

## High Signal Intensity on T1-Weighted MR Image Related to Vacuum Cleft in the Intervertebral Disk: Clinical and Phantom Study<sup>1</sup>

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**Purpose:** To determine the possible mechanism by which an area of high signal intensity appears on T1-weighted MR images adjacent to a vacuum cleft in intervertebral disks.

**Materials and Methods:** We analyzed a total of 14 disks in nine patients in whom a vacuum cleft with T1-signal hyperintensity was observed. Lesions were present from T11-12 to L5-S1 using a 1.5-T whole-body imager, sagittal spine-echo T1-weighted and gradient-echo images (flip angle, 20° and 60°) were obtained. In order to identify the vacuum cleft, using plain radiographs in all patients and CT scans in two were also obtained. A 3% agar-gel block containing empty slits to form a magnetic susceptibility difference, a phantom was designed. The air spaces were 1.6 mm in thickness, 25 mm in width, and 20 to 25 mm in depth with 1.6-mm spacing.

**Results:** In all patients, vacuum clefts were confirmed by plain radiographs and CT scans. At the level containing air, T1-weighted images (both spin-echo and gradient-echo) showed a signal void resulting from the intervertebral disk vacuum cleft. A hyperintense band adjacent to the vacuum cleft was, however, observed. A gradient-echo image with a 60° flip angle showed a brighter signal intensity than one with a 20° angle. Our phantom study gave the same results.

**Conclusion:** The magnetic susceptibility artifact may be responsible for the T1-signal hyperintensity observed adjacent to the vacuum cleft in intervertebral disks. In addition, in order to generate signal hyperintensity, the desiccating disk material must contain a certain amount of water molecules.

**Index words :** Magnetic resonance (MR), artifact  
Spine, intervertebral disks  
Spine, MR

High signal intensity within the disk space on T1-weighted images is a unique and unusual feature of MR

imaging. Calcification of the disk is known to be responsible for this phenomenon, and protein contained in fluid within the disk space is another cause (1, 2). Within this space, vacuum cleft-associated high signal intensity was revealed by T1-weighted imaging, a phenomenon which could not be explained by the mechanism mentioned above.

It is well known that vacuum clefts in the disk space appear as signal voids on both T1 and T2-weighted im-

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ages (3). One recent study reported that a vacuum cleft can demonstrate T2-signal hyperintensity (4), though on T1-weighted images, no such hyperintensity has been reported.

Based on the observation that due to magnetic susceptibility, high signal intensity often occurs adjacent to the paranasal sinus, we hypothesized that T1-signal hyperintensity adjacent to a vacuum cleft in the intervertebral disk was due to the magnetic susceptibility artifact.

In this study, we aimed to determine whether this artifact was responsible for the high signal intensity observed adjacent to a vacuum cleft in the intervertebral disk, and to explain why this intensity did not appear constantly.

### Materials and Methods

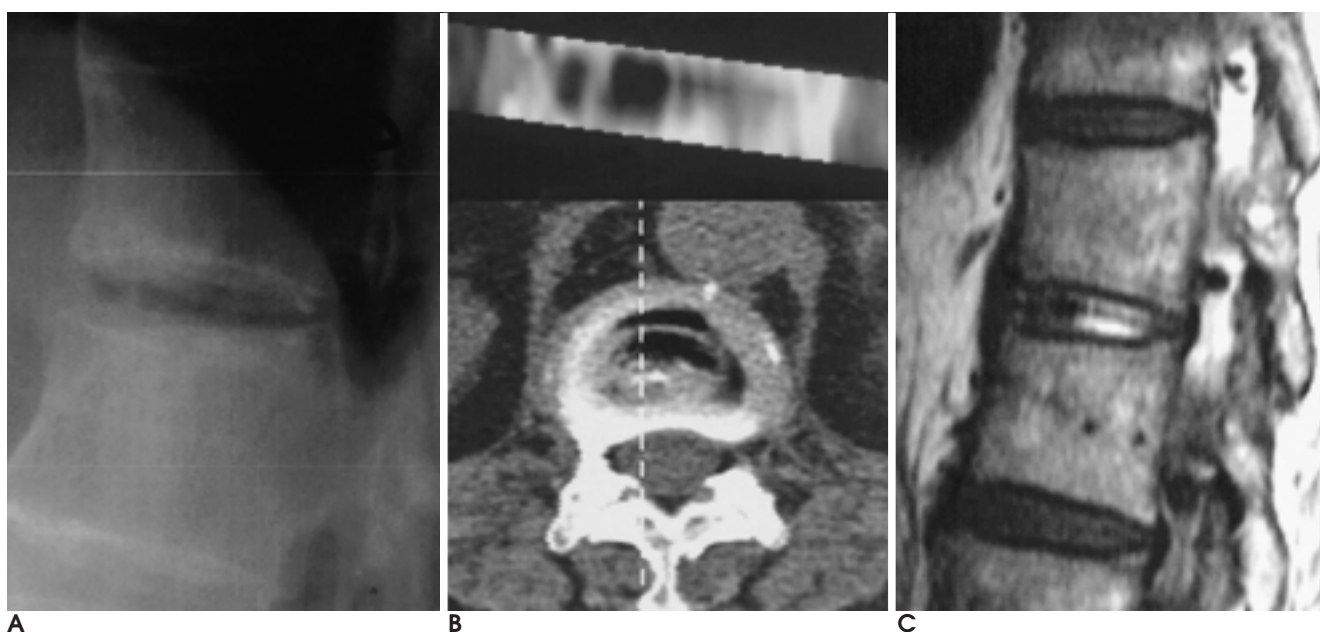
**Clinical study:** We analyzed the findings of nine patients (M: F = 4:5, aged 51 - 80 years) with vacuum clefts in whom T1-signal hyperintensity was observed. Among 23 disk spaces containing a vacuum cleft, 14 disks showed T1-signal hyperintensity adjacent to the cleft. Disk levels at which this hyperintensity was observed were T11 - 12 (n=2), T12 - L1 (n=3), L1 - L2 (n=2), L2 - 3 (n=2), L3 - 4 (n=1), L4 - 5 (n=2), and L5 - S1 (n=2).

