MS AND PHYSICAL TRAUMA

V. Brinar
University Hospital Zagreb, School of Medicine, Zagreb, Croatia

In spite of many published cases of MS exacerbation after trauma (1,2,3,4,), in epidemiological studies such correlation was not found (5,6,7,8,). Causal relationship between MS and trauma confirmed in many clinical case reports and MRIs, was explained through the breakdown of the blood-brain barrier (3,4). Trauma in particular mild concussive injury to the head, neck or upper back my result in an increase in BBB permeability by impinging on the brain and spinal cord. In such situations trauma acts as facilitator of the pathogenetic mechanism of lesion formation. It is well documented that trauma may cause hyperintense lesions demonstrated on T2 images, often misdiagnosed as MS in non-MS patients, and in many individuals such changes are found accidentally. Why should it be different in MS patients?
Poor correlation between size and site of MS lesions and clinical manifestations of MS is the reason that there is many time lack of appearance of new MS symptoms after traumatic episodes. It is well known that many demyelinating plaques occur so often in non-eloquent areas without provoking clinical symptoms, and in such situations the correlation between traumatic injury and MS could not be found. Therefore epidemiologic data comparing various severity of traumatic lesions and clinical symptoms in general population, or even in MS patients are not appropriate. It is reasonable to count that traumatic break down of BBB occurs after severe traumatic CNS injury. If it happens in eloquent area it will cause clinical symptoms in MS patients or even trigger initiation of MS in genetically prone individuals.

Conclusion is yes; brain trauma may cause new lesions in MS patient, and may even trigger initiation of MS in geneticaly susceptible patients. The unpredictability and variability of the clinical manifestations of the disease, the difference in the genetic and immunologic backgrounds of individuals as well as their degree of clinical and pathological involvement and level of MS activity, point that epidemiological studies are inappropriate in this specific area.

References:
Chaudhuri A & Behan PO Acute cervical hyperextension-
hyperfl exion injury may precipitate and/orexacerbate symptomatic multiple sclerosis. *European Journal of Neurology*, 2001 **8**, 659–64.
Pluchino S, Vallone I, Annunziata P severe clinical relapse and secretion of pro-inflammatory cytokines in a multiple sclerosis patient treated with IFN B after head injury ital. J. neurol Sci 1999 ;20 (suppl No 4) :S 189
Poser C.M. Physical trauma and psychological stress in multiple sclerosis
Rev Neurol (Paris). 1980;136(12):807-14.
Poser CM: The role of trauma in the pathogenesis of multiple sclerosis: a review.
Clin Neurol Neurosurg. 1994 May;96(2):103-10.
Siva A, Radhakrishnan K, Kurkland LT *et al*. Trauma and multiple sclerosis: a population based
cohort study from Olmsted County. *Minnesota Neurology*, 1993 43, 1878–82.
Sibley WA, Bamford CR, Clark K, Smith MS & Iaguna
JF A prospective study of physical trauma and multiple sclerosis. Journal of Neurology, Neurosurgery
and Psychiatry, 1991 54, 584–9
Kurland L.T.: Trauma and Multiple sclerosis Ann Neurol. 1994;36:S33-S37
M.J Goldacre, J.D.Abisgold,D.G.R Yeats,V. Seagroatt: Risk of multiple sclerosis after head injury:record linkage study J neurol. Neurosurg. Psychiatr. 2006,77:351-353