Central Nervous System Involvement in Chronic Inflammatory Demyelinating Polyradiculoneuropathy: A Clinical, Neurophysiological and Radiological Study

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ABSTRACT

Background: The frequency of the association between CIDP and CNS lesions is probably underestimated. Objective: To assess the frequency of CNS involvement in CIDP patients, and to study the characteristics of this possible association. Methods: Forty patients (20 males, and 20 females) aged between 19 and 50 years (mean 33.12±9.3 years) fulfilling the clinical, neurophysiological and CSF criteria of INCAT for the diagnosis of CIDP were submitted to complete general and neurological assessment, laboratory investigations, CSF analysis, neurophysiological evaluation [NC studies, evoked potentials (VEPs, BAEPs, SSEPs)], and MRI brain and spinal cord. Results: Clinical evidences of CNS involvement were recorded in 12 patients (30%) of CIDP patients, abnormally delayed VEPs latencies were recorded in 16 patients (40%), abnormal BAEP latencies in 12 patients (30%), abnormal SSEP latencies in 22 patients (55%), and abnormal latencies in more than one modality in 13 patients (32.5%), MRIs brain and spinal cord were abnormal in 10 patients (25%). CIDP patients with clinical and/or radiological evidences of CNS involvement had a significantly younger age of disease onset, more frequent relapsing-remitting pattern of the disease course, more prolonged disease duration, and less favorable response to therapy than those without evidences of CNS involvement. CIDP patients with delayed evoked potentials' latencies and/or MRI demyelinating lesions were more frequent in CIDP patients with clinical evidences of CNS involvement. Moreover, MRI lesions were more frequent in those having abnormal visual evoked potential responses. Finally, there was a percentage of CIDP patients who showed a subclinical central neurophysiological and/or radiological abnormalities. Conclusion: CIDP is frequently associated with various clinical, neurophysiological and radiological evidences of CNS involvement. MRI and evoked potentials are useful non-invasive techniques for demonstrating this association. (Egypt J. Neurol. Psychiat. Neurosurg., 2008, 45(2): 637-646)

INTRODUCTION

Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is an acquired, immune-mediated, slowly progressive, sometimes relapsing, steroid-dependent, demyelinating sensorimotor polyneuropathy, primary affecting the limbs.¹ The exact mechanisms that underlie the pathogenesis of CIDP remain unclear. However, based on the observations that a significant proportion of patients with CIDP are responsive to immunotherapy and that an inflammatory response is observed at sites of active disease, suggested its immune-mediated pathogenesis.²,³

Chronic inflammatory demyelinating polyradiculoneuropathy is regarded to be restricted to the peripheral nervous system (PNS). The usual explanation for this localization is that the immune response in CIDP is directed against an antigen confined to the PNS, analogous to experimental allergic neuritis, where lesions in the PNS induced form of the disease are confined to the PNS and P2 protein occurs. However, there are occasional pathologic and clinical reports⁴⁻⁹ of the coexistence of CNS and PNS lesions in cases resembling CIDP.
The frequency of this association between CIDP and central nervous system (CNS) demyelinating lesions is probably underestimated\textsuperscript{10,11}. Chronic inflammatory demyelinating polyradiculoneuropathy with central nervous system demyelinating lesions has recently been reported to mimic multiple sclerosis (MS). However, it is unclear whether sporadic reports of concurrent multiple sclerosis and chronic inflammatory demyelinating polyradiculoneuropathy represent coincidence or whether these two demyelinating disorders are pathologically related\textsuperscript{12,13}.

The main purpose of this study was to assess the frequency of the central nervous system involvement in CIDP patients, and to study the clinical, neurophysiological and radiological characteristics of the association between CNS involvement and CIDP.

