the images, some technical challenges persist for DWI of the SC [26].

Quality of echoplanar DWI is reduced due to susceptibility artifacts and those related to spine magnetic field, which may result in false positive results [28]. Throughout the long echo sequence, phase errors will accumulate, resulting in spatial inconsistency in the reconstructed image. The longer the echo sequence and the higher the resolution, the more pronounced the distortions will be, which will be also amplified due to differences in the susceptibility of various spinal tissues (bones, intervertebral discs, cerebrospinal fluid, etc.) [16, 27].

Susceptibility distortions around the SC can lead to “pile-up” artifacts (hyperintensities) that can mimic cord infarction [23, 29]. To maintain sensitivity to ischemia, it was proposed to use higher b-factor values (> 600) [28].

Due to possible false-positive DWI results, it is recommended to supplement subsequent control MR studies with standard sequences (T2-WI, T2-STIR) [11].

According to M.X. Wang et al., diffusion weighted images at the spinal level are preferred in sagittal planes, as this view allows for larger coverage, shorter acquisition time, and less artifact [12].

Key MR features of vascular myeolpathy

Due to small size of the SC, its lesions are relatively small and visually indistinguishable on MRI, which definitely complicates the differential diagnosis of myelopathies. However, for some demyelinating disorders (multiple sclerosis, neuromyelitis optica spectrum disorder, acute disseminated encephalomyelitis), systemic inflammatory disorders (sarcoidosis), dysmetabolic disorders (vitamin B12 deficiency) and other processes, specific MR patterns have been described, which, together with the clinical and laboratory findings, allow establishing the correct diagnosis [30–33]. Accuracy of imaging can be improved by considering MR patterns typical for the acute and subacute stages of vascular myelopathy of arterial or venous origin.

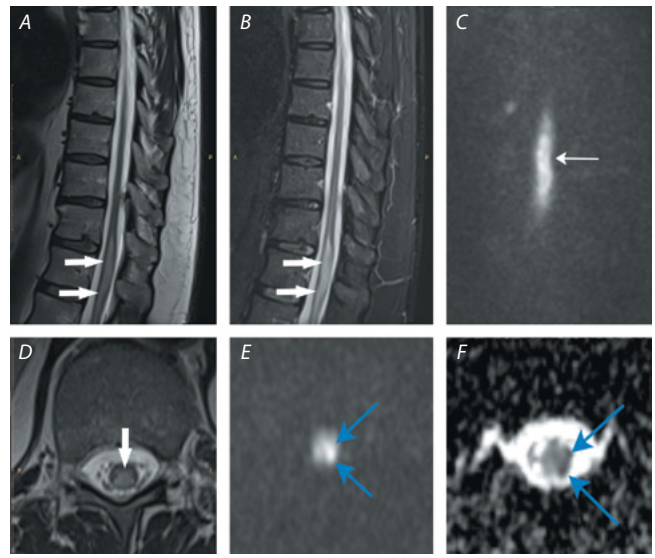

Fig. 1. MR image of patient P. with SC infarction at Th11–Th12.
A) T2-weighted image, sagittal plane: intramedullary hyperintense elongated lesion (white arrows) at Th11–Th12;
B) T2-STIR, sagittal plane: intramedullary hyperintense elongated lesion (white arrows) at Th11–Th12;
C) DWI, coronal plane: intramedullary hyperintense lesion of irregular shape at Th11–Th12 (white arrow), b = 800;
D) T2-weighted image, axial plane: hyperintense intramedullary lesion at Th11–Th12;
E) DWI, axial plane: intramedullary hyperintense lesion on the right at Th11–Th12 (blue arrows), b = 800;
F) DWI, axial plane, ADC map: diffusion restriction corresponding to the lesion on DWI (blue arrows), b = 800.

MR features of spinal cord arterial infarction

Arterial infarction of SC tends to occur in “watershed” areas where collateral circulation is poor, which is likely to explain delayed signal increase on T2-WI and T2-STIR in the acute phase of SCI [34]. However, a literature review demonstrated the lack of consensus on the most common location of such areas (lower cervical segments, middle, lower thoracic segments, conus medullaris).

