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REVIEW

Encefalitis de Rasmussen: una encefalitis autoinmune rara

Rasmussen's Encephalitis: a rare Autoimmune Encephalitis

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ABSTRACT

Introduction: Rasmussen's encephalitis is a type of autoimmune encephalitis unusual in adults, an inflammatory, chronic and neurodegenerative brain disorder that manifests itself with treatment-resistant neocortical focal motor seizures. The pathophysiology of this disease has involved viral infections and the existence of antibodies, among other possible etiologies. Due to pharmacological resistance to anticonvulsants, management with immunoglobulin, in most patients, demonstrates clinical improvement and decreased progression. However, the only therapeutic management that seems to give accurate results to date is hemispherectomy.
Objective: to describe the risk factors, symptoms and treatments of Rasmussen's Encephalitis.
Method: as it is a disease with scarce bibliography and occasionally rare diagnosis, the study was carried out under a cross-sectional, historical, statistical search supported by a comparative review in the various national and international databases.
Conclusions: the authors found a certain discrepancy in certain aspects, an element that, together with the magnitude that this pathology can have and its consequences, reflects the need for study and knowledge on the part of professionals and the health team for an accurate diagnosis in time.

Keywords: Hemispherectomy; Glutamate; Rasmussen's Encephalitis.

RESUMEN

Introducción: la Encefalitis de Rasmussen es un tipo de encefalitis autoinmune inusual en los adultos, un trastorno cerebral inflamatorio, crónico y neurodegenerativo que se manifiesta con crisis focales motoras neocorticales resistentes al tratamiento. Dentro de la fisiopatología de esta enfermedad se han involucrado infecciones virales y la existencia de anticuerpos, entre otras posibles etiologías. Debido a la resistencia farmacología ante los anticomiciales, el manejo con inmunoglobulina, en la mayoría de los pacientes, demuestra una mejoría clínica y disminución de la progresión de la misma. Sin embargo, el único manejo terapéutico que hasta la actualidad parece dar resultados certeros, es la hemisferectomía.
Objetivo: describir los factores de riesgo, síntomas y tratamientos de la Encefalitis de Rasmussen.
Método: al ser una enfermedad con escasa bibliografía y diagnóstico ocasionalmente raro el estudio fue realizado bajo una búsqueda transversal, de corte histórico, estadístico apoyado en una revisión comparativa en las diversas bases de datos tanto nacionales como internacionales.
Conclusiones: fue encontrado por los autores cierta discrepancia en ciertos aspectos elemento que junto a la magnitud que puede llegar a tener dicha patología y sus consecuencias refleja la necesidad de estudio y conocimiento por parte de los profesionales y el equipo de salud para un diagnóstico certero a tiempo.

Palabras clave: Hemisferectomía; Glutamato; Encefalitis de Rasmussen.

INTRODUCTION

Rasmussen's syndrome was first described in 1958, by a physician who named the entity after his surname, studying patients who presented with continuous partial seizures and a neurological deficit. It is an autoimmune encephalitis that mostly affects only one cerebral hemisphere. The nomenclature of the disease has always been controversial, but the latest proposal is to call it Kozhevnikov-Rasmussen syndrome, as an equivalent of the old Kozhevnikov syndrome. Typically, it usually starts in childhood with an incidence of up to 2,4 cases per 10 million children per year. Onset in adolescence or adulthood is rare, with this variant known as late-onset Rasmussen's encephalitis.[1,2,3]

A multitude of etiopathogenic hypotheses have been elaborated in the development of the disease such as infection by different neurotropic viruses or the possibility of an autoimmune process as it has been demonstrated in some patients the existence of antibodies against the brain glutamate receptor 3 (anti-GluR3) or anti-glutamic acid decarboxylase antibodies (anti-GAD), or the increase of T cell mediation in the cerebral inflammatory process.[1,2]

The only currently effective treatment is functional hemispherectomy, which is usually applied in early stages of the disease, prior to neurological deterioration. Due to the implications of autoimmune phenomena in the development of the disease, treatments aimed at modulating the cerebral inflammatory reaction, such as corticoids, immunoglobulins, plasmapheresis or interferon, began to be applied, with good results in some cases, but always characterized by the disappearance of the effect when the treatment is withdrawn.[1,2,3]

Scientific Problem: to reach an accurate description about this rare disease by means of a comparative bibliographic research.

Objectives: to describe the risk factors, symptoms and treatments of Rasmussen's Encephalitis; furthermore, the long-term effects of hemispherectomy in patients with the disease were explored, including the evolution of quality of life, cognitive effects and the emotional and social impact of the surgery.

