Research Article

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Assessment Of Patients With Polyneuropathy Hospitalized In The Neurology Ward Of The Provincial Hospital Of Bialystok.

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ABSTRACT

Polyneuropathies define syndromes of clinical symptoms resulting from extensive damage to peripheral nerves, manifested by motor deficits, sensory deficits, and autonomic disorders. The morphological basis is axonal degeneration of the fibers or their demyelination.
The medical records of patients hospitalized in the Neurology Ward of the J. Śniadecki Provincial Hospital in Bialystok were subjected to a retrospective analysis in the period from January 2014 to December 2019.
The study includes 100 patients (55 women and 45 men). The criterion for inclusion in the study was the admission to the Neurology Ward of patients suspected of symmetrical distal polyneuropathy.
Demographic data, time of hospitalization and clinical course of the disease were analyzed.
In more than 50% of patients the disease had a chronic course, 25% of patients experienced the disease with symptoms developing in an acute manner.
91% of patients - from sensory symptoms - reported paresthesia located mainly in the lower limbs. The feeling of weakness in the lower limbs was reported by 61% of patients.
It was found that congenital polyneuropathy accounted for 5% of all polyneuropathy, while acquired polyneuropathy - 95%. Diabetic polyneuropathy turned out to be the most common acquired polyneuropathy with an established cause, followed by acute inflammatory demyelinating polyneuropathy, and third - polyneuropathy induced by oncological treatment.

Keywords: *polyneuropathy, diabetic polyneuropathy, oncological treatment..*

INTRODUCTION

Polyneuropathy defines a set of clinical symptoms resulting from extensive damage to peripheral nerves, usually symmetrical and diffuse, and affecting the distal sections of peripheral nerves, at least in the initial phase of the disease [1]. Morphologically, this pathology involves axonal degeneration of fibers or their demyelination, although this type of damage frequently assumes a mixed form. The disease manifests itself with motor deficits, sensory losses and autonomic disorders [2, 3]. The etiological factors of polyneuropathy are usually systemic - metabolic, toxic, deficient or inflammatory [2].
Polyneuropathy may lead to a significant reduction in the quality of life. The incidence of this disease is 5-8% and increases with age, to 13% over 55 years.
The major factors in the diagnostic process include:

- medical history - especially family history of polyneuropathy, social history, presence of comorbidities, treatment applied and exposure to toxic factors,
- physical examination to determine the anatomical pattern and location of the pathological process and to what degree motor, sensory and autonomic neurons are involved,
- electroneurographic examination enabling determination of the distribution and type of damage to nerve fibres,
- cerebrospinal fluid examination (protein-cell splitting, signs of neuroinfection),

The Journal of Clinical Medicine (ISSN 2995-6315)

- histopathological examination of the nerve, laboratory tests and imaging investigations.

Treatment of polyneuropathy includes causal therapy (if it is amenable to such treatment) or treatment of the diseases responsible for the development of polyneuropathy as well as symptomatic treatment.

The aim of our study was epidemiological assessment of patients with polyneuropathy hospitalized in the Neurology Ward of the Provincial Hospital in Bialystok.

MATERIAL AND METHODS

The medical records of patients hospitalized in the Neurology Ward of the J. Sniadecki Provincial Hospital in Bialystok were subjected to a retrospective analysis in the period from January 2014 to December 2019. The study involved 100 patients (54 females and 40 males). The inclusion criterion for the study was admission to the Neurology Ward of patients suspected of symmetric distal polyneuropathy.

The following factors were analyzed:

1) **Demographic data** - age, gender, place of residence (town/village),

2) **Duration of hospital treatment** - expressed in days.

3) **Clinical course of the disease:** acute - from a few days to 3 weeks, subacute - from 3 weeks to 3 months, chronic - over 3 months.

4) **Medical history** - family history of polyneuropathy, comorbidities and relevant therapies in the etiopathogenesis of polyneuropathy, reported complaints: sensory (neuropathic pain, paresthesia, hypoesthesia, sensory ataxia), motor (paresis/paralysis, muscle cramps, muscle atrophy), autonomic (impaired sweat secretion, hypersensitivity to heat, gastrointestinal motility disorders, orthostatic drops in blood pressure).

5) **Physical examination** - sensory symptoms (touch, pain, temperature, vibration and position sensation disorders, ataxia), motor symptoms (paresis/paralysis, weak/absence of deep reflexes, muscle atrophy), autonomic (circulatory system disorders and at least one of such symptoms as changes in skin color and temperature, swelling of the limbs, sweating disorders).

