Early Clinical and Electrodiagnostic Assessment of Guillain Barré Syndrome: Predictors for Prognosis

Ashraf M. Zaitoun, Amr Kamel, Sawsan Abdel Aziz, Hanan Abdel Azem, Hanan Salah
Department of Neurology, Zagazig University

ABSTRACT

Background and purpose: GBS is an important cause of acute neuromuscular paralysis. Our study was design to determine the early pattern of neurophysiological findings within the first week of GBS that guide to the diagnosis, to evaluate the relationship of these electrodiagnostic tests with both clinical findings and laboratory investigation, and to postulate the early findings suggestive of prediction of the prognosis. Subjects and Methods: Thirty-four patients who fulfilled the clinical criteria for diagnosing GBS were selected. Clinical evaluation with Hughes scale, serum TNF-α levels and electrodiagnostic tests were performed within the first week of onset. Results: The study included 34 patients with GBS, 21 males and 13 females with a mean age of 38.4±14.1 years. Abnormalities of motor conduction were detected in two or more nerves in 25 (73.5%) patients, of sensory nerve conduction study in 17 (50%) patients, of F-wave response in 27 (79.4%) patients, and H-reflex in 22 (64.7%) patients. A raised serum concentration of TNF-α was detected in 15 (44.1%) patients. Serum levels of TNF-α correlated with the degree of disease severity, as measured by Hughes scale. As a group, patients with raised TNF-α levels had significantly abnormal electrodiagnostic feature consistent with peripheral nerve demyelination. Using multivariate analysis, the variables significantly related to poor prognosis were Hughes grade ≥ 3, age ≥ 50 years, no antecedent infections, presence of autonomic symptoms, axonal EMG and elevated TNF-α serum levels. Conclusion: These findings suggested that EMG studies and TNF-α serum levels could aid in the diagnosis of early GBS as well as predict the prognosis. (Egypt J. Neurol. Psychiat. Neurosurg., 2009, 46(1): 169-176)

INTRODUCTION

Guillain Barré syndrome (GBS) is an acute post-infectious immune mediated peripheral neuropathy characterized by rapidly progressive weakness and sensory loss, usually followed by slow clinical recovery. GBS is heterogeneous in severity of neurological deficits and prognosis. Some patients develop paralysis of ocularmotor, facial, oropharangeal, respiratory, and limb muscles within days and remain bedridden or wheelchair bound. Others have mild limb paresis from which they recover spontaneously within weeks. Advances in general care facilities and the availability of specific treatments have improved the outcome of patients with GBS.1

GBS is an important cause of acute neuromuscular paralysis. Molecular mimicry and a cross-reactive immune response play a critical part in its pathogenesis, at least in those cases with a preceding campylobacter jejune infection and with antibodies to gangiliosides. The type of preceding infection and patient–related host factors seem to determine the form and severity of the disease.2

Electrodiagnostic study is very important in the diagnosis of inflammatory neuropathy, including GBS, with clinical, biological or histobiological criteria. It initially affirms the existence of a neuropathy and then to define if the pathological process is demyelinating, axonal or more rarely mixed. It is also specifies if it concerns only sensory fibers, motor fibers or both. This exploration thus will make it possible to define sub-groups with, for each one a possible etiology guidelines.3

Nerve conduction studies are the most important ancillary diagnostic test in GBS. Electrodiagnostic studies often show evidence of patchy demyelination, however, axon loss variants has been described.4 While the electrical abnormalities may not be sufficiently widespread for
definite diagnosis in the first two weeks, early diagnosis is important, because treatment shortens the course of GBS, reduces the time required to receive mechanical respiratory assistance, and lessens the overall severity.5

Tumor necrosis factor (TNF-α) plays an important role in many aspects of immune system development, immune response regulation, and T-cell mediated tissue injury. Histological studies have shown that demyelination is associated with inflammatory infiltration T-lymphocytes and macrophages, both release TNF-α, a pleotropic proinflammatory cytokine that exerts toxic effects on myelin, Schwann cells, and endothelial cells.6

Our study was design (1) to determine the early pattern of neurophysiological findings within the first week of GBS that guide to the diagnosis, (2) to evaluate the relationship of these electrodiagnostic tests with both clinical findings and laboratory investigation, (3) to postulate the early findings suggestive of prediction of the prognosis.