Studies by A.A. Skoromets et al. [1], J. Novy et al. [18], S. Weidauer et al. [24, 31] illustrated various models (types) of arterial ischemia of the SC, which reflect the vascular territory involved: the area of the anterior spinal artery (ASA) limited by the anterior horns and adjacent white matter on

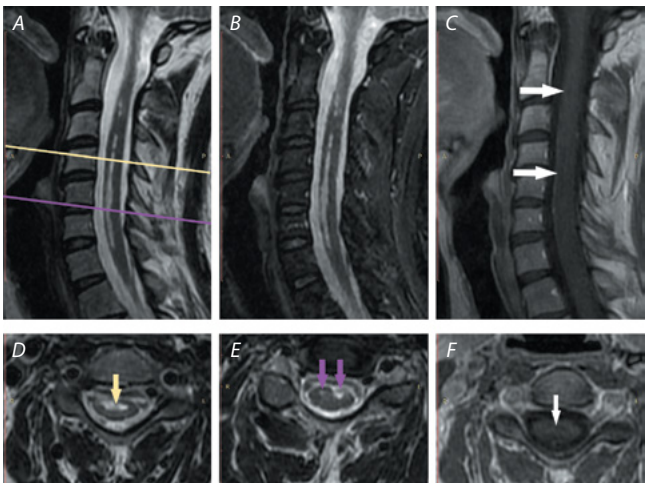

Fig. 2. MR image of patient A. with arterial infarction of spinal cord at C2–C3, C3–C7.
A) T2-weighed image, sagittal plane: multifocal intramedullary hyperintense elongated lesion at C2–C3, C3–C7; the light-yellow line indicates the section at C3 (*D*), the purple line indicates the slice at the C5–C6 intervertebral disc level (*E*);
B) T2-STIR, sagittal plane: intramedullary hyperintense elongated lesion (white arrows) at C2–C3, C3–C7;
C) T1-WI, sagittal plane: multifocal intramedullary hypointense elongated lesion at C2–C3, C3–C7 (white arrows);
D) T2-weighed image, axial plane: intramedullary hyperintense lesion at C3 occupying the gray matter area (hologrey phenomenon, light yellow arrow);
E) T2-weighed image, axial plane: intramedullary hyperintense lesions at the intervertebral disc at C5–C6 (snake eyes phenomenon, purple arrows);
F) T1-WI, axial plane: intramedullary hypointense lesion at C3 (white arrow).

both sides (anterior type); the area of the posterior spinal arteries limited by the posterior columns, adjacent areas of the lateral columns and part of the posterior horns (posterior type); rarer sulcocommissural, central and transverse types.

In some cases of reduced collateral blood supply to the SC, the ischemic lesion can involve only the gray matter of the anterior horns due to a greater sensitivity of motor neurons to anoxia and the presence of a "watershed" area between the pial and sulcocommissural arteries. Over time, this morphological pattern leads to a typical MRI pattern described as "owl eye" or "snake eye" appearance on axial T2-weighed images [27, 31]. On sagittal T2-weighed images, this hypointense lesion corresponds to a pin-like or pencil-like appearance and usually involves more than 2 spine segments (Figure 2) [19].

However, the "snake eye" appearance is not typical for SCI. Some other diseases that involve anterior horn motor neurons, such as motor neuron disease, spinal muscular atrophy [35], Hirayama disease [36], poliomyelitis and tick-borne encephalomyelitis, may also be associated with this MR sign [31–33].

It should be noted that the identification of specific MR patterns may have limitations in differential diagnosis, for example, with demyelinating lesions of the SC [34, 37]. In such cases, i. v. contrast enhanced brain MRI must be performed [24].

N.L. Zalewski et al. in their series of articles evaluated incidence of different MR patterns in 75 SCI patients [10, 17]. Signs of ischemia in the territory of the ASA with involvement of the anterior 2/3 of the SC and the "snake eye" or "pin" appearance was quite common (63–70% of cases). In almost half of the cases (46%), the hologrey phenomenon was observed on axial images, which corresponds to an increase in the T2 signal from the gray matter of the SC and, probably, reflects its greater vulnerability to hypoxia. Atypical T2-hyperintense anteromedial U- or V-shaped lesions were seen in 15–20% of cases. Lesions were often (68%) vertically extended (⩾ 3 vertebral segments), extending from the thoracic region to the conus and were accompanied by edema of the SC substance (25%). In some cases, a perifocal increase in T2-weighted signal from the ASA indicated the presence of a thrombus or slow blood flow [10, 17].

N. Yasuda et al. demonstrated different MR patterns depending on the location and extent of aortal surgery (thoracic/abdominal/aortic arch replacement) [38], thus showing the importance of neuroimaging monitoring in this population.