Additional tests

a) Electrophysiological tests in polyneuropathy were performed using the Keypont Dantec device. Supramaximal stimulation was used to evaluate motor nerve conduction and sensory nerve conduction, except for the investigations performed with the antidromic method in which submaximal stimulation was applied. Depending on the type of nerve fibers involved, polyneuropathies were divided electrophysiologically into three groups: motor, sensory and sensorimotor. Taking into account the type of damaged fibers, demyelinating, axonal and demyelinating-axonal polyneuropathies were distinguished.

b) Examination of the cerebrospinal fluid - especially important when acquired primary polyneuropathy (protein-cell splitting) and polyneuropathy developing as a result of neuroinfection are suspected.

c) Basic laboratory blood tests - blood count, Erythrocyte Sedimentation Ratio (ESR), C-Reactive Protein (CRP), fasting blood glucose/glucose tolerance test, vitamin B12 level.

d) Additional laboratory tests: onconeuronal antibodies, tumor markers, serological tests for hepatitis B and C, HIV, TBE, anti-Borrelia antibodies, anti-ganglioside antibodies (GM1, GD1a, GD1b, GD3, GQ1b, GT1b).

The following statistical measures were used:

- number, percentage,
- mean, median, standard deviation,
- quartiles.

Statistical tests included:

1. The two sample t-test (Student's t-test) - t-test function in R;
2. The Chi-square test of independence – chisq function test in R.

Statistical calculations were performed in Microsoft Excel and R.

RESULTS

The greatest number of people suffering from polyneuropathy was found in the age group between 50 and 80 years. The mean age of these patients was 62.26 years; median - 62.5± 13.7 years.

In terms of gender, women constituted a larger group (55%), men made up a 10% smaller group (45%).

The mean age of men was 62.9 years, median age - 62 ± 10.1 years. The average age of women was calculated to be 61.7 years, with the median of 64 years ± 16 years.

In the study, 67 patients lived in towns (39 women; 28 men), while 33 came from rural areas (16 women; 17 men). The average time that passed to the beginning of hospital treatment was 263 days (8 months 23 days) and the median was 90 days. Fifty percent of patients started hospitalization within 3 months of the onset of symptoms, 25% entered hospital treatment within 3 weeks after the symptoms had appeared, and 75% of patients were hospitalized within 6 months of the onset of symptoms.

The standard deviation of the time that elapsed until treatment was as high as 498 days. This was probably due to the different course and severity of symptoms that vary greatly in polyneuropathy, being a heterogeneous group of diseases of the peripheral nervous system.

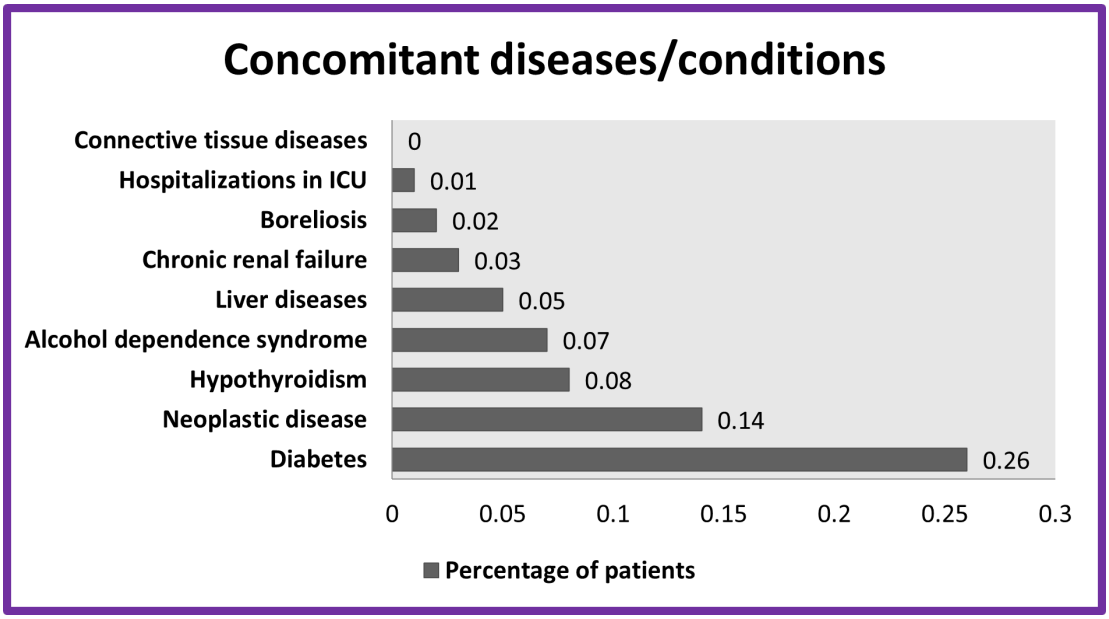
In over 50% of patients (67 patients: 32F and 32M), the disease had a chronic course (lasting over 3 months), in 25% of patients (13F and 12M) it was acute (from several days to 3

The Journal of Clinical Medicine (ISSN 2995-6315)

weeks), and in the remaining patients (8 people: 7K and 1M) - subacute (from 3 weeks to 3 months).

