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EDITORIAL

Advances in clinical neurology through the journal "Neurological Sciences" (2015–2016)

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In the years 2015–2016, several important advances in neurology and neurosciences have been published in Neurological Sciences. The Journal continues to attract an ever-increasing number of article submissions. Here, we report a brief review of the journal's content in the 2015–2016 years.

Vascular disorders

Stroke pathophysiology, treatments and outcomes are an active field of interest. Copy number variants, specific genetic polymorphisms, toll-like receptors, inflammatory citochines, and chemokines represent factors of susceptibility for stroke [1–6]. Moreover, microRNAs appear to play a role in post-stroke excitotoxicity [7], while Brain-derived neurotrophic factor Val66Met polymorphism is associated with functional and cognitive outcomes of stroke [8]. A specific ACE gene polymorphism has been reported to predispose to hemorrhagic stroke [9]. Acid uric and antioxidants act as a neuroprotective agent for the ischemic stroke [10]. Granulocyte-colony stimulating factor (G-CSF) combined with repetitive transcranial magnetic stimulation (rTMS), administered in the early subacute phase of ischemic stroke, may exert a hazardous effect on functional recovery, possibly due to impaired angiogenic mechanism, decreased cell survival, and increased inflammation [11]. Single small subcortical infarction has been reported associated to early neurological deterioration [12]. Investigations on the influence of cognition changes during post-stroke rehabilitation is relevant [13], associated to post stroke depression and to the degree of neurological deficit [14], while long-term mortality after stroke is higher than after myocardial infarction [15]. Several articles discuss epidemiology and diagnosis. The incidence of hemorrhagic stroke in Japan has been reported higher than in the western countries [16]. Glial fibrillary acidic protein test is a promising technique for diagnosis of intracerebral hemorrhage from ischemic stroke and prediction of short-term functional outcomes [17]. In mice, lithium treatment exerted a neuroprotective effect on learning and memory by potentiating the Akt/GSK3β cell-signaling pathway [18].

Antiplatelet treatment is useful both in primary and secondary prevention, but poor response to aspirin or clopidogrel is a not rare condition [19, 20].

A public education campaign or health-related applications (app) could potentially reduce pre-hospital delay for ischemic stroke patients [21], and also a web-based telemedicine system for thrombolysis could give a growing number of patients access to treatment [22]. Stroke awareness in general population could improve public behavior in terms of prevention, symptom recognition, and timely response [23]. Creation of hospital-based registers may help to ameliorate stroke management [24]. Endovascular treatment (ET) has shown to be safe in acute stroke, but its superiority over intravenous thrombolysis is debated [25]. In murine models, the adenosine A2A receptor antagonist, administered soon after ischemia, has been shown to protect from neurological deficit in the first days but not later [26].

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Stem cell-based therapy hold extensive potential in treating intracerebral hemorrhage, which should be further evaluated with more evidence-based animal studies [27].

Carotid ultrasounds and transcranial Doppler remain the most important diagnostic tools [28]. Chronic cerebral hypoperfusion due to carotid arteries stenosis leads to axonal damage over time [29]. Carotid endarterectomy implies a reduced blood flow to the brain, but protective mechanisms such as brain release of erythropoietin and nitric oxide represent an endogenous self-activated neuroprotective mechanism aimed at the prevention of ischemia/reperfusion damage [30].

