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Epidemiological, Clinical, Electroneuromyographic, And Etiological Profile Of Ataxic Neuropathies: A Study Of 18 Cases

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Abstract:
Objective*: This study aims to delineate the epidemiological, clinical, electroneuromyographic, and etiological characteristics of ataxic neuropathies based on an analysis of 18 cases.*
Methods*: A retrospective review was conducted on 18 patients diagnosed with ataxic neuropathy from Mars 2023 to Mars 2024 in the neurology department at the University Hospital Center of Tanger. Data on demographics, clinical presentation, neurological examination, electroneuromyographic findings, and etiological investigations were collected and analyzed.*
Results*: The cohort comprised 18 patients (8 males and 10 females) with a mean age of 60 years (range: 45-78 years). The predominant clinical feature was progressive gait ataxia, reported by 15 patients (83.3%), followed by sensory disturbances in 14 patients (77.8%) and limb weakness in 10 patients (55.6%). Neurological examinations consistently revealed sensory ataxia across all cases. Electroneuromyographic studies indicated axonal neuropathy in 16 patients (88.9%), characterized by reduced nerve conduction velocities and absent sensory nerve action potentials. Etiological assessment identified autoimmune causes in 7 cases (38.9%), metabolic disorders in 5 cases (27.8%), and idiopathic origins in 6 cases (33.3%).*
Conclusion*: Ataxic neuropathies present a heterogeneous group of disorders marked by progressive gait instability and sensory deficits. A thorough clinical and electroneuromyographic evaluation is essential for accurate diagnosis and management. Identifying the underlying etiology is crucial for tailoring appropriate therapeutic strategies, which may significantly impact patient outcomes. This study underscores the importance of a systematic approach in the assessment of ataxic neuropathies to enhance diagnostic accuracy and treatment efficacy.*

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I. Introduction

Ataxic neuropathy is a complex and heterogeneous condition characterized by progressive gait instability and sensory deficits, which significantly impair patients' quality of life. Despite its clinical relevance, ataxic neuropathy remains under-recognized, partly due to its varied presentations and the diagnostic challenges it poses[1]. Unlike more common neuropathies, ataxic neuropathy often involves a combination of motor and sensory symptoms, making clinical assessment and diagnosis particularly intricate[2].

The accurate diagnosis of ataxic neuropathy relies heavily on a multidisciplinary approach that includes detailed clinical evaluation, comprehensive electrophysiological studies, and thorough etiological investigations. Electroneuromyography (ENMG) is a critical tool in this diagnostic process, as it helps differentiate between axonal and demyelinating patterns of neuropathy. Additionally, identifying the underlying etiology is paramount for determining the appropriate treatment and improving patient outcomes[3].

This article aims to elucidate the epidemiological, clinical, electroneuromyographic, and etiological characteristics of ataxic neuropathies by examining a cohort of 18 patients. By providing a detailed analysis of these cases, we hope to enhance understanding and awareness of ataxic neuropathy, facilitate earlier diagnosis, and promote more effective management strategies. Through this study, we seek to contribute to the growing body of knowledge on this debilitating condition and underscore the importance of a systematic approach to its evaluation and treatment.

II. Methods:

A retrospective review was conducted on 18 patients diagnosed with ataxic neuropathy at the neurology department of our University Hospital between Mars 2023 to Mars 2024. The inclusion criteria were patients

presenting with clinical signs of ataxia and confirmed diagnosis of neuropathy through electroneuromyographic studies.

Patient Demographics: Data on patient age, gender, and duration of symptoms were collected.

Clinical Assessment: Comprehensive clinical evaluations were conducted, focusing on gait analysis, sensory examination, and motor strength testing. Specific attention was given to identifying signs of sensory ataxia, including proprioceptive deficits and Romberg's test results.

Electroneuromyographic Studies: All patients underwent standard electroneuromyography (ENMG) to assess nerve conduction velocities (NCVs) and sensory nerve action potentials (SNAPs). Both motor and sensory nerve studies were performed to identify axonal or demyelinating neuropathies.

Etiological Investigations: A detailed etiological workup was performed, including autoimmune markers (e.g., anti-neuronal antibodies), metabolic panels (e.g., vitamin B12 levels, thyroid function tests), and other relevant diagnostic tests (e.g., genetic testing, lumbar puncture for cerebrospinal fluid analysis).

III. Results:

Patient Demographics: The study cohort consisted of 18 patients (8 males and 10 females) with a mean age of 60 years (range: 45-78 years). The duration of symptoms prior to diagnosis ranged from 6 months to 5 years, with a median duration of 18 months.

Clinical Findings: The predominant clinical feature was progressive gait ataxia, observed in 15 patients (83.3%). Sensory disturbances, including numbness and tingling, were reported by 14 patients (77.8%). Limb weakness was noted in 10 patients (55.6%), primarily affecting the lower extremities. All patients exhibited sensory ataxia on neurological examination, with positive Romberg's test in 16 patients (88.9%).

Tableau 1: Prodeminent clinical features

Clinical features	Percentage
Progressive Gait Ataxia	83.3%
Sensory Disturbances	77.8%
Limb Weakness	55.6%
Positive Romberg's Test	88.9%

Electroneuromyographic Findings: ENMG studies revealed evidence of axonal neuropathy in 16 patients (88.9%), characterized by reduced NCVs and absent SNAPs. Two patients (11.1%) exhibited features suggestive of demyelinating neuropathy. These findings were consistent across multiple nerves tested, including the peroneal, tibial, median, and ulnar nerves.

Tableau 2: Types of Neuropathy

Type of neuropathy	Percentage
Axonal Neuropathy	88.9%
Demyelinating Neuropathy	11.1%

Etiological Findings: The etiological assessment identified the following causes:

Autoimmune etiologies in 7 patients (38.9%), including chronic inflammatory demyelinating polyneuropathy (CIDP) and paraneoplastic syndromes.

Metabolic disorders in 5 patients (27.8%), such as diabetes mellitus and vitamin B12 deficiency.

Idiopathic origins in 6 patients (33.3%), where no definitive cause could be determined despite extensive workup.

Tableau 3: Etiological findings

Etiology	Percentage
Autoimmune	38.9%
Metabolic Disorders	27.8%
Idiopathic	33.3%

Treatment and Outcomes: Patients received individualized treatment based on the identified etiology. Those with autoimmune neuropathies were treated with immunomodulatory therapies, including intravenous immunoglobulins (IVