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Review Article

Brief Review of the Etiopathogenics Aspects in Multiple Sclerosis

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Abstract

Multiple Sclerosis is an autoimmune, inflammatory and degenerative disease of the central nervous system. Three hypotheses have been postulated in order to explain their etio pathogenesis: 1) The existence of a persistent viral infection; 2) The presence of an autoimmune process with loss of the to-lerance toward antigens of the myelin; and 3) The presence of a phenomenon of molecular mímic between virales antigens and proteins of the myelin. Exists a generalized consensus of which this secondary mechanism is probably found to be represented by an autoimmune reaction that attacks some of the protein components of the myelin. The first event in the pathogenesis of multiple sclerosis is the activation of autoreactive T cells outside the central nervous system, either specify, through molecular mechanisms as a consequence of infections, or else nonspecifically through mechanisms middle by cito-quins or other T cells.

Keywords: Multiple Sclerosis; T Cells; Autoimmune; Pathogenesis

Introduction

Multiple Sclerosis (MS) is an autoimmune, inflammatory and degenerative disease of the central nervous system. In its etiology intervene environmental and genetic factors [1]. It is one of the most frequent neurological disorders in the young adult of non-traumatic etiology, that affects 2.5 million people in the world. The majority of the patients show their first symptoms between 20 and 40 years, very few cases are developed in the puberty or pasts the 60 years [2-5].

MS affects the female sex in a proportion of 3: 1 in comparison with the masculine one. Furthermore, the disease is 5 times most frequent in temperate climates than in the tropical ones. In Europe are diagnosed approximately 10000 annual cases [6].

Even though the exact cause of the MS is unknown, many scientists think that the destruction of the myelin is due to certain immunity altered in genetically susceptible individuals, this immunity is probably induced by environmental exposure to specific pathogens, particularly virus and among them Epstein-Barr.

Discussion

Three hypotheses have been postulated in order to explain the etio¬pathogenesis of MS: 1) The existence of a persistent viral infection; 2) The presence of an autoimmune process with loss of the tolerance toward antigens of the myelin; and 3) The presence of a phenomenon of molecular mímic between vi-rales antigens and proteins of the myelin. Even though numerous indirect evidence favors the existence of a viral cause as the one responsible for the onset of the disease, up to the no present virus has been isolated from tissues of the totality of patients with MS [7-9].

On the other hand, some agents viral postulates inclement as causes of the disease have been isolated in individuals that did not suffer MS. If actually occurs a viral infection of the central nervous system (CNS) during the infancy, then during the puberty or adult

age some other secondary factor it should trigger the disease or else to exacerbate it.

Exists a generalized consensus of which this secondary mechanism is probably found to be represented by an autoimmune reaction that attacks somes of the protein components of the myelin [9]. Different observations support this possibility: 1) The anatomicopathological characteristics of the demyelinating injuries. In them is possible to observe: the presence of perivascular inflammatory, presence of lymphocytes and macrophages activated, complement united to the macrophages, phagocytosis of fragments of myelin, expression of the complex greater of histocompatibility class II in astrocitos and endothelial cells, existence of plasmatic cells and finally presence of lymphocytes T associated to stress proteins in the chronic injuries; 2) The similarity of the post-vaccinates injuries observed in MS and the ones that become evident in encephalomyelitis and experimental allergic ence-falitis; 3) The presence in these patients of anomalies both in lymphocytic T and in the immunoglobulins; 4) The increase in the activity of the disease in patients that have received interferon-γ (IFN-γ); 5) the presence of an immunogenetic «background» in patients that present MS [10].

The evidence previously presented permit pustular the following model physio pathogenic: Is accepted that specific potentially autogenesis's T cells against antigens of the myelin, exist normally in the immune system. Such cells have escaped from thymic mechanisms of control for example the deletion clonal. The first event in the pathogenesis of MS is the activation of these autoreactive cells outside the CNS, either specify, through mechanisms of molecular mímic as a consequence of infections, or else nonspecifically through mechanisms middle by cytokines or other T cells. As a consequence of the activation, the lymphocytes T acquire the capacity to be expanded, cause different cytokines, and increase the expression of adhesion molecules on its surface. This alternative permits to T lymphocytes to adhere to the endothelial cells that express the adequate against-receptors, to pass through the perivascular space and to reach thus the CNS [11,12].

The T cells that reach the `CNS` recognize the antigen specific united to the greater complex of present histocompatibility in astrocytes or cells of the microglia, and are in this way re-activated. This phenomenon of re-activation implies the production of different cytokines and inflammatory mediators such as prostaglandins, free radicals, or nitric oxide [13].

The certain cytokine secretion during the course in MS is found to be implied both in the induction and in the regulation of the disease. Different evidence suggest that IL-2, IFN-γ, y TNF-α/β can mediate inflammatory responses and the tissue damage observed in MS. Inversely IL-4, IL-10 and TGF-ß are associated with an inhibition of the immune response in the CNS. The secretion of such cytokines can come from cells CD4+ that have entered the CNS from the periphery after specific activation, of cells recruited secondaries, or else of living cells glial [14].

Adicional cells CD4+ can contribute to the activation of B Cells and consequently to the production of antibodies against different components of the myelin. Different studies have demonstrated that the macrophages not only produce DE myelinization through mechanisms of phagocytosis of the myelin but also through the liberation of complement and inflammatory mediators such as cytokines, toxic metabolites of the oxygen and eicosanoids. These factors can in turn stimulate other cells and to contribute to local tissue damage affecting both the myelin and the oligodendrocytes and increasing the permeability of the hematoencephalic barrier, allowing thus the influence of greater number of cells which increase the inflammatory reaction.

Which are the mechanisms by which the immunological factors, and consequently the clinical manifestations, can be limited in EM? Exists sustanciales evidence that suggests that during the recuperation phase in MS exists an increase in the anti-inflammatory cytokine secretion: IL-10 y TGF-β. It has in addition been suggested that the local apoptosis of the T cells is an important factor in the confinement of the immunological response. The principal physiological effect of the phenomenon of demyelination is the limitation in the management of the electric impulse saltatorial from a node of Ranvier, to the next node. Such limitation in the transmission of the nerve impulse can be manifested either as a decrement in the speed of management, deficiency in order to transmit potentials for action to frequencies discharges or else by a blockade total of the management [11].

Other alterations in the management of the Tambien nerve impulse are feasible to be observed in de myelinated fibers. For example the generation of ectopic potentials for action, or else the existence of fiber-fiber stimulations anomalous (efatic transmission). The dysmyelinated fibers also are capable of generating the

reflection of certain impetus that collisional with incentives orthodromic and produce the abolition of the normal traffic of the nerve impulse. This series of defects in the transmission of the nerve impulse mediate the majority of the clinical anomalies observed in demyelinating diseases [13,15].

In spite of the foregoing other authors in addition describe the possibility of the action of a related secondary factor with an antiaging gene that is associated with autoimmune disease and multiple sclerosis and the diets (high calorie, xenobiotics) that suppress the gene with relevance to autoimmunity [16,18].

Conclusions: The current studies emphasize the presence of an autoimmune phenomenon in the etiopathogeneses of multiple sclerosis, resulting in the symptomatic variability, the type of disease and the evolution of the disease.

Conflict of Interest

None to declare.

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