Multiple sclerosis

Also multiple sclerosis (MS) represents an active field, with high social and economic impact worldwide [31]. Grey matter damage in MS is an acquired data, with relevant clinical implications [32]. Genetic and environmental factors are involved in the risk of developing the disease [33–36]; while in other cases this susceptibility has not been demonstrated [37–39]. Other factors, as alcohol consumption, present potential protective effect [40]. Elevated fibrinogen levels are associated with a worse prognosis [41]. Higher C-reactive protein values were associated with pregnancy-related comorbidities but not with MS disease activity [42]. The management of disease-modifying or symptomatic therapies represent an active field of interest in MS. Autologous hematopoietic stem cell transplantation might represent a therapeutic possibility in MS patients unresponsive to approved therapies [43]. High-dose interferon-β therapy is associated with thrombocytopenia [44]. Natalizumab is extremely effective in reducing disease activity in MS patients but its long-term use has been reported associated with the risk of progressive multifocal leukoencephalopathy (PML). Early detection of PML is related to a favourable outcome [45]. Recurrence of disease activity Natalizumab discontinuations despite the treatment with immunomodulant or more aggressive therapy has been demonstrated [46]. New diagnostic tools for assessment of urinary dysfunction lead to identify lower urinary tract symptoms also in asymptomatic patients [47]. Efficacy and safety of nabiximols on spasticity in MS have been largely demonstrated [48]. Alternative and complementary medicines, such as herbal remedies, appears to be scares and presents potential risk of adverse reactions or interference with conventional treatments [49]. Cognitive and psychiatric aspects have been better defined with differentiation of various profiles [50–54]. Fatigue, quality of life, and working status have recently arisen great interest in MS patients [55–57]. Asymmetrical visual and brainstem auditory evoked potentials (VEP and BAEP) abnormalities were found in fatigued MS patients, with no relationships to disease-related variables, inducing to consider them as an electrophysiological marker of fatigue in MS patients [58]. Physical activity should be encouraged in all stages of disease [59, 60].

Migraine and headache

Headache is a very common neurological problem with a high impact on quality of life, also among children and adolescent [61, 62]. Physiopathology of cephalic pain is not clearly understood [63–67]. Studies of resting-state functional magnetic resonance imaging have detected a brain dysfunction affecting intrinsic connectivity of brain networks [68]. Headache represents also a critical problem in the emergency setting [69–72]. The elevated high-sensitivity C-reactive protein level and low Retinol-binding protein-4 level in migraine patients suggest that vitamin A might play a major role in the pathogenesis of migraine [73]. An association between inflammation and atherosclerosis in patients with migraine, even in children, have been hypothesized [74], but the absolute risk of ischemic stroke in migraineurs is relatively low and an antithrombotic primary prevention is not indicated [75]. Several non-controlled studies suggest that closure of the foramen ovale significantly reduces attack frequency in migraine patient, but the only prospective placebo-controlled trial does not support these results [76]. Natural menopause is associated with a lower incidence of migraine as compared with surgical menopause [77]. Idiopathic intracranial hypertension without papilledema should be considered in all patients with almost daily migraine pain, with evidence of sinus stenosis and unresponsive to medical treatment [78, 79]. Ocular pain, in same case, requires the exclusion of ophthalmologic diseases, as uveitis, angle closure glaucoma, neuritis [80]. Pathophysiology of vestibular migraine has been better defined with functional neuroimaging techniques [81]. The combination of cinnarizine and dimenhydrinate in the prophylactic therapy has been reported effective [82]. Acute confusional migraine still has an unclear pathophysiology, but dysfunction of dorsal anterior cingulated cortex probably plays a pathogenic role [83]. Women with migraine and phonophobia exhibited deficits in otoacoustic emissions suppression, which points to a disorder affecting the medial olivocochlear efferent system [84]. The choroidal thickness has been found to decrease significantly not only in patients with migraine with aura, but also in those without aura during the attack-free period [85]. Treatment of chronic migraine with medication overuse requires withdrawal from acute medications [86]. Triptans represent the most specific and effective therapy option for migraine attacks. Nevertheless, in clinical practice, they are often underused [87, 88]. Topiramate is an effective drug in

migraine prophylaxis but paresthesia is a frequent adverse reaction in patient with migraine more than in epileptic patients [89, 90]. Onabotulinumtoxin A treatment is efficacious in refractory chronic migraine [91, 92]. Recently, several neuromodulatory surgical techniques have been developed for the management of headaches that are unresponsive to medical treatment [93]. Transcutaneous neurostimulation with the Cefaly(®) device has shown efficacy for migraine therapy [94] while both deep brain stimulation and occipital nerve stimulation are utilized in refractory migraine [95]. Lymphatic drainage has been suggested as a therapeutic option in the prophlaxys of migraine [96].

Tumors

Cerebral tumors remains an important cause of death and disability [97]. The investigation of genetic and molecular mechanism on proliferation, apoptosis, invasion, and angiogenesis may help to focus potential therapeutic targets [98, 99]. As a class of small non-coding RNAs, microRNAs (miRNAs) has been discovered to be closely involved in carcinogenesis and might also be connected with glioma diagnosis and prognosis [100–102]. Metastasis-associated protein 3 (MTA3) expression was decreased in human glioma and negatively associated with prognosis of patients, suggesting that MTA3 may play a tumor suppressor role in glioma [103].

Alzheimer's disease

Alzheimer's disease (AD) is the principal cause of dementia in elderly, with high cost of treatment and care [104].

Accumulating evidence has indicated the role of insulin deficiency and insulin resistance as mediators of AD neurodegeneration, calling AD as "type 3 diabetes" [105], while a negative association between AD and cancer is an evidence that needs to be clarified [106]. Vitamin D deficiency presents a greater risk for ApoEε4 non-carrier AD patients than for ε4 carriers [107]. *N*-methyl-D-aspartate receptors (NMDARs) play a pivotal role in the synaptic transmission and synaptic plasticity thought to underlie learning and memory and have been recently implicated in Alzheimer's disease [108]. Retinal nerve fiber layer (RNFL) thickness by optical coherence tomography (OCT) has been evaluated and correlated with cognitive impairment, but further studies are needed to optimize the utility of this method as an ocular biomarker in AD [109].

Edaravone, a potent free radical scavenger with antioxidant effects, may be developed as a novel agent for the treatment of AD for improving cholinergic system and protecting neurons from oxidative toxicity [110]. The mammalian target of rapamycin (mTOR) pathway has been reported to mediate Aβ clearance through autophagy and may represent an important therapeutic target for AD [111].

Parkinson's disease

Parkinson's disease (PD) is a major worldwide public health problem with a prevalence that is expected to increase dramatically in the coming decades [112]. Inflammatory markers as carcinoembryonic antigen, high-sensitivity C-reactive protein (hs-CRP), and Neutrophil/lymphocyte ratio (NLR) are significantly higher in the PD patients than in the normal controls [113]. Oxidative stress is considered as a contributing factor to the development of PD. Decreased nitric oxide (NO) level and negative correlation observed between NO level and disease rating scale implicated a role for NO in the disease process [114]. An inverse relation between uric acid levels and L-Dopa treatment and PD stages may be due to the fact that high serum uric acids levels may decrease the oxidative stress taking part in the pathogenesis of PD [115]. The levels of oligomeric form of α-synuclein of red blood cells in ischemic stroke and in Parkinson's disease patients were both significantly higher than in normal people [116]. Corneal thickness may decrease in patients with PD [117], and colour and contrast dysfunction are present as the earliest symptoms of disease [118]. Minor salivary gland biopsy is a potential pathological biomarker for PD, but with a lower diagnostic accuracy than DAT-PET scan [119]. [(18)F] fluorinated-N-3-fluoropropyl-2-β-carboxymethoxy-3-β-(4-iodophenyl)nortropane (FP-CIT) positron emission tomography (PET) scans are able to differentiate PD and drug-induced parkinsonism [120]. Robertson dysarthria profile may be a valuable tool to detect speech/voice disturbances in Parkinson's disease [121]. Indoor and outdoor falls among people with PD often result in activity limitations, participation restrictions, social isolation or premature mortality [122]. Oropharyngeal bradykinesia may be responsible for drooling in PD [123]. Botulinum toxin is a safe and effective therapy for the treatment of sialorrhea, applied without the requirement of ultrasound guidance [124]. Non-motor symptoms of idiopathic Parkinson's disease, specifically pain, olfactory dysfunction, fatigue, depression, anxiety, and sleep disturbances, are important contributors for worsening the quality of life and poor patient outcomes [125–129].