A 1.5-T MR scanner (Signa, GE Medical System,

Milwaukee, U.S.A.) was used to obtain sagittal spin-echo T1-weighted (TR/TE: 500/25) and gradient-echo images (TR/TE: 80/4.4 - 12.1) with flip angles of 20° and 60°. (512 × 256 matrix, 4 mm slice thickness, 0.4 mm in-



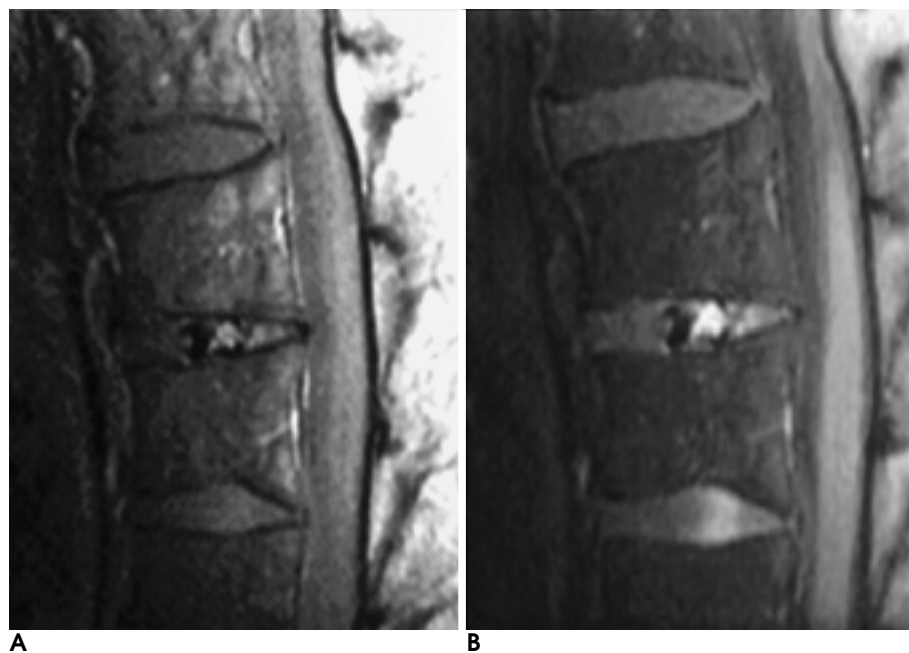
**Fig. 1.** Phantom was designed with 3% agar-gel block containing empty slits to form magnetic susceptibility difference. The air spaces were 1.6 mm in thickness, 25 mm in width, and 20 to 25 mm in depth with 1.6 mm spacing.