**PATIENTS AND METHODS**

**Patients**

This study was included forty Egyptian patients (20 males and 20 females) fulfilling the clinical, neurophysiological and CSF criteria for the diagnosis of CIDP according to research criteria of diagnosis of CIDP reported from Inflammatory Neuropathy Cause And Treatment (INCAT)\textsuperscript{14} which are symptomatic motor and/or sensory neuropathy of unknown origin affecting more than one limb with an onset duration of two months or more. CSF analysis revealed that cell count <10/mm\textsuperscript{3} with or without elevated proteins. Electrophysiological data were consistent with a demyelinating polyradiculoneuropathy. No etiology of neuropathy was detectable. Patients with monoclonal gammopathy, HIV infection, metabolic, paraneoplastic, ischemic disorders or dysimmune disease such as sarcoidosis, lupus erythematosus or angiitis were excluded.

The age of the patients ranged from 19 to 50 years with a mean age of 33.12±9.3 years. Patients were randomly collected from the Neurology Outpatient Clinics, Cairo University, between October 2003 to October 2005.

**Methods**

Patients in this study were subjected to:

1. Complete general and neurological assessment.

2. Laboratory investigations including routine blood tests (CBC, ESR, blood sugar, liver and kidney function tests, lipid profiles, uric acid and calcium level). Serum protein electrophoresis, immune electro-phoresis, HIV and hepatitis C and B serology, tests for antinuclear antibodies were also done. CSF was analyzed for the presence of elevated proteins with normal other parameters (glucose, chloroid, cells).

3. Neurophysiological investigations:
   a. Nerve conduction velocities were tested in all patients using surface electrodes, motor and sensory conduction velocities, sensory and motor potentials, distal latencies and F-wave were all taken into account. Electrophysiological criteria of the INCAT for CIDP diagnosis were used.
   b. Evoked responses: including patterns reversal visual evoked potential (VEP), brain stem auditory evoked potential (BAEP), and short latency somatosensory evoked potential (SSEP), were done for all patients.

4. Radiological investigations: MRI of the brain and spinal cord with and without contrast were done using General Electric Signa Advantage 1.0-Tesla (T) system were carried for all patients at the Diagnostic Radiology Department of Kasr El-Aini Hospitals, Cairo University.

T\textsubscript{1} weighted images were obtained in the sagittal and axial plane, T\textsubscript{2} weighted images and fast-fluid acquisition and inversion-recuperation (fast-flair) images were obtained in the axial plane. MRI images were carefully analysed for the presence of any demyelinating plaques and were analysed for their number, pattern, size and distribution.

**RESULTS**

**Clinical Results**

**Disease Characteristics:**

The symptoms duration ranged from 6 months to 5 years with a mean of 2.3±1.9 years, the disease was gradual progressive in 26 patients (65%) and acute relapsing-remitting in 14 patients (35%). preceding infection was reported in 8 patients (20%), the number of the attacks was one in 26
patients (65%), and more than one attack in 14 patients (35%), first symptoms was sensory-motor in 28 patients (70%), motor only in 8 patients (20%), and sensory only in 4 patients (10%). 22 patients (55%) received steroids only, while 18 patients (45%) received steroids and others (plasmapharesis, immunosuppressant). 16 patients (40%) reported good improvement on treatment, 20 patients (50%) reported partial improvement, while 4 patients 10% showed no improvement.

**Clinical CNS involvement:**

Clinical criteria of C.N.S involvement were illustrated in Fig. (1) which revealed that clinical CNS involvement in 12 CIDP patients (30%), where 12 patients had cranial nerves affection [5 patients had optic atrophy (12.5%), 4 patients (10%) had nystagmus and 3 patients (7.5%) had unilateral 6th nerve palsy]. Pyramidal signs were observed in 10 patients (25%) [extensor planter in 4 patients (10%), and lost abdominal reflexes in 8 patients (20%)], sensory tract signs in 6 patients (15%) [4 patients (10%) with sensory level, 5 patients (12.5%) with posterior column signs], cerebellar manifestations (either alone or in association with sensory ataxia) were observed in 3 patients (7.5%). Finally precipitancy of micturition was reported by 6 CIDP patients (15%).