SUBJECTS AND METHODS

Thirty- four patients who fulfilled the clinical criteria for diagnosing GBS7 were selected from Neurology Department, Zagazig University Hospitals in the period from September 2006 to October 2007. Clinical evaluation and electrodiagnostic tests were performed within the first week of onset.

Inclusion criteria

Patients were included if finding from the clinical, radiographic imaging, laboratory and electrodiagnostic studies combined were suggestive of GBS and no other disorder.

Exclusion criteria

Excluded from the study were patients with other causes of nerve conduction abnormalities, such as renal failure or diabetes mellitus, and patients with evidence of another neuromuscular diagnosis, such as myopathy, familial polyneuropathy or chronic polyneuropathy. We excluded also patients with neuropathies associated with exogenous toxic agents, metals, or drugs.

Disabilities

The disabilities of the patients were evaluated on the Hughes Functional grading Scale.8 It composed of six grades; grade (0)= healthy; grade (1)= minor signs or symptoms of neuropathy but capable of manual work; grade (2)= able to walk without support of a stick but incapable of manual work; grade (3)= able to walk with a stick appliance or support; grade (4)= confined to bed or chair bound; grade (5)= requiring associated ventilation.

Laboratory studies

Serum samples were filtered and then frozen at -80ºC until the time of assay. Serum TNF-α levels were determined by enzyme linked immunosorbant assay (ELISA). Cytokines concentrations were only regarded as raised if they were higher than 25 pg/ml.9

Electrodiagnosis

Nerve conduction studies (NCS) were done with Nicoli EMG machine within one week of onset, Motor studies were made of median and ulnar nerves in the upper limbs and common peroneal and posterior tibial nerves in the lower limbs. Sensory nerve conduction studies were performed using antidromic techniques, on the median and ulnar nerves in the upper limbs and sural nerve on the lower limbs. F- waves were measured with each motor NCS for which a compound motor action potential (CMAP) result was obtained and H- reflex was recorded from gastrocnemius and soleus muscles after stimulation of the posterior tibial nerve. A value was defined as abnormal if it fell outside a 2.5 SD from the control. Patients were classified as having polyradiculoneuropathy consistent with GBS if there was a combination of prolonged distal motor latency (>150% of the upper limit of normal), conduction velocity (CV) slowing (<70% of the lower limit of normal), prolongation of F-wave latency (>150% of the upper limit of normal), low CMAP amplitude or proximal CMAP drop suggestive of conduction block, or abnormal temporal dispersion in two or more nerves.5

Follow up

Follow up studies were performed after one month for all patients. Hughes scale was used to evaluate the clinical state of the patients. The predefined outcome measure was improvement at
four weeks by at least one grade on the scale of motor function\textsuperscript{19}.

**Statistical analysis**

Data were collected and statistically processed using Epi Info software state package. Descriptive statistics was presented as mean ± SD. The relationship between clinical severity and TNF-\(\alpha\) were analyzed using ANOVA test, whereas t-test was used to assess the relation between TNF-\(\alpha\) and EMG findings. For the assessments of associated factors with poor prognosis after one month, Odd ratio (OR) (95% CI) were calculated. The value of \(P <0.05\) was considered statistically significant.

**RESULTS**

The study included 34 patients with clinically diagnosed GBS according to diagnostic criteria, 21 males and 13 females. Age ranged from 15 to 58 years with a mean age of 38.4±14.1 years. Fifteen (44.1%) patients had history of infection, either respiratory or gastrointestinal infection, in the prior month. Most of our patients (73.5%) were in grade II and III of Hughes disability scale with a mean of 2.4±0.9 (Table 1).

Table (2) demonstrated EMG findings of our patients in the first week of affection. Abnormalities of motor conduction were detected in two or more nerves in 25 (73.5%) patients, nine of them had absent M response, whereas the other had prolonged distal latencies, decreased conduction velocities, or/and low amplitude motor action potentials. Temporal dispersion and/or conduction block was detected in seven (20.6%) patients. Sensory nerve conduction study revealed abnormalities in two or more nerves in 17 (50%) patients, ten of them showed absent response. The most common EMG abnormalities in our patients was F-wave response in 27 (79.4%) patients, absent in 11 (32.4%) patients and delayed in 16 (47.1%) patients. H-reflex was absent in 15 (44.1%) patients and delayed in 7 (20.6%) patients.

A raised serum concentration of TNF-\(\alpha\) was detected in 15 (44.1%) patients. Serum levels of TNF-\(\alpha\) in the whole cohort correlated with the degree of disease severity, as measured by Hughes scale (Table 3). As a group, patients with raised TNF-\(\alpha\) levels had significantly abnormal electrodiagnostic feature consistent with peripheral nerve demyelination compared to patients with normal TNF-\(\alpha\) levels. The amplitude of motor action potentials were also significantly reduced in patients with raised TNF-\(\alpha\) (Table 4).

Regarding the prognosis of our patients, using multivariate analysis the variables significantly related to poor prognosis were Hughes grade ≥ 3, age ≥ 50 years, no antecedent infections, presence of autonomic symptoms, axonal EMG and elevated TNF-\(\alpha\) serum levels (Table 5).

Table 1. Demographic data of our patients (34) at admission.

<table>
<thead>
<tr>
<th>Data</th>
<th>No</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (years)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>15 – 58</td>
<td></td>
</tr>
<tr>
<td>Mean±SD</td>
<td>38.4 ± 14.1</td>
<td></td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>21</td>
<td>61.8</td>
</tr>
<tr>
<td>Female</td>
<td>13</td>
<td>38.2</td>
</tr>
<tr>
<td><strong>Preceding events:</strong> (either respiratory infection or diarrhea)</td>
<td>15</td>
<td>44.1</td>
</tr>
<tr>
<td><strong>Disability score (X±SD)</strong></td>
<td>2.4 ± 0.9</td>
<td></td>
</tr>
</tbody>
</table>
Hughes grades

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>5</td>
<td>14.7</td>
</tr>
<tr>
<td>II</td>
<td>14</td>
<td>41.2</td>
</tr>
<tr>
<td>III</td>
<td>11</td>
<td>32.3</td>
</tr>
<tr>
<td>IV</td>
<td>4</td>
<td>11.8</td>
</tr>
</tbody>
</table>

Table 2. EMG studies at the first week of onset of GBS.

<table>
<thead>
<tr>
<th>EMG findings</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor conduction abnormalities (in ≥ 2 nerves)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absent response</td>
<td>25</td>
<td>73.5</td>
</tr>
<tr>
<td>Slow CV</td>
<td>9</td>
<td>26.5</td>
</tr>
<tr>
<td>Prolonged latency</td>
<td>14</td>
<td>41.1</td>
</tr>
<tr>
<td>Low amplitude</td>
<td>16</td>
<td>47.1</td>
</tr>
<tr>
<td>Sensory conduction abnormalities (in ≥ 2 nerves)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absent response</td>
<td>17</td>
<td>50.0</td>
</tr>
<tr>
<td>Abnormal response</td>
<td>10</td>
<td>29.4</td>
</tr>
<tr>
<td>Sensory conduction abnormalities (in ≥ 2 nerves)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>F-response abnormalities</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absent response</td>
<td>27</td>
<td>79.4</td>
</tr>
<tr>
<td>Delayed response</td>
<td>11</td>
<td>32.4</td>
</tr>
<tr>
<td>H-response abnormalities</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absent response</td>
<td>16</td>
<td>47.1</td>
</tr>
<tr>
<td>Delayed response</td>
<td>7</td>
<td>20.6</td>
</tr>
<tr>
<td>Temporal dispersion and/or conduction block (in ≥ 2 nerves)</td>
<td>7</td>
<td>20.6</td>
</tr>
</tbody>
</table>

Table 3. Relations between TNF-α and Hughes scale of GBS patients at admission.

