IMMUNOTHERAPY IN NEUROMUSCULAR DISORDERS GLADY JACOB pdf

1: Electrophysiological study in neuromuscular junction disorders

TY - JOUR. T1 - Immunotherapy in neuromuscular disorders. T2 - Muscle and Nerve. AU - Drachman,Daniel B. PY - Y1 - N2 - Autoimmune mechanisms have recently been implicated in the pathogenesis of an increasing number of neuromuscular diseases.

Neurologists must first obtain an undergraduate pre-medical degree, and the next step in their education involves four years at medical school. Upon completing medical school, neurologists typically enter a one-year internship program, and this is followed by a three-year residency program in neurology. After this residency is completed, neurologists may receive advanced training via a fellowship program, and these typically last one to two years. How much does a neurological exam cost? The cost of a neurological exam will depend on the service that is being provided. Other types of diagnostic tests include a nerve biopsy, a spinal tap, and magnetic resonance imaging MRI. How are neurological disorders treated? A wide range of options are available for the treatment of neurological disorders. Multiple sclerosis may be addressed with disease-modifying therapies that are offered in injection, intravenous, and oral forms, and emotional, cognitive, and physical therapies are often recommended to support the process. Stroke treatments include blood thinners, blood pressure control, and thrombolytic therapy to break up blood clots. Dementia refers to a set of symptoms, and these include language difficulty, confusion, and memory loss. What are the signs of a concussion? The signs of a concussion may include headache, lack of coordination, memory loss, vomiting, nausea, dizziness, ringing in the ears, confusion, and severe fatigue. A concussion is typically caused by an injury that causes your head and brain to be quickly shaken back and forth. A concussion can be caused by a fall or a car accident, and it may also occur during participation in impact sports. What is a migraine? A migraine is a severe headache that causes pressure or pain in the head. Migraines are recurrent, and they are often preceded by signs such as nausea, vomiting, tingling in the limbs, and heightened sensitivity to light. A migraine headache often lasts for a few hours, and severe cases may last for days. Migraines may be triggered by stress, hormonal changes, and certain foods and drinks. What is a neurological examination like? This is followed by a physical examination in which the neurologist measures your pulse and listens to your heart and your lungs. What are the most common neurological disorders? What conditions does a neurologist treat? Neurologists provide care for patients who have suffered a stroke, and those who have been struck by seizure disorders such as epilepsy. Neurologists also treat infections that affect the nervous system, such as meningitis and encephalitis. What is a neurologist? A neurologist is a physician who is focused on the care and treatment of diseases that affect the nerves and the nervous system. The nervous system is comprised of both the central and the peripheral nervous system, and it includes the spinal cord and the brain.

IMMUNOTHERAPY IN NEUROMUSCULAR DISORDERS GLADY JACOB pdf

2: Learn about Concussions from Neurologist Glady Jacob, MD

Neuromuscular Disorders presents a multi-disciplinary approach to the management and therapeutic treatment of the full range of neuromuscular disorders and resulting complications.