**Evoked potential results:**

The evoked potentials results were illustrated in Fig. (2) which revealed that 16 patients (40%) with CIDP had abnormal VEP latency, 12 patients (30%) had abnormal BAEP latency, 22 (55%) had abnormal SSEP latency, and 13 CIDP patients (32.5%) had abnormality in more than one modality.

**Radiological results:**

MRI brain and spinal cord were normal in 30 patients (75%), and abnormal in 10 patients (25%) in the form of hyperintense lesions in T2 weighted image (demyelinating plaques). Brain lesions were observed in 5 patients (12.5%), and spinal cord lesions in 2 patients (5%), combined brain and spinal cord lesions were observed in 3 patients (7.5%) (Table 1).

All brain lesions were bilateral, small, multiple, and periventricular in location, in addition to bilateral small, multiple, subcortical hyperintense lesions in 4 patients (10%), brain stem lesions in 3 patients (7.5%), (2 pontine and one mid brain), and cerebellar lesions in 2 patients (5%).

**CNS involvement and disease characteristics:**

CIDP patients with clinical and/or radiological evidences of CNS involvement had significantly earlier age of disease onset, more prolonged disease duration, more relapsing-remitting course, and less favorable disease outcome when compared with those without CNS affection.

**Clinical CNS involvement and evoked potential results:**

CIDP patients with clinical evidence of CNS involvement had a significantly more frequent abnormality in all modalities of evoked potentials studied (75%, VEP, 50% BAEP, 91.3% SSEP, and 75% more than one modality) (Fig. 3).

So a percentage of CIDP patients with clinical CNS involvement had normal evoked potential response, and on the other hand another percentage had a subclinical neurophysiological evidence of CNS involvement. Cranial nerves signs (optic nerve atrophy and nystagmus) were related mainly to abnormal VEP latency. Pyramidal signs, sensory tract signs and precipitancy were related mainly to abnormal SSEP latency.

**Clinical CNS involvement and radiological results:**

CIDP patients with clinical evidence of CNS involvement had significantly more patients with radiological evidence of CNS involvement than those without CNS involvement (6/12 (50%) and 4/28 (14.3%) respectively, P<0.01**. So [6/12 (50%) of patients with clinical CNS involvement had normal MRI, and 4/10 (40%) of patients had subclinical MRI findings (Table 2).

**Radiological CNS involvement and evoked potentials results:**

CIDP patients with MRI lesions had more significantly frequent abnormal visual evoked potentials latencies than those without MRI lesions. However a non-significant difference was observed regarding the BAEP, SSEP latencies. These results are illustrated in Table (3). So, a percentage of patients with radiological evidences of CNS involvement had a normal neurophysiological results and vice versa.
**Fig. (1):** Clinical CNS involvement in CIDP patients.

**Fig. (2):** CIDP patients with abnormal evoked potential responses.

**Table 1.** MRI results of the brain and spinal cord in CIDP patients.

<table>
<thead>
<tr>
<th>MRI results</th>
<th>CIDP patients (N= 40)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
</tr>
<tr>
<td>Normal</td>
<td>30</td>
</tr>
<tr>
<td>Abnormal</td>
<td>10</td>
</tr>
<tr>
<td>- Brain only</td>
<td>5</td>
</tr>
<tr>
<td>- Spinal cord only</td>
<td>2</td>
</tr>
<tr>
<td>- Both brain and spinal cord</td>
<td>3</td>
</tr>
</tbody>
</table>
Fig. (3): Clinical CNS involvement and results of evoked potentials.