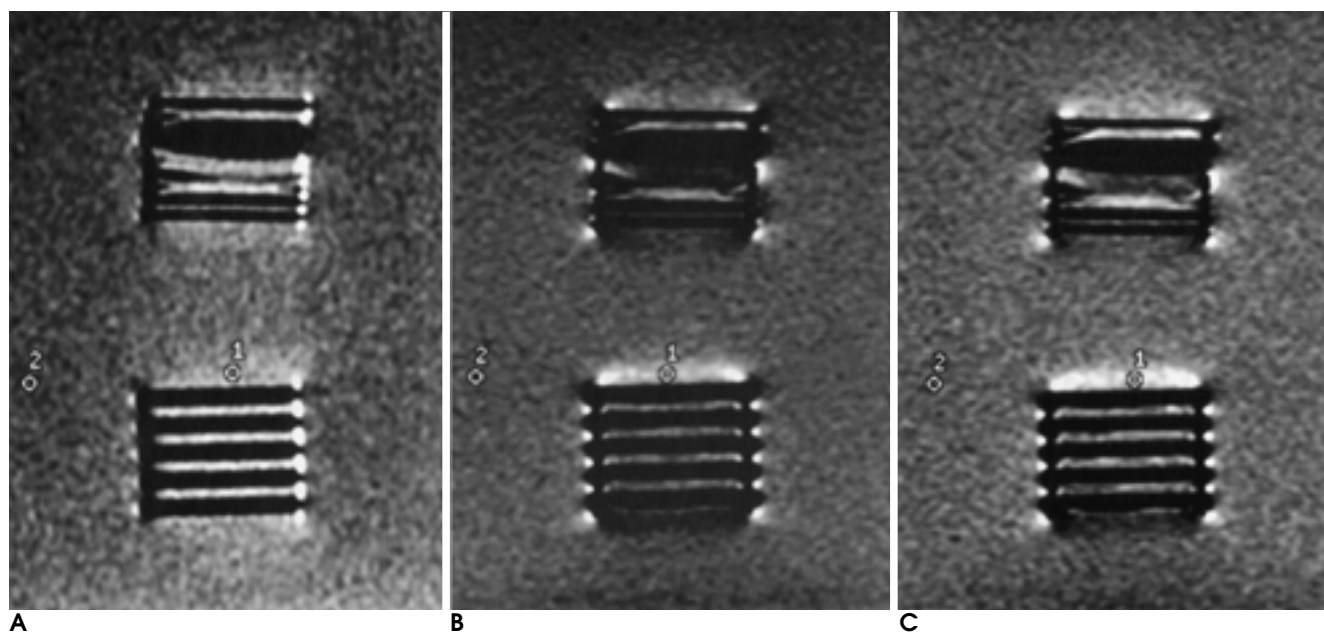
**Fig. 2. A.** Plain radiograph, and **B.** Axial CT scan with sagittal reconstruction image show vacuum cleft in the T12-L1 intervertebral disk. There is no demonstrable calcification in the disk. **C.** Spine-echo sagittal T1-weighted image shows bands of bright signal intensity adjacent to vacuum cleft in the T12-L1 intervertebral disk.

tersection gap, 10.67 kHz bandwidth, and 280 mm FOV). In order to maximize the magnetic susceptibility artifact, the frequency encoding gradient was perpendicular to the long axis of the vacuum cleft (5). In order to exclude the presence of calcification, the plain radiograph of all patients were reviewed, and for the same purpose, two also underwent CT scanning (HiSpeed Advantage, GE Medical System, Milwaukee, U.S.A.).

Phantom study: To simulate a solid tissue structure containing water, a 3% agar-gel block with slits containing air was devised. We thus achieved a magnetic susceptibility difference between air and agar-gel without any intervening material. The air spaces were 1.6 mm in thickness, 25 mm in width, and 20 to 25 mm in depth, with 1.6-mm spacing (Fig. 1). The same imaging protocols as in the clinical study were applied.



**Fig. 3.** **A.** Gradient-echo sagittal images with flip angle 20°, and **B.** flip angle 60° show patchy bright signal intensity adjacent to vacuum cleft in the T11-T12, more pronounced in the gradient-echo with flip angle 60°.



**Fig. 4.** **A.** Spin-echo T1-weighted image shows bands of bright signal intensity at the air-agar interface toward the frequency encoding direction. Signal voids are seen in the air spaces. **B.** Gradient-echo images with flip angle 20°, and **C.** flip angle 60° show brighter signal intensity than spin-echo T1-weighted image, especially gradient-echo with flip angle 60°. The signal intensity ratio of air-agar interface to background agar-gel is 1.24 in figure A, 1.45 in the figure B, and 1.51 in the figure C.

During each sequence, vacuum cleft-related signal hyperintensity was assessed in patients and in the phantom study in terms of intensity and direction. To quantitatively analyse the degree of high signal intensity, the region of interests (ROI) were drawn both in the high signal area adjacent to the vacuum cleft and in the underlying disk. The signal intensity ratio of the high signal area to the underlying disk was then determined by clinical study. The ROI method was also employed in the phantom study to estimate the signal intensity ratio of the high signal area at air-agar interface to background agar-gel.

