Assessment of Nerve Growth Factor and Nerve Conduction Velocity in Diabetic Patients with Neuropathy

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ABSTRACT

Introduction: Neuropathy is one of the most debilitating complications of both type 1 and type 2 diabetes, with estimates of prevalence between 50-90%. There is variable involvement of large myelinated fibers and small, thinly myelinated fibers. Many of the neuronal abnormalities in diabetes can be duplicated by depletion of specific neurotrophic factors, their receptor or their binding proteins. Nerve growth factor (NGF) is a neurotrophic polypeptide. It selectively promotes the differentiation and maintenance of small fiber sensory and sympathetic neurons in the peripheral nervous system. Aim of the work: to find out the changes in the level of NGF and nerve conduction velocity (NCV) in diabetic neuropathy and the relation between serum level of NGF and each of degree of glycemic control, duration and type of diabetes in diabetic patients. Patients and Methods: This study included 10 controls and 40 patients with diabetic peripheral neuropathy (P.N) 20 of them with type 1 diabetes mellitus 20 with type II diabetes mellitus. All were subjected to: history taking and clinical examination, laboratory investigation including liver function tests, kidney function tests, complete blood picture, fasting and 2 hours postprandial blood glucose level, glycosylated haemoglobin (HBA1c), serum NGF level and NCV measurement. Results: There was a significant decrease in the serum NGF levels and NCV in the diabetic patients compared to the control; also, there was a significant decrease in the NGF levels and NCV in type I diabetic patients as compared to type II. A highly significant negative correlation was found between both of NGF and NCV to each of fasting blood glucose, glycosylated haemoglobin, duration of diabetes, symptoms and examination scores in diabetic patients with P.N; while, there was a significant positive correlation between neuropathy scores and each of fasting blood glucose, glycosylated haemoglobin, and duration of diabetes, while there was a highly significant negative correlation with NCV of four measured nerves in diabetic patients with P.N. In conclusion: Nerve growth factor deficiency may be responsible for the pathogenesis of neuropathy which occurs in such patients. Therefore modulation of nerve growth factor may offer hope for patients with P.N and will open new therapeutic era in management of P.N: also, good diabetic control and foot care are very important in improvement of diabetic P.N. in diabetic subjects. (Egypt J. Neurol. Psychiat. Neurosurg., 2009, 46(1): 101-109)

INTRODUCTION

Neuropathy is the frequent symptomatic complication of most metabolic diseases such as diabetes mellitus¹. Diabetic peripheral neuropathy encompass a group of clinical and subclinical syndromes each characterized by diffuse or focal damage to peripheral nerve fibers². The incidence of diabetic neuropathy approaches 50% in most diabetic population, and its consequences in the form of foot ulceration and leg amputation may be mandatory as there is no curative therapy for diabetic neuropathies.³ Distal symmetrical sensory or sensorimotor polyneuropathy (DSP) affects 30% of the hospital-based population and 20% of community-based samples of diabetic patients. The incidence of DSP is 2% per year. The most important etiological factors that have been associated with DSP are poor glycaemic control, diabetes duration, with possible roles for hypertension, age, smoking, hypoinsulinemia and dyslipidaemia⁴. Many of the neuronal abnormalities in diabetes can be duplicated by experimental depletion of specific neurotrophic factors, their receptors or their binding proteins. These neurotrophic factors are required for the maintenance of the neurons, the
ability to resist apoptosis and regenerative capacity. The best studied of the neurotrophic factors is nerve growth factor (NGF) that stimulate the growth and survival of neurons in the nervous system. NGF is trophic to sensory and sympathetic fibers.

In animal models, NGF is depleted in diabetic nerves. Exogenous NGF can reverse some of the pathological changes in diabetic nerves. Diabetes may affect the body's ability to produce nerve growth factor, which occur naturally. It may also affect the body's ability to transport the naturally occurring nerve growth factor from the skin tissue back to the nerve cell.

So this study is aimed (1) To find out how much the changes in the level of nerve growth factor and nerve conduction velocity in diabetic neuropathy. (2) To find out any relation between serum level of nerve growth factor and, degree of glycemic control, duration and type of diabetes in diabetic patients.

