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The Correlation of Clinical Course and Magnetic Resonance Image Findings of Radiation Myelopathy: A Case Report

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We described a case of radiation myelopathy (RM). The correlations of evolutional changes in magnetic resonance image (MRI) findings in three years with clinical symptoms and treatment responses in different stages of the disease were discussed. We also describe some special rehabilitative consideration for RM patients. Cord enlargement with contrast-enhancement showed in MRI findings corresponding to rapid neurological deterioration. Some neurological improvement after anti-coagulation and steroid therapy was observed in this stage. Cord atrophy without contrast-enhancement was showed in following MRI. The neurological deficits still progressed slowly in this stage and almost did not response to the treatment. We conclude that MRI is a powerful diagnostic tool for RM and may predict the treatment responses. (J Rehab Med Assoc ROC 2003; 31(2): 99 - 104)

Key words: radiation complication, magnetic resonance image, spinal cord

CASE REPORT

This is a 47-years-old gentleman who was well before. In September 1997, nasopharyngeal carcinoma was diagnosed by biopsy with initial presentation of primary neck mass. The tumor stage was T2N2M0. He received radiotherapy (7200 cGy in 40 fractions) from December 22, 1997 to February 20, 1998 via bilateral opposing fields. He was fairly well without tumor recurrent sign during follow-up in outpatient clinic. However, he noted left buttock and left lower leg paresthesia since April 1999. The paresthesia progressed to left side body and he noted right limb weakness since June, 1999. These symptoms became much worse. Weakness of right extremities developed gradually since July 1999 and progressed to bilateral sides. He was admitted for further workup from August 13, 1999 to August 21, 1999. MRI of cervical spine (Figure 1) showed cord enlargement with contrast-enhancement at C1-3 segments. He received a course of anti-coagulant therapy and corticosteroid during admission under the impression of RM. The muscle power got about one degree improvement and he was discharged and followed up in outpatient clinic. He still could ambulate with walking aid at that time. However, the muscle weakness became worse again during

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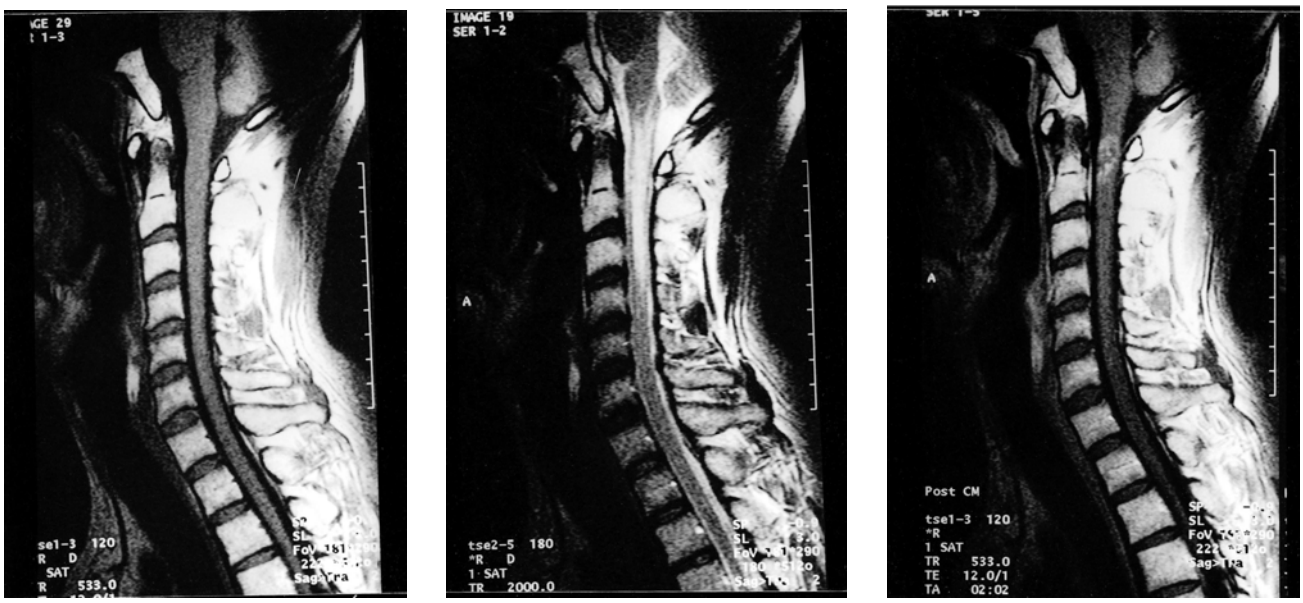
follow-up. He was admitted from January 5, 2000 to January 12, 2000. Follow-up MRI of cervical spine (Figure 2) showed cord atrophy without contrast-enhancement at C1-4 segments. Another course of corticosteroid with anticoagulant was given again but no obvious neurological improvement was observed this time. His symptoms got worse and worse. He lost the ability of ambulation gradually and relied on the wheelchair for locomotion. He was discharged and stayed at home without active treatment. Slowly but progressive muscle weakness still bothered him. He was admitted from May 7, 2001 to May 30, 2001 due to short of breath. Follow-up MRI (Figure 3) showed more remarkable cervical cord atrophy at the same level compared with that of previous study. Pulmonary function test showed moderately to severely restrictive lung disease corresponding to high level cervical cord injury. Breathing exercise technique, including deep breathing exercise and abdominal muscle strengthening were instructed to him. He was discharged and followed up in outpatient clinic. The neurological deficits still progressed slowly during follow-up.