As **Figure 1** shows, the percentage of oncological patients was 0.14%. In the etiopathogenesis of polyneuropathy, the type of oncological therapy was much more important than the type of proliferative process. Chemotherapy is the most vital factor in the development of polyneuropathy and in the current study, this form of oncological treatment was applied in the highest percentage of patients - individually (0.08%) or in combination with radiotherapy (0.03%), accounting for a total of 0.11% of patients.

Figure 1. Review of concomitant diseases in the presented percentage of patients.



Sensory symptoms reported by patients.

Most patients reported skin sensory disturbances such as paresthesia (0.91%). Hypoesthesia was the second most common symptom among patients (0.58%).

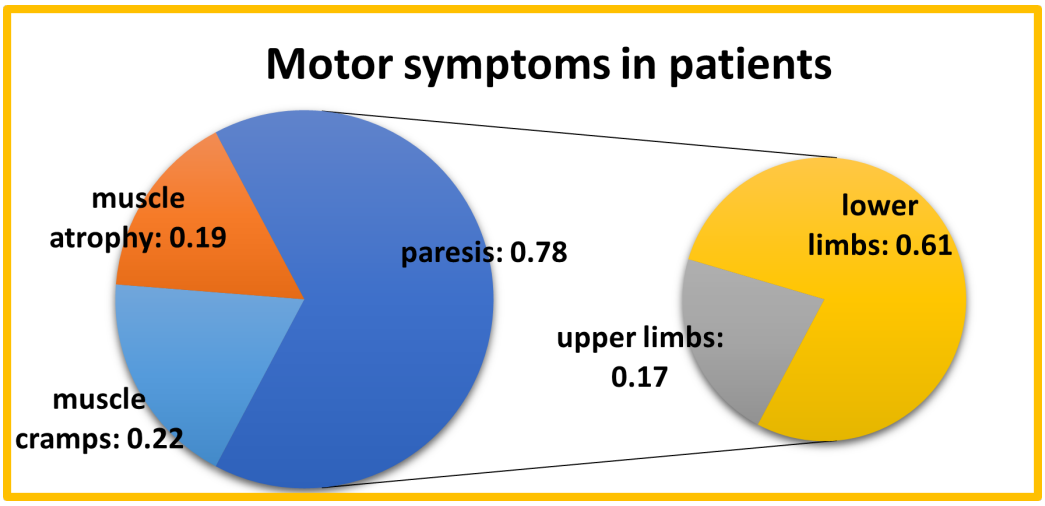
Neuropathic pain, was found to accompany 0.40% of patients. The smallest percentage of patients (0.03%) complained of sensory ataxia.

Most patients experienced neuropathic pain in the lower limbs (24%). Fewer than 10% of patients reported pain in the upper limbs (9%) and spine (7%).

The motor symptoms reported by patients upon admission to hospital were shown in **Figure 2**.

The following autonomic symptoms were considered: impaired sweat secretion, hypersensitivity to heat, gastrointestinal motility disorders, and orthostatic drops in blood pressure. Such ailments were reported by only 2 out of 100 patients participating in the study. The examined sensory symptoms are displayed in **Table I A**.

Figure 2. Percentage of patients reporting motor symptoms.



The Journal of Clinical Medicine (ISSN 2995-6315)

Table 1. Types of sensory disorders (A) and motor deficits (B) found in physical examination.

Table A

Sensory symptoms	Percentage of patients
Touch sensation disorders	1.33
In the „gloves" area	0.47
In the „socks" area	0.82
In another location and/or asymmetrical	0.04
Pain sensation disorders	0.17
Temperature sensation disorders	0.13
Vibration sensation disorders	0.19
Position sensation disorders	0.05
Sensory ataxia	0.03

The physical examination revealed the predominance of touch sensation observed in 1.33% of patients. They were most frequently examined in the "socks" area, followed by the "gloves" area. In a small percentage of patients (0.04%), the symptoms were located asymmetrically. The motor symptoms (**Table I B**)

Table B

Motor symptoms	Percentage of patients
Paresis/paralysis	1.33
Upper limbs	0.27
Lower limbs	0.56
Oculomotor muscles	0.02
Facial muscles	0.03
Deep reflexes– weak/missing	0.85
Muscle atrophy	0.21

In 88% of patients, physical examination revealed muscle weakness.

