GRETA GEMCHELU 15/11/21 PROVA NON ESTRATTA

14_CONCORSO PUBBLICO, PER TITOLI ED ESAMI, PER LA COPERTURA A TEMPO DETERMINATO, DELLA DURATA DI CINQUE ANNI PER N. 1 POSTO DI COLLABORATORE PROFESSIONALE DI RICERCA SANITARIA - CAT. D, DA ASSEGNARE ALLA UOC NEURORADIOLOGIA

PROVA 1

1. In un paziente con lesione espansiva frontale posteriore-sinistra, quale paradigma fMRI sarebbe più indicato e quali tratti di sostanza bianca sarebbero da esaminare mediante trattografia?

2. Cos'è Excel e a cosa serve?

3. Si traduca l'abstract dell'articolo sottostante

Amyotrophic lateral sclerosis

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Abstract | Amyotrophic lateral sclerosis (ALS), also known as motor neuron disease, is characterized by the degeneration of both upper and lower motor neurons, which leads to muscle weakness and eventual paralysis. Until recently, ALS was classified primarily within the neuromuscular domain, although new imaging and neuropathological data have indicated the involvement of the non-motor neuraxis in disease pathology. In most patients, the mechanisms underlying the development of ALS are poorly understood, although a subset of patients have familial disease and harbour mutations in genes that have various roles in neuronal function. Two possible disease-modifying therapies that can slow disease progression are available for ALS, but patient management is largely mediated by symptomatic therapies, such as the use of muscle relaxants for spasticity and speech therapy for dysarthria.

Amyotrophic lateral sclerosis (ALS) is a heterogeneous neurodegenerative disease that is characterized by the degeneration of both upper motor neurons (that is, neurons that project from the cortex to the brainstem and the spinal cord) and lower motor neurons (that is, neurons that project from the brainstem or spinal cord to muscle), leading to motor and extra-motor symptoms (FIG. 1). The initial presentation of ALS can vary between patients; some present with spinal-onset disease (that is, the onset of muscle weakness of the limbs), but other patients can present with bulbar-onset disease, which is characterized by dysarthria (difficulty with speech) and dysphagia (difficulty swallowing). In most patients, the cause of ALS is unknown, although some individuals have familial disease, which is associated with mutations in genes that have a wide range of functions, including roles in non-motor cells. In familial ALS, some of the implicated genes are incompletely penetrant, and with rare exceptions, genotype does not necessarily predict phenotype[1].

The classification of ALS can vary depending on the criteria used. The traditional definitions of ALS subgroups are based on the extent of involvement of upper and lower motor neurons, although other classification systems include different parameters, such as the site of onset (that is, bulbar-onset or spinal-onset disease), the level of certainty of diagnosis according to the revised El Escorial criteria and heritability (sporadic or familial disease)[7]. To date, none of these classification systems have incorporated the cognitive or behavioural symptoms, and within each classification system, a range of sub-phenotypes and clinical trajectories can be observed.

This Primer reviews the aspects of ALS that contribute to disease heterogeneity and looks to the future of new therapeutic trials that incorporate recent advances in our understanding. For new therapies, the challenge is to define mechanisms of disease that are amenable to drug targeting and to define patients who are likely to respond to these therapeutic agents.

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PROVA 2

1. Ci descriva quale sarebbe il mapping preoperatorio corticale e della sostanza bianca per l'area motoria della mano

2. Cos'è Matlab e a cosa serve?

3. Si traduca l'abstract dell'articolo sottostante:

Abstract

Objective To explore structural and functional changes of the brain and cervical cord in patients with amyotrophic lateral sclerosis (ALS) due to mutation in the superoxide dismutase (*SOD1*) gene compared with sporadic ALS.

