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Effects on the vertebral end-plate of uncomplicated lumbar discography: an MRI study

Received: 13 May 1997
Revised: 15 August 1997
Accepted: 14 October 1997

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Abstract The study aimed to identify and characterise changes occurring in the vertebral end-plate on MRI following uncomplicated lumbar discography. MRI was performed immediately before and within 2 h after uncomplicated lumbar discography in 20 consecutive patients undergoing the study as a precursor to possible spinal fusion. Of these, seven patients underwent a further MRI study at a mean of 72 days after discography (range 19–183 days). The MRI scans were assessed for the presence of any changes in the end-plates prior to and following discography. End-plate changes were identified in

eight patients (40%) prior to discography. No new changes in end-plate signal intensity (SI) were identified in either the immediate or delayed MRI studies. The study suggests that any changes occurring in the vertebral end-plate following discography should be considered due to infectious discitis. There is no support for the concept of chemical discitis, chemical irritation of bone or microfracture of subchondral trabeculae as a cause of pain at discography.

Key words Discography · Lumbar intervertebral disc · Back pain · Discitis · MRI

Introduction

Lumbar discography is considered a valid technique for the diagnosis of 'discogenic' low back pain [6]. Post-discography discitis is a recognised complication and is considered to be initiated by bacterial contamination [3], although it has been suggested that it is chemically induced [1, 12]. The overall incidence of this complication is quoted as 0.25% of patients [6]. We have recently seen a case of suspected post-discography discitis in which MRI demonstrated reduced signal intensity (SI) in the end-plates adjacent to the injected disc on T1-weighted spin echo (SE) sequences and increased SI on T2-weighted SE sequences. These features are similar to the type I end-plate changes seen in association with degenerative disc disease [11] and also the end-plate changes associated with vertebral osteomyelitis/infectious discitis [10]. However, the possibility that these changes were due

to the discography per se rather than any complication had to be considered, since the potential for end-plate changes secondary to uncomplicated discography has not been formally investigated. Gibson et al. [4] described the changes in lumbar intervertebral discs on MRI following chemonucleolysis and showed that transient type 1 end-plate changes were relatively common. In six control subjects who underwent discography only, the end-plate appearances were not commented upon. The major aim of the present study was therefore to identify whether uncomplicated lumbar discography was associated with end-plate changes identifiable by MRI.

It has also been suggested that the pain of discography may be due in part to a chemical effect on subchondral bone from the injection [3] or due to microtrabecular fracture from end-plate deflection [7]. The second aim of the study was to identify any evidence in support of these hypotheses.

Fig. 1A–C MRI of the lumbar spine. Discography performed at L2/3, L3/4, L4/5 and L5/S1. **A** T1-weighted spin echo (SE), sequence prior to discography (*left*) and immediately following discography (*right*). There is a mixture of type I and II end-plate changes at the L5/S1 level. No change is seen following discography. **B** Short tau inversion recovery (STIR) sequence prior to (*left*), and immediately following (*right*) discography. The L3/4, L4/5 and L5/S1 discs are degenerative. End-plate changes are seen at L5/S1 but are not altered by the disc injection. Note the increased disc signal in L3/4 following discography. **C** Lateral view lumbar discogram at the L3/4 level. There is early nuclear degeneration with a posterior tear and contrast leak

Fig. 2 MRI of the lumbar spine. STIR sequence prior to (*left*), immediately following (*centre*), and 60 days following (*right*) discography. There is multilevel disc degeneration but no end plate changes. Immediately following discography, increased signal intensity is seen in the L2/3 and L3/4 discs. The disc signal has returned to the pre-discogram state on the delayed MRI scan. No new end-plate changes are seen on the immediate or delayed post-discogram MR images. Discography was performed at the L2/3, L3/4, L4/5 and L5/S1 levels

Materials and methods

The study group comprised 20 consecutive patients who were referred for lumbar discography prior to lumbar fusion for chronic non-radicular low back pain. MRI was performed immediately prior to the discogram and within 2 h following discography. An early post-discography MRI examination was performed to investigate the possibility of pain reproduction at discography being due to chemical irritation of subchondral bone or microtrabecular fracture of subchondral bone. A delayed post-discogram MRI examination was performed to identify whether new end-plate changes could occur as a consequence of uncomplicated lumbar discography in the absence of discitis.

Each MRI study consisted of a sagittal T1-weighted SE sequence (TR 500 ms; TE 20 ms) and a sagittal short tau inversion recovery (STIR) sequence (TR 2250 ms; TE 30 ms; TI 160 ms). A STIR sequence was chosen because of its increased sensitivity to marrow oedema compared to a conventional T2-weighted SE sequence. All studies were performed on the same 1.5-T machine, with the exception of one delayed post-discogram MRI scan, which was performed on a 0.5-T scanner.

The T1 and STIR sequences were imaged at identical window settings, allowing direct comparison between the pre- and post-discography MRI scans. The pre-discography MRI scan was assessed for lumbar disc degeneration and the presence of vertebral end-plate changes as described by Modic et al. [11]. However, the

type II (fatty) end-plate change differed in that it demonstrated hypointensity on the STIR sequence due to the suppression of fat signal. Post-discography MRI scans were assessed for the presence of new end-plate changes. All MRI scans were assessed without knowledge of the discogram findings.

Immediately prior to discography, 1.5 g cefuroxime was given intravenously as prophylaxis against infection. Discography was performed via a conventional posterolateral injection technique using non-ionic contrast medium (iopamidol 240 mg I/ml). In the presence of a major total annular tear with leak, the pressure in the system is low, and theoretically there is no limit to the amount of contrast that can be injected. If the annulus is intact peripherally, then a high pressure system exists and only a limited amount of contrast can be injected. The injection was stopped either when there was a firm end-point and further contrast medium could not be injected, or when a positive pain response was obtained from the patient. Discograms were recorded as being either normal, degenerative and non-painful, degenerative and painful (concordant) or degenerative and painful (non-concordant). The discograms were reported without knowledge of the MRI findings.

Results

The study group comprised 13 men and 7 women with a mean age 38.7 years (range 23–59 years). Pre-discogram MRI scans revealed a total of 40 degenerative disc levels. End-plate changes were identified at eight levels (three at L4/5; five at L5/S1). These were type I changes in three

cases, type II changes in three cases and a combination of types I and II in two cases. Immediate post-discogram MRI scans revealed no new end-plate changes and no alteration of previous end-plate changes. In several patients, increased signal intensity was identified in the injected disc on the STIR sequence, consistent with increased water content (Fig. 1).

Seven patients returned for delayed post-discogram MRI scans, at a mean time interval of 72 days after discography (range 19–183 days). No new or altered end-plate changes were identified. Those discs that were of increased SI on the immediate post-discogram study had reverted to their pre-discogram state (Fig. 2).

Discography was performed at 61 disc levels, of which 18 were normal, 3 were degenerative but not painful, 33 were degenerative and reproduced the patient's back pain, while 4 injections produced non-concordant pain in degenerative discs. Three injections at L5/S1 were thought to be into the disc annulus rather than the nucleus.

At clinical review following lumbar discography, no patient suffered progressively worsening back pain to suggest the development of discitis.

Discussion

Vertebral end-plate changes identified on MRI and classified by Modic et al. [11] are seen in a variety of clinical situations and may be considered to represent a non-specific response of the cartilaginous end-plate and subchondral bone. Such changes have been noted in 50% of patients with degenerative disc disease [2]. Type I changes seen in active infectious discitis may convert to type II changes following appropriate antibiotic therapy [10]. Similarly, transient type I changes have been identified within 3 months following chemonucleolysis and type II changes 2 years after treatment [9]. The former change was considered to represent hyperaemia of the end-plate secondary to inflammation in the nucleus, whereas the latter change was thought to be secondary to degenerative spondylosis in the treated segment. We have also observed end-plate changes adjacent to Schmorl's nodes. In the acute stage they are of type I and may convert to type II as the subchondral bone heals.

Transient exacerbation of back pain is relatively common immediately following discography. It has been suggested that this may be due to a mechanical or chemical effect [3]. Post-discography discitis is characterised clinically by progressive worsening of back pain accompanied by an elevated ESR occurring several weeks after the procedure [5]. Planar bone scintigraphy (PBS) may be normal within 3 weeks of the onset of symptoms, but is typically positive at 5 weeks [5]. The ability of single photon emission computed tomography (SPECT) to demonstrate post-discography discitis has not been assessed, although it is to be expected that SPECT would be more sensitive

to end-plate abnormalities than PBS. However, SPECT could not be expected to demonstrate changes isolated to intervertebral discs prior to development of osteomyelitis within the subchondral bone. MRI has also been used to identify post-discography discitis and may show abnormalities related to the disc space alone, although the adjacent vertebral end-plate is usually also affected [5]. We suggest therefore that MRI is the investigation of choice for suspected post-discography discitis.

It has been suggested that such discitis is chemical or aseptic in nature based on the inability to isolate organisms from biopsy material in the majority of cases [1, 12]. However, the clinical experience of Fraser et al. [3], combined with experimental evidence in sheep, suggests that post-discography discitis is initiated by infection.

