Ruolo determinante della sostanza bianca nella sindrome dell'eminegligenza visuo-spazilale

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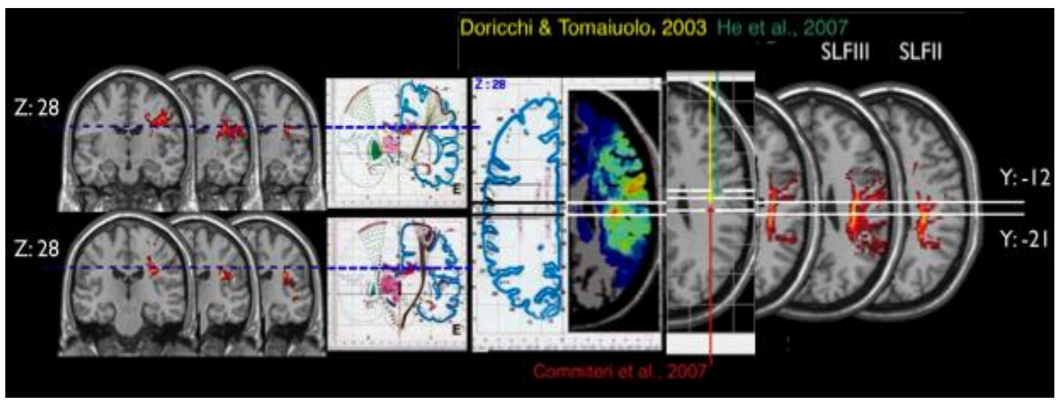
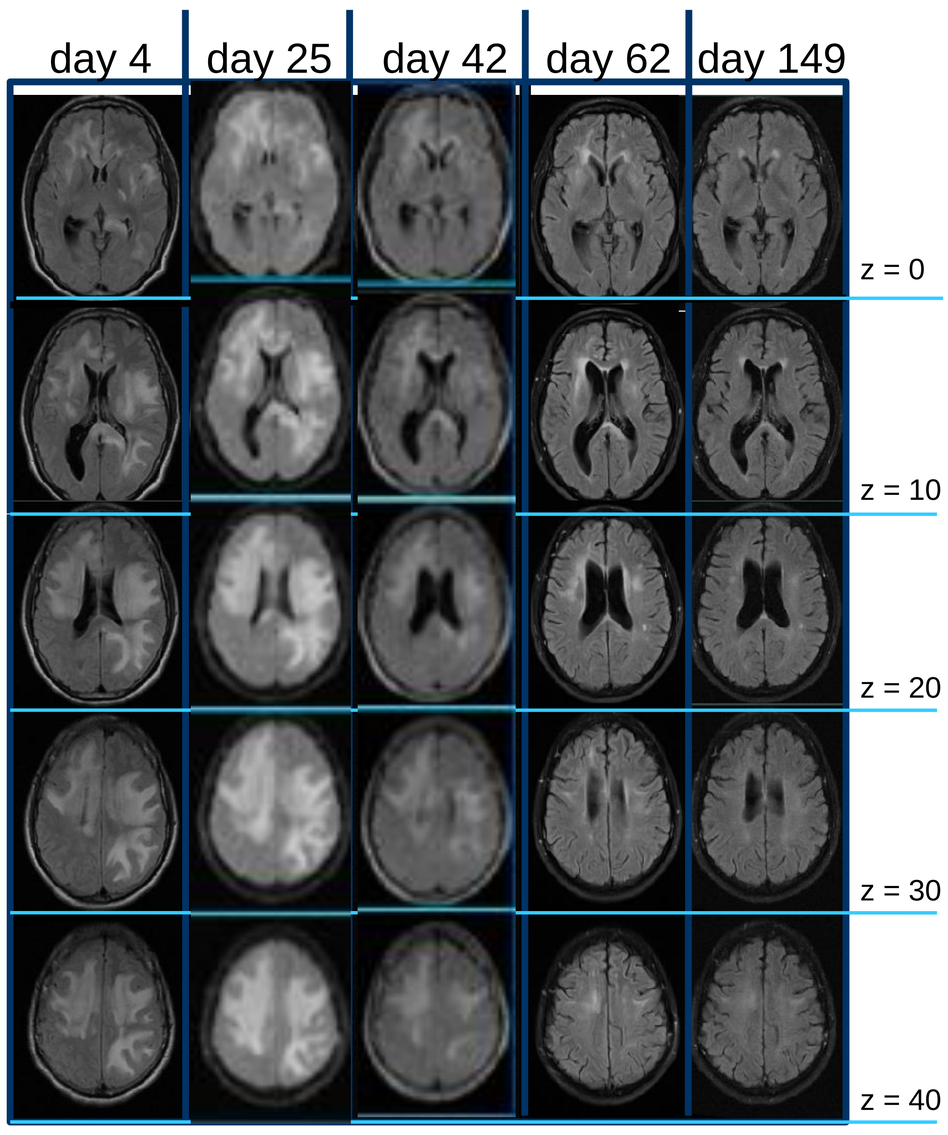
INTRODUZIONE