Besides medullary ones, we should note extramedullary MR signs of arterial ischemia of the SC, such as vertebral body infarction, which is manifested by abnormal high signal from the bone marrow on T2-weighed images. This phenomenon can be explained by proximal occlusion of the artery leading to the vertebral body, intervertebral disc, and spinal cord [26, 37, 39]. According to S. Weidauer, this MR sign can appear from 8 hours to several days or weeks after the onset of clinical symptoms [31]. Its prevalence ranges from 14% to 44%, and it is more common with lesions in the territory of the ASA [27].

MR features of spinal venous insufficiency

Spinal arteriovenous fistulas are the most common vascular malformations of the spine (70%). Among those, the most common are spinal dural arteriovenous fistulas (SDAVFs) of thoracolumbar localization, which account for 70–85% of cases with an annual incidence of 5–10 cases/1 million [32].

The fistula drains directly into the intradural radicular vein and then into the perimedullary venous plexus. Enlarged serpentine perimedullary veins ascend along the SC. Due to insufficient venous egress into the epidural plexus, venous congestion develops, followed by medullary edema, which later can lead to decreased arterial perfusion and subacute/chronic ischemia with very non-specific clinical manifestations such as gait disturbances (myelogenous intermittent claudication), "saddleback" hypoesthesia, pain in the lower extremities, and dysfunction of pelvic organs [1, 19, 40].

MR signs of SDAVF reflect the pathophysiological pattern of venous hypertension: key signs include swelling of the lower thoracic and caudal segments of the SC with hyperintensity on T2-WI and hypointensity on T1-WI together with tortuosity of the dilated perimedullary veins of the SC usually on its dorsal surface. These serpentine veins appear as linear areas of flow void phenomenon on T2-WI or contrast-enhancing structures on post-contrast T1-WI (Figure 3). In case of severe edema of the SC, the veins may not be visualized due to the mass effect [40]. Limited, often well-defined lesions with a hypointense rim due to hemosiderin deposits can also be detected, and they are characterized by a heterogeneous hyperintense intralesional signal depending on the stage of hemorrhage on T2-weighted images [31, 41].

N.L. Zalewski et al. demonstrated an additional MR pattern for SDAVF (termed the missing-piece sign), which is defined as an area of missing contrast enhancement in at least one or several SC segments amidst an intense area of contrast enhancement [42]. According to the authors, missing contrast enhancement in individual segments was likely to be related to the intact blood-cord barrier and better venous egress routes.

MR imaging of spinal cord vessels

Based on characteristics of the spinal cord ischemic lesion obtained using standard (T2, STIR) and diffusion MRI sequences, it should be considered whether the patient needs MR angiography of the CS.

In this context, selective spinal angiography (SSA) remains the diagnostic gold standard. SSA allows visualization of both normal angioarchitecture and various abnormalities of the arteries and veins of the SC. This method allows dynamic assessment of arterial inflows to the vascular myelopathy lesion, condition of the vessels directly in the lesion, and the features of the venous egress [1, 43, 44]. However, this invasive procedure can only be carried out in specialized centers by specialists in X-ray surgical diagnostics and treatment. Due to its technical difficulty and potential complications, the indication for SSA should be well considered and reserved for the cases where a vascular malformation is suspected (flow void phenomenon, i.e. a typical SC edema on T2, STIR), or secondly for preoperative imaging of the vascular anatomy or supply in cases of spinal or medullary tumors, or aortic disease [44].

Contrast spinal MR angiography (CMRA) can be an alternative option or a method preceding SSA that allows clarifying the origin, location, and volume of previously identified vascular change [45]. With comparable sensitivity to SSA, this method can be used for dynamic SC MR-angiography, including perioperative setting [44]. Angiodynamic analysis of the vascular myelopathy lesion is an advantage of CMRA. Dynamic CMRA is also useful in detecting dissection and thrombosis of large (aorta) and small (vertebral) arteries (Figure 4) [21, 45].

CMRA allows visualization of blood flow in the arterial, venous and delayed phases. Therefore, 3D and 4D CMRA is an effective technique for visualization of vascular malformations and arteriovenous fistulas, such as SDAVFs [45]. These sequences allow determining the exact location of the arteriovenous fistula, its interaction with other vascular structures, and the course of serpentine perimedullary vessels [33]. In a study by A. Lindenholz et al., CE-MRA correctly localized the SDAVF in 43 of the 53 cases (81%) [46].

In some cases, such as in patients with artificial pacemakers, other MR-incompatible devices, or severe claustrophobia, MRI cannot be performed for diagnostics of vascular myelopathy. These patients should have CT angiography of the aorta and its branches, which allows identifying atherosclerotic damage to the walls of the major vessels, the presence of local narrowings and dissections that impede the SC blood supply [12, 43, 44].