Concurrent Paraspinous Myopathy and Myasthenia Gravis. Leading clinical features include a bent spine or dropped head antecollis. In myasthenia gravis MG , patients may have camptocormia secondary to neuromuscular junction dysfunction of the paraspinal muscles, and this condition usually responds to acetylcholinesterase inhibitors or immunosuppressive treatments. However, concomitant MG and paraspinous myopathy with histologic and electrophysiologic evidence of myopathic changes of the paraspinal muscles has only been reported twice in the literature Journal of Clinical Neuromuscular Disease https: Chronic graft-versus-host disease GVHD appears several months following allogenic hematopoietic stem cell transplantation HSCT and is clinically analogous to autoimmune disorder. Hence, its pathophysiology and treatment have not been elucidated. A year-old man with a history of chronic GVHD presented with ptosis, dropped head, and dyspnea on exertion, which had worsened over the previous several months The electromyogram confirmed the diagnosis of amyotrophic lateral sclerosis. After 3 years the patient remained able to walk unassisted and without significant bulbar manifestations or upper neuron signs. The concomitant presence of dropped head syndrome and man-in-barrel syndrome in an amyotrophic lateral sclerosis patient makes our case unique. Oxford Medical Case Reports https: Age-related sarcopenia may cause physical dysfunction. We investigated the involvement of sarcopenia in dropped head syndrome DHS. Our study subjects were ten elderly women with idiopathic DHS mean age Twenty age- and sex-matched volunteers mean age Scoliosis and Spinal Disorders https: Clinical features include early-onset weakness of limbs and oculobulbar muscles resulting in hypotonia, bulbar paresis, ptosis, and hypoventilation. We report a month-old boy with dropped head and limb-girdle weakness, who had no ptosis or ophthalmoplegia at presentation Marija Cauchi, Eleanor Marsh Head drop, or having a dropped head, is an uncommon condition in which patients present with a disabling inability to lift their head. It may arise in many neurological conditions that can be divided into those with neuromuscular weakness of neck extensors and those with increased tone of neck flexors. The most common neuromuscular causes include myasthenia gravis, motor neurone disease and myositis, while neck dystonia secondary to movement disorders can cause an increased tone. Investigations should include blood tests, imaging, muscle biopsy and neurophysiological studies

3: neuromuscular junction Publications | PubFacts

The pathogenesis of autoimmune inflammatory disorders of the neuromuscular system is multifactorial. In the past decade, substantial progress in understanding the basic cellular and molecular processes has opened the way for more target-oriented immunotherapy.

Hematopoietic SCs have been among the first used in the clinic, mainly to treat blood disorders and restore hematopoietic function after radiation and chemotherapy. Afterwards, they have also been considered to treat other diseases, such as AIDs [50]. In , seven patients with refractory and severe MG who received autologous hematopoietic SC transplants have experienced long-term remission, and remained symptom and treatment free for many years. This therapeutic approach can only be applied to severe and treatment-resistant patients, as the procedure is not without risk [51]. Mesenchymal SCs are distinct from the hematopoietic SCs and have the capacity to differentiate into multiple cell types including adipocytes, myocytes, but not in the hematopoietic cells. Mesenchymal SCs display regenerative and trophic features, as well as anti-inflammatory and immunosuppressive properties. Using a new humanized mouse model of MG, Sudres et al. They observed that conditioned mesenchymal SCs act by several mechanisms, including reduction in the expression of costimulatory molecules and proliferation, and activation of complement regulator components [54]. Mesenchymal SC-based therapy has not yet been tested in MG patients and the mechanisms by which mesenchymal SCs exert their regulatory effects in other AIDs remain largely unknown. Targeting the CD40L pathway to inhibit B-cell activation CD40 is expressed on antigen-presenting cells, such as macrophages, dendritic cells and B cells, while its ligand CD40L is expressed on activated T cells. CD40L induces activation of B cells, immunoglobulin class switch, plasma cell differentiation, as well as GC formation. Moreover the use of an anti-CD40L antibody is an effective treatment to suppress EAMG even when it is given during the chronic stage of disease [56]. Novartis has developed, a fully human IGg1 anti-CD40 monoclonal antibody CFZ that inhibits B-cell activation without B-cell depletion, for diverse autoimmune conditions. Other monoclonal antibodies against CD40 have been developed, such as Bleselumab Astellas Pharma but it has not yet been evaluated in clinical trial for AIDs. Antibodies targeting CD40L have also been developed and were promising in experimental models. Its use has been approved in different countries for the treatment of systemic lupus erythematosus SLE with mixed results [65]. In EOMG patients, CXCL13 is overexpressed in the thymus leading to the abnormal B-cell recruitment [67] and a higher frequency of circulating follicular T cells was observed in the peripheral blood of MG patients [68]. This new observation supports the potential interest of this therapeutic approach. Targeting IL pathway to inhibit pro-inflammatory Thcell effects In recent years, the development of AIDs has been linked to the imbalance between Treg cells and pro-inflammatory Th17 cells, in favor of these lasts. Treg cells possess immunomodulatory properties indispensable to terminate a normal immune reaction, maintain peripheral tolerance to self-antigens and avoid autoimmunity. In contrast, Th17 cells are highly inflammatory. They are defined by the production of different IL cytokines that are involved in inflammation and AIDs. The overexpression of cytokines of the IL family has been observed in T cells from MG thymuses together with the up-regulation of IL receptor expression [41]. Bimekizumab UCB is another monoclonal antibody against both ILA and ILF that demonstrated positive results in early development in patients with psoriatic arthritis [73]. IL inhibition can be also achieved by blocking the IL receptor with Brodalumab a human IgG2 monoclonal antibody. Brodalumab AstraZeneca is licensed for the treatment of psoriasis. Other therapeutic approaches aiming at blocking Th17 cell differentiation can also be envisaged with the use of molecules targeting IL These observations suggest that targeting the IFN-I signaling pathway could shut down the chronic inflammation characteristic of many AIDs. In this way, different therapeutic approaches are investigated but not yet in MG. Targeting the IL-6 pathway to overcome its pro-inflammatory effects IL-6 is a pleiotropic cytokine that has context-dependent pro- and anti-inflammatory properties. IL-6 is produced by various cell types and not only immune cells. IL-6 actively stimulates T lymphocytes and favors the switch of Treg cells towards a pathogenic Th17 cell phenotype. IL-6 also induces B-cell differentiation into antibody-secreting cells and promotes the survival of long-lived plasma