**Table 2.** Relation between clinical CNS involvement and radiological results.

<table>
<thead>
<tr>
<th>Radiological results</th>
<th>Patients with clinical CNS involvement (N=12)</th>
<th>Patients without clinical CNS involvement (N=28)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MRI +ve patients (n=10)</td>
<td>6 (50%)</td>
<td>4 (14.3%)</td>
<td>&lt;0.01**</td>
</tr>
<tr>
<td>MRI -ve patients (n=30)</td>
<td>6 (50%)</td>
<td>24 (85.7%)</td>
<td>&lt;0.01**</td>
</tr>
</tbody>
</table>

**highly significant**

**Table (3):** Relations between MRI results and Evoked potentials results

<table>
<thead>
<tr>
<th>Evoked potential results</th>
<th>With MRI lesions (N=10)</th>
<th>Without MRI lesions (N=30)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abnormal VEP latency (16)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No (%)</td>
<td>6 (60%)</td>
<td>10 (33.3%)</td>
<td>&lt;0.05*</td>
</tr>
<tr>
<td>Mean ± S.D</td>
<td>121±6.7</td>
<td>119.3±7.1</td>
<td></td>
</tr>
<tr>
<td>Abnormal BAEP latency (12)</td>
<td></td>
<td></td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>No (%)</td>
<td>3 (30%)</td>
<td>9 (30%)</td>
<td></td>
</tr>
<tr>
<td>Mean ± S.D</td>
<td>3.9±0.4</td>
<td>3.7±0.3</td>
<td></td>
</tr>
<tr>
<td>I-III wave</td>
<td>4.1±0.31</td>
<td>3.9±0.29</td>
<td></td>
</tr>
<tr>
<td>III-V wave</td>
<td>6.2±1.2</td>
<td>5.9±1.3</td>
<td></td>
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<tr>
<td>Abnormal SSEP latency (22)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No (%)</td>
<td>6 (60%)</td>
<td>16 (53.3%)</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Mean ± S.D</td>
<td>5.1±3.7</td>
<td>5.9±3.9</td>
<td></td>
</tr>
<tr>
<td>EP-P/N13</td>
<td>8.2±1.4</td>
<td>8.7±4.2</td>
<td></td>
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<tr>
<td>Combined abnormal evoked potentials (13)</td>
<td>5 (50%)</td>
<td>8 (26.7%)</td>
<td>&lt; 0.05*</td>
</tr>
</tbody>
</table>

*Significant
DISCUSSION

The main purpose of this study was to assess the frequency of CNS involvement in CIDP patients.

Clinical evidences of central nervous system involvement in the present study were recorded in 12 patients (30%), a finding which is supported by many authors, with a frequency ranging from 4% to 64%.

In the pyramidal signs, sensory tract signs, precipitancy of micturition and cerebellar manifestations were due to CNS involvement (spinal cord and/or brain lesions). A finding which was previously reported by Stojkovic et al., Rodrigues-Casero et al., and Feasby et al.

On the other hand, there is a controversy about cranial nerves involvement in CIDP. In our series, cranial nerves involvement was found in 30% of patients, a figure which is similar or higher than that in previous series (5-30%). On clinical ground it was not always possible to be certain whether this cranial nerves affection was the result of brain stem or extra-axial involvement as reported by many authors.

In this study, optic atrophy, nystagmus and unilateral 6th nerve palsy were mostly due to CNS involvement as proved by their strong correlation with the abnormal visual evoked potentials latency, and MRI abnormalities in the brain stem.

The results of evoked potential responses were in agreement of that of Stojkovic et al., Ormerod et al., Giali et al., Pakalnis et al., and Uncini et al., who recorded either complete absence or delayed latency of VEP and BAEP responses in CIDP patients suggesting slow conduction in the visual or auditory pathways. Moreover, Stojkovic et al. found that demyelination of visual pathways, as evidenced by prolonged latencies of VEPs were identified in nearly half of the patients with CIDP.

Prolonged VEP latency were found in 44% among 18 CIDP patients reported by Pakalnis et al. and Uncini et al. found abnormal VEPs in 6 out of 7 patients with CIDP. Altered motor or brain-stem auditory evoked potentials were also reported in CIDP patients.