Promising approaches in MR imaging of vascular myelopathy

Analysis of intramedullary lesions is challenging if standard MR sequences are used. Determining the nature of myelopathy and distinguishing acute ischemic lesions from hyperacute and subacute ones remains challenging from the clinical and radiological points of view.

Use of ultra-high-field MRI scanners (⩾ 3 T) has an obvious advantage of more detailed imaging of small structures, which is especially valuable in visualization of the SC and its vessels for primary or preoperative diagnostics [20]. However, the choice of a 3 T MR scanner for SCI is controversial, as more detailed visualization of the lesion may be attenuated by the presence of the aforementioned “imaging barriers” [47].

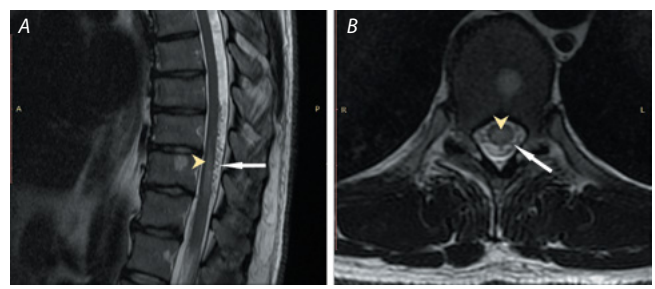

Fig. 3. MR image of patient V. with spinal venous insufficiency at Th9–Th11.
A) T2-WI, sagittal plane; *B)* T2-WI, axial plane: intramedullary hyperintense lesion of predominantly dorsal location at Th9–Th11 (light yellow arrow), hypointense dilated perimedullary vessels, mainly along the posterior surface of the SC at Th8–Th11 ("flow voids" phenomenon, white arrow).

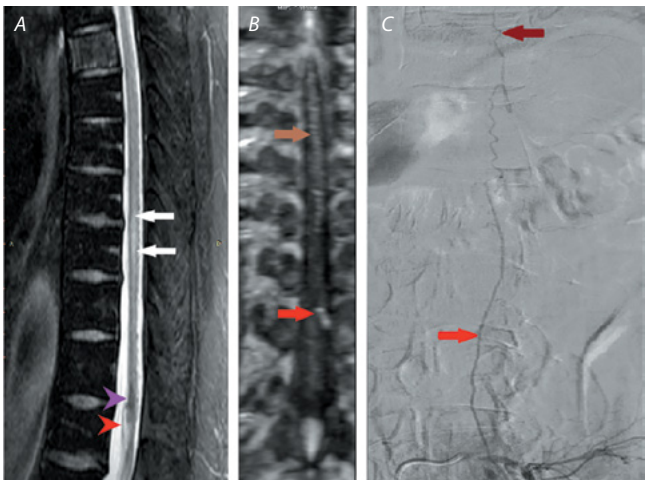

Fig. 4. MRI and angiography of patient S. with spinal venous insufficiency at Th8–Th10, Th11–Th12.
A) T2-SPAIR (SPectral Attenuated Inversion Recovery), sagittal plane: centromedullary hyperintense lesion at Th8–Th10 (white arrows), sharply hypointense lesion probably caused by hemosiderin deposition at Th11–Th12 (purple arrow), dilated straight vessel adjacent to the spinal cord conus (red arrow);
B) SMRA, coronal plane: under contrast enhancement along the entire posterior surface of the SC, a dilated convoluted vessel (brown arrow) is seen, into which the wide radicular vein flows from the left intervertebral foramen Th10–Th11 (red arrow);
C) selective spinal angiography of the left common lumbar artery (L4, L5): early arteriovenous discharge from the radiculospinal artery into the dilated vein of the cauda equina (red arrow) with contrasting of the spinal veins in the cranial direction (brown arrow).

Diffusion tensor imaging (DTI) is an additional sequence that can be performed at 1.5 and 3 T. This method allows non-invasive mapping of molecular diffusion in biological tissues. Similarly to DWI, DTI uses a b-factor range of 600–800 mm/sec, mainly because of the cranial-caudal direction of water molecule diffusion in the SC [21, 29, 48].

The relatively small transverse dimensions and elongated shape of the SC is a major diagnostic challenge for DTI in SCI imaging. The limited volume of fiber tracts naturally leads to the need for high spatial resolution [47].