Neuromuscular disorders

Amyotrophic lateral sclerosis (ALS) is the most common degenerative disease of the motor neuron system. Genetic and epigenetic factors play a role on the pathogenesis and the evolution of the clinical course [130–132]. Despite scientific efforts, pathophysiological mechanisms are not fully understood [133]. Modern MR techniques are helpful in ALS diagnosis, in assessment of clinical course, or even in the effects of new drugs [134, 135].

Spinal muscular atrophy (SMA) is a hereditary neuromuscular disorder with genetic heterogeneity [136]. Duchenne muscular dystrophy (DMD) and Becker muscular dystrophy (BMD) are the most frequent muscular dystrophies with different genetic profiles [137].

Genetic and clinical heterogeneity in genetic neuropathies and in Charcot–Marie–Tooth disease is notable [138].

Electrophysiological study is a fundamental diagnostic tool for all neuromuscular disorders [139–141]. For Guillain–Barrè syndrome (GBS), a correct and prompt diagnosis may be sometimes hard [142, 143]. A correlation between ubiquitin carboxy-terminal hydrolase-L1 (UCH-L1), a neuron-specific protein, in the cerebrospinal fluid of patients with GBS and severity of the disease at the acute phase has been suggested [144]. Facial onset sensory and motor neuronopathy (FOSMN) is a recently defined slowly progressive motor neuron disorder [145]. Brachial plexus injury (BPI) causes functional changes in the brain, but the structural changes resulting from BPI remain unknown [146]. Neuromyotoxicity due to therapy with hydroxychloroquine has been reported in a case [147].

Epilepsy

Epilepsy is a frequent cause of hospitalization, especially in pediatric age [148]. Seizure outcome in patients with juvenile absence epilepsy is not clear [149], and cognitive deterioration is not rare [150]. A prospective case–control study has shown that pregnancy does not affect seizure frequency in women with epilepsy [151]. Psychogenic nonepileptic seizures are more frequent among women and have possible biological basis [152, 153].

Electroencephalogram (EEG) is a fundamental diagnostic tool in epilepsy [154]. Focal changes in EEG have been reported in idiopathic generalized epilepsies [155].

Interesting articles report data on treatment. Phenytoin mainly metabolized by hepatic cytochrome P450 enzymes (CYP) and genetic polymorphism of CYP is related to therapeutic response [156]. The best withdrawal rate of antiepileptic monotherapy in seizure-free adult patients with epilepsy is questionable [157]. Ketogenic diet (KD) is one of the most effective therapies for intractable epilepsy, and, even if rich in olive oil, high-fat KD causes significant increase in LDL-cholesterol and triglyceride levels [158].

Vagus nerve stimulation therapy is the most frequently used neurostimulation modality for patients with drug-resistant epilepsy who are not eligible for seizure surgery [159].

Neurogenetics

Molecular genetics has an important role in all neurological diseases with the possibility to discover new mutations and new phenotypic presentations [160–166] also with investigations related to eye movements [167, 168]. Enzyme replacement therapy (ERT) is possible in a growing number of diseases: an open pilot study has been reported in late onset Pompe disease [169]. Allogenic hematopoietic stem cell transplantation and, more recently, liver transplantation are therapeutic options for mitochondrial neurogastro-intestinal encephalomyopathy (MNGIE) [170]. Llama single domain antibodies (VHH) directed against mutant huntingtin are interesting candidates as therapeutic agents or research tools in Huntington disease because of their small size, high thermostability, low cost of production, possibility of intracellular expression, and potency of blood–brain barrier crossing [171].

Conclusions

In summary, this 2 years review of what we learned by Neurologic Sciences Journal is an useful update of the main data published in the journal in the different fields of neurology, confirming the good quality of the journal, the modern and the international approach reporting all the new diagnostic, therapeutic and research strategies.

Compliance with ethical standards

Conflict of interest The authors declare no conflict of interest with the publication of this article.

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