cells [79]. IL-6 expression is increased in different cell types in MG patients: Moreover, antibodies against IL-6 suppress EAMG in the rat model when administrated either during the acute or the chronic phase of disease. Compounds have been developed that target IL-6, its receptors or downstream signaling molecules. None has been considered so far for MG patients. Targeting the proteasome to reduce antibody-producing cells Bortezomib VelcadeTM, Millennium Pharmaceuticals is an inhibitor of the proteasome. The proteasome is a large complex of proteases involved in the degradation of abnormal or unneeded proteins. The immunoproteasome is formed by the replacement of certain subunits of the proteasome and is abundantly expressed in cells of hematopoietic origin. It actively participates in the degradation of intracellular proteins for presentation of antigens by the major histocompatibility complex molecules [87]. Bortezomib was initially used as an anti-cancer drug in particular in hematological malignancies. However, Bortezomib has been reported to reduce autoantibody titers improving clinical condition in a SLE mouse model [88]. Since Bortezomib has been efficiently used in other experimental autoimmune models. It depletes both short-lived and long-lived plasma cells that usually survive the standard immunosuppressant treatments. In the mouse EAMG model, Bortezomib efficiently reduces the rise of anti-AChR autoantibodies, prevents ultrastructural damage of the postsynaptic membrane, improves neuromuscular transmission, and decreases MG symptoms [89]. It was also demonstrated that in vitro Bortezomib can kill long-lived plasma cells derived from the thymus of EOMG patients [90]. A selective inhibitor of the immunoproteasome ONX has also proved to be efficient in the classical EAMG model ameliorating disease severity [91]. Complement inhibition to attenuate the consequences of the autoantibody attack Binding and activation of complement at the NMJ is the predominant pathogenic mechanism of anti-AChR antibodies [92]. In the MG thymus, the activation of the complement cascade has also been observed on myoid cells and on thymic epithelial cells [93]. Changes in complement concentration can influence the severity of MG: Moreover, increased complement consumption was detected in MG patients with high AChR antibody concentrations [98]. Altogether, these data suggest that molecules aiming at decreasing complement deposition at the NMJ could represent a therapeutic approach of major interest [99]. Nevertheless, only Eculizimab SolirisTM, Alexion Pharmaceuticals but no other molecules aiming at inhibiting the complement cascade activation have been considered for MG patients. Eculizumab is a humanized monoclonal antibody that inhibits the cleavage of the complement protein C5 acting at the terminal complement activation cascade. The primary endpoint based on the measure of a change in MG activities of daily living was not reached due to the use of prespecified worst-rank criteria. However, secondary end points and sensitivity analyses suggested efficacy of Eculizumab leading to a reduction in disease severity [ ]. After this clinical trial, in October , the US Food and drug administration has extended the indication for this molecule as a potential treatment for patients with refractory and generalized AChR-MG. Nevertheless, as C5 terminal complement activation is necessary in the defense against encapsulated bacterial infections, meningococcal vaccines and antimicrobial prophylaxis must be considered to reduce the risk of meningococcal disease [ ]. Conclusion Various therapeutic strategies have been developed for MG patients, but MG is still a chronic disease and most patients will need treatments for the rest of their life. Our current strategies have some limitations, mainly due to the potential toxicity of the drugs over time, and to the fact that some patients are or become refractory to their treatment. There is a real need for new therapeutic approaches that could be envisaged earlier in the course of the disease and not only in refractory forms, often after complications linked to the burden of immunosuppressant treatments. Conflict of interest There is no conflict of interest to declare pertaining to this study. Humoral antibodies to acetylcholine receptor in patients with myasthenia gravis. Auto-antibodies to the receptor tyrosine kinase MuSK in patients with myasthenia gravis without acetylcholine receptor antibodies. Autoantibodies to low-density lipoprotein receptor-related protein 4 in myasthenia gravis. Diagnostic and clinical classification of autoimmune myasthenia gravis. Passive transfer of experimental autoimmune myasthenia gravis by monoclonal antibodies to the main immunogenic region of the acetylcholine receptor. Autoantibody mechanisms and new developments on immune regulation. Quantitative immunocytochemical analysis of inflammatory cells and detection of complement membrane attack complex at the end-plate in 30 patients. CCL21 overexpressed on lymphatic vessels drives thymic hyperplasia in myasthenia. Role of the thymus in the physiopathology of myasthenia. Muscle autoantibodies