<table>
<thead>
<tr>
<th>Disability Score</th>
<th>No.</th>
<th>TNF-α X±SD</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade I</td>
<td>5</td>
<td>34.6 ± 11.4</td>
<td>38.18</td>
<td>0.001*</td>
</tr>
<tr>
<td>Grade II</td>
<td>14</td>
<td>50.7 ± 12.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade III</td>
<td>11</td>
<td>250.4 ± 82.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade IV</td>
<td>4</td>
<td>272.5 ± 95.3</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* = significant

Table 4. Relations of nerve conduction studies and TNF-α in 34 patients with GBS.

<table>
<thead>
<tr>
<th>Motor nerve conduction</th>
<th>Median n. (upper limb)</th>
<th>CPN n. (lower limb)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>+ve TNF-α</td>
<td>-ve TNF-α</td>
</tr>
<tr>
<td>Amplitude</td>
<td>2.2±2.0*</td>
<td>6.1±1.1</td>
</tr>
<tr>
<td>Conduction velocity</td>
<td>35.5±12.0</td>
<td>41.5±10.0</td>
</tr>
<tr>
<td>Distal latency</td>
<td>7.2±1.6*</td>
<td>3.6±0.9</td>
</tr>
<tr>
<td>F-response</td>
<td>44±4.3*</td>
<td>38.0±8.9</td>
</tr>
<tr>
<td>Sensory nerve conduction</td>
<td>Median n. (upper limb)</td>
<td>Sural n. (lower limb)</td>
</tr>
<tr>
<td>Amplitude</td>
<td>+ ve TNF- α</td>
<td>-ve TNF- α</td>
</tr>
<tr>
<td>1.6±2.1*</td>
<td>4.3±1.1</td>
<td>3.5±2.3</td>
</tr>
<tr>
<td>Peak latency</td>
<td>4.0±0.7</td>
<td>3.8±0.9</td>
</tr>
</tbody>
</table>

Table 5. Variables significantly associated with worse clinical outcome.

<table>
<thead>
<tr>
<th>Variables</th>
<th>OR (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 50</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>≥ 50</td>
<td>9.71 (1.33 - 89.45)</td>
<td>0.009**</td>
</tr>
<tr>
<td>Hughes grades</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 3</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>≥ 3</td>
<td>8.0 (1.29 – 56.89)</td>
<td>0.007**</td>
</tr>
<tr>
<td>Autonomic symptoms</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- ve</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>+ ve</td>
<td>7.44 (1.02 -67.68)</td>
<td>0.023*</td>
</tr>
<tr>
<td>TNF-α</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- ve</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>+ ve</td>
<td>11.2 (1.77 – 83.3)</td>
<td>0.0018**</td>
</tr>
<tr>
<td>EMG findings</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Demyelinating</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Axonal</td>
<td>5.96 (1.08 – 36.34)</td>
<td>0.015*</td>
</tr>
</tbody>
</table>

**DISCUSSION**

Diagnosis of GBS within the first week is often difficult because the elevation of the cerebrospinal fluid protein level and motor nerve conduction study (NCS) changes may not evolve until later, yet early diagnosis is desired since several important treatments have been shown to lessen the disease severity and improve outcome. We tried to identify the earliest electrodiagnostic changes (within seven days of onset of motor weakness) in our patients based on a constellations of findings including, slowed motor conduction velocities, delayed latencies, low distal compound motor action potential (CMAP) amplitude, dispersion of response and conduction block, in-excitible nerves, and absent or prolonged F or H – response. Different studies on patients with GBS have included patients with symptoms for less than one week. In the study done by Ropper et al., 41 patients underwent electrodiagnostic studies in patients with GBS within one week of symptom onset. Sixteen of these patients had multiple abnormalities of CMAP, including dispersion, delayed latency, low amplitude, CV slowing, conduction block, or abnormal F- waves. Thomaides et al. found that the commonest early abnormality in GBS patients was delayed distal motor latency, F-wave abnormality and abnormal thermal threshold measurements in one or more nerves. Clauston et al. assessed CMAP in 47 patients with GBS, 20 of whom were evaluated less than one week from symptoms onset, thirteen had at least one nerve with low amplitude CMAP with conduction block in ten of them.