**Subjects and Methods**

**Subjects:**
This study was carried on 50 subjects which were divided into the following groups.

- **Group I:** control group: it comprised 10 apparently healthy subjects with no evidence of peripheral neuropathy clinically (5 males and 5 females) their ages ranged from 36-55 years and their mean age±SD was (42.8±8.17 y).

- **Group II:** it included 40 patients with diabetic peripheral neuropathy which was diagnosed according to, and by electrophysiological study of nerve conduction. Twenty of them with type I diabetes mellitus and the other twenty with type II diabetes mellitus, (20 males and 20 females) their ages ranged from 25-65 years and their mean age±SD was (36.8±6.79), the duration of diabetes mellitus varied between 5-15 years, fasting blood glucose ranged from 215-265 mg/dl with the mean of (220), and 2 hours postprandial blood glucose ranged from 190 -274 mg/dl with the mean of (272) and they had normal kidney function.

**Exclusion criteria:**
All patients of this study were excluded from:

1. Other causes of neuropathies other than diabetes, such as uraemic neuropathy, vitamin deficiencies (B1, B6, B12) thyroid dysfunction, inflammatory disorders, and toxins, liver disease.
2. Any diseases that may affect the level of nerve growth factor such as allergic rhinitis, allergic asthma, autoimmune diseases, leukaemia, parasitic infestation and any inflammatory disorders. and also not taking any drugs known to cause peripheral neuropathy (colchicine-phyntoin- metronidazo-amidarone) or affect the level of nerve growth factor such as cortisone, and cytotoxic drugs.

**Methods:**
All subjects of this study were subjected to the following:

1. **Complete history taking, clinical examination:**
Both general and neurological examinations with special stress on type and duration of diabetes, lines of treatment and clinical symptoms of neuropathy (pain, numbness, paraesthesia, abnormal sensation of heat and cold, and weakness. Assessment of peripheral neuropathy by symptoms and examinations scores according included:
   a. Semi quantitative symptoms scoring for pain, paraesthesia, numbness, and abnormal sensation for heat and cold, that scored as follows: absent “1”, present”2”, and severe “3” giving an over all semi quantitative symptoms score ranging from 5-15.
   b. Semi quantitative sensory examination to pin prick, touch and vibration with assessment of ankle and supinator reflexes. Sensory examination results by examination of vibration by 128-Hz tuning fork on the index finger in the upper limb and on the big toe in the lower limb. For each the modalities tests were scored as: No change “1”, a change below wrist and or below ankle “2” and a change above wrist and or above ankle “3”.Supinator and ankle reflexes were assessed and scored on the dominant side, with scoring of normal “1”,

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presence on reinforcement only "2" and absent "3".

Giving an over all semi quantitative sensory examination scores and reflexes ranging from 4-12.

2. Routine investigations:
The following laboratory investigations were performed for every patients: Complete urine and stool analysis, complete blood count, fasting and 2 hours postprandial blood glucose levels using Biomereax kits, Liver functions tests, renal function tests. Lipid profile (Serum total cholesterol, Serum triglycerides, LDL-cholesterol and HDL-cholesterol), Erythrocyte sedimentation rate (ESR), Serum thyroxin level (TSH ) and (T4) by ELISA, Chest X ray and electrocardiography (ECG) and fundus examination.

3. Standard electrophysiological study of nerve conduction: particularly, nerve conduction velocity (NCV) that measures the response in nerves or muscles following electrical stimulation of sensory receptors, sensory nerves, or motor nerves, peroneal and sural nerves in the lower limb and median nerve in the upper limb.

- Median nerve (MN) motor conduction: It performed with active recording electrode placed one-half the distance between the metacarpophalangeal joint of the thumb and the midpoint of the distal wrist crease. The reference electrode is placed distally on the thumb, and the ground electrode is placed over the palmer aspect of the ulnar border of the hand. The normal motor nerve conduction velocity ranged from 46 to 67 m/sec.

- Peroneal nerve (PN) motor conduction: The active recording electrode is placed over the main bulk of the extensor digitorum brevis muscle. The reference electrode is placed distally over the small toe and the ground electrode is placed over the medial portion of the foot.

