LETTER TO THE EDITOR

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OXFORD

Increased Nuclear T2 Signal Intensity and Improved Function and Pain in a Patient One Year After an Intradiscal Platelet–Rich Plasma Injection

Dear Editor,

Rich in fibrin, platelets, and anabolic growth factors that play central roles in IVD homeostasis, platelet-rich plasma (PRP) has been demonstrated in both a prospective cohort study [1] and a double-blind randomized controlled trial (DBRCT) to have therapeutic value for discogenic pain [2]. However, the relationship between patient outcomes and radiological changes remains incompletely understood in the context of this intervention [1–3]. We present here a case of recalcitrant two-level DDD that was effectively managed with intradiscal PRP, evidenced by long-term clinical follow-up and positive magnetic resonance imaging (MRI) changes.

A 42-year-old male presented to our clinic with a chief complaint of axial low back pain persisting for one year and ranging in severity from 3/10 to 7/10. He stated that his pain was exacerbated after running and standing for prolonged periods of time but sitting did not provoke his symptoms. He also complained of morning stiffness, which would improve as the day progressed. His pain was not associated with radiculopathy, paresthesia, or constitutional symptoms (fever, chills, weight loss). Aside from an arthroscopic removal of a loose body from his left hip 13 years prior, his past medical history was unremarkable. Upon clinical examination, the patient demonstrated normal stance, gait, posture, toe and heel walk, and squat. His range of lumbar spine motion was painful with extremes of extension, while flexion was full and did not reproduce pain. Hip range of motion was full and pain free. His neurologic examination revealed no focal motor or sensory deficits. His deep tendon reflexes were physiologic and symmetric. He exhibited no dural tension signs.

Radiographs showed moderate to severe disc space narrowing at L5–S1 with associated retrolisthesis and traction spurs, but there were no other abnormalities. A subsequent MRI revealed disc degeneration along with a crescentic fissure in the outer annulus adjacent to the left neural foramen, reduced T2 nuclear signal intensity, and obscuration of the normal horizontal intranuclear cleft at L4–L5. At L5–S1, disc degeneration was also present along with moderate decreased disc height, reduced T2 nuclear signal intensity, obscuration of the normal horizontal intranuclear cleft, and mild/moderate endplate degeneration associated with type I Modic changes. A distinct annular fissure was not detected at L5–S1 (Figure 1A).

With consideration of clinical and radiological findings, it was decided between the treating physician and patient to proceed with a caudal epidural steroid/lidocaine injection combined with a spine rehabilitation regimen as an initial course of treatment. The patient reported pain relief during the anesthetic phase of the injection. However, one month after the caudal epidural steroid injection and physical therapy, the patient reported only a transient improvement of his symptoms.

The patient then underwent a fluoroscopically guided, contrast-enhanced L4–L5 and L5–S1 discography for confirmation of pain origin. Discography is invasive and has the potential to be associated with negative effects on intervertebral discs [4]. Thus, discography was limited only to the suspected symptomatic levels and did not include a control disc. Using a double needle technique and lateral extrapedicular approach, 25-gauge needles were advanced through 20-gauge introducer needles into the midposition of the L4–L5 and L5–S1 discs. Proper needle placement was confirmed with anteroposterior and lateral fluoroscopic projections. After contrast was instilled, a grade III–IV annular fissure at L4–L5 and grade IV annular fissure at L5–S1 were identified, with concordant pain at both levels. After considering these findings, it was then decided to inject, into each disc, 1.5 cc of autologous platelet-rich plasma graft (magnitude of concentration by volume of whole blood: 20x) prepared using the Arteriocyte Magellan Autologous Platelet Separator (Hopkinton, MA, USA). The patient tolerated the discogram and concomitant PRP injections well without any adverse reactions other than a temporary increase in pain and was discharged in stable condition following the procedure.

Six weeks after the discogram and PRP injections, the patient returned for a follow-up visit where he demonstrated improvements in pain and range of motion. At a one year, the patient indicated a significant improvement in his low back pain and a return to participation in several athletic activities including running. A follow-up MRI at one year utilizing imaging parameters/sequences consistent with the pre-injection MRI revealed increased T2 nuclear signal intensity and a normal horizontal intranuclear cleft at L4–5, but no change in the crescentic fissure in the outer annulus adjacent to the left neural foramen. At L5–S1, there was increased T2 nuclear signal intensity and a reduction of type I Modic changes compared with the pre-injection MRI (Figure 1B).