## Results

**Clinical study:** In all patients, the levels of vacuum cleft were confirmed by plain radiographs, and in two patients by CT scans. Disc spaces contained no demonstrable calcification or fat (Figs. 2A, 2B).

At the level containing air, T1-weighted images showed a signal void resulting from the intervertebral disk vacuum cleft. In all patients, a hyperintense band adjacent to the vacuum cleft was, however, also found (Fig. 2C). Gradient-echo images with a 60° flip angle showed brighter signal intensity than those with a 20° angle (Fig. 3). The signal intensity ratio of the high signal area adjacent to the vacuum cleft to underlying disk was 2.04 on 20° flip angle images, and 3.41 on 60° images.

**Phantom study:** On all sequences, high signal intensity was observed at the air-agar interface. In contrast with the background signal from agar-gel, gradient-echo images, especially those with a 60° flip angle, showed brighter signal intensity than spin-echo T1-weighted images. The signal intensity ratio of the air-agar interface to background agar-gel was 1.24 on spin-echo T1-weighted images, 1.45 on 20° flip angle images and 1.51 on 60° images. High signal intensity occurred adjacent to the empty slits in all directions, being especially prominent in the frequency encoding direction. Signal voids, on the other hand, were observed in the empty slits containing air (Fig. 4).

## Discussion

Hyperintensity on T1-weighted images is observed in cases involving hemorrhage, or where material containing high levels of protein, fat, or calcification is present. An intervertebral disk is one of the least vascular structures in the body, and in the absence of trauma, is not

expected to be a site of hemorrhage. Similarly, proteinaceous material does not accumulate there. Degenerative disk changes, on the other hand, are common, and calcification of a degenerated disk is a well-known phenomenon (6). Major et al (1) reported that calcification of an intervertebral disk results in increased signal intensity on T1-weighted images, and calcification can therefore explain most of the hyperintense disks seen on T1-weighted images. Bangert et al (2), however, reported that approximately 28 - 48% of intervertebral disks which showed increased signal intensity on T1-weighted images did not demonstrate calcification on CT scans or plain radiographs. Their report therefore suggested that calcification alone could not fully explain the hyperintense signals seen on T1-weighted images. We also observed disks with a vacuum cleft which showed high signal intensity on T1-weighted images but no demonstrable calcification on plain radiographs or CT scans. We have already encountered some cases that due to the magnetic susceptibility artifact showed high signal intensity adjacent to paranasal sinus and mimicked an intraocular pathologic condition. We believe that this artifact can explain the high signal intensity shown by a disk with vacuum clefts.

The magnetic susceptibility artifact occurs when material becomes partially magnetized in the presence of an applied external magnetic field. When two separate tissues with great difference in magnetic susceptibility are placed within a uniform magnetic field, the susceptibility artifact occurs at an interface such as the tissue-air or tissue-fat plane, as found in paranasal sinuses, the skull base, and sellae. These differences in susceptibility lead to distortions in the local magnetic environment, causing altered spin relaxation properties which may generate signal loss, misregistration and accelerated T1 relaxation (7). Because there is more time for protons to dephase and a lack of refocusing of the 180° pulse, magnetic susceptibility artifacts are more prominent in images acquired with longer TE or a gradient-echo sequence with increased flip angle (8).

We hypothesized that if the magnetic susceptibility artifact was responsible for the high signal intensity seen on T1-weighted images adjacent to the vacuum cleft, high signal intensity should be more pronounced on gradient-echo images with increased flip angle and longer TE. In our study, all disks, as well as the phantom, showed a more pronounced high signal intensity on gradient-echo images, especially on those with a 60° flip angle. Moreover, our phantom study showed that a high

signal intensity occurred in all directions, being particularly prominent in the direction of the frequency encoding gradient. This finding is consistent with those reported by Frazzini et al (5).

In summary, our phantom study successfully simulated a vacuum cleft in the intervertebral disk space and demonstrated high signal intensity on T1-weighted images. These results thus suggested that the magnetic susceptibility difference might be responsible for the observed high signal intensity adjacent to the vacuum cleft.

On the other hand, we have encountered several cases in which vacuum cleft-related high signal intensity was not revealed by T1-weighted imaging, and have tried to explain why it is that not all disks with a vacuum cleft create high signal intensity on such images. We currently conjecture that in order to create high signal intensity on T1-weighted images, a desiccating disk must contain an adequate amount of free protons, in one which is completely desiccated, the absence of high signal intensity is due to a lack of protons.

In conclusion, the magnetic susceptibility artifact may be responsible for the high signal intensity seen on T1-

weighted images adjacent to a vacuum cleft in the intervertebral disks.

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