Methods Twenty patients with *SOD1* ALS, 11 with sporadic ALS, and 33 healthy controls underwent clinical evaluation and brain MRI. Cortical thickness analysis, diffusion tensor MRI of the corticospinal tracts (CST) and corpus callosum, and resting-state functional connectivity were performed. Patients with ALS also underwent cervical cord MRI to evaluate cord cross-sectional area and magnetization transfer ratio (MTR).

Results Patients with *SOD1* ALS showed longer disease duration and slower rate of functional decline relative to those with sporadic ALS. No cortical thickness abnormalities were found in patients with ALS compared with controls. Fractional anisotropy showed that sporadic ALS patients had significant CST damage relative to both healthy controls ($p = 0.001–0.02$) and SOD1-related ALS ($p = 0.05$), although the latter showed alterations that were intermediate between controls and sporadic ALS. Functional hyperconnectivity of the motor cortex in the sensorimotor network was observed in patients with sporadic ALS relative to controls. Conversely, patients with *SOD1* ALS showed lower cord cross-sectional area along the whole cervical cord relative to those with sporadic ALS ($p < 0.001$). No cord MTR differences were found between patient groups.

Conclusions Patients with *SOD1* ALS showed cervical cord atrophy relative to those with sporadic ALS and a relative preservation of brain motor structural and functional networks. Neurodegeneration in *SOD1* ALS is likely to occur primarily in the spinal cord. An objective and accurate estimate of spinal cord damage has potential in the future assessment of preventive *SOD1* ALS therapies.

GRETA DEMICHELIS 15/11/21 PROVA NON ESTRATTA

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PROVA 3

1. Descrivere un possibile quadro clinico e di planning pre-chirurgico nel caso di un paziente con afasia di Wernicke

2. Cos'è un software Open Source e in cosa si distingue dagli altri?

3. Si traduca l'abstract dell'articolo seguente:

ABSTRACT

Background: Imaging studies have revealed cortical thinning and subcortical atrophy occurring in Parkinson's disease (PD); however, the topographical distribution and clinical associations related to advancing stages of PD remains unclear.
Objective: We aimed to investigate the topographical distribution of cortical and subcortical morphometric changes, and their clinical associations, related to increasing disease severity.
Methods: In this cross-sectional imaging study, T1-weighted structural magnetic resonance imaging data for 80 non-demented PD patients and 30 age-matched healthy controls were analysed using FreeSurfer software suite to derive morphometric changes using whole-brain vertex-wise analysis, and surface-based (cortical) and volume-based (subcortical) parcellation maps. PD patients were divided into three groups of mild ($n = 27$), moderate ($n = 27$), and severe ($n = 26$) PD based disease duration and Hoehn and Yahr and Unified Parkinson's Disease Rating Scale Part-III motor severity scores.
Results: Whole-brain vertex-wise analysis revealed cortical thinning in the orbitofrontal cortex in early PD ($P = .011$), and in the superior frontal ($P = .002$), caudal middle frontal gyrus ($P = .001$) and inferior parietal cortex ($P = .006$) in moderate PD. Severe PD patients showed additional cortical thinning in temporal and occipital cortices ($P < .005$). Subcortical volume loss was detected in the thalamus ($P = .012$) and hippocampus ($P = .032$) in moderate PD, which extended to the caudate ($P = .012$), putamen ($P = .042$) and amygdala ($P = .008$) in severe PD. Increasing disease duration and motor severity scores, correlated with cortical thinning in frontal, temporal, parietal and occipital cortices, and subcortical volumetric loss in the thalamus, caudate, putamen, amygdala and hippocampus. Lower global cognitive status, measured with MMSE, correlated with cortical thinning in temporal, parietal, frontal and cingulate cortices, and with volumetric loss in the hippocampus ($r = 0.31$; $P = .009$); suggesting subclinical pathogenic changes occur prior to the onset of cognitive impairment.
Conclusion: In conclusion, in more severe disease stages PD patients exhibit progressive cortical thinning and subcortical volume loss which could have relevance to the development of cognitive impairment.