The frequency of CNS involvement on MRI examination is highly variable, in different studies it ranges from 3-23%, while Wokke and VanDenBerg and Thomas et al. reported evidence of demyelination of the CNS in up to one half of the patients with CIDP as shown by MRI. CNS lesions were not observed in the series of 92 patients reported by McCombe et al. and series of 19 patients reported by Ormerod et al.

The controversy of these findings may be partially due to the method of case selection, as some studies like that of Thomas et al. studied patients with clinical features of both CIDP and MS. So, it was expected that most of these cases scanned showed MRI lesions mostly similar to MS, while that of Mendell et al. reported positive scans in 6 of 16 CIDP patients clinically diagnosed as having CIDP.

So these studies imply that central demyelinating lesions may be common in CIDP, but each author acknowledged a selection bias in favour of CNS involvement in at least some cases, consequently the frequency of central lesions in CIDP can't be estimated. The estimated frequency in this study is more or less accurate as there was no bias in our patients' selection.

In previously reported CNS demyelinating lesions, no specific criteria to define demyelination were used. A study by Stojkovic et al. reported CIDP patients with high signal intensity lesions on T2 weighted images in a cohort of CIDP patients, none of whom had a history of CNS involvement. Moreover, the number and the distribution of these lesions fulfilled the MS criteria.

As observed in this study when CNS involvement in CIDP patients appears on MRI, the brain lesions are mostly multiple, small, periventricular high signal intensity lesions of the white matter.

Similarly, Mendell and Colleagues reported 6 patients with CIDP who had CNS lesions, all had lesions contiguus with the ventricles and five also had subcortical white matter lesions. Also the five patients reported by Thomas et al. all had periventricular lesions and some had additional cerebral white matter lesions. Moreover, Stojkovic et al. reported that the distribution of T2 weighted areas of increased signal in CIDP patients were periventricular and similar to those observed in MS.

Although the frequency of brain lesions in CIDP patients were extensively investigated, spinal cord demyelination has not been investigated. In this study 5 patients (12.5%) had a spinal cord demyelinating lesion mainly in the cervical region.

Laura et al. reported that the presence of high signal intensity in the posterior columns of the thoracic
Spine in one patient with mainly sensory CIDP is of interest and it may by a sign of severe involvement of the dorsal root ganglia with proximal axonal degeneration in the dorsal column. However spinal cord demyelination was not found. Moreover, Freitas et al. reported cervical spinal cord atrophy in CIDP using MRI, a finding which has previously been described and may be due to a dying-back mechanism. Axonal loss is well recognized in CIDP, suggesting proximal motor axonal damage.

In agreement with present results Bouchard et al. reported that Patients with symptomatic CNS lesions had a more severe course, while Ormerod et al. and McCombe et al. reported a significantly earlier age of onset in patients with CNS involvement. On the contrast Pineda et al. reported a favorable response to immunotherapy in CIDP patients with CNS demyelination.

In this study, although patients with clinical CNS signs had more frequent evoked potentials abnormalities there is some discrepancy evident between the presence of clinical CNS involvement and the neurophysiological abnormalities.

The previously described findings are in agreement with that of Uncini et al., who observed prolonged evoked potential latencies in high percentages among CIDP patients in those having clinical signs of CNS involvement. However, Pakalnis et al. reported that CIDP patients with abnormal VEP had no clinical signs of CNS involvement. Moreover, Stojkovic et al. observed subclinical visual pathway abnormalities in 47% of CIDP patients.

In spite of this discrepancy between clinical CNS involvement and the neurophysiological abnormalities, there were some good correlations between clinical signs and the neurophysiological abnormalities, as 4/5 (80%) patients with optic atrophy, and 3/4 (75%) with Nystagmus had delayed latency of p100 of VEP. Moreover long tracts signs (Pyramidal signs, Sensory tracts signs, precipitancy) correlated well with the abnormality of the SSEP latencies in 8/10 (80%), 5/6 (83.3%) and 5/6 (83.3%) respectively.