In the present study, new end-plate changes were not demonstrated by MRI in either the immediate post-discography phase or at various intervals after uncomplicated lumbar discography in the seven patients who underwent delayed MRI. The latter finding would suggest that the presence of 'inflammatory' type I end-plate changes following discography, combined with the appropriate clinical symptoms, should be taken as evidence of disc space infection and treated appropriately. This viewpoint would be strengthened by confirming such findings in a large group of patients. However, the observation of increased SI within injected discs in the absence of end-plate changes cannot be considered as evidence of discitis, since this occurred in six of the patients in this study and is easily explained by the increased water content within the degenerative disc due to the injection of water-soluble contrast medium. It is not possible to say for how long this change persists, since only two of these patients had delayed MRI scans (at 60 and 183 days respectively). In both cases, increased disc SI was not appreciable on the delayed scans.

There is no support in this study for the concept of 'chemical' irritation of bone as a pain source following discography, since chemical irritation of subchondral bone would be expected to produce bone oedema, manifest on MRI as reduced SI on T1-weighted SE sequences and increased SI on STIR sequences, i.e. type I end-plate changes. Although one may not expect such changes to be present immediately following discography, one could expect them to be present within 3 months of discography since this is the time interval within which such changes are identified following chemonucleolysis [9].

Another suggested cause of pain reproduction is increased pressure at the vertebral end-plates, since it has been shown experimentally that intradiscal injection can result in deflection of the end-plate [7]. These authors suggested that such end-plate deflection may stimulate pain by changes in intraosseous pressure or by microtraumatic fractures. This concept is distinct from the painful lumbar end-plate disruptions described by Hsu et al. [8]. In the present study, there is no support for the concept of

pain being due to microtrabecular fractures, since such trauma might be expected to produce haemorrhage or oedema within the adjacent bone marrow similar to the 'bone bruise' demonstrated following occult microscopic intraosseous fractures in the knee [13].

Conclusions

We have performed MRI on patients immediately prior to and following uncomplicated lumbar discography and

have not demonstrated changes in end-plate signal intensity. We have found no support for the concept of chemical irritation or microtrabecular fracture in subchondral bone as being a cause of discogenic pain provocation. We also suggest that the development of end-plate changes following discography should be considered as a sign of infectious discitis and should be treated appropriately.

References

1. Brodsky AE, Binder WF (1979) Lumbar discography: its value in diagnosis and treatment of lumbar disc lesions. *Spine* 4: 110–120
2. De Roos A, Kressel H, Spritzer C, Dalinka M (1987) MR imaging of marrow changes adjacent to end plates in degenerative lumbar disc disease. *Am J Roentgenol* 149: 531–534
3. Fraser RD, Osti OL, Vernon-Roberts B (1987) Discitis after discography. *J Bone Joint Surg [Br]* 69: 26–35
4. Gibson MJ, Buckley J, Mulholland RC, Worthington BS (1986) The changes in the intervertebral disc after chemonucleolysis demonstrated by magnetic resonance imaging. *J Bone Joint Surg [Br]* 68: 719–723
5. Guyer RD, Collier R, Stith WJ, Ohnmeiss DD, Hochschuler SH, Rashbaum RF, Regan JJ (1988) Discitis after discography. *Spine* 13: 1352–1354
6. Guyer RD, Ohnmeiss DD (1995) Contemporary concepts in spine care: lumbar discography. Position statement from the North American Spine Society Diagnostic and Therapeutic Committee. *Spine* 20: 2048–2059
7. Heggeness MH, Doherty BJ (1993) Discography causes end-plate deflection. *Spine* 18: 1050–1053
8. Hsu KY, Zucherman JF, Derby R, White AH, Goldthwaite N, Wynne G (1988) Painful lumbar end-plate disruptions: a significant discographic injury. *Spine* 13: 76–78
9. Kato F, Ando T, Kawakami N, Matsu K, Iwata H (1993) The increased signal intensity at the vertebral body endplates after chemonucleolysis demonstrated by magnetic resonance imaging. *Spine* 18: 2276–2281
10. Kramer J, Stiglbauer R, Wimberger D, Imhof H (1992) MRI of spondylitis (abstract). *Bildgebung* 59: 147–151
11. Modic MT, Steinberg RM, Ross JS, Masaryk TJ, Carter JR (1988) Degenerative disc disease: assessment of changes in vertebral body marrow with MR imaging. *Radiology* 166: 193–199
12. McCulloch JA (1977) Chemonucleolysis. *J Bone Joint Surg (Br)* 59: 45–52
13. Yao L, Lee JK (1988) Occult intraosseous fracture: detection with MR imaging. *Radiology* 167: 749–751