The above-mentioned studies demonstrated the increasingly important diagnostic role of DTI owing to its ability to assess the white matter microstructural integrity via measurements of quantitative diffusion indices, such as cross-sectional area as an indicator of SC atrophy, fractional anisotropy to assess axonal integrity, magnetization transfer coefficient as an indicator of demyelination, and mean diffusion in the ischemic lesion and perifocal areas [49]. At 3T or higher field strengths, T2-weighted imaging of the SC provides high resolution and strong contrast between gray matter and white matter, allowing segmentation between these structures and calculation of their cross-sectional area (Figure 5) [50].

For the axial imaging of SC several authors suggested supplementing the MRI protocol with T2*-gradient recalled echo,

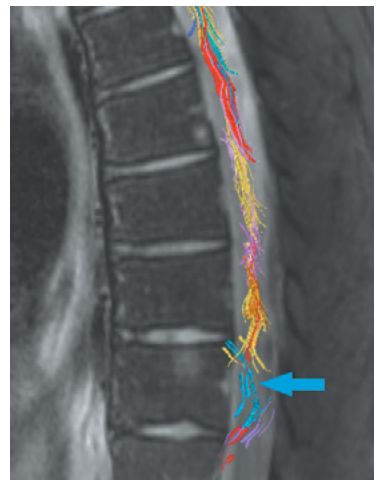

Fig. 5. 3D DTI color-coded tractography map of patient V. with spinal venous insufficiency at Th9–Th11.
Conducting fibers at Th9–Th11 are highlighted in blue, demonstrating abnormal spatial orientation of the fibers in the ischemic lesion (blue arrow), fractional anisotropy 0.46 ± 0.18, apparent diffusion coefficient 0.79 ± 0.15 × 10^3 mm^2/sec.

which has a high sensitivity to paramagnetic blood products (hemosiderin) [19].

Conclusion

Despite the lack of uniform diagnostic protocols, MRI is the method of choice for the diagnosis and differential diagnosis of vascular and other myelopathies. Diagnostic efficacy of MRI directly depends on spatial resolution and signal/noise ratio of the scanner.

Patterns described do not sufficiently increase the specificity of the radiological picture of the acute period of SC infarction, so the MR examination protocol should be elaborated. DWI is an additional sequence that allows establishing the ischemic nature of myelopathy. Average time to normalization of DWI signal is 2 to 3 weeks, which is essentially a "diagnostic window" for confirming/excluding the diagnosis and starting treatment.

If included in a comprehensive diagnostic protocol, dynamic angiographic methods, such as CMRA, allow visualization of thrombosis of the aorta and its large branches or SDAVF and other spinal arteriovenous fistulas, contributing to their early surgical correction.

Thus, the optimal technical protocol for MRI for 1.5 and 3 T scanners in patients with suspected arterial vascular myelopathy is the following [19, 21, 44]:
- Sagittal spin-echo T2-WI;
- Axial spin-echo T2-WI;
- Sagittal spin-echo T2 STIR;
- Sagittal and axial DWI (b = 600–800);
- Axial T2*-gradient recalled echo;

- Pre-contrast sagittal spin-echo T1-WI;
- 3D dynamic three-phase contrast CMRA;
- Post-contrast sagittal spin-echo T1-WI.

Slice thickness is 3 mm.

Differential diagnostic aspects of identifying vascular lesions necessitate comparison of sagittal and axial images of the SC. The advantage of the sagittal plane is greater coverage with shorter acquisition times. The axial plane allows visualizing the specific location of the lesion across the diameter of the SC and the symmetry of MR signal change in the case of ischemia in the territory of the ASA.

We should highlight the issue of introducing mandatory neuroimaging monitoring including diffusion and angiodynamic MR scanning modes, in particular, during surgery on the aorta. This approach seems to be extremely important, since it will allow timely preventive measures to be taken against potential myeloischemic disorders in this patient population.

Investigation of microstructural ischemic lesions of the SC with DTI is of great clinical interest. Assessing the integrity of the SC tracts under the influence of various causes of myeloischemia will facilitate the selection of an appropriate treatment. Additional studies to investigate capabilities of DTI with substantiated results will allow including this method in an expanded examination protocol.

In general, there are still many unresolved issues regarding the visualization of ischemic lesions of the SC. There are no generally accepted approaches and protocols for MRI diagnostics of vascular myelopathies, and differential diagnostics is challenging. The information presented in this review can be a guideline in routine clinical practice and a basis for further research and development of a unified approach to diagnosing vascular myelopathy in Russia.

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