DISCUSSION

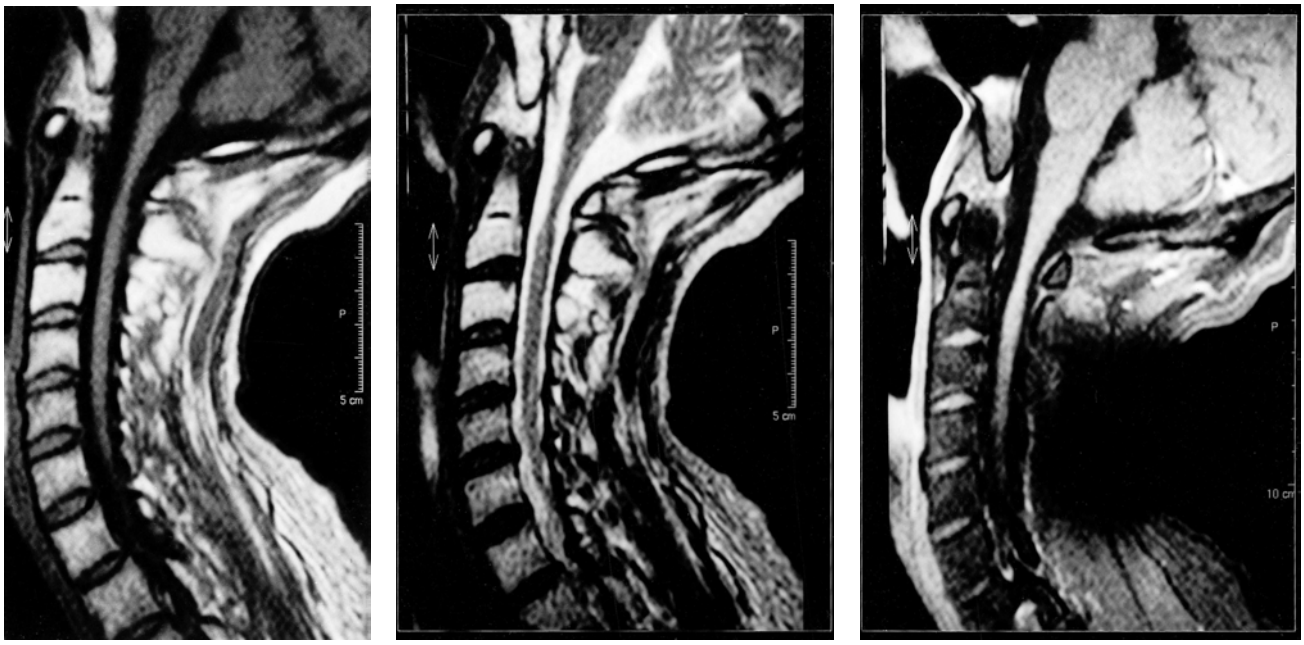
Radiation myelopathy (RM), also known as post-irradiation myelopathy, is a rare but serious complication after spinal irradiation. It related to the radiation dose and fraction size direct to the spinal cord as well known.[1-3] Pallis et al. suggested three criteria for diagnosis of RM: 1) The spinal cord must be irradiated. 2) The damaged segment of the spinal cord must be in the irradiated area. 3) Cord compression or neuropathy must be excluded.[4] RM presented as a spectrum of syndromes that may associate with different underlying pathogensis. There are four different syndromes have been recognized including acute complete paraplegia or quadriplegia, lower motor neuron disease, acute transient radiation myelopathy, and chronic progressive radiation myelitis (CPRM).[5,6] The former two syndromes are associated with secondary to spinal cord infarction and selective anterior horn cell damage; both types are very rare conditions. Acute radiation myelopathy clinically presented as tingling, shock-like feelings radiating from neck to the extremities that will be induced by neck flexion (Lhermittees's sign). Underlying pathogenesis may be due to transit demyelination of ascending sensory tract as a consequence of loss of oligodendroglial cells by radiation damage. These symptoms start after radiation therapy around 10 to 16 weeks and may as early as 6 weeks and late as 6 months or even later. The outcome is good and most symptoms resolve gradually in several months.[5,6] However, motor deterioration may follow these symptoms in some case as the onset of CPRM. Several hypotheses have been stated for underlying pathogenesis. White matter destruction as demyelination, necrosis, focal hemorrhage and vascular changes including endothelial damage with fibrinosis, and increased permeability were noted in histological examination.[7]