The most common electrodiagnostic abnormalities in the first week of GBS in two or more nerves in our study was F-wave response abnormalities in (79.4%), followed by abnormalities
of motor nerve conduction (73.5%), then H-
response abnormalities (64.7%). Sharief et al.\textsuperscript{14}, on
studying GBS patients within 4-12 days of onset, found abnormalities of motor conductions in 84%,
sensory nerve conduction abnormalities in 72%, and F-wave abnormalities in 69%. Furthermore, Gordon
and Wilbourn\textsuperscript{5}, on studying 31 patients with GBS in the first week of the disease, found absent H-
reflex (in all patients with 100%), absent F-wave (84%), reduced CMAP amplitude (71%), prolonged distal
latency, temporal dispersion, conduction block, slowed motor CV, low amplitude or absent sensory
nerve action potential in upper extremity.

Tumor necrosis factor-\(\alpha\) (TNF-\(\alpha\)) plays an
important role in many aspects of immune system, immune-
response regulation, and T- cell- mediated
tissue injury. The evidence that TNF-\(\alpha\), released by
autoreactive T-cell and macrophages, may contribute to
the pathogenesis of immune- mediated
demyelinating neuropathies, including GBS is
postulated by Stubgen\textsuperscript{6}. In our study, we found a
high serum concentration of TNF-\(\alpha\) in patients with
GBS. Previous studies in patients with GBS, reported high serum concentration of TNF-\(\alpha\)\textsuperscript{5,13,17}.
Furthermore, our data, suggested that TNF-\(\alpha\) may be
a critical factor in the pathogenesis of GBS as plasma TNF-\(\alpha\) concentrations correlated with
disease severity and this is in accordance with
Sharief et al.\textsuperscript{15} and Exley et al.\textsuperscript{16}.

A putative role for TNF-\(\alpha\) in peripheral
demyelination can be mediated by several
mechanisms that include direct toxic effects on
Schwann cells and myelin, and indirect effects
through the disruption of the blood- nerve barrier,
and the release of toxic oxygen radicals,
phospholipases, and nitric oxide.\textsuperscript{14} Stubgen\textsuperscript{6}
suggested the proposed pathogenesis of TNF-\(\alpha\)
associated neuropathies that include both a T-
cell and humoral immune attack against peripheral
nerve- myelin, vasculitis- induced nerve ischemia,
and inhibition of signaling support for axons.

Our results demonstrated a consistent
relationship between high circulating TNF-\(\alpha\) levels
and electrodiagnostic parameters of peripheral
demyelination. Sharief et al.\textsuperscript{14} found that high serum
level of TNF-\(\alpha\) was correlated with electrodiagnostic
studies of patients with GBS, whereas other
cytokines (IL-1B and sIL-2R) did not correlate with
electrodiagnostic parameters. The correlation
between increased TNF-\(\alpha\) levels and electrodiagnostic abnormalities suggested that TNF-
\(\alpha\) may be important in the pathogenesis
demyelinating neuropathy in GBS. It is worth
mentioning that the association between the high
TNF-\(\alpha\) levels and reduced CMAP amplitude in our
study as well as the study of Sharief et al.\textsuperscript{14} further
suggested that serum TNF-\(\alpha\) may have a prognostic
significance, as CMAP amplitude is a significant
predictor of outcome in patients with GBS.\textsuperscript{15}

