01, 02 e 03 de outubro de 2015 - Dourados - MS - Brasil

Natureza do trabalho: Resumo Revisão de Literatura

TÍTULO

OVERVIEW OF MULTIPLE SCLEROSIS AND ITS TREATMENT USING INTERFERON BETA

RUBIANI ANDRESA PARIZOTTO[,] ANGÉLICA CRISTINA MILAN MARESE, LEANDRO SILVA PIVATO, PRISCILLA MILAN MORENO MAZUROSKI, JÉSSICA VENCATTO SENEM

FACULDADE ASSIS GURGACZ, FAG, CASCAVEL, PR, BRASIL

HOSPITAL CARDIOLÓGICO COSTANTINI, CURITIBA, PR, BRASIL

RESUMO

Introduction: Multiple sclerosis (MS) is a chronic and progressive inflammatory disease of the central nervous system. It is most frequently found in women aged between 20 and 40. In Brazil, there are about 35,000 people with MS; the highest incidence is in the south and southeast of the country. Objectives: This paper outlines the general aspects of MS and highlights the improvements in treatment which have been achieved. Materials and methods: The data were collected from scientific articles published between 2000 and 2015 through the CAPES, MedLine and PubMed platforms of national and international research. The following keywords were used: multiple sclerosis, interferon beta, and treatments. Results and discussion: It is believed that MS is the result of a combination of genetic predisposition and an unknown environmental factor, which causes a self-injurious character dysfunction of the immune system in relation to white matter. This results in the loss of oligodendrocytes and myelin and, consequently, failures in the conduction of nerve impulses, which leads to the appearance of symptoms. This demyelination affects multiple regions of the neuraxis, which explains the variety of clinical manifestations of MS. Diagnosis is performed by imaging and clinical manifestations. The relapsing-remitting form of disease is prevalent among patients with MS; it occurs in 70-80% of cases and is characterized by exacerbations followed by a variable degree of improvement of the neurological deficit, which may be complete or which may evolve as a residual symptomatic dysfunction. Treatment with beta interferon (IFN β) has shown success in the remission of symptoms. These proteins act in cellular, antiviral and antiproliferative functions and also in immunoregulation. IFNB continues to be one of the most used medicines in relation to the relapsing-remitting form of MS due to its ability to modulate the activity of T and B cells, its effects on the blood-brain barrier, and its neuroprotective role, which induces the release of growth factor of astrocytes or enhances the protection of the neurons themselves. IFN β is used to treat outbreaks; to prevent future exacerbations and the subsequent progression of disease; and also in the treatment of complications. Although the mechanisms by which IFN β achieves its therapeutic effects are poorly understood, this drug has a good safety profile, transient side effects, and it is well tolerated by patients. Studies have shown that the use of IFNB in patients with the relapsing-remitting form of MS showed a tendency to reduce the frequency and severity of outbreaks and a slower progression of the disease in a significant proportion of treated patients. Final considerations: MS is a treatable disease and neurological disabilities can be prevented or delayed with the use of specific drugs. Due to the increasing use of IFN β in the treatment of MS, it is recommended that further research and more case studies are performed to gain additional information about progress in treatments using IFNβ, as well as carrying out close monitoring of these patients in order to prevent the complications described in the literature.

Acknowlegments

The authors appreciate the assistance and support of the Faculdade Assis Gurgacz (FAG), Cascavel, Brazil.

Anais do 3º Simpósio Internacional de Neurociências da Grande Dourados - SINGraD - 2015

01, 02 e 03 de outubro de 2015 – Dourados – MS - Brasil

References

ABREU, P; MENDONÇA, M.T; GUIMARÃES, J; SÁ, M.J. Esclerose múltipla: Epidemiologia, etiopatogenia, fisiopatologia e diagnóstico diferencial. Revista Sinapse, nov. de 2012; 2 (12), (supl. 1): 5-14.

AXTELL R.C; RAMAN C; STEINMAN L. Interferon- β exacerbates Th17-mediated inflammatory disease. Trends Immunol. 2011; 32(6): 272-277.

BICHUETTI, D.B; FALCÃO, A.B; BOULOS, F.C; MORAIS, M.M; LOTTI, C.B.C; FRAGOMENI, M.F.C; SOUZA, N.A; OLIVEIRA, E.M.L. The profile of patients followed at the Neuroimmunology Clinic at UNIFESP: 20 years analysis. Arq. Neuropsiquiatria 2015; 73 (4).

BICHUETTI, D.B; OLIVEIRA, E.M.L; OLIVEIRA, D.M; SOUZA, N.A; GABBAI, A.A. Neuromyelitis optica treatment: ana.lysis of 36 patients. Arch Neurol. 2010; 67(9): 1131-1136.

COMPSTON A; COLES, A. Multiple sclerosis. Lancet 2008; 372(9648): 1502-1517.

CREE B.A; LAMB S; MORGAN K; CHEN A; WAUBANT E; GENAIN C. An open label study of the effects of rituximab in neuromyelitis optica. Neurology. 2005; 64: 1270 -1272.

CRUZ, B.A.; QUEIROZ, E.; NUNES, S.V.; CRUZ FILHO, A.; CAMPOS, G.B.; MONTEIRO, E.L.C.; CRIVELLARI, H. Fenômeno de Raynaud grave associado a terapia com interferon beta para esclerose múltipla. Relato de caso. Arq Neuropsiquiatr 2000; 58(2-B): 556-559.

DHIB-JALBUT, S.; MARKS, S. Interferon beta mechanisms of action in multiple sclerosis. Neurology 2010; 74(Suppl 1): S17-S24.

MENDES, A.; SÁ, M.J. Classical immunomodulatory therapy in multiple sclerosis. How it acts, how it Works. Arq Neuropsiquiatr 2011; 69(3): 536-543.

MOREIRA, M.A; FELIPE, E; MENDES, M. F; TILBERY, C. P. Esclerose múltipla: Estudo descritivo de suas formas clínicas em 302 casos. Arq. Neuropsiquiatria 2000; 58 (2B): 460-466.

POPPE, A.Y; LAPIERRE, Y; MELANCON, D; LOWDEN, D; WARDELL, L; FULLERTON, L.M; BAR-OR, A. Neuromyelitis optica with hypothalamic involvement. Mult Scler 2005; 11: 617–621.

ROSA, D.J.F; MATIAS, F.A.T; CEDRIM, S.D; MACHADO, R.F; SÁ, A.A.M; SILVA, V.C.P. Erupção acneiforme aguda induzida por interferon beta-1b durante tratamento para esclerose múltipla. An Bras Dermatol. 2011; 86(2): 336-338.

TILBERY, C.P; FELIPE, E; MOREIRA, M.A; MENDES, M.F; FRANÇA, A.S. Interferon beta 1-A na esclerose múltipla. Experiência de um ano em 62 pacientes. Arq Neuropsiquiatr 2000; 58(2-B): 452-459.