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Sequential Changes of Traumatic Vertebral Compression Fracture on MR Imaging

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The purpose of this study was to evaluate the sequential signal intensity changes in post-traumatic vertebral compression fractures of varying ages. Sixty-six patients with 115 post-traumatic vertebral compression fractures underwent MR imaging. The ages of fractures at the time of MR images ranged from 1 day to 6 years. Sequential follow-up MR imagings were obtained in 4 patients for 2 years after initial MR examination.

The fracture sites in all 52 fractures with traumatic events less than 3 months prior were hypointense on T1-weighted images and hyperintense on T2-weighted images(type I). A type I fracture could be subdivided into 3 patterns depending on its morphologic appearance: diffuse(type Ia); patchy(type Ib); and bandlike(type Ic). In 12 fractures of 3 to 5 months after trauma, six showed focal hypointensity(type II) in all pulse sequences, and six showed isointensity(type IV). Four of 51 fractures with trauma over 5 months showed focal hyperintensity on T1-weighted images and isointensity on T2-weighted images(type III); and the remaining 47 fractures showed isointensity on all sequences(type IV). In conclusion, MR imaging is useful in predicting the age of known traumatic compression fractures, so familarity with these sequential MR findings would be helpful in distinguishing benign from malignant fractures.

Key Words: *MR imaging, Spine, Compression fracture, Trauma*

INTRODUCTION

The distinction of pathologic fracture from non-traumatic collapse or posttraumatic fracture may not be possible on MR imaging(Baker et al., 1990). A vertebral compression fracture without obvious radiologic evidence of malignancy in an elderly patient may be troublesome in the differential diagnosis when the causes are concerned. It is, therefore, important to be aware of the various signal intensity characteristics in post-traumatic compression fractures. However, to our knowledge, chronologic MR studies of traumatic vertebral compression fractures have been limited(Bennett, 1966; Kim et al., 1979), and prior studies have focused on differentiating benign from pathologic compression fractures on MR imaging(Bennett, 1966; Kim et al., 1979; Baker et al., 1990). The purpose of

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this study was to evaluate the sequential signal intensity changes of traumatic vertebral compression fractures according to their ages.

MATERIALS AND METHODS

We retrospectively studied 66 patients with 115 post-traumatic vertebral compression fractures. Among these, 24 patients had multiple compression fractures. There were 37 males and 29 females, ranging in age from 21 to 76 years(mean, 42 years). The ages of their fractures at the time of MR imaging varied from 1 day to 6 years : less than 3 months in 31 patients(52 compression fractures), 3 to 5 months in 7(12), and more than 5 months in 28(51). None of the patients had any known history of primary neoplasm. Seventeen patients underwent surgery. We also excluded patients whose previous trauma date was uncertain. A diagnosis of traumatic fracture was made when there was a well-documented history of trauma and an improvement on the clinical and radiologic follow-up studies for 2 years. Four patients underwent follow-up MR imaging for 2 years following initial examination : i.e., 3 MR examinations in two patients and 2 in the other two.

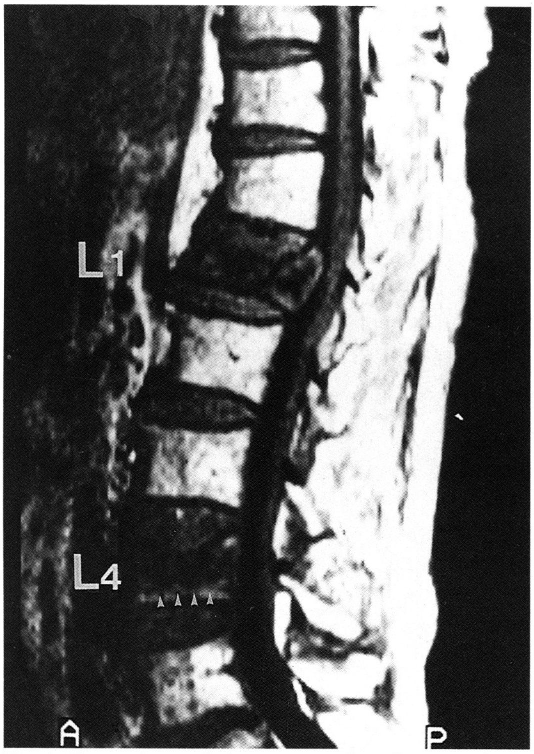


Fig. 1. Types Ia and Ic compression fractures in a 51-year-old man with a history of trauma 1 month prior. T1-weighted MR image shows diffuse hypointensity(type Ia) in L1 and band-like hypointensity(type Ic) compression fracture in L4(arrow heads).

MR imaging of the spine was performed on a 0.5-T superconducting magnet(Gyroscan T5, Philips, Netherlands). Sagittal and axial images were obtained with T1-weighted images(T1WI) (TR/TE, 450/20), proton-and T2-weighted images(T2WI) (TR/TE, 1800/25,90). Section parameters were 4mm thickness with a 2mm interslice gap, 256X256 aquisition matrix, 350mm field of view and two excitations were used in all sequences.

MR images were evaluated for the signal intensity patterns of compression fractures. Signal intensities were described as hypointense, hyperintense or isointense, as compared to the signal intensity of normal vertebral marrow in the same patient. The signal intensities of vertebral compression fractures on MR imaging were arbitrarily classified into 4 types. Type I was characterized by hypointensity on T1WI with hyperintensity on T2WI throughout the fracture sites. Depending on the morphologic appearances of signal intensity abnormalities, type I could be subdivided as diffuse in form(type Ia) (Fig. 1), patchy and focal involvement with preservation of peripheral portion (type Ib) (Fig. 2), or a bandlike configuration parallelling the end plate(type Ic) (Fig. 1). Type II was focally hypointense in the compressed vertebral body on all pulse sequences(Fig. 3A, B). Type III was hyperintense on T1WI(Fig. 3C) and isointense on T2WI. Type IV was isointense on all sequences.

RESULTS

The signal intensities at the fracture sites had changed as time passed(Table 1). All 52 fractures

Table 1. MR Signal Intensity Patterns of Traumatic Spines Acording to Fracture Age

	fracture age		
MR pattern	0-3m	3-5m	5m-6yrs
Type I	52		
Type II		6	
Type III			4
Type IV		6	47
Total	52	12	51*

* Half of these cases had a fracture age of over 1 year.

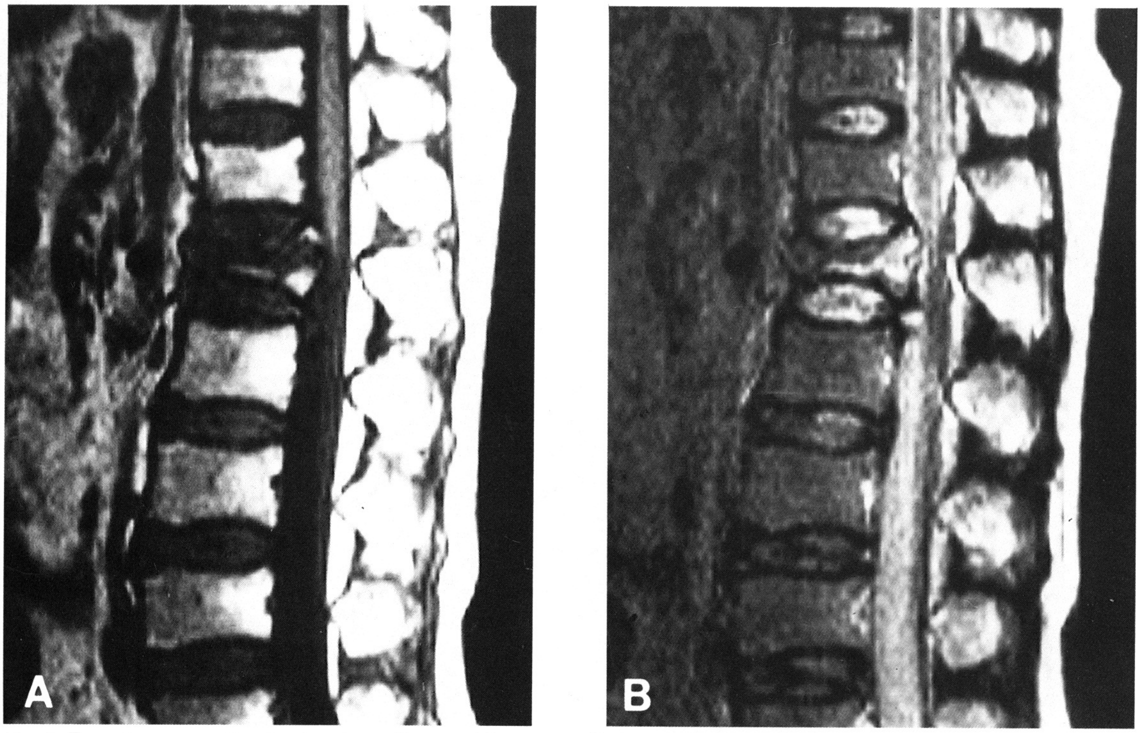


Fig. 2. Type Ib compression fracture in a 41-year-old woman with trauma 2 days prior.
A, T1-weighted MR image shows patchy hypointensity with preservation of peripheral portion in compressed L1 vertebra.
B, T2-weighted image shows hyperintensity in the corresponding area.

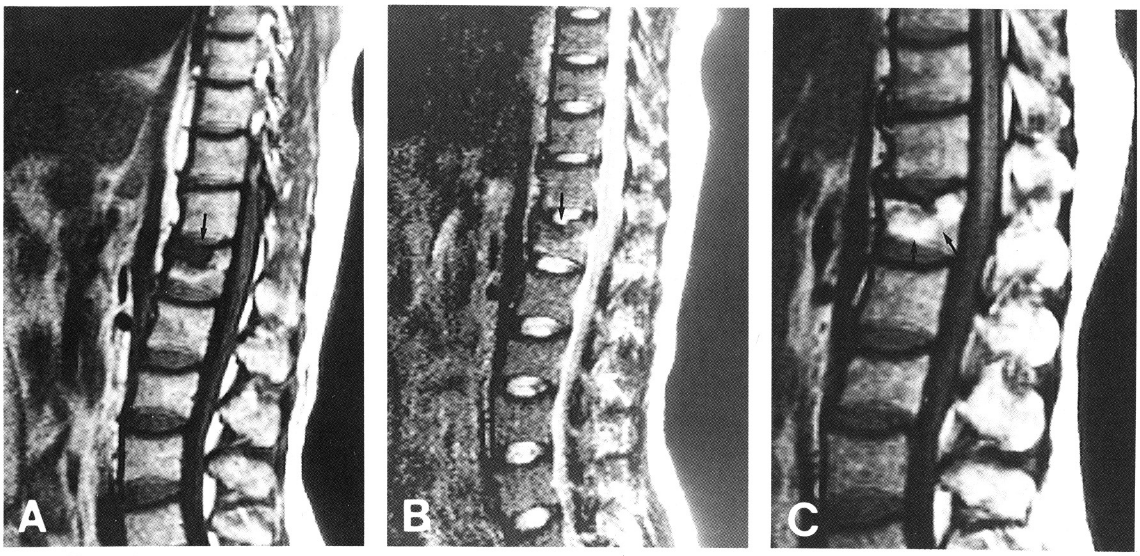


Fig. 3. Types II and III compression fractures in a 38-year-old man.
A, B, T1- and T2-weighted MR images obtained 5 months after a motor vehicle accident, demonstrate focal hypointensity (arrow) in the fractured T12 vertebral body (Type II).
C, Follow-up MR image 2 years after trauma shows hyperintensity (arrows) on T1-weighted image and isointensity on T2-weighted image (not shown), representing type III fracture.

with trauma less than 3 months past showed hypointensity on T1WI with hyperintensity on T2WI throughout the fracture sites(Type I). Such findings were not observed in patients with fractures over 3 months of age. Of 52 fractures with type I abnormalities, type Ia(Fig. 1) was noted in 14 fractures, type Ib(Fig. 2) in 17, and type Ic in 12; two types of abnormalities were combined in five patients.

In 12 fractures of 3 to 5 months, six demonstrated focal areas of hypointensity at the compressed vertebral bodies on all pulse sequences(type II) (Fig. 3), while six were isointense(type IV). Of 51 fractures with fracture ages over 5 months, the signal intensities of the fractures were isointense on all pulse sequences in 47(type IV) (Fig. 4); the remaining 4 showed hyperintensity on T1WI, and isointensity on T2WI(type III) (Fig. 3).

Four patients were imaged sequentially after the initial MR examination. In patients with multiple compression fractures who underwent follow-up MR imaging, initial abnormal vertebral signal intensities had progressively resolved within 3 months at the less extensively involved sites. However abnormal signal intensity lasted at the most compressed site until approximately 5 months after trauma(Fig. 4). We could observe that the time for the normalization of the signal intensity of the injuried spine appeared to be affected in part by the severity of the compressive forces applied to the fracture sites.

On sequential MR images, abnormal marrow signal intensity appeared to return to normal as early as approximately 3 months after trauma(Table 2). The serial changes of the marrow signal intensity did not vary with patient age without statistical evaluation.

On the basis of our study, the expected natural course of marrow signal intensity change in traumatic vertebral compression fractures is shown on Table 3.

Table 2. Correlation of Fracture age with Patient's Age in Presenting MR Signal Pattern

	fracture age				
pt. age	-1m	-2m	-3m	3-5m	5m-6yr
21-30	15/I*	2/I	4/I	2/II, 6/IV	3/IV
31-40	12/I			1/II	2/III, 8/IV
41-50	2/I			1/II	2/III, 18/IV
51-60	6/I	4/I		1/II	13/IV
61-76	5/I	2/I		1/II	5/IV
Total	40	8	4	12	51

*n/I, II, III, IV : number of patient/type of marrow signal intensity

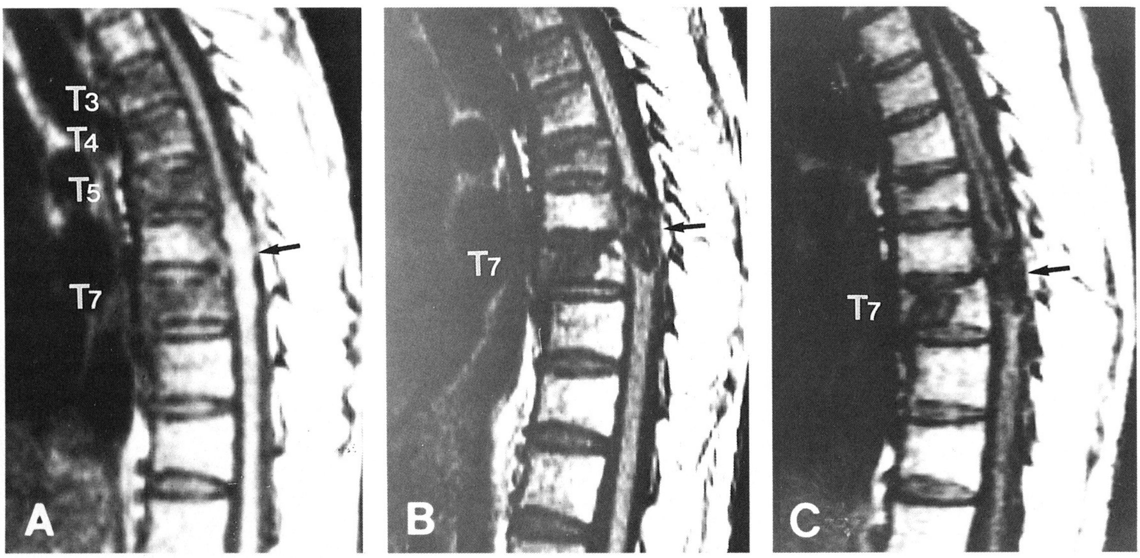
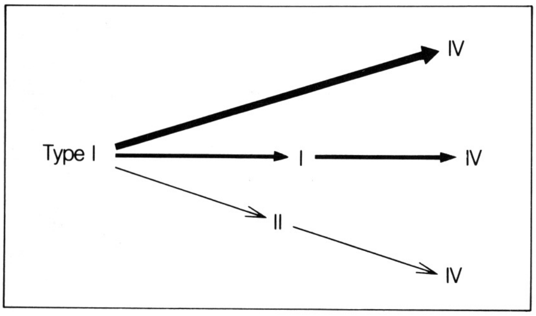


Fig. 4. Type I compression fracture in a 30-year-old man. A, Initial MR image 20 days after trauma shows diffuse hypointensity on T1-weighted image throughout the fractured T3, 4, 5, 7 vertebrae. Note the spinal cord injury (arrow).
B, Follow-up MR image obtained 3 months after trauma. Abnormal signal intensity is localized to the fracture sites. Note cystic myelomalacia of the spinal cord (arrow).
C, MR image 5 months after trauma shows return of abnormal signal intensities to normal marrow signal intensities in all compressed vertebrae except for T7. Post-traumatic syrinx (arrow) is noted.