- Median nerve sensory conduction: It was measured by the antidromic technique: The active recording electrode and the reference electrode (wire ring electrode) were placed 4cm apart over the digital nerve branches of the index and middle fingers. The stimulating cathode (surface electrode was placed exactly 14 cm proximal to the active recording electrode. The ground recording electrode was placed over the palmar aspect of the ulnar border of the hand.

- Sural nerve (SN) sensory conduction studies: The active recording electrode and reference electrode are mounted 4cm apart on a plastic block and placed over the sural nerve as it passes around the lateral malleolus.

4. Special investigations:

  * Determination of glycosylated hemoglobin: It was done by stanbio glycohemoglobin (pre-fil) kit (sand Antonio, Texas).
  * Detection of Nerve growth factor (NGF): Enzyme immunoassay (ELISA) for in vitro determination of human nerve growth factor-β (NGF- β) with two monoclonal antibodies against the β- subunit human NGF- β standards allow the construction of calibration curves.

Statistical analysis:
All data were coded, entered and analyzed using Epi-Info software computer package. Arithmetic mean (X ± SD), The student’s t-test (for comparison of means in two independent samples), Chi-square (χ²) test (test of association between a factor and an outcome. Analysis of variance (ANOVA of F test) used for comparison of means of more than two groups. P value < 0.05 was considered Significant.

RESULTS
Table (1) shows that NGF (pg/dl) was found to be significantly decreased in all diabetic patients with P.N. compared to control group.

There was no statistical significant difference as regard the age of the examined patients (53.8±5.4 years) and the control group (52.1±5 years). On the other hand, male patients (17 patients, 85%) were
statistically significantly more than female (3 patients, 15%).

A highly significant decreased (P<0.001) was found serum NGF (pg/dl) level, while a highly significant increased was found in both FBG (mg/dl) and HbAlc (%) in Type I compared to Type II diabetic patients with P.N. as shown in table (2).

Comparison of NCV (m/s) of four nerves between diabetic group with P.N. a highly significant decreased (p<0.001) in NCV was found in Type I compared to Type II diabetic patients with P.N. as shown in table (3).

Table (4) shows a significant positive correlation (p<0.05) between neuropathy score (symptoms score and examination score), and each of fasting glucose, glycosylated haemoglobin, and duration of diabetes, while there was highly significant negative correlation (p<0.001) with NCV of four measured nerves.

A highly significant negative correlation (p<0.001) was found between NGF level and each of fasting glucose, glycosylated haemoglobin, duration of diabetes, and symptoms score and examination score, while there was significant positive correlation (p<0.05) with NCV of four measured nerves in diabetic patients (Table 5).

A highly significant negative correlation (p<0.001) was found between NCV of four nerves and each of duration of diabetes, and symptoms score and examination score, fasting blood glucose, and glycosylated haemoglobin (Table 6).

Table 1. Comparison of NGF (pg/dl) between diabetic group with peripheral neuropathy and control group.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control group N=10 (mean±SD) (Range)</th>
<th>diabetic patients with P.N N=10 (mean±SD) (Range)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>NGF (pg/dl)</td>
<td>554 ± 98.8 (275-609)</td>
<td>391 ± 85.2 (221-530)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Table 2. Comparison of serum NGF (pg/dl), FBG (mg/dl) and HbAlc (%) between Type I and Type II diabetic patients with peripheral neuropathy.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Type I diabetic patients with P.N</th>
<th>Type II diabetic patients with P.N</th>
<th>T test</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>NGF (mean±SD)</td>
<td>385.4 ± 75.2</td>
<td>462.1 ± 74.5</td>
<td>31.1</td>
<td>**&lt;0.001</td>
</tr>
<tr>
<td>FBG (mean±SD)</td>
<td>235 ± 25.4</td>
<td>190 ± 20.5</td>
<td>0</td>
<td>**&lt;0.001</td>
</tr>
<tr>
<td>HbAlc % (mean±SD)</td>
<td>12.9 ± 2.3</td>
<td>11.7 ± 2.4</td>
<td>90.7</td>
<td>**&lt;0.001</td>
</tr>
</tbody>
</table>