MRI is a very helpful tool in RM. It may help exclude other cause of cord lesions and indicates the exact lesions site. However, there is still no large series of studies available in MRI findings of RM due to rare cases. Typical MRI findings in RM include: (1) spinal cord swelling on T1-weighted images (T1WI) and intramedullary high signal on T2-weighted images (T2WI) , and (2) a ring-like enhancement on post-contrast enhanced T1WI.[8-13] These findings may suggest the inflammatory process in spinal cord and breakdown of blood-brain barrier (BBB) after radiation damage corresponding to microscopic histopathological findings. However, Komachi et al found the most predominant histopathological findings that correlate with high signal intensity on T2WI are proliferation of the small vessels without marked edema in an autopsy case of CPRM.[14] Alfonso et al. reported MRI findings of eight patients with RM. They outlined that MRI findings varies depending on the stage of CPRM. MRI findings may be normal in acute phase even if the patients have already presented severe neurological deficit.[8] Melki et al. reported MRI findings of ten cases of CPRM and the relationships among MRI findings, clinical course and prognosis. They found that MRI findings usually demonstrated cord enlargement if the onset of symptoms was within eight months. After the symptom began more than eight months, cord atrophy was always demonstrated in MRI. An enlarged cord was often associate a neurological deterioration and a fatal outcome. In the cases of cord atrophy, neurological deficit was more stable and had better survival rate.[9] No treatment was proved to be effective for RM currently, although anticoagulant or hyperbaric oxygen therapy had some improvement in small number patients reported.[11,15]



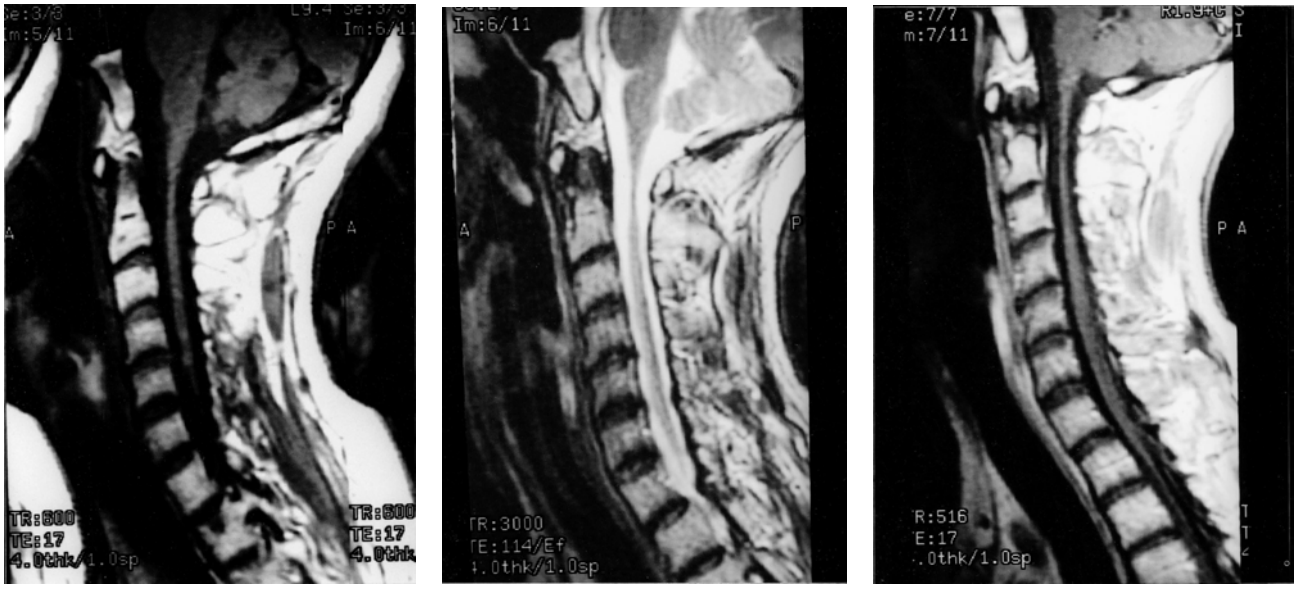
(a) (b) (c)