Inspite of more frequent clinical CNS signs in CIDP patients with CNS lesions. The present results observed another discrepancy between clinical and radiological signs of CNS involvement. These findings were supported by Rezania et al., who reported a subclinical CNS lesions in 5-100% of CIDP patients in different series. Again one explanation of these results was the methods of patients selection and bias, that favour features of CNS involvement, and the absence of some radiological techniques as of optic nerve MRI study which might have shown demyelinating lesions.

Some clinical signs of CNS involvement correlated well with CNS lesions. Cranial nerve involvement reported in 6/10 (60%) of patients with MRI lesions, moreover 3/6 of those with optic nerve atrophy had brain lesions, those with nystagmus and/or 6th nerve palsy had brain stem lesions in 2 patients. Similarly Wokke and VanDen Berg reported CIDP patients who developed unilateral 6th nerve palsy associated with pontine white matter lesions. Moreover, it is suggested that cranial nerve lesions may be related to CNS lesions and enhancing brainstem lesions is suggestive of active CNS demyelination and therefore probably responsible for a central origin of the ocular palsies.

CIDP patients with clinical signs of long tracts involvement (pyramidal, sensory, precipitancy) correlated well with their pathology location in the spinal cord alone or in addition to brain. Moreover in three patients with cerebellar manifestations, one had cerebellar lesions and another one had brain stem lesion.

Not all patients with MRI lesions recorded abnormal neurophysiological records. So there is incomplete concordance between evoked potentials and MRI results in CIDP patients, a finding which was supported by many authors.

This is not surprising as the two investigations demonstrate qualitatively different abnormalities within the nervous system. Furthermore, there is only a partial overlap in the anatomical territory encompassed by the two techniques. MRI demonstrates lesions where there are changes in the amount of tissue water and also alterations in the physical behaviour of water protons. Abnormalities of evoked potentials demonstrate a functional disturbances in the same central pathways. This technique has the additional advantage of including the spinal cord but of course will not demonstrate lesions within the brain which are out side their pathways.

This lack of agreement between MRI and clinical or neurophysiological features has also been suggested in MS patients and could be related to the absence of an optic nerve MRI study, which might have shown demyelinating lesions. Moreover demyelinating lesions in MS patients are often transient and thus undetectable on MRI. These rapid transient demyelinating lesions are thought to be the consequence of blood-brain barrier.
abnormalities which could be reversed by intravenous steroids.22,26.

On the other hand, there was a good anatomical correlation between abnormal evoked potentials and central MRI lesions. 5/6 patients with abnormal VEP latency had MRI brain lesions, 2/3 patients with abnormal BAEP had brain stem lesions, while 5/6 patients with abnormal SSEP had spinal cord lesions.

There may be several explanations for the combined occurrence of central and peripheral myelin dysfunction. Firstly, coincidence seems unlikely, as so many patients with CIDP show evidences of involvement of white matter, secondly the process of demyelination itself may lead to ongoing generalized demyelination through activation of cytokines, finally central and peripheral myelin may share a common antigen which trigger an immune response.5,21

The mechanisms of peripheral and central nervous involvement in the same patient is not known, this may be due to a chance association or alternatively the same pathophysiological mechanism that may underlie both conditions central and peripheral myelinate share common antigens and immunological mechanisms and thought to be important in producing demyelination in both MS and CIDP. Antigenic cross reaction between central and peripheral myelin has been put forward as a possible mechanism by which central and peripheral nerve demyelination may occur in a subset of MS and CIDP patients 6,8,15 and a grossly deranged blood brain barrier (BBB) could facilitate this process.8

Indeed, it has been shown that both myelin basic protein and myelin-associated-glycoprotein, proteins which are common to the CNS and PNS, have both encephalitogenic and neuritogenic properties.7,8,39. The post immune response induced by these antigens may vary depending on the haplotype background. Different responses to the same antigen could explain the variety of the spectrum of central and peripheral inflammatory demyelinating diseases. The susceptibility to develop MS, CIDP or both syndromes suggests a general predisposition to autoimmunity. This could be related to a breakdown of natural self-tolerance which potentiates pathogenic cell-reactive T cells against a common but as yet unidentified autoantigen7,9,28,31,40.