Peripheral neuropathies like GBS may cause
imperfections (such as weakness and sensory
deficits), which may lead to problems in daily life
and social functioning with a possible decrement in
quality of life expectations. Choosing the proper
outcome measure to evaluate the therapeutic efficacy
of an intervention at one of these levels of outcome
should therefore be considered as fundamental to the
design of trials in peripheral neuropathies.\textsuperscript{18} Despite
numerous studies, there is still some uncertainty
concerning GBS outcome. Thus one important aim
in our study was to achieve the early clinical,
laboratory and electrodiagnostic findings that could
predict the prognosis. We found that older age \(\geq\) 50
Ys, Hughes grades \(\geq\) 3, serum level of TNF-\(\alpha\),
autonomic symptoms, and axonal form of nerve
conduction study were predictive of worse clinical
prognosis in our patients. Several factors related to a
worse recovery have been described, in different
studies. Sharief et al.\textsuperscript{14} suggested that TNF-\(\alpha\) may
have a prognostic significance in cases with GBS. In
multivariate analysis on 120 GB patients, a worse 2-
years outcome was found to be related to higher
Hughes grade at nadir, axonal or mixed EMG, age \(\geq\)
50 years, and absence of respiratory infections
preceding GBS.\textsuperscript{19} Hadden and Hughes\textsuperscript{20}
postulated that older age, preceding diarrhoeal illness, more
severe weakness, rapid onset, electrically inexcitable
nerves, and muscle wasting are factors associated
with poor outcome. Nagarajan and Al-
Shubaili\textsuperscript{21} found that the presence of predominant distal
weakness in lower limbs, proximal weakness in
upper limbs, autonomic disturbances, and axonal
pattern in NCS predict a poor outcome, hence they
recommended early immunomodulatory therapy in
patients presenting with these features. In studying
motor recovery after GBS in childhood, Ortiz-
Corredor et al.\textsuperscript{22}, using univariate analysis, found
that cranial nerve impairment, requirement of
assisted ventilation, presence of quadriplegia and presence of non-exci
table motor nerves were associated with delayed motor recovery time. In the
multivariate analysis, muscular strength, assessed at day 10 of the disease was the most important
predictor to determine motor recovery. Van-Koningsveld et al. assessed potential predictors for
their association with the inability to walk independently at 6 months, and they found that age,
preceding diarrhea, and GBS disability score at two weeks after entry are the prognostic predictors. Dhar et al. assessed the morbidity and outcome of patients with GBS admitted to the intensive care unit, over an average 3 years follow up recovery of independent ambulation was seen in 75%, with advanced age being the most powerful predictors of poor outcome. Kalita et al. demonstrated that outcome of GBS was related to severity of illness, and CMAP amplitude.

**Conclusion**
The diagnosis of GBS is difficult in the first week of motor onset, yet early recognition is necessary to begin appropriate treatment and avoid potential medical complications. Nerve conduction studies and TNF-α serum level play an important role in the early diagnosis of GBS and throw some light on the prognosis.

**REFERENCES**

المؤثر العربي
التقييم السريري والكهروفسيولوجي المبكر لمتلازمة جيلان باري وعلاقته بمثال المرض

يعتبر التهاب الأعصاب الحاد (متلازمة جيلان باري) سابقاً دوسياً لاعتلال الأعصاب الطرفية. تهدف الدراسة إلى تحديد أوضاع التغيرات الكهروفسيولوجية المبكرة لمرضى متلازمة جيلان باري وذلك تقييم العلاقة بين هذه التغيرات والфункциة السريرية للمرضى وكذلك الفحوصات العملية.

تمت هذه الدراسة على 34 مريضاً بمتلازمة جيلان باري تم إدخالهم في لقسم الأمراض العصبية بمستشفى الزقازيق الجامعي وقد خضع هؤلاء المرضى للتحاليل الهيكلية، التي تتضمن التذبذب الدماغي في الجهاز العصبي الشمالي والدفوقي باستخدام مقياس هافس، قياس نسبة TNF-α بعمل الدم. اختبار كهروفسيولوجي لأمراض تشير إلى الأسباب الأول من الإصابة بالمرض.

أظهرت نتائج الدراسة الآتي:

* تراوحت أعمار المرضى بين 15-58 سنة، وكانوا 21 من الذكور و 13 من الإناث. تم تحديد الاعتلال في التوصيل العصبي الحركي لائي أو أكثر من الأعصاب ب 25 مريضاً (73.5%) بينما كان الاعتلال في التوصيل العصبي الحسوي في 17 مريضاً (50%).
* ارتفعت نسبة TNF-α بعمل الدم في 15 مريضا (44.1%) ونسبة TNF-α المرضا، و التغيرات الكهروفسيولوجية.

المراجع: