Role of CISS MR sequence in detection of spinal dural arteriovenous fistula

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Introduction

Spinal dural arteriovenous fistulas (SDAVFs) are the most common spinal vascular malformations. They represent a specific type of spinal arteriovenous malformation, in which a small low-flow fistula (abnormal communication between the arteries and veins) is located within the dura of the spinal canal.1,2 The diagnosis and treatment of SDAVFs has received considerable attention in the literature over the last few years.1–6 In current clinical practice, patients with myelopathy are initially examined by magnetic resonance imaging (MRI). It is important to recognize this lesion on MRI images because surgical and endovascular treatments are relatively straightforward and associated with low morbidity and mortality.1,2,7 MRI findings reported in conjunction with SDAVF are cord swelling, increased T2 signal within the spinal cord, and parenchymal enhancement, none of which is specific.1–3 Although the additional MRI finding of flow voids in the subarachnoid space improves the diagnostic accuracy considerably, these are not always present.1,3,4

We report a case of non-compressive progressive myelopathy in which the constructive interference in steady state sequence (3D-CISS) helped in making the diagnosis, whereas standard MRI findings were non-specific.

Case report

A 40-year-old man presented with a history of progressive ascending weakness for 1 year and progressive ascending numbness of both lower limbs for 8 months duration. He had suffered from constipation and excessive straining during micturition for 6 months, and impotence for the last 2 months. At the time of admission, neurological examination revealed increased tone in both lower limbs. There was decreased power in the bilateral hip, knee and ankle flexor and extensor groups of muscles and ankle and knee jerks were exaggerated. Plantar reflexes were raised. A sensory examination showed 10% sensory loss below the level of T12, 10–30% between L1 and L3, 30–50% between L3 and L5, and 60% loss below L5.

MRI was performed on a 1.5 T superconductive unit (Sonata, Siemens, Erlangen, Germany) and revealed a swollen cord with increased T2 signal intensity in the central portion of the cord from T6–12 (Fig. 1(a)). The spinal cord was hypointense on the T1-weighted image (Fig. 1(b)). The periphery of the spinal cord was hypointense on the T2-weighted image (Fig. 1(c)). No flow voids were seen within spinal cord or in the intra-dural space. In the present case, gadolinium-enhanced MRI was not performed because of financial constraints.

The 3D-CISS sequence was performed (10.58/5.29/1 repetition time (TR)/echo time (TE)/excitations; flip angle 70°; matrix 256 £ 256; field of view 350 £ 210 mm; in-plane resolution 0.8 mm; acquisition time 4 min 30 s). A sagittal 32-mm slice with 42 partitions was used to cover the entire spinal canal. The phase-encoding direction was right to left. The raw data were reviewed on an independent console with multiplanar reconstruction (MPR) into the axial, coronal and sagittal planes. Sagittal and coronal reconstructed images revealed serpentine flow voids on the posterior aspect of cord in the dorsiolumbar region, suggesting spinal vascular malformation (Fig. 1(d) and (e)).

Spinal angiography revealed an SDAVF supplied by the radiculomeningeal branch of the right first lumbar artery, which was drained by medullary vein draining rostrally into the coronal venous plexus (Fig. 2(a)). The dilated venous plexus correlated with the flow voids seen on 3D-CISS sequence. The left T12 intercostal artery injection filled the artery of Adamkiewicz and the previous fistula at the level of L1 via retrocorporeal anastomosis (Fig. 2(b)).

Because of the danger of embolization of the anterior spinal artery, embolization of the SDAVF was not attempted. As a result surgical treatment was undertaken wherein the arterialized vein was coagulated and interrupted as it entered the dura. This produced partial collapse of engorged vessels of the coronal venous plexus and stasis of flow within them. The patient experienced progressive improvement in neurological function after surgery.
Figure 1  (a) Sagittal unenhanced T1-weighted (TR/TE/NEX-400/20/02) MR image shows spinal cord hypointensity from T6-12. (b) Sagittal FSE T2-weighted (TR/TE/NEX-4500/234/2) MR image shows central spinal cord hyperintensity and rim of peripheral hypointensity from T6-12. No intra-dural or intra-medullary flow voids are seen. (c) Axial FSE T2-weighted (4500/90) MR image shows peripheral hypointensity with increased spinal cord signal. (d) Sagittal CISS sequence shows flow voids in the intra-dural space. (e) Coronal CISS sequence shows flow voids in the intra-dural space.
Discussion

SDA VF s seem to represent an acquired abnormal communication between arterial and venous channels within the dura, and produce dilated, tortuous, and elongated veins on the cord surface resulting in venous hypertension and chronic venous congestion of the spinal cord.\(^1,2\) The ensuing spinal cord dysfunction is usually localized to the lower cord and conus.\(^1,2\) Prominent symptoms include para-paresis, local pain, sensory disturbance and bowel and bladder dysfunction.\(^1,2\)

Establishing the correct diagnosis early in the course of the disease, before irreversible neurological deficits occur, is a challenge for the neuroradiologist. Findings suggestive of an SDAVF using conventional spin-echo (SE) MRI have been well documented.\(^1-3\) These findings include mild enlargement of the cord, scalloping of cord contours, central hyperintensity of the cord on T2-weighted images, intra-dural serpentine and punctate low signal intensity (flow void) on T2-weighted images; and patchy, diffuse or serpentine gadolinium enhancement.\(^1-3\) Hurst and Grossman\(^8\) suggested peripheral hypointensity of the spinal cord on T2-weighted images, in the absence of spinal haemorrhage, as a reliable imaging sign of venous hypertensive myelopathy.