in subgroups of myasthenia gravis patients. A higher than expected incidence in the elderly. Distinct roles of nerve and muscle in postsynaptic differentiation of the neuromuscular synapse. Clinical correlates with anti-MuSK antibodies in generalized seronegative myasthenia gravis. Antibodies against muscle-specific kinase impair both presynaptic and postsynaptic functions in a murine model of myasthenia gravis. Lrp4 is a receptor for Agrin and forms a complex with MuSK. Autoantibodies to lipoprotein-related protein 4 in patients with double-seronegative myasthenia gravis. A comprehensive analysis of the epidemiology and clinical characteristics of anti-LRP4 in myasthenia gravis. Antibodies against low-density lipoprotein receptor-related protein 4 induce myasthenia gravis. The thymus and the pathogenesis of myasthenia gravis. The role of the thymus in myasthenia gravis: Immunohistological and immunological studies in cases. Ann N Y Acad Sci. Expression of acetylcholine receptor genes in human thymic epithelial cells: Implications for myasthenia gravis. In vitro anti-acetylcholine receptor antibody synthesis by myasthenia gravis patient lymphocytes: Correlations with thymic histology and thymic epithelial-cell interactions. Thymic B cells from myasthenia gravis patients are activated B cells. Phenotypic and functional analysis. Thymus in myasthenia gravis. Isolation of T-lymphocyte lines specific for the nicotinic acetylcholine receptor from thymuses of myasthenic patients. Anti-AChR antibodies, thymic histology, and T cell subsets in myasthenia gravis. Anti-acetylcholine receptor antibodies decrease after thymectomy in patients with myasthenia gravis. Randomized trial of thymectomy in myasthenia gravis. N Engl J Med. Expert Opinion on Biological Therapy. Acetylcholine receptor-reactive T cells in myasthenia gravis:

4: Doctor draws attention to Brain Injury Awareness Month

disorders of the peripheral nerve, the neuromuscular junction, and skeletal muscle generally start slowly or subacutely and follow a chronic or chronic undulating course with persistent.