In 85% of cases, flaccid paresis was accompanied by weakening or absence of deep reflexes. Muscle atrophy was present in 21% of patients.

Only 10 patients had autonomic symptoms, such as circulatory system disorders and at least one of the following symptoms: changes in skin color, changes in skin temperature, swelling of the limbs, and sweating disorders.

Based on electroneurographic (ENG) examination, 64 patients were diagnosed with sensorimotor polyneuropathy. In 14 patients, only sensory polyneuropathy was diagnosed, while pure motor polyneuropathy was diagnosed in only 7 people. ENG testing was not performed in 15 people.

The electroneurographic examination revealed axonal polyneuropathy in 46 patients. Demyelinating-axonal polyneuropathy was diagnosed in 31 patients, and demyelinating polyneuropathy in 8 patients.

Cerebrospinal fluid examination was performed in 55% of patients. In 35% the result was correct. Protein-cell cleavage was found in 17% of patients, which, in correlation with the clinical picture and ENG results, allowed for the diagnosis of inflammatory demyelinating polyradiculoneuropathy. Neuroinfection resulting in polyneuropathic symptoms was diagnosed in 3% of patients.

Ninety-eight blood counts were performed, anemia was found in 20 people and leukocytosis in 7 patients.

Inflammatory markers were assessed. ESR were examined in 51 patients and were found elevated in 11 of them. CRP tests were performed in 78 patients and in 16 of them they were increased.

Fasting blood glucose levels were determined and, when elevated, they were verified in the same patients with an oral glucose tolerance test (OGTT) in 98 patients.

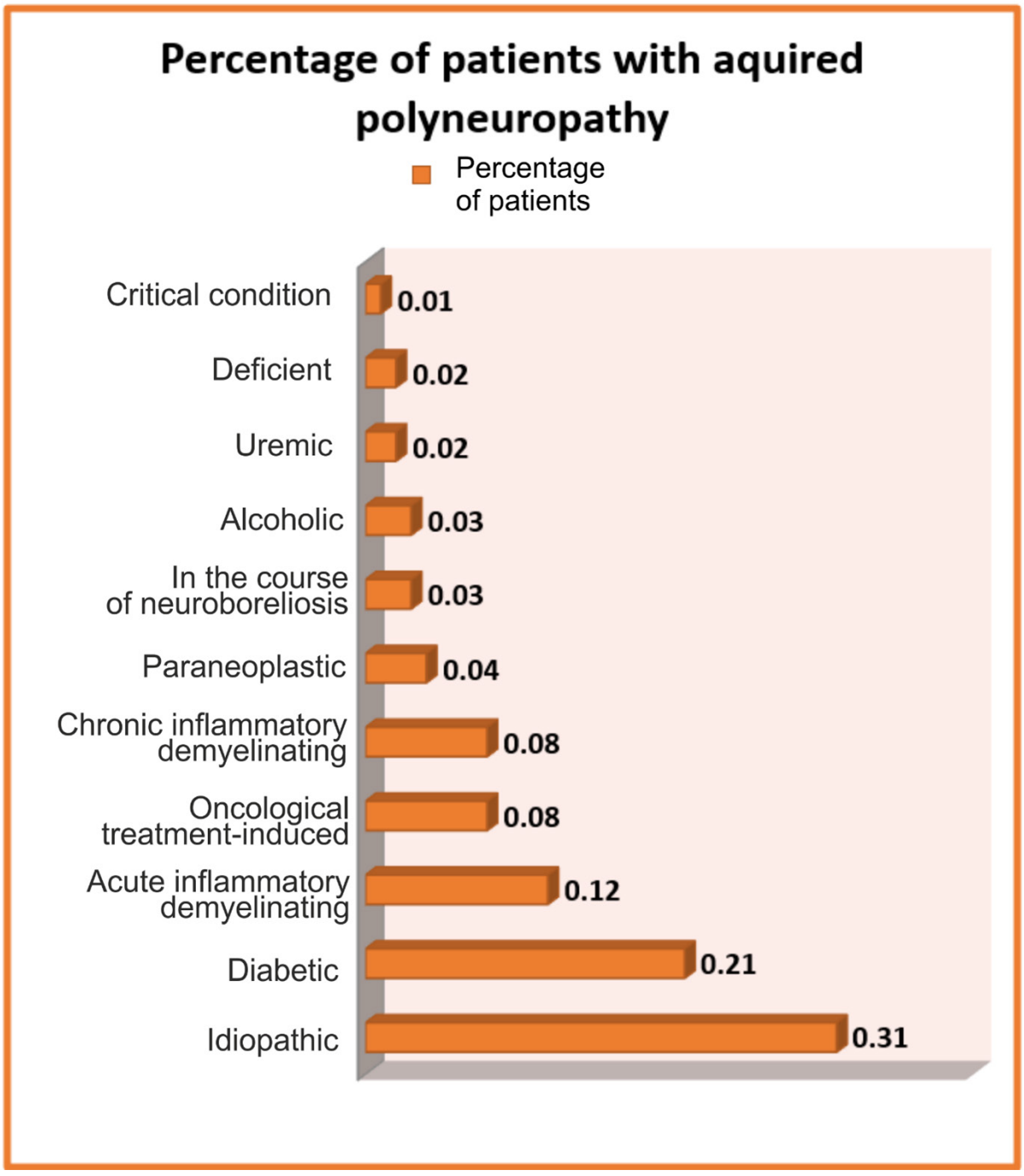
Vitamin B12 levels were assessed in 56 patients and were decreased in 10 cases.