The most common findings on MRI are increased intra-medullary signal abnormality on T2-weighted images occurring in 95–100% cases.\(^2,3\) The most consistent sign of an SDAVF on conventional MRI is intra-dural perimedullary tortuous flow voids, which can be identified in only 35% of patients on T1-weighted images and 45% of patients on T2-weighted images.\(^3\) An imaging technique,

![Figure 2](image_url)

Figure 2  (a) Anteroposterior view of spinal angiogram shows early filling of pial venous drainage from the SDAVF supplied by the radiculomeningeal branch of the right first lumbar artery. (b) Anteroposterior view of spinal angiogram shows filling of the artery of Adamkiewicz and the SDAVF at the level of L1 via retrocorporeal anastomosis with the left T12 intercostal artery injection.
hydrocephalus and syringomyelia. MRI images may show enhancement of the spinal cord or serpiginous flow voids. Preferably non-invasive, which improves the detection of intra-dural perimedullary flow voids, will help in making an early diagnosis: post-gadolinium MRI images may show enhancement of the spinal cord or serpiginous flow voids.

The CISS is a true fast imaging sequence with steady-state free precession. Pulsatile cerebrospinal fluid (CSF) flow is minimized by acquiring the sequence with flow compensation applied over each TR cycle, rather than over each echo as in the case of conventional compensation techniques. Turbulent flow, however, is not suppressed, with phase dispersion resulting in signal loss. Two data sets are acquired successively with alternating and non-alternating radio frequency pulses, which are subsequently combined to produce a myelographic image with excellent CSF-to-cord contrast. CISS sequence is now routinely used for the diagnosis of CSF-rhinorrhea, cerebellopontine angle lesions, hydrocephalus and syringomyelia.

In the present case, vascular flow voids, which were not visualized on routine SE sequences, were visualized on CISS sequence for several possible reasons, as follows. (1) The 3D-CISS sequence can provide higher spatial resolution, which is essential for delineation of small structures such as vascular channels. The comparatively thick slices employed for two-dimensional Fourier transform SE and turbo SE (TSE) images (3–4 mm) result in partial volume averaging and decreased spatial resolution in comparison with the submillimetre capability of the three-dimensional Fourier CISS sequence. (2) The short TE employed in the CISS sequence results in limited signal loss from magnetic susceptibility effects, and the low flip angle reduces T1 weighting. (3) The 3D-CISS sequence provides better contrast between the spinal cord and dilated vascular channels. In general, the signal-to-noise ratio of three-dimensional gradient-echo images is lower than that of SE images because of the small voxel size and cross-excitation between adjacent imaging sections. However, the contrast-to-noise ratio of the 3D-CISS sequence was significantly higher than that of the SE sequences, which is probably due to the inherent heavily T2-weighted contrast induced by the 3D-CISS sequence itself. (4) The loss of signal from pulsatile CSF motion, an inherent problem with two-dimensional T2-weighted fast SE (FSE) techniques, is minimized by acquiring a free induction with steady-state precession (FISP) sequence with flow compensation applied over each TR cycle, rather than over each echo as in conventional compensation techniques. The well recognized flow voids, particularly in the dorsal thecal sac in the cervicothoracic region or in individuals with capacious thecal sacs, are therefore diminished. Turbulent flow, such as that seen in a complex hydrosyrinx, is not, however, suppressed using this technique, resulting in signal loss due to phase dispersion.

Detection of flow voids is not specific to spinal vascular malformations, as they have been described in association with paraganglioma and ependymomas. Flow voids and vascular enhancement greater than three vertebral levels in length are strongly associated with SDAVFs. Conversely, flow voids and enhancement less than three vertebral levels in length are strongly associated with absence of SDAVFs. The dominant medullary vein draining a fistula has been reported to show increased tortuosity on MR angiograms and may help to differentiate from normal spinal veins. However, in view of central cord T2-weighted hyperintensity, mild cord expansion and a progressive clinical history in a middle-aged or older patient, the diagnosis of SDAVF should be considered.

Several studies have used MR angiography to image a dilated venous plexus in the spine by phase contrast and three-dimensional time-of-flight or first-pass post-gadolinium techniques. The addition of MR angiography to standard imaging protocol does not significantly alter the sensitivity, specificity or accuracy of prediction of an SDAVF for a given observer. However, it does help improve localization of the vertebral level of the fistula, which potentially expedites subsequent IADSA. The CISS sequence can demonstrate the features of a spinal vascular abnormality without the use of contrast agents, and is a useful adjunct for localizing the nidus and possibly the site of fistulation. The present authors are unaware, however, of any published articles reporting detection of flow voids on 3D-CISS sequences when standard MRI failed to show flow voids. We believe that the 3D-CISS sequence could be added to the MR sequence armamentarium to detect SDAVF flow voids, which may not be seen on routine SE imaging. Nevertheless, spinal angiography remains the gold standard technique for assessing vascular malformations of the spine.

In conclusion, the 3D-CISS sequence should be employed during MR examination of patients with a strong clinical suspicion of vascular myelopathy and having cord signal alteration on SE sequences, without evidence of vascular flow voids. Additional studies are required to determine whether the addition of the 3D-CISS sequence to the standard imaging protocol will increase the sensitivity and specificity of conventional MR. The CISS sequence may also be explored to diagnose intra-cranial dural arteriovenous malformations.
References