**P value < 0.001 = Highly significant (HS)**

Table 3. Comparison of the mean±SD of NCV (m/s) of four nerves between diabetic group with peripheral neuropathy.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Type I diabetic patients with P.N</th>
<th>Type II diabetic patients with P.N</th>
<th>T test</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MNCV of MN (mean±SD)</td>
<td>33.9 ± 6.2</td>
<td>34.03 ± 6.3</td>
<td>34.7</td>
<td>**&lt;0.001</td>
</tr>
<tr>
<td>MNCV of CPN (mean±SD)</td>
<td>33.7 ± 6.3</td>
<td>33.09 ± 3.5</td>
<td>59.1</td>
<td>**&lt;0.001</td>
</tr>
<tr>
<td>SNCV of MN (mean±SD)</td>
<td>34.5 ± 3.7</td>
<td>34.2 ± 3.9</td>
<td>56.9</td>
<td>**&lt;0.001</td>
</tr>
<tr>
<td>SNCV of SN (mean±SD)</td>
<td>31.03 ± 4.6</td>
<td>31.2 ± 4.6</td>
<td>43.09</td>
<td>**&lt;0.001</td>
</tr>
</tbody>
</table>

MNCV of MN = Motor nerve conduction velocity of median nerve.
MNCV of CPN  = Motor nerve conduction velocity of common peroneal nerve  
SNCV of MN  = Sensory nerve conduction velocity of median nerve.  
SNCV of SN  = Sensory nerve conduction velocity of sural nerve  
m/s = meter/second  
**P value < 0.001 = HS**

### Table 4. Correlation coefficient between neuropathy score and other parameters in all diabetic group with peripheral neuropathy.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Symptoms score of P.N</th>
<th>Examination score of P.N</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>r</td>
<td>p</td>
</tr>
<tr>
<td>Age (y)</td>
<td>-0.028</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>FBG</td>
<td>0.014</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>HbAlc</td>
<td>0.215</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Duration of diabetes(y)</td>
<td>0.109</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>MNCV of MN</td>
<td>-0.099</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>MNCV of CPN</td>
<td>-0.34</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>SNCV of MN</td>
<td>-0.100</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>SNCV of SN</td>
<td>-0.084</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

### Table 5. Correlation coefficient between NGF and other parameters in all diabetic group with peripheral neuropathy.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>NGF</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>r</td>
</tr>
<tr>
<td>Age (y)</td>
<td>0.269</td>
</tr>
<tr>
<td>FBG</td>
<td>-0.122</td>
</tr>
<tr>
<td>HbAlc</td>
<td>0.385</td>
</tr>
<tr>
<td>Duration of diabetes(y)</td>
<td>-0.441</td>
</tr>
<tr>
<td>Symptoms score of P.N</td>
<td>-0.431</td>
</tr>
<tr>
<td>Examination score of P.N</td>
<td>-0.111</td>
</tr>
<tr>
<td>MNCV of MN</td>
<td>0.032</td>
</tr>
<tr>
<td>MNCV of CPN</td>
<td>0.177</td>
</tr>
<tr>
<td>SNCV of MN</td>
<td>0.049</td>
</tr>
<tr>
<td>SNCV of SN</td>
<td>0.198</td>
</tr>
</tbody>
</table>

### Table 6. Correlation coefficient between neuropathy score and other parameters in all diabetic group with peripheral neuropathy.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>MNCV of MN</th>
<th>MNCV of CPN</th>
<th>SNCV of MN</th>
<th>SNCV of SN</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>r</td>
<td>p</td>
<td>r</td>
<td>p</td>
</tr>
<tr>
<td>Age (y)</td>
<td>-0.073</td>
<td>&gt;0.05</td>
<td>-0.039</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>FBG</td>
<td>-0.048</td>
<td>&lt;0.05</td>
<td>-0.064</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>HbAlc</td>
<td>-0.307</td>
<td>&lt;0.05</td>
<td>-0.39</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Duration of diabetes(y)</td>
<td>-0.077</td>
<td>&lt;0.001</td>
<td>-0.329</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Symptoms score of P.N</td>
<td>-0.099</td>
<td>&lt;0.001</td>
<td>-0.034</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Examination score of P.N</td>
<td>-0.033</td>
<td>&lt;0.001</td>
<td>-0.336</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>
DISCUSSION

Neuropathy is the frequent symptomatic complication of most metabolic diseases such as diabetes mellitus. Diabetic neuropathy, which affects 50% of those with diabetes mellitus is one of most debilitating complications of both type 1 and type 2 diabetes.

Although several pathogenic mechanisms have been proposed none has been clearly demonstrated. The most important etiological factors that have been associated with Distal symmetrical sensory or sensorimotor polyneuropathy (DSP) are poor glycaemic control, diabetes duration, with possible roles for hypertension, age, smoking, hypoinsulinaemia and dyslipidaemia.

Nerve growth factor (NGF) is a neurotrophic protein that play a major role in the development, maintenance, and regeneration of neurons in the nervous system through its high affinity receptor tropomyosin receptor kinase A (TrkA). Later data suggest that low serum level of NGF may play a significant role in the pathogenesis of diabetic polyneuropathy.

This had been confirmed in this study as all the studied parameters of the serum nerve growth factor (NGF) levels and nerve conduction velocity (NCV) showed a highly significant decrease (p<0.001), in all groups of diabetic patients with peripheral neuropathy compared to control group, this may be explained by the fact that peripheral neuropathy is associated with decreased nerve conduction velocity values, which may be due to reduction of nerve growth factor and it is further supported in our study by finding a highly significant positive correlation between nerve growth factor and nerve conduction velocity. This was in agreement with Dipaolo et al., who found that nerve conduction velocity is decreased in over 90% of patients with peripheral neuropathy, and Goss et al., also confirmed that reduced availability of NGF may play a significant role in the pathogenesis of peripheral neuropathy.

The present study showed that nerve growth factor and nerve conduction velocity were lowered in type 1 diabetic patients than type II, this may be attributed to marked insulin deficiency and glucotoxicity. This goes hand in hand with Faradji, who reported that insulin and nerve growth factor share several properties concerning physical activity, molecular structure and site of synthesis, but these findings are not in agreement with Azar et al., who found that an elevated level of NGF has been reported in certain autoimmune diseases. A role of this cytokine has been proposed in the pathogenesis of type-1 diabetes mellitus (IDDM), but no clinical studies have yet measured its serum level in this disease.

In our study glycemic control was a good parameter for diabetic neuropathy. A highly significant increased in fasting blood glucose and HbA1c was found in type I diabetic patients with Peripheral neuropathy compared to type II diabetic patients with Peripheral neuropathy.

Also, it was found there was a positive correlation between both of fasting blood glucose and HbA1c with symptoms score and examination score of diabetic peripheral neuropathy, while, a highly significant negative correlation (p<0.001) was found between NGF level and each of fasting blood glucose and glycosylated haemoglobin indicating that the severity of neuropathy is related to glycemic control. This is in agreement with Jager et al., who studied the role of fasting plasma glucose in diabetic patients with complications, and also with Tkac and Bril, who found that the prolongation glycemic control is related to improvement of the diabetic complications.

There are also a positive correlation between symptoms and examination scores of peripheral neuropathy and duration of diabetes reflecting the relation between the duration of diabetes and presence and severity of diabetic neuropathy and this is in agreement with Ziegler et al., who studied the role of the duration of diabetes in diabetic patients with complications, while there was a significant negative correlation between duration of diabetes and level of nerve growth factor and nerve conduction velocities. This was in agreement with Pfeifer et al., who found that symptoms or signs of neuropathy usually occur after a prolonged duration of diabetes.

From the previous data it is clear that the severity of neuropathy as evidenced in this study by abnormalities in nerve conduction velocities and worse neurological status is related to low nerve growth factor levels. It is unknown whether nerve growth factor should be expected to improve neuronal function or just prevent progression of neuropathy in a clinical setting. Preclinical experimental models demonstrated that nerve
growth factor has the ability to prevent neuropathy not reverse it. So, Decreased level of nerve growth factor may play a key role among pathophysiological mechanisms involved in peripheral neuropathy.

Prior studies have demonstrated deficits in production and retrograde axonal transport of nerve growth factor released from sensory and sympathetic target tissues in diabetic. Systemic administration of rhNGF may bypass the need for retrograde axonal transport and allow the protein to reach sensory ganglion cells directly.

Conclusions and recommendations

From the previous results, it is clear that serum nerve growth factor is decreased in patients with peripheral neuropathy and this reduction is closely related to control, duration, and type of diabetes in diabetic patients.

Therefore, modulation of nerve growth factor may offer hope for patients with peripheral neuropathy and will open new therapeutic era in management of peripheral neuropathy in new future, and also both good diabetic control and foot care are very important in improvement of diabetic peripheral neuropathy in diabetic subjects.

REFERENCES

تعاملن العصبي في مرضى الاعتلال العصبي الحساسين والحساسين بالسكري

يعتبر مرضى السكري المزمن من أهم الآسياح التي تؤدي إلى الإصابة بالاعتلال العصبي. وسبب مرضى السكري مضاعفات على الجهاز العصبي الطرف، والتي قد تؤدي إلى إعاقة وزيادة نسبة الوفيات. حوالي 50% من مرضى السكري يعانون من الاعتلال العصبي الذي يحدث في القدم وقد يصل إلى بتر القدم حيث إنه لا يوجد علاج حاسم لعلاج الاعتلال العصبي الناجم عن السكري.

يستند عامل النمو العصبي إلى مجموعة النيتروفسين التي تساعدهم نمو وتحدد الخلايا العصبية والأعصاب الحسية والحركية لكل من الجهاز العصبي الطرفي والمركزي. وقد أثبتت الدراسات الأخيرة بأن فقص عامل النمو العصبي يساهم بدرجة كبيرة في حدوث الاعتلال العصبي. وقد استعرضت هذه الدراسة اكتشافات التغيرات التي تحدث في مستوى عامل النمو العصبي وسرعة التوصيل العصبي في مرضى السكري المصابين بالاعتلال العصبي ويجاد العلاقة بين مستوى عامل النمو العصبي في الدم وكلا من درجة تظاهر السكر بالدم زمن الإصابة ونوع مرض السكري في مرضى الاعتلال العصبي المصابين بالسكري.

وقد انتهت هذه الدراسة على 50 شخصاً وتم تقسيمهم إلى المجموعات الآتية: المجموعة الأولى: عشرة من المتطوعين الأصحاء ظاهرية كمجموعة ضابطة؛ المجموعة الثانية: أربعون من مرضى السكري الذين يعانون من اعتلال العصب (عشرون منهم يتم علاجهم بالإنسولين، والعشرون الآخر تتم نظافة ابتدائية عن طريق القناطر خارجة.)

وقد أسفرت نتائج هذا البحث عن:

* انخفاض مستوى عامل النمو العصبي في الدم وكذلك سرعة التوصيل العصبي في المرضى الذين يعانون من الاعتلال العصبي بالمقارنة بالمجموعة الضابطة.

* انخفاض مستوى عامل النمو العصبي في الدم وكذلك سرعة التوصيل العصبي في مرضى السكري الذين يعانون من الاعتلال العصبي، يتم علاجهم بالإنسولين، بالمقارنة بالذين يتم علاجهم بأدوية خاصة في حالات السكر بالدم عن طريق القناطر خارجة.

* يعتبر حدوث الاعتلال العصبي بدرجة كبيرة على أن يتطور مستوى السكر بالدم وعلى الفترة الزمنية لمرض السكري في هؤلاء الأشخاص.

وقد خلص البحث أنه يوجد علاقة وطيدة بين الاعتلال العصبي لمرضى السكري ومستوى عامل النمو العصبي في الدم، والذي قد يكون مسؤولاً عن فترة الإصابة بالسكتة ونوعية وضغوطها، لذلك فإن انخفاض مستوى عامل النمو العصبي في مرضى السكري قد يكون مسؤولاً عن الأمراض التي تحدث لعوامل السكر، مما يعزى إلى دور الريزي في حدوثها.

ومن هنا تأتي الحاجة إلى توفر عامل النمو العصبي كعقار يمكن أن يساهم في حل مشاكل المرضى الذين يعانون من الاعتلال العصبي، وهو أيضاً يجدي المضاعفات الخطيرة التي قد تنتج عنه.