The most frequently used drug in symptomatic treatment was lignocaine (58% of patients), accompanied by physical rehabilitation in 57%.

The Journal of Clinical Medicine (ISSN 2995-6315)

The average number of hospitalization days was 14.9. The shortest hospital stay lasted 2 days, and the longest was 33 days. The median number of days of hospital treatment was 13.50, which means that 50% of patients were hospitalized for 13 days or shorter. Hospitalization of 25% of patients lasted 10 days or less, and hospitalization of 75% of patients was no longer than 18 days.

Figure 3. Percentage of patients with the diagnosis of all acquired polyneuropathies (primary and secondary).



Division of polyneuropathies into congenital and acquired.

In our study, congenital polyneuropathy was diagnosed in only 5% of patients, while 95% of patients were diagnosed with acquired polyneuropathy.

Acute inflammatory demyelinating polyradiculoneuropathy (AIDP) was diagnosed in 60% of patients with primary polyneuropathy, while chronic inflammatory demyelinating polyradiculoneuropathy (CID) was diagnosed in 40% of patients.

Secondary polyneuropathy that occurred in the highest percentage of patients (0.41) was idiopathic polyneuropathy (cryptogenic). .

The Journal of Clinical Medicine (ISSN 2995-6315)

Taking into account the percentage of patients with acquired secondary polyneuropathy and the percentage of patients with all acquired polyneuropathies (both primary and secondary), the following conclusions can be drawn:

- idiopathic polyneuropathy was still the most common polyneuropathy in the studied group of patients;
- diabetic polyneuropathy was the second most common diagnosis;
- acute inflammatory demyelinating polyradiculoneuropathy was the third type of polyneuropathy diagnosed.

Idiopathic polyneuropathy was diagnosed in 31% of all patients. Statistical data indicate a female predominance among patients with idiopathic polyneuropathy.

Patients with unknown cause of polyneuropathy were aged 50 years and older in 80.65% of cases. The oldest patient was a woman aged 88.

The type of damaged nerve fibers is illustrated in **Figure 4A** and the type of damaged nerve fibers is shown in **Figure 4B**.

In the group of diagnosed secondary acquired polyneuropathies, 81% of patients had motor symptoms. Movement disorders were examinable in 86 out of 100 patients analyzed.

Sensory symptoms detected during neurological examination in respective polyneuropathies were found in a total of 88 patients.

Figure 4 A. Percentage of patients suffering from idiopathic polyneuropathy, taking into account the variety of nerve fibre damage.

