The relationship of white matter lesion and contract enhanced lesion development courses in radiation induced brain injury: an MRI based study

H. Huang^{1,2}, M. Deng¹, S. F. Leung³, Y. L. Chan¹, D. K. Yeung¹, H. C. Chan¹, A. T. Ahuja¹, and Y. X. Wang¹

Department of Diagnostic Radiology and Organ Imaging, The Chinese University of Hong Kong, Prince of Wales Hospital, Shatin, NT, Hong Kong, Department of Radiology, The Shenzhen Third People's Hospital, Shenzhen, China, People's Republic of, Department of Clinical Oncology, The Chinese University of Hong Kong, Prince of Wales Hospital

Introduction:

The natural course of radiation induced brain injury remains poorly understood. Descriptions of radiation injury frequently originate from patients treated for brain tumors. In these cases analysis of the effects of radiation can be hampered by the underlying tumour. This is not so problematic with imaging radiation injury in patients treated for nasopharyngeal carcinoma (NPC), where the primary cancer rarely involves the brain and direct invasion by recurrent tumour or by haematogeneous metastases is uncommon. The radiation field for NPC includes the skull base and so often the inferior and medial aspects of the temporal lobes are irradiated, resulting in an effective radiation dose that exceeds the tolerance limit for neural tissue and leading to a substantial risk of temporal lobe injury (TLI). Patients with NPC frequently survive this cancer so providing the opportunity for the long term follow up of the effects of radiation. Among the TLI abnormalities identified by MRI, white matter edema-like lesions (WML) and contrast enhanced necrotic lesions (CEL) have been most commonly reported (1). TLI had been generally regarded as a progressive and irreversible process, however, with NPC patients data, it was recently reported that radiation induced brain injury was not always an irreversible and progressive process, but one that could show regression and resolution (2). The aim of this study was to investigate the relationship of WML and CEL during the course of their development. Based our clinical observational experiences, our hypothesis was that the development of WML and CEL would follow the same direction in the same period, i.e. if WML increases, then co-existing CEL in the same lobe would also increase, or develop new CEL, or remains stable, but would not decrease or resolve.

Materials and Methods:

This is a retrospective study. Patients with TLI were identified from MRI of the head and neck performed in patients with a history of NPC treated with radiotherapy. Patients in whom there was evidence of tumor invading the cavernous sinus or floor of the middle cranial fossa adjacent to the injured temporal lobes were excluded. Only the patients with MRI displayed both WML and CEL in the same temporal lobe and had at least one follow up MRI examination were analysed. Patients comprised of 22 patients (males = 17, females = 5, age range 38 to 73 years, mean 55 years). In total 36 lobes with TLI and 53 MRI examinations were analysed. The median interval between two consecutive MR exams was 11.6 months. MRI was performed at 1.5T scanner, including axial T1 and T2-W sequences; coronal T1 and T2-W sequences, and contrast-enhanced axial and coronal T1-W sequences. FOV was 22×22 cm, or 25.6×25.6 cm, matrix was 256 × 202 or 512 × 512, section thickness was 4 mm with no gap. For each patient the two temporal lobes were assessed separately, and lesion volume of WML and CEL was measured on each MRI examination. The lesion volume change over two consecutive MRI of WML and CEL were divided into five categories (1) new lesion (2) increasing (3) static (4) decreasing (5) resolved. The measured lesion volume change of less than 5% was arbitrarily regarded as lesion being static.

Results:

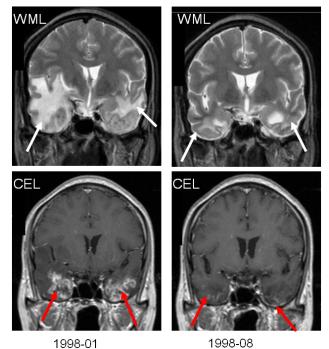
The results of the quantitative measurement are shown in table 1 and one example is shown in Fig 1. The results showed in most the cases, WML and CEL developed in the same pattern. It was possible when WML increase, CEL could increase, develop new lesion, remain static, but not decrease or resolve. When WML remained stable, CEL could either increase or decrease.

Table 1	WML	WML	WML	WML	WEL
	inc	dec	new	sta	res
CEL inc	16	0	0	1	0
CEL dec	0	14	0	1	0
CEL new	2	0	8	0	0
CEL sta	1	1	0	3	0
CEL res	0	1	0	0	0

Table 1. Number of WML and CEL changes seen on two consecutive MRI examinations. inc: increase, dec: decrease, new: new lesion, sta: static, res: lesion resolved.

Fig 1. WML (white arrows) and CEL (red arrows) follow the same trend of lesion decrease during the follow-up.

<u>Discussion</u>: Due to the scarcity of available histological specimen from human subjects, the exact histopathology of WML and CEL remain unclear. WML is believed to represent demyelination, gliosis and oedema. The close relation between contrast enhancement and radionecrosis has been recognized. Focal disruption of the blood-brain barrier has been reported to correspond to histological findings of necrosis in irradiated pigs (3), while contrast enhancement on CT or MRI has been associated with radiation necrosis at histologic analysis (4, 5, 6). The preliminary results from study suggest that the development of WML and CEL tend to follow the same direction, and not develop in the opposite direction.



References: [1] Chan YL, et al. Radiology 1999;213:800-807 [2]. Wang YX et al. Radiology in press [3] Miot E, et al. Int J Radiat Oncol Biol Phys. 1995;32:121-8. [4]. Lee AWM, et al. Cancer 1988; 61:1535-1542 [5]. Valk PE, et al. Am J Roentgenol. 1991; 156:45-62. [6]. Norris AM, et al. Clin Radiol 1997; 52:356-362