METHOD

Systematized search protocol: reproducible search algorithm in electronic databases, in guideline producing or compiling centers, systematic reviews, clinical trials, diagnostic test studies, observational studies on specialized websites and manual literature search. Emphasizing a time limit of about 5 years in the publication of the literature consulted, which guaranteed the novelty of the information collected. The review was cross-sectional, historical, statistical, supported by a comparative research model, through the consultation of national and international databases, such as: Scopus, ENFISPO, Cuiden, MEDES, Scielo, Cochrane Plus and PubMed with no defined consultation limit.

DEVELOPMENT

Autoimmune encephalitis (AE) is a group of inflammatory diseases of the central nervous system in which the immune system mistakenly attacks the body's own proteins, in this case neurotransmitter receptors or neuronal surface proteins.[4] The clinical syndromes are complex and are associated with manifestations that vary according to the type of associated antibody. The autoimmune response may be triggered by the presence of a tumor or a viral infection, but in most cases the cause is unknown. In children and adolescents the symptoms are usually different from adults and the disease is rarely associated with tumors. The form of clinical presentation varies according to age; in children the predominant symptoms at the onset are epileptic seizures, abnormal movements, irritability, sleep disturbances, memory problems and behavioral disturbances.[4]

The diagnosis of AD in children is a real challenge and is delayed by several factors inherent to the clinical picture and laboratory diagnosis, as well as to the characteristics of children such as the behavioral changes of age and the difficulty in describing their complaints, and the fact that not in all cases of patients with a clinical picture of AD autoantibodies are detected in blood or cerebrospinal fluid.[4]

In adolescents and young adults, behavioral alterations and psychosis predominate; in patients over 45 years of age, memory deficits and behavioral alterations predominate.[1,2,4]

In general, the treatment of AD is based on immunotherapy with corticosteroids, plasma exchange or immunosuppressants, which in most cases is administered empirically since there are no specific biomarkers to guide therapy and there are no biomarkers to predict the short or long term outcome.[4]

Rasmussen's syndrome

An inflammatory, chronic, progressive brain disorder manifesting with treatment-resistant neocortical motor focal seizures and culminating in severe impairment with hemiparesis, cognitive delay and aphasia with

autoantibodies against R3 glutamate receptors. The disease mainly affects children and debuts with a prodromal period with mild hemiparesis or infrequent seizures lasting up to several years.[5] The most typical seizure is continuous partial epilepsy characterized by uncontrolled movements of a hand or a foot for days, weeks or months, although other types of focal seizures or simply convulsions may occur, the seizures are daily and repeated, especially numerous during the day and rebellious to any treatment. Slowly progressive weakness of one side of the body appears leading to paralysis of an arm or a leg, accompanied by homonymous hemianopsia in half of the cases, dysarthria and dysphasia, behavioral and learning problems and often mental retardation. Histologically it is characterized by perivascular lymphocyte leakage, microglial nodule proliferation, neuronal loss and gliosis.[6,7]

Glutamate receptors

Recall first that glutamate, a nonessential amino acid, is involved in several metabolic pathways. However, in the central nervous system, its role is to facilitate and expedite communication between the various nerve cells through contacts known as synapses.[8]

Receptors are a type of molecules specialized in capturing the main excitatory neurotransmitter of the brain, glutamate, are classified into two families ionotropic and metabotropic, and mediate most of the excitatory synapses of the Central Nervous System, It is the main mediator of sensory, motor, cognitive and emotional information and is involved in the formation of memories and their retrieval. As if all this were not enough, it is also involved in neuroplasticity, learning processes and is the precursor of GABA. The process of glutamate synthesis starts in the Krebs cycle whose main precursor will be alpha-ketoglutarate, which will receive an amino group to become glutamate.[9] It is important to highlight that most neurodegenerative disorders are associated with alterations in the concentration of certain neurotransmitters, among them glutamate. For this reason, a finely coordinated bidirectional glial/neuronal communication is necessary for the proper transduction of signals by this neurotransmitter.[9]

Another significant precursor should also be mentioned: glutamine. When the cell releases glutamate into the extracellular space, the astrocytes recover this glutamate which, by means of an enzyme called glutamine synthetase, becomes glutamine. Then the astrocytes release this glutamine, which is recovered again by the neurons to be transformed into glutamate. The receptors are located in the cell membrane, they are frequently located in the postsynaptic cell and as I mentioned before there are two main types: ionotropic and metabotropic. The ionotropic ones are those in which when their ligand binds they open channels that allow the passage of ions into the cell. Metabotropics, when their ligand binds, cause changes in the cell by means of second messengers.[9]

Epidemiology

This is a very rare disease with slightly more than 200 cases described in the literature. There is one record reported in the literature that corresponds to Germany, with an annual incidence of 2,4 cases/10 million people; on the other hand, another study shows that there are only about 100 case reports and that they are somehow related to infection or brain malformations. The onset of the disease is rare in adulthood and there are no gender differences. It occurs predominantly during childhood, the usual age of onset is 1 to 10 years, with a peak at 6-7 years of age and with an estimated frequency of 2,4 cases per 10 million people under 18 years of age. About 50 % of patients have a history of an infectious or inflammatory episode within the six months prior to disease onset.[10,11] This element is much discussed throughout the literature consultation, there is discrepancy between the various professionals and the studies found.