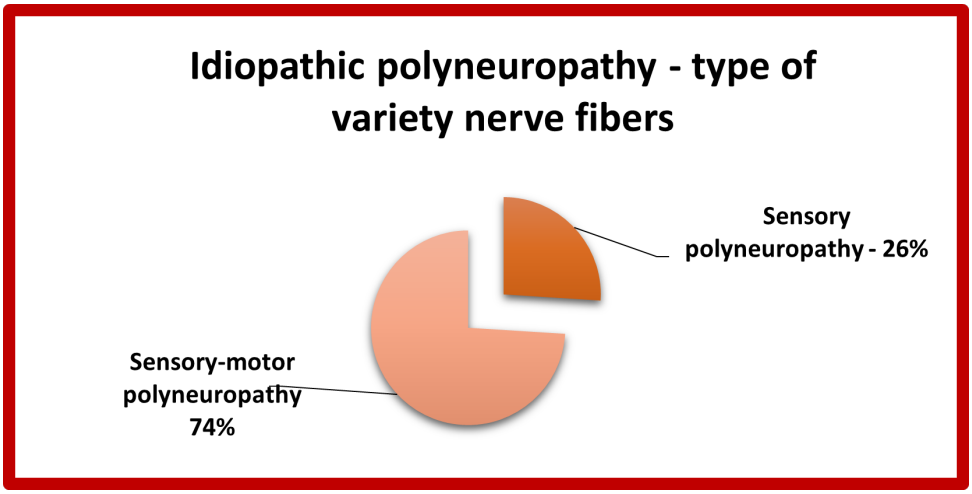
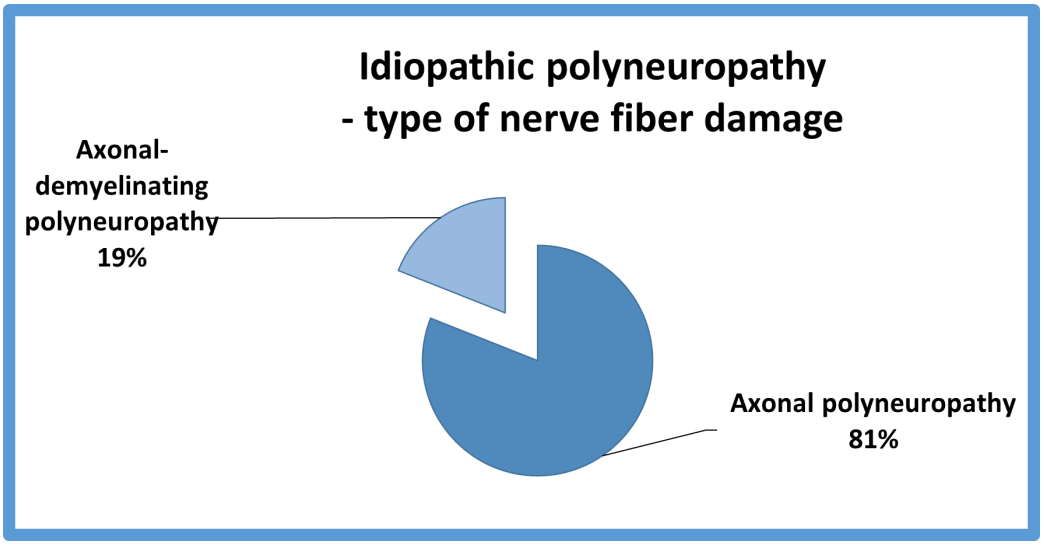


Figure 4 B. Percentage of patients suffering from idiopathic polyneuropathy, with regard to the type of nerve fibre damage.



The Journal of Clinical Medicine (ISSN 2995-6315)

DISCUSSION

This study analyzed the causes of polyneuropathy in patients hospitalized in the Neurology Ward of the J. Sniadecki Provincial Hospital in Bialystok in the years 2014 - 2019. The study included 100 patients with symmetric distal polyneuropathy (55 women and 45 men).

Acquired polyneuropathies were diagnosed in 95% of patients - primary in 21%, secondary in 79%. The primary ones include GBS and CIDP. According to scientific reports, the incidence of GBS is 0.9 - 1.9/100,000 healthy people, and it is more common in men (M:F = 1.5:1) and in the elderly [4]. This type of polyneuropathy was the third most frequently diagnosed polyneuropathy among the analyzed acquired polyneuropathies (12%). However, the incidence of chronic inflammatory polyneuropathy, considered to be the most common polyneuropathy undergoing treatment worldwide is 0.46 - 1.83/100,000 inhabitants [5]. CIDP ranks fourth in terms of the frequency of polyneuropathies diagnosed in the analysis.

The most common defined secondary polyneuropathy is diabetic polyneuropathy, accounting for 21% of the analyzed polyneuropathies. Diabetes contributes to 32-53% of peripheral neuropathy cases, making it the most common polyneuropathy [6, 7 , 8]. The risk of developing neuropathy has been shown to increase by 10 - 15% for each additional 1% of glycosylated hemoglobin. Determinants of diabetic neuropathy include both hypoglycemia and hyperglycemia. Diabetic neuropathies are divided into several types, among which chronic symmetric distal polyneuropathy is the most common, accounting for up to 70% of all cases [9].