Introduction: Visual neglect has been associated with focal damage of parietal, frontal or temporal lobe, as well as basal nuclei and thalamus. Recent investigations suggest that visual neglect could also be related to lesions of the white matter (WM) tracts connecting the frontal and parietal/temporal lobes in the right hemisphere, or damage to the visual pathways up to the calcarine cortex in addition to the posterior part of the corpus callosum (Doricchi et al., 2008). However, these investigations have mostly evaluated patients suffering from WM injury associated with grey matter damage (Doricchi & Tomaiuolo, 2003; Thiebaut de Schotten et al,. 2005; He at al.,2007; Committeri et al., 2007; Tomaiuolo et al., 2010).



In this study: We present the case of patient LB, affected with acute disseminated encephalomyelitis (ADEM; a monophasic, inflammatory demyelinating disease causing specific WM damage) who showed signs of neglect that decreased and then disappeared concomittantly to the WM damage induced by the ADEM as observed on his brain MRIs.
No grey matter damage was detected.

Caratteristiche Anatomiche



LB's brain MRI showed multiple areas of altered WM signal in the right temporal and parietal lobes, in the left corona radiata, around the calcarine sulcus, and in the callosal splenium.

Evident diminuition of the WM damage was detectable on the MRI on day 149.

Caratteristiche Cliniche

LB was a 34-year old male crane operator affected by specific WM damage caused by acute disseminated encephalomyelitis (ADEM), a monophasic, inflammatory demyelinating disease.

ADEM: The distinctive clinical feature of the disorder is the development of a focal or multifocal neurological disorder after a prodromal phase of several days, characterized by fever, malaise and myalgias. The onset of the CNS disorders is usually rapid (abrupt or up to several hours), reaching peak dysfunction within several days. Neurological signs are variable and depend upon the location of the CNS involvement, usually diffuse or multifocal.

After about 10 days of a febrile episode, LB was treated with antibiotics and antipyretics, then he began to suffer from loss of concentration, mental confusion, and later showed left upper limb weakness.

3rd day: onset of respiratory failure, coma and marked bradycardia. The patient is connected to a mechanical ventilator.

30th day: the patient comes to our unit for observation, still under corticosteroids. He is alert, breathing spontaneously through the tracheotomy tube; responds verbally to questions with latency and not constantly; shows localization of pain, severe weakness in all 4 limbs, diffuse muscle hypotonia, mild muscle wasting, mild dysphagia, and sphincter incontinence.

51st day: the patient has been discharged from the Severe Acquired Brain Injury Unit, without tracheotomy tube, with excellent recovery of strength, autonomous walking, feeding, sphincter control.

He showed:

- perceptual-visual neglect;
- representational-topographical neglect;
- very mild signs of personal-body neglect;
- dressing apraxia of the left part of the body.

Cognitive neglect and dressing apraxia slowly disappeared after 20 more days.

Risultati e Conclusioni

A complete recovery from Neglect and dressing apraxia was observed simultaneously to an almost complete remission of the WM damage as detected from brain MRI.

White Matter damage *per se* may cause visual neglect, disrupting the integrated functioning of fronto-parietal or temporo-parietal networks and/or WM visual pathway in addition to the posterior part of the corpus callosum.

References:

. Committeri G., Pitzalis S., Galati G., Patria F., Pelle G., Sabatini U., Castriota-Scanderbeg A., Piccardi L., Guariglia C., Pizzamiglio L. *Brain*. 2007; 130:431-41.

. Doricchi F. & Tomaiuolo F. *Neuroreport* 2003; 14(17): 2239-2243.

. Doricchi F., Thiebaut de Schotten M., Tomaiuolo F., Bartolomeo P., *Cortex,* 2008; 44:983-995.

. He B. J., Snyder A. Z., Vincent J. L., Epstein A., Shulman G. L., Corbetta M., *Neuron*, 2007; 53: 905-918.

. Thiebaut de Schotten M., Urbanski M., Duffau H., Volle E., Lévy R., Dubois B., Bartolomeo P. *Science*; 2005; 30;309:2226-8. Erratum in: Science. 2007; 3;317:597.

. Tomaiuolo F., Voci L., Bresci M., Cozza S., Posteraro F., Oliva M., Doricchi F., *Exp Brain* Res. 2010; 206:209-17.

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