Conclusion

CIDP is frequently associated with various clinical, neurophysiological and radiological evidences of CNS involvement. Although the mechanism of this association is not clearly understood, and inspite of incomplete concordance between these evidences of CNS involvement, still MRI and evoked potentials are useful non-invasive techniques for demonstrating CNS involvement in cases of CIDP.

REFERENCES


الملخص العربي
تأثر الجهاز العصبي المركزي في التهاب الأعصاب ووجدناها المزمن متجد النخامية

تهدف هذه الدراسة إلى تحديد معدل تأثر الجهاز العصبي المركزي في مرضى التهاب الأعصاب وزجورها المزمن ودراسة مميزات هذه العلاقة المحتملة.

انتمت هذه الدراسة على 40 مريضاً مرضي التهاب المزمن للأعصاب ووجدواها (20 ذكر و 20 إمرأة) وقد تم إجراء الفحوصات التالية لكل المريض: قياس إلإتريكيا، فحص مستويات فحوصات ميالينية صببة الشبكة على رسم عصابات، سرعة توصيل للأعصاب ووجدناها، جهد مثار (صرحي، سمعي وحسى) أنثى بالنورين المغايني على المخ والجلد الشوكي.

وقد أظهرت النتائج ما يلي، وجود الواضح الإلإتريكيا للدالة على تأثر الجهاز العصبي المركزي في 30% من مرضى التهاب المزمن للأعصاب، تتباطئ الجهد المثار أو تغيرات غير طبيعية في الجهد البصري المثار في 40% من المرضى والجهد السعوي المثار في 50% من المرضى ولكن هناك 30% من المرضى يعانون من تغيرات غير طبيعية في لمعة مريض المغايني على المخ والجلد الشوكي أو تغيرات في 50% من المرضى، وجد أن مرضى التهاب المزمن للأعصاب ووجدناها الذين يعانون من تأثر الجهاز العصبي المركزي أصغر سنًا وقت حدوث المرض كما أنهم يعانون أكثر من النوع المترددة من المرض، ولم تذكر تاريخ مرضي أطول، كما أنه كان استخدام لوساطة العلاج عن غيرهم الذين يعانون من مظاهر تأثر الجهاز العصبي المركزي، التغيرات غير الطبيعية في استجابات الجهد المثار المختلفة والتغيرات في لمعة مريض المغايني أكثر حدوثًا في مرضى التهاب المزمن للأعصاب ووجدناها الذين يعانون من تأثر إلإتريكيا لتأثر الجهاز العصبي المركزي عن وعاء بدون هذا التأثر، التغيرات في لمعة مريض المغايني على المخ والجلد الشوكي أكثر حدوثًا في مرضى التهاب المزمن للأعصاب ووجدناها الذين يعانون من تغيرات غير طبيعية في الجهد البصري المثار. توجد نسبة من مرضى التهاب الأعصاب ووجدناها لا تظهر عليهم أعراض إلإتريكيا لتأثر الجهاز العصبي المركزي بالرغم من وجود تغيرات ميالينية عصبية، ونفترض أن العلاقة بالرنين المغايني على المخ والجلد الشوكي لتآثر الجهاز العصبي المركزي.

ومن هنا نستنتج أنه بالرغم من عدم وجود مسبب فأن التهاب الأعصاب ووجدناها المزمن متجد النخامية كثيرًا ما يكون مصحوبًا بظاهرة تأثر الجهاز العصبي المركزي، وبالرغم من عدم اتفاق الكثیر بين هذه الملاحظة فإن أنثى بالنورين المغايني والجهد المثار هو من الوسائط المفيدة لتحديد تأثر الجهاز العصبي المركزي في حالات التهاب الأعصاب ووجدناها المزمن متجد النخامية.

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