Idiopathic polyneuropathy was diagnosed in 31 patients. This type is highly controversial, since with such high availability of diagnostic tests, it is not possible to determine the etiopathogenetic factor. The distinctive feature of this polyneuropathy was the age of onset - the percentage of patients over 50 years of age was 80.6% , with the predominance of females. The oldest patient was a woman aged 88. The main symptoms reported by patients concerned sensory experiences: paresthesia (tingling, burning, pricking sensation in the feet) in 87% of patients, hypoesthesia in the toes (48%), and neuropathic pain mainly in the feet (32%). Patients also complained of weakness of the toe extensors (16%). Electroneurographic examination showed sensorimotor polyneuropathy in 74% of patients, and sensory polyneuropathy was diagnosed in 26%. The types of nerve fiber damage found included axonal polyneuropathy (81%) and axonal-demyelinating polyneuropathy (19%). Idiopathic polyneuropathy was demonstrated among 26% of patients in the study population in the Netherlands. The incidence rate was estimated at 30.3/100.000 healthy people aged 40 years and older [10]. In other specialist centers, the cause of polyneuropathy could not be determined in 10-40% of cases; however, the course of the disease was always similar to the clinical picture of chronic idiopathic axonal polyneuropathy [11, 12].

The presented retrospective analysis shows an axonal-demyelinating lesion that does not fully meet the criteria for diagnosing this exact type of polyneuropathy. We also encountered, although more seldom, the neuropathic pain, described in the literature as a frequently accompanying pain. However, some scientists observe axonal-demyelinating disorders (most likely secondary) and classify the polyneuropathy as chronic idiopathic [13]. However, we believe that, based on the analysis performed, the diagnostic panel should be more often extended to include MRI with contrast of the nerve roots in order to exclude their swelling and differentiate it from inflammatory polyneuropathy (especially CIDP).

CIDP, according to some scientists, is more common in men, although more important is to determine the potential cause of polyneuropathy, which is difficult to diagnose. The considered factors that may have a significant impact are: metabolic predispositions, such as impaired glucose tolerance, hypertension, dyslipidemia and obesity [12, 13].

Numerous clinical studies have proven that people suffering from GBS have a genetic predisposition to susceptibility to this type of polyneuropathy after exposure to various pathogens [14, 15].

Claudia Vellozzi and Shahed Iqbal examined the complex relationship between influenza illness, influenza vaccination and the occurrence of GBS. Hospitalization rates for pneumonia and influenza illness were significantly correlated with hospitalization rates for GBS. GBS hospitalizations showed seasonality, with the winter months having higher rates compared to June [16]. Worth mentioning is the axonal subtype of GBS - acute motor axonal neuropathy (AMAN), which predominates in China and among Mexican children. It has been shown that significant severity of the disease from July to September (in summer) and significant deterioration of the course of polyneuropathy during hospitalization are characteristic features of AMAN cases [17].

Borrelia garinii has the greatest affinity for the nervous system involvement. The intensity of radicular pain clinically resembles discopathic pain, with the difference that it is more severe especially at night, poorly responds to analgesic treatment, and quickly subsides after the implementation of antibiotic therapy [18]. In most cases, peripheral polyneuropathy, like encephalomyelitis, is a late form of neuroborreliosis.

The presented analysis shows that the time elapsed from the onset of symptoms to the start of hospitalization ranged from 90 days in 50% to 180 days in 75% of patients, and the standard deviation was up to 498 days.

Polyneuropathy manifests itself with a wide range of

The Journal of Clinical Medicine (ISSN 2995-6315)

symptoms. In our opinion, it was the motor symptoms, such as paresis, or severe neuropathic pain that forced patients to use specialist medical services.

Worthy of attention is paraneoplastic polyneuropathy, a specific type of polyneuropathy, belonging to the paraneoplastic neurological syndromes and found in approximately 1% of cancer patients. In the current analysis, it was diagnosed in 4% of patients. However, numerous studies concerning PNS show that up to 15% of all oncological patients can be affected [19, 20].

Subacute sensory neuropathy is a typical neurological syndrome with a high risk of paraneoplastic etiology, accounting for 6% of all neuropathies [21]. The most common cancer associated with sensory neuropathy is small cell lung cancer. Other oncological diseases include breast cancer, ovarian cancer, Hodkin's lymphoma and sarcoma. The most characteristic antibody for this type of polyneuropathy is anti-Hu, followed by anti-CV2 and anti-amphiphysin [22].

The polyneuropathy presented above is closely related to polyneuropathy induced by oncological treatment, which occurred in 8% of the subjects with acquired polyneuropathy. Eight percent of the patients received only chemotherapy, 6% only radiotherapy and 3% underwent combined treatment.