Neural Disorders of Skeletal Muscle Overactivity: Neuropathies Associated with Systemic Disease. Neuropathies Associated with Infections. Neuropathies Related to Nutritional Deficiencies. Neuropathies Associated with Malignancy. Neuropathies Associated with Endocrinopathies. Radiculopathies, Plexopathies, and Mononeuropathies of the Lower Extremity. Disorders of Neuromuscular Transmission. Nondystrophic Myotonias and Periodic Paralysis. Myopathies Associated with Systemic Disease. Neuromuscular diseases are among the more common neurological disorders, and gratifying advances in our understanding of them have occurred in recent years. In particular, advances in genetics, immunology, epidemiology, neurophysiology, imaging, histopathology, and pharmacology have led to new approaches to diagnosis and treatment. Many disorders once considered distinct entities are now recognized to be heterogeneous, with different subgroups having different prognostic implications and requiring different therapeutic strategies. Anthony Amato and Dr. They describe an approach to diagnosis, management, treatment, and prognostication that is practical but is based on their extensive clinical experience and wide knowledge of the literature. I have little doubt that this book will become an indispensable resource for clinicians involved in the diagnosis and management of patients with neuromuscular diseases, as well as for scientists working on the nature and pathogenesis of these disorders. It is a much-needed and welcome addition to the neurological literature and will rapidly gain acceptance as a standard work of reference. I welcome its publication and congratulate Dr. Russell for producing such an outstanding volume. Click here for terms of use. Our knowledge regarding the various neuromuscular disorders, their pathogenic bases, and treatment options has rapidly expanded over the past decade, thus making the practice more challenging. There are several outstanding reference textbooks devoted to myology, neuropathies, electrodiagnostic medicine, or neuromuscular pathology and this book is not meant to replace any one of these classic texts. However, our experience as mentors to medical students, residents, and fellows in the neuromuscular clinic and hospital wards as well as referrals from practicing clinicians suggested to us the need for a neuromuscular textbook that was all inclusive and practical in regards to how to approach and treat individuals with the broad range of neuromuscular diseases. There is no standard approach or treatment for most of these disorders and experienced clinicians may disagree with our recommendations. Nevertheless, we hope this book lays the groundwork for how to evaluate and manage patients with all types of neuromuscular disease. Amato, MD James A. Accurate diagnosis requires consideration of individual patient and disease differences. Confounding variables that are part of the human experience may be missed or overemphasized by testing algorithms. This textbook will repeatedly emphasize the strongly felt philosophy of its authors, i. These hypotheses should be formulated based on the principles of neurological localization, the chronological course of symptom development, and application of risk factor analysis. Ideally, the tests described in the subsequent two chapters and throughout the text would be ordered with the primary intent of resolving a clinically established differential diagnosis or, if possible, in an attempt to prove a working diagnosis. As all tests are potentially fallible, the credibility of their results diminishes when they are used as screening procedures. They are of great value when placed in the hands of a skillful artisan, but are potentially damaging if used injudiciously. The differential diagnosis of disorders of the neuromuscular system neuromuscular varies to a certain extent with age. Infants, children, and adolescents share both similarities and differences in their differential diagnosis with their adult counterparts Tables 1â€”1 to 1â€”3. The neuromuscular system will also be considered to include the sensory nerves at or distal to the dorsal root ganglion. Disorders affecting the peripheral autonomic system or cranial nerves will be discussed only as necessary to better understand diseases affecting their somatic and spinal counterparts. There is another key point in history taking that will be repeatedly emphasized in this text. Many patients with heritable disorders will not recognize the hereditary nature of their disease. This may be due to a recessive inheritance pattern, spontaneous mutation, false paternity, or incomplete or delayed penetrance. Frequently, it is due to a lack of

familiarity with the medical issues of other family members. In the pediatric population, parents must be questioned with great care and sensitivity. Parents may also bring a considerable amount of guilt to the examination, which may limit their willingness to share information. If necessary, professional counseling should be offered in addition to treating the patient. Often, when a child is ill, the entire family is affected, which can in turn have profound repercussions on more than just the patient from both a physical and a psychological standpoint. Electrodiagnostic Medicine, 2nd edn. In our opinion, adherence to these principles will improve the diagnostic accuracy of both the neurologist and the referring physician. This chapter will attempt to focus on information that is important to elicit, and also on an organizational framework to interpret it accurately. Sensory symptoms may also manifest in either a positive e. During history acquisition, there is considerable value in identifying both the location and the nature of the initial symptom s , including the circumstances during which that symptom developed. The subsequent evolution of symptoms should then be developed in a chronological fashion. The value of this approach can best be illustrated in the setting of patients with multifocal neuropathy. Identifying that the initial symptom occurred in a focal nerve distribution may be a key element in diagnostic success. As muscle weakness is usually the most objective manifestation of neuromuscular disease, emphasis is often placed not only on its existence but on its type and pattern as well. The existence of weakness may be apparent either through history taking or, more commonly, by examination. Even though muscle weakness is the hallmark of neuromuscular disease, patients uncommonly use the word weakness in their symptom description. The complaint of weakness is more commonly used by patients as a synonym for asthenia—a more pervasive, generalized complaint due to a number of different pathologies. Patients with true muscular weakness more commonly speak in terms of the sequelae of their weakness, i. Muscle pain is also a common complaint brought to the attention of the neuromuscular clinician. Patients with weakness of hip abductors will waddle as a compensatory maneuver to maintain their center of gravity and balance. Patients with chronic weakness of hip extension will tend to have exaggerated lumbar lordosis resulting from posterior displacement of the shoulders for the same compensatory reasons. These patients may hyperextend their knees in order to prevent this while standing or walking, i. Wrist and digit weaknesses interfere with grip and dexterity, which may impair multiple activities of daily living, including opening of bottles and cans, turning ignition keys, or buttoning buttons. Neuromuscular disorders often affect the motor and to a lesser extent sensory functions of cranial nerves. Extraocular muscle involvement is a key discriminating factor in working through the differential diagnosis of neuromuscular disorders. Patients typically become aware of ptosis by personal or family observation Table 1—4. Extraocular muscle involvement is typically expressed as diplopia, although patients with slowly progressive, symmetric involvement of the extraocular muscles such as in chronic progressive external ophthalmoplegia may be amazingly asymptomatic. Patients with the acute onset of unilateral facial weakness are usually very aware of the existence and nature of their problem. Again, this is commonly due to their appearance in a mirror prompted by an abnormal feeling that they may describe as numbness. They may be bothered by the saliva that drains from their drooping mouth, the tears that drain from their sagging lower eyelid, or an initial tendency to be slightly dysarthric. Symptomatic jaw weakness is infrequent in most neuromuscular disease. When present, it is often overshadowed by symptoms referable to muscles concomitantly affecting speech, swallowing, and breathing. Symptoms referable to tongue weakness are common in many neuromuscular disorders. Patients may become aware of it with the development of dysarthria or through their inability to manipulate food properly. This kind of detail is rarely volunteered by the patient and is more commonly elucidated by detailed questioning. Weakness of the neck muscles may be noticed by patients or their families with the development of head drop. This is often accompanied by nuchal discomfort, presumably due to the constant and unaccustomed traction on posterior cervical ligamentous structures. It is possible that this same head drop may contribute to dysphagia as well. Trapezius weakness is most commonly symptomatic when acute and unilateral and is usually a result of a mononeuropathy of the accessory nerve. These are usually painful disorders, due again presumably to the shoulder drop and resultant traction on pain-sensitive structures. These can be easily missed unless the patient is viewed from the rear, with the shoulders exposed. Symptoms of ventilatory muscle weakness represent a fairly common, ominous, and occasionally initial

manifestation of a selective group of neuromuscular disorders. Dyspnea on exertion is the typical symptom of hypoventilation. It may result in airway collapse, obstruction, and snoring, with resultant excessive daytime fatigue and sleepiness. Nocturnal hypercarbia may also interrupt normal sleep cycling and promote nocturnal restlessness. Early morning headache due to carbon dioxide retention is usually a late symptom but one that should be inquired about. If the diaphragms are preferentially involved in the disorder, orthopnea may be a prominent and even initial symptom. Other organ systems may be affected in neuromuscular disorders, and a careful system review is important in an attempt not only to achieve a diagnosis but also to fully anticipate the scope of its potential morbidity. Symptoms referable to cardiomyopathy or cardiac conduction defects, impaired GI motility, cutaneous change, and discolored urine from myoglobinuria or porphyria may provide valuable insight. UMN involvement is usually noted at an earlier stage in the illness in comparison to its lower motor neuron counterpart, as it interferes with the synergistic functions of multiple muscle groups simultaneously. As a result, coordinated activities are impaired early in the course and positive motor symptoms that are more readily recognized are more likely to occur. Patients with UMN lesions will lose the ability to run early in the course if their legs are affected. They may complain of stiffness or a tendency to drag one or both lower extremities. They may notice an impaired ability to perform many activities that require rapid coordinated movements in the upper extremities. If the corticobulbar tracts are affected, swallowing and articulation are affected early and prominently, as these functions are dependent on the coordinated interplay of many muscle groups. Patients may lose their ability to effectively sniff or blow their nose. Patients with corticobulbar tract involvement may also develop lability of affect known as pseudobulbar palsy. In the motor system, one could argue that it is the examination rather than the history that produces the most useful information in the majority of cases. Inquiry regarding the existence of sensory symptoms on the other hand is frequently far more rewarding than the sensory examination.

5: Papers with the keyword "dropped head" (Page 2) | Read by QxMD

A new immunotherapy appears to be well tolerated and possibly of benefit in patients with amyotrophic lateral sclerosis. Epilepsy Next Article in Neuromuscular Disorders.

Funding information and disclosures deemed relevant by the authors, if any, are provided at the end of the article. The Article Processing Charge was paid by the authors. Received Sep 19; Accepted Dec 4. The work cannot be changed in any way or used commercially. This article has been cited by other articles in PMC. To show that immunotherapy with medications such mycophenolate mofetil MMF can cause serious complications in patients with neuromuscular disorders. Two patients with neuromuscular disorders on immunotherapy with long-term MMF who developed toxoplasmic encephalitis TE were included in this case series. One patient with myasthenia gravis and one patient with inflammatory myopathy on immunotherapy with long-term MMF developed severe TE. Both patients were treated with pyrimethamine, sulfadiazine, and leucovorin for 2 months without clinical improvement, and both died. Early consideration and recognition of this complication is important to possibly prevent unfavorable outcomes. The utility of screening and prophylaxis against toxoplasmosis in individuals with neuroimmunologic disorders and other autoimmune disorders who receive immunosuppressive therapy requires future study. Mycophenolate mofetil MMF is increasingly used in the treatment of neuromuscular autoimmune diseases such as myasthenia gravis. Toxoplasma gondii encephalitis occurs most often in AIDS patients and posttransplantation and rarely is reported as a complication of immunosuppressive treatment of autoimmune disease. No previous cases of toxoplasmic encephalitis TE occurring in association with MMF in the treatment of neuromuscular autoimmune disease have been reported. We report 2 cases of TE associated with immunosuppressive treatment of neuromuscular disease with long-term MMF therapy. Examination was notable for expressive aphasia, right-sided hemiparesis, and right-sided hyperreflexia. CT of the chest, abdomen, and pelvis was unremarkable. Laboratory tests showed normal leukocyte count. T gondii immunoglobulin Ig G and IgM serum antibodies were positive. Serum HIV test was negative. CSF showed scattered, mature-appearing lymphocytes. CSF Gram stain was unremarkable. CSF acid-fast bacilli smear and culture were negative. CSF bacterial and fungal culture was negative. CSF histoplasmosis and cryptococcus serologies were negative. MMF was discontinued and he was treated with pyrimethamine 50 mg daily, sulfadiazine 1, mg 4 times per day, and leucovorin 25 mg daily for 2 months without significant improvement. He died 2 months after the onset of his symptoms.