Table 3. Expected Natural Course of MR Signal Patterns in Compression Fracture



DISCUSSION

Although MR imaging of vertebral compression fractures has been studied previously by some researchers(Bennett, 1966 ; Kim et al., 1979), studies of time-related signal intensity changes in vertebral fracture have been limited. It has been suggested that post-traumatic compression fractures less than 1 month showed homogeneous or heterogeneous diffuse hypointensity on T1WI and hyperintensity on T2WI(Bennett, 1966). However, further long-term chronologic studies appear to be necessary for the accurate definition of vertebral compression fractures and for the evaluation of compression fracture especially in obscure trauma history in elderly patients. Our study has extended our knowledge regarding sequential changes in compression fractures. For example, fractures less than 3 months of age show a diffuse, patchy or band-like hypointensity on T1WI and hyperintensity on T2WI with signal intensities gradually declining and localizing over time. This type I abnormality was not identified in all patients over 3 months after trauma.

Two different patterns of marrow signal intensity have been described in fractures more than 1 month after trauma(Bennett, 1966), i.e., focal fat deposition and decreased signal intensity due to bone sclerosis or fibrosis. Although precise time correlation was lacking in this study, we could confirm both findings at different time periods following the fractures. Six fractures with trauma of 3 to 5 months showed hypointensity on all sequences(type II), and four of the 51 fractures over 5 months showed type III pattern probably due to increased fat deposition.

The differentiation between benign and malignant forms of fracture in vertebral bodies has not been considered possible using conventional methods(Mirvis et al., 1988). Observation of intensity, appearance and resolution of radiotracer uptake in serial bone scans is also unreliable for this purpose(Sartoris et al., 1986). Uptake at the sites of hypertrophic spurring also limits the ability of radionuclide bone scans to distinguish between acute and chronic fractures(Sugimura et al., 1987). The period varies for increased radioactivity on bone scans at a fracture site. Sixty and 90 percent of vertebral fractures will return to normal within 1 and 2 years, respectively, but they do not return to normal until at least 7 months after vertebral fracture(Sugimura et al., 1987). On the basis of our results, we believe that the time for the reversal of abnormal signal intensities to normal on MR imaging takes 3 months. The apparent discrepancy(7 vs 3 months) between these studies using these two modalities may be due to the different sensitivity for the composition of the tissues making up the vertebral body.

Recognition of the chronologic stages of a benign traumatic vertebral compression fracture would prevent confusion with malignant lesion, since healing and remodelling of a fracture can last for months or even years(Wiener et al, 1989). If types II, III, or IV signal intensities are seen in patients with fracture age over 3 months, MR imaging easily distinguishes benign compression from pathologic fractures. As most malignant lesions exhibit homogeneous hypointensity on T1WI and hyperintensity on T2WI due to total replacement of the fatty marrow by tumor infiltration. However, if the history of trauma to the spine is within 3 months, MR differentiation of acute traumatic from pathologic fracture may not always be possible using marrow signal intensity alone. Among type I in acute fractures less than 3 months, type Ia appeared to be diffusely homogeneous or heterogeneous on MR imaging, resembling a pathologic fracture caused by tumor infiltration, but both types Ib and Ic differed in appearance from those cases associated with tumors. Observation of progression of the lesions and analysis of associated secondary MR findings were helpful in differentiating acute traumatic from pathologic fracture. In metastatic fractures, the loss of bone marrow signals is persistent and progressive, while signal intensity abnormalites in traumatic fractures are restored to normal marrow signal on follow-up MR imaging. In addition, disc disruption, vertebral body fragmentation, angulation of fragments, and associated soft tissue changes such as paraspinous hematoma or posterior ligamentous disruption strongly favor the traumatic compression fracture(Kim et al., 1979 ; Yuh et al., 1989).

Our study had several limitations. The ideal study should be done with the same patients being imaged sequentially at regular intervals after trauma. However such a study would be costly and not easily accomplished. Only four patients were able to undergo sequential follow-up MR imaging in our series. In addition, our study did not include pediatric aged patients in whom different time sequential changes were expected on MR imaging due to more rapid bone remodelling and healing processes than those of adults.

In conclusion, MR imaging findings of fracture in post-traumatic spine varied, depending mainly on the age of the fracture. Our study showed that MR imaging was useful in distinguishing between acute and chronic post-traumatic vertebral compression fractures.

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