According to literature data, the incidence of chemotherapy-induced neuropathy is approximately 38% [22]. Regardless of the type of the drug used, the clinical picture of neuropathy is the same, although the mechanisms of neurotoxicity are different. Factors contributing to the occurrence of this type of polyneuropathy include smoking, the presence of neuropathy before the start of treatment, genetic factors, abnormal creatinine clearance values, duration of treatment and cumulative dose of the drug. Electromyography examination (EMG) is of great importance, as it allows the assessment of the type and extent of damage to the peripheral nervous system in order to possibly modify chemotherapy. Polyneuropathy is most often induced by platinum derivatives, mitotic spindle poisons and immunomodulatory drugs [23].

Radiotherapy does not cause specific polyneuropathy; it causes damage mainly to the brachial or lumbosacral plexuses.

Alcoholic polyneuropathy is one of the most common complications of alcohol abuse and, according to literature data, may account for up to 66% of such side effects [24]. In our study, this type of polyneuropathy accounted for 3% of all acquired polyneuropathies.

In the current study, polyneuropathy resulting from vitamin B12 deficiency was found in 2% of patients. As the disease progresses, sensory (posterior cord) ataxia and sensory-motor polyneuropathy develop. It is difficult to separate neuropathic symptoms from spinal symptoms [25]. This type of neuropathy may develop when vitamin B12 values are at the lower limit of the normal range, and then the diagnosis can be made based on the measurement of increased concentrations of methylmalonic acid and homocysteine [26].

Uremic polyneuropathy is probably the most common complication of chronic renal failure according to literature data [27]. In our analysis, uremic polyneuropathy accounted for 2% of all acquired polyneuropathy cases and met the criteria for end-stage renal disease treated with hemodialysis.

Critical illness polyneuropathy (CIP) was the least common in our study. It was found in one out of 100 patients, namely in an individual transferred from the Intensive Care Unit to the Neurology Department. CIP is an acute axonal sensorimotor polyneuropathy usually suspected in patients who cannot be weaned off the ventilator after several weeks despite the absence of pulmonary or cardiac causes of respiratory failure. Bioenergetic failure is believed to be an important pathophysiological mechanism explaining both CIP and multi-organ failure [28].

The most important test used in the diagnosis of polyneuropathy and constituting an extension and complement to the clinical examination is the electrodiagnostic examination, which includes ENG and EMG. ENG examination is a very useful tool in the diagnosis and monitoring of treatment of nerve damage during chemotherapy, metabolic syndromes, deficiency syndromes, in alcohol abusers, with hereditary or inflammatory neuropathy [29]. Research data published in 2018 in Muscle Nerve showed that the results of electrodiagnostic tests contributed to a change in the type of therapy in 63.4% of patients [29].

Neuropathic pain most frequently occurs in the first sensory neuron of the nervous system as a result of its repeated damage. National and international guidelines consider new generation antiepileptic drugs (pregabalin and gabapentin) to be the most effective in the treatment of neuropathic pain [30]. Second-line drugs include lamotrigine, opioids and selective serotonin reuptake inhibitors, as well as tramadol [30].

In the current analysis, intravenous lidocaine was the most frequently used symptomatic drug, followed by gabapentin, which was replaced by pregabalin as the first-line drug in the following years.

In patients diagnosed with type 2 diabetes, the first screening test for polyneuropathy should be performed immediately after diagnosis, while in the case of type 1 diabetes - after 5 years from the onset of the disease symptoms. The tests should be repeated at least once a year [31, 32].

Data availability statement

The data that support the findings of this study are available in the Neurology Ward of the J. Sniadecki Provincial Hospital in Bialystok on request.

Author Contributions Statement

J.D.W. contributed to clinical and laboratory data acquisition

The Journal of Clinical Medicine (ISSN 2995-6315)

and analyzed and drafted manuscript. A.H. analyzed and extensively reviewed manuscript.

A.G. and D.M.-J. contributed to clinical and laboratory data acquisition.

All authors have read and agreed to the published version of the manuscript.

Disclosure of interest

The authors declare no conflict of interest.

Declaration of Funding

The research did not receive specific funding but was performed as a part of the work of the authors at the Neurology Ward of the J. Sniadecki Provincial Hospital in Bialystok.

Informed Consent Statement

Written informed consent has been obtained from patients to publish this paper,

Institutional Review Board Statement

The Bioethics Commission of Medical University of Bialystok approved our research. Approval code: R-I-002/209/2019; approval date: 28 March 2019.

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The Journal of Clinical Medicine (ISSN 2995-6315)

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