Etiology

The etiology of Rasmussen's encephalitis is unknown, although there is evidence of cytotoxic T lymphocyte-mediated reactions against neurons in its pathogenesis. There are no reports of genetic, seasonal or clustering causes for its occurrence.[11]

Recognizing that over time some authors have reported the possibility of a viral origin, but in situ hybridization and polymerase chain reaction studies have failed to identify a viral agent. However, Rasmussen proposed a viral etiology for the syndrome based on the immune reaction found in the necropsies, subsequently no virus was isolated and serological studies were inconclusive. Subsequently the autoimmune hypothesis began to gain strength, specifically that of anti-glutamate receptor (GLuR3) antibodies that were shown to be able to provoke a response similar to that of Rasmussen's encephalitis in rabbits, but nevertheless there are patients in whom it has not been possible to demonstrate a relationship with them and this antibody has also been detected in patients suffering from other epileptic syndromes, so the autoimmune response has not been ruled out but other antibodies continue to be sought. Serum IgG autoantibodies against a7 nicotinic acetylcholine receptors (a7nAChR) have also been described in a subset of patients with Rasmussen's encephalitis, which produce a specific blockade of neuronal function that is a regulator of the blood-brain barrier, which is probably impaired

in patients with encephalitis.[13]

Other ideas have been proposed such as genetic origin, encephalopathy secondary to a baseline focal epilepsy, or even focal epilepsy as a gateway for a focal immune response to be triggered.[11,12,13,14] Research shows high discrepancy in the literature.

Atypical presentation

When we refer to atypical forms, it is worth highlighting the occurrence of this disease in adolescents and young adults as the disease seems to acquire a much more prolonged process, with fewer residual lesions, more occipital onset seizures and lesser degree of hemiatrophy.[15,16] An article found reported an atypical presentation where the case was initially presented with unilateral complete cortical hemispheric edema (CUBE - Cortical Unihemispheric Brain), which has been described in the context of hemiconvulsion-hemiplegia epilepsy syndrome (HEE) or in Rasmussen's encephalitis.[15] On the clinical side, focal status epilepticus classified as continuous partial epilepsy was evidenced, as was a focal neurological deficit, in this case it is right hemiplegia and with some degree of language involution.[15]

Diagnosis

Different elements are collected, throughout the literature reviewed, which yield positivity and increase diagnostic suspicion. However, in a more generalized way they can be summarized as follows: clinical: focal seizures (with or without continuous partial epilepsy) and unilateral cortical deficit. Electroencephalogram: hemispheric slowing with or without epileptiform activity and unilateral onset seizures. MRI: with focal hemispheric cortical atrophy and at least one of the following: hyperintense T2 / FLAIR signal of gray or white matter, or hyperintense signal or atrophy of the ipsilateral caudate head.[17,18]

At the time of performing an MRI on these patients we can observe an increased signal on the affected side of the brain, as well as a decrease in the volume of the cerebral cortex.[17]

As for the clinical presentation, 3 stages are described, a prodromal period in which mild hemiparesis and frequent crises occur, which may precede the acute stage with an average of 7 months, but may last for years. Then follows the acute stage which is characterized by progressive focal seizures, about 50 % develop continuous partial epilepsy and as the disease progresses, focal seizures of different semiology appear, being the most frequent the motor focal seizures that can extend to be bilateral, they can also originate in the supplementary motor area and in the somatosensory cortex.[17,19]

In adult and adolescent cases in which this disease is atypical should be considered in case patients with pharmacoresistant focal epilepsy evolve to continuous partial epilepsy, considering that focal neurological deficits, cognitive impairment and typical neuroimaging findings might not be present or be of scarce presentation.[19,20]

Treatment

Treatment is generally aimed at reducing the severity and frequency of crises. Studies were found that show positive results with the use of injected butolytic toxin. Antiepileptic drugs are usually ineffective and surgery with hemispheric disconnection is the treatment of choice to control epilepsies and prevent cognitive impairment, case studies and retrospective analyses have established a multimodal treatment, as in other autoimmune encephalopathies involving high doses of corticosteroids or immunoglobulins, plasmapheresis or immunoadsorption have also been used in few. Therapeutic apheresis may also offer a rapid response for severe neurological symptoms. Immunoadsorption is increasingly replacing plasmapheresis because of its safety and greater selectivity in replacing plasma products, increasing knowledge about the pathogenic relevance of autoantibodies.[17,21]