6: Best 18 Neurosurgeons in Oneida, NY with Reviews - www.enganchecubano.com

Myasthenia gravis is an autoimmune neuromuscular disorder caused by circulating antibodies that block acetylcholine receptors at the postsynaptic neuromuscular junction.

7: Papers with the keyword myasthenia gravis recognition (Page 2) | Read by QxMD

Two patients with neuromuscular disorders on immunotherapy with long-term MMF who developed toxoplasmic encephalitis (TE) were included in this case series. Results: One patient with myasthenia gravis and one patient with inflammatory myopathy on immunotherapy with long-term MMF developed severe TE.

8: Toxoplasmic encephalitis during mycophenolate mofetil immunotherapy of neuromuscular disease

Electrophysiological studies in Neuromuscular Junction Disorders Single-fiber electromyography The selectivity of the technique results from the small recording surface (25 Î¼m in diameter), which is exposed at a port on the side of the electrode, which is 3 mm from the tip [Figure 2].[23].

9: Clinical Trials | BioPharma Dive

Myasthenia gravis is the most common disorder of neuromuscular junction transmission and is also one of the best characterized autoimmune diseases. However, its symptomsâ€”primarily weaknessâ€”vary from patient to patient, and in the same patient, by time of day and over longer time periods.

IMMUNOTHERAPY IN NEUROMUSCULAR DISORDERS GLADY JACOB pdf

Sap hr security material Upton Sinclair finds God Program review of past and existing global payment programs. Subnautica guide xbox one Mathematical reasoning Chris Ninness . [et al.] Get Ready for First Grade! Water for every farm V. 14 : Innovations in technique The glitch in sleep Longman English-Portuguese/Portuguese-English School Dictionary for Brazil Pursuit of Meaning The peace wheel : nonviolent activism in the Buddhist tradition Christopher S. Queen The exhibitions at Harvard College founded prior to 1800. The lost musicians. Zebra Finches Complete Owners Manual Mausoleum of Henry and Arabella Huntington Changing Genders in Intercultural Perspectives (Studia Anthropologica) Encyclopedia of american religion and politics Epilogue : falling down, reaching for the stars, and coping with feelings Travels with a Primate Tables for the computation of the Jupiter perturbations of the group of small planets whose mean daily mo Managing Organizational Behavior Fifth Edition Experiences in Organizational Behavior Third EDI A nice day for murder Outlines Highlights for Human Development Across the Lifespan Hawaiian money 2nd edition Adele sheet music piano Product of two binomials worksheet How To Mesmerize Animals Pamphlet Part IV: Wisdom in society Three men and Jennie Law and the Social Order Student study guide with solutions for vector calculus Rs aggarwal maths book class 12 solutions The Handbook of Chemistry and Physics on CD-ROM Version 2004 Jack in the water, or, The ladder of life Radiology typists handbook Automated systems based on human skill The Twin Pillars of the Christian Life Smoke Rings and Roundelays Pipes and Tobacco Andy mcnab nick stone series