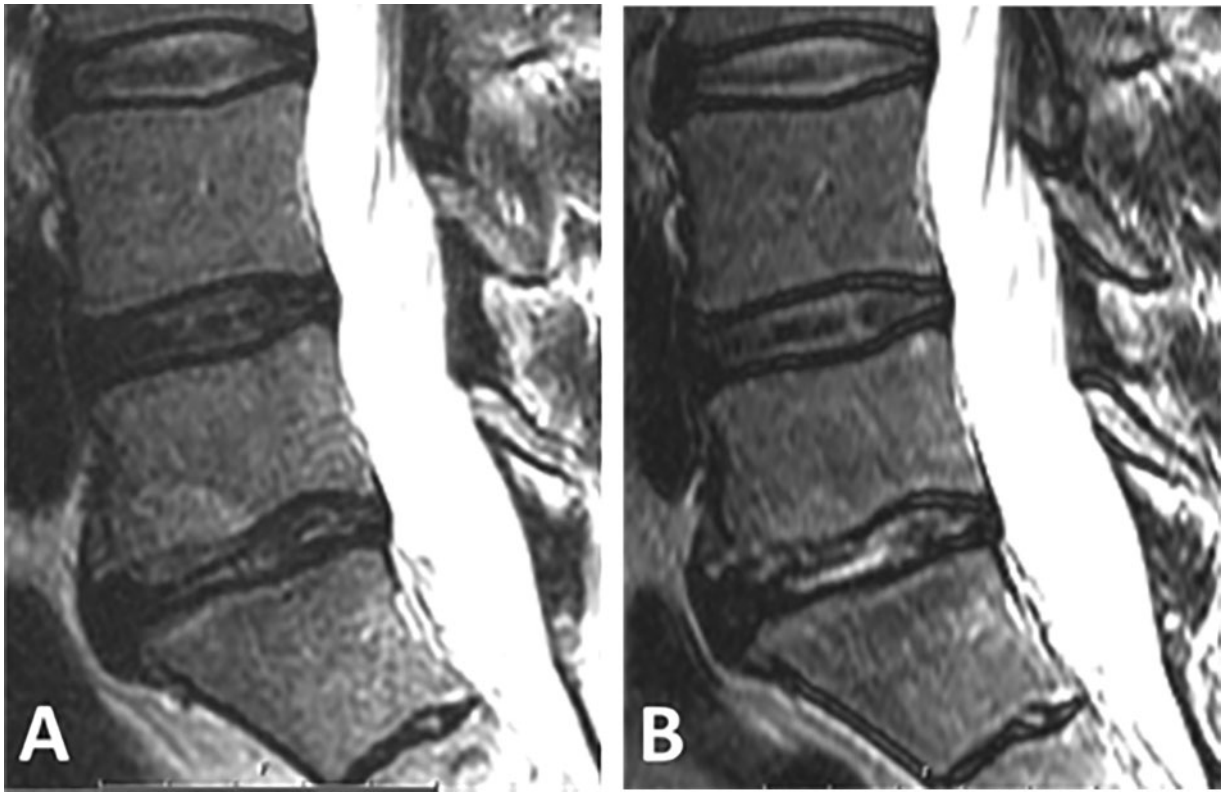

Figure 1 Sagittal MRIs depicting L3-4 through L5-S1 intervertebral discs prior to caudal epidural and intradiscal PRP injections (a) and one year post-PRP injections (b).

The negative correlation between the progression of DDD and T2 signal intensity is well established [5,6]. Healthy IVDs are associated with high T2 signal intensity, while degenerative IVDs exhibit decreasing T2 signal intensity over time as the discs dehydrate [7–9]. While investigating diurnal changes in nuclear T2 signal intensity, Karakida et al. found that degenerated discs had lower signal intensity compared with normal discs at all time points studied [10]. Additionally as DDD progresses, the demarcation between the annulus fibrosus [AF] and nucleus pulposus [NP] on MR imaging is eventually lost [11]. While several studies have reported degenerative changes in asymptomatic volunteers, a recent meta-analysis demonstrated that MR imaging evidence of disc degeneration is more prevalent in adults age 50 years or younger with back pain than asymptomatic individuals [12].

While it is our clinical impression that the positive MRI changes were directly attributable to the PRP injection, other possible causes of increased nuclear signal intensity include disc space infection and diffusion of fluid into vacuum discs following prolonged supine positioning. An imaging study of 44 IVDs with confirmed spondylodiscitis found that 93% exhibited hyper or "fluid-equivalent" nuclear signal intensity on T2 MR imaging [13]. In a clinical study of 31 discs affected by vacuum phenomena, 81% exhibited progressive replacement of the vacuum content with hyperintense fluid after prolonged MR imaging in a supine position [14]. Given the favorable clinical course, pronounced intranuclear cleft at L4–L5, and reversal of Modic endplate changes at L5–S1, discitis can be considered an unlikely cause of this patient's increased nuclear signal intensity.

To our knowledge, this is the first reported case of a degenerative disc exhibiting increased nuclear T2 signal intensity after intradiscal PRP. While ex vivo analysis would be necessary for true confirmation, the clinical and radiological findings described above strongly suggest that this patient's degenerated L4–L5 and L5–S1 discs exhibited increased T2 signal changes after intradiscal injection of PRP. Well-designed prospective investigations are necessary to further elucidate the relationship between clinical outcomes and MRI changes of degenerated IVDs following this treatment.

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OXFORD

Cervical Spinal Cord Stimulation with Concomitant Serotonin Norepinephrine Reuptake Inhibitor Therapy Leading to the Serotonin Syndrome

Dear Editor,

For the past almost 50 years, a form of therapeutic neuromodulation known as neurostimulation of the central and peripheral nervous system has been utilized in the management of various painful conditions. Spinal cord stimulation (SCS), in particular, has been reported as an effective treatment modality for complex regional pain syndrome (CRPS) [1]. One of the several proposed mechanisms of the antinociceptive effect of SCS is increased release of serotonin in the spinal dorsal horn and activation of serotonergic descending pathways [2,3]. In the presence of existing serotonin norepinephrine reuptake inhibitor (SNRI) pharmacotherapy, could cervical SCS lead to development of the serotonin syndrome (SS)?

We describe a case of SS in a patient who received cervical SCS and SNRI for CRPS of the upper limb. A 25-year-old male with no previous medical history suffered a construction site soft tissue injury to his left forearm and hand. Within months, despite conservative therapy, he developed increasingly worsening pain, hyperalgesia, allodynia, and decreased range of motion.