It is important to highlight that, although the studies consulted disagree among themselves, it is certain that antiepileptic drugs interfere in the brain maturation process, as well as in the frequency of seizures, which is why the development of refractory epilepsy at early ages, with polytherapy schemes, is a solid indication for early surgical treatment.[22,23]

Surgical Treatment

The surgical treatment option, especially hemispherotomy, is currently the most effective therapeutic option to achieve seizure control. The decision to perform hemispherotomy in patients with RD lies primarily in the need to control seizures refractory to pharmacological treatment.[24]

Hemispherectomy

Consisting of the removal or disconnection of one of the cerebral hemispheres, it is highly invasive and is only performed in very particular situations. It is mainly used to treat the most extreme cases of epilepsy, as it is so invasive it cannot be used in all types of patients, there are requirements and one of them is not to have

improved with the previous treatment or the rest of available treatments. The age is key to decide whether to carry out this operation or not, the younger the patient is, the more likely he/she is to present fewer sequels, this is due to brain plasticity, that is why the best candidates are children. Since young children have not yet completed many neuronal connections, they start with certain advantages, since the remaining hemisphere will be able to assume a large part of the tasks that, in the general population, would be distributed in both hemispheres of the brain.

In spite of the invasiveness of this technique, the average success rate is very high, which is logical since the cerebral hemisphere that is causing the seizures is removed. It is also observed that, in patients so affected by the severity of epilepsy, Hemispherectomy generates an important improvement in their capacity to carry out cognitive processes, the first time a Hemispherectomy was performed was in 1928.

There are two types of hemispherectomy, anatomical and functional. Anatomical hemispherectomy consists of directly removing the damaged hemisphere. Functional hemispherectomy, instead of removing the entire hemisphere, the parts of the brain tissue where the origin of the epilepsy is located are sectioned. Logically, this option has certain advantages, such as the non-elimination of brain structures that may be performing crucial tasks.

But these are not the only ways to perform such intervention, in recent times a methodology known as peri-insular hemispherectomy has been perfected. This technique involves the removal of a minimal part of the brain where the seizure focus is located.

After a hemispherectomy, children suffer from neurological complications, so a plan must be made to evaluate the impact of the procedure on the child.

In general, and based on a study we found, we reflect that hemispherectomy is a reliable and effective technique for the management of pediatric and adult patients with refractory epilepsy who present cerebral hemispheric damage. Postoperative results demonstrate low morbidity and mortality in selected patients. Our data show positive effects on postoperative evolution in terms of seizure control.[25]

Possible negative results after surgical treatment

The most significant visual impairment a child will have following hemispherectomy will be homonymous hemianopsia, a type of cortical visual impairment, the loss of half of the visual field on the same side in both eyes opposite the hemisphere removed. Visual processing is also affected because, due to the nature of the surgery, occipital lobe connections are disconnected from other parts of the brain, which can cause visual processing impairments.[26]

Hearing challenges after hemispherectomy are not fully studied, most children will pass a typical hearing test because the remaining hemisphere can tone separately in each ear during the test, however, when only one hemisphere is required to process those sounds that go to both ears there can be problems, many children face central auditory processing disorders, hyperacusis or misophonia.[26]

Since surgery disconnects or removes the upper motor neurons on one side of the brain that are responsible for approximately 90 % of the movements on the opposite side of the body, hemiparesis will occur on the opposite side. Existing connections to motor neurons in the remaining part of the brain make it possible to regain some movement, including the ability to walk or use the affected hand as an aid or for some bimanual activities.[26]

Our senses help us to understand the world around us, the brain through its elaborate connections processes sensory input from the environment, as the process disconnects or removes the sensory area on one side of the brain, proprioception, sensation and kinesthesia can be profoundly affected.

According to studies show that the IQ of a child after hemispherectomy is in the range of 70, after this procedure infants may have learning problems in specific fields of information processing, such as problems in understanding complex and abstract concepts, difficulties in retrieving stored information, while growing up they may have a slow development in certain fields of language.[25]

When patients are properly selected, hemispherectomy is an effective treatment for pediatric epilepsy resulting in marked dysfunction of a single cerebral hemisphere, not only providing the patient with a high probability of seizure freedom, but also providing an improvement in motor and cognitive skills.[26]

CONCLUSIONS

Rasmussen's syndrome is a rare autoimmune encephalitis, an underdiagnosed disease due to its infrequency of presentation and the few documented cases. It is a disease that tends to incite debate because there are evident discrepancies in several important points of the disease. These elements plus the sum of the possible complications and their fatal outcomes are more than enough to emphasize the need for the study of the same and its deepening, by the team of health professionals in anxious for an accurate diagnosis in time from childhood.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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