Figure 1. C-spine MRI in August 1999. a: Cord enlargement at the level of C1-3 was showed on sagittal T1WI. b: Bright signal intensity was showed at the same level on T2WI indicating cord edema. c: Intramedullary contrast-enhancement was showed on post-contrast enhanced T1WI.



(a) (b) (c)

Figure 2. C-spine MRI in January 2000. a: Cord atrophy was showed at C1-3 level on sagittal T1WI. b: No obvious signal change at atrophic cord on T2WI. c: No enhancement at atrophic cord on post-contrast enhanced T1WI.



(a) (b) (c)

Figure 3. C-spine MRI in May 2001. a-c: Similar findings compared with that of previous MRI (Figure 2) but much more remarkable cord atrophy was demonstrated.

In our case, MRI demonstrated all similar evolutional changes as previous published reports. In the stage with cord enlargement shown in MRI, although the patient had more rapidly neurological deterioration, there were some favorable responses to the treatment. However, this patient still had slowly neurological deterioration even in the stage of cord atrophy presented in MRI. No response to further treatment was observed in this stage. The cord atrophy in MRI findings may not indicate the stable neurological deficit. These findings are consistent with the report of Wang et al.[13] Slow progression of disease suggest that the neuron and axon loss may persist in chronic stage of CPRM. These findings signify that the pathogenesis of neurological deterioration is different in early and late stage of RM resulting in different responses to treatment. It is well-established that early mega-dose steroid administration is beneficial for treatment of acute traumatic spinal cord injury. However, it is still controversial that steroid or other medication is effective for RM at present. Schultheiss suggested that earlier intervention with steroid may improve treatment for some high risk patients.[16] We concluded that MRI findings reflect the underlying pathogenesis in different stage of RM that may associate with different treatment responses. Further study should be conducted for understanding treatment response according to MRI findings and different stage of the disease.

The physiatrist should involve the management in early stage of disease. The rehabilitative goal is to keep the patient at the highest functional level and to prevent the complications such as contracture, skin problem and cardiopulmonary deconditioning, etc. Instruction to these patients including adequate daily range-of-motion (ROM) exercise, stretching, and muscle strengthening exercise should be provided. As disease progress, proper orthosis and assistive devices for improving functional status should be prescribed. General guidelines applied in traumatic SCI patients are useful for RM patients. In our experience, there are two features for RM patients following head-neck cancer irradiation. First, spasticity may affect the neck and shoulder muscle due to very high cervical spinal cord involvement. The range of motion of neck and shoulder may be limited severely. This condition will interfere the bed and wheelchair mobility profoundly. Early intervention for improving neck and shoulder ROM should be given for preventing secondary disability. Second, involvement of respiratory muscle leads to progressive ventilatory dysfunction. It is important to emphasis daily breathing exercise and monitor pulmonary function regularly. Mechanical respiratory

assistance should be indicated if significant respiratory decompensation developed.

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放射治療後脊髓病變臨床病程與核磁共振影像之相關：病例報告

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本報告提出一位放射治療後脊髓病變的病例。在追蹤三年中，對於臨床症狀及治療反應，與核磁共振的影像之間的相關加以討論。同時對於此類病患所須的特別復健考量加以討論。

結果顯示核磁共振出現脊髓腫大及出現不正常顯影劑顯影，和臨床症狀快速惡化有明顯相關，在此時期若給予抗凝血劑及類固醇治療可以觀察到某些神經學上的進步。當追蹤的核磁共振下出現脊髓萎縮，同時不正常顯影消失，此時期仍有緩慢的神經學惡化，並且對於治療幾乎沒有反應。我們認爲核磁共振對於放射治療後脊髓病變是一個極有用的診斷工具，並且可用來預測治療的反應。（中華復健醫誌 2003; 31(2): 99 - 104）

關鍵詞：放射治療併發症(radiation complication)，核磁共振(magnetic resonance image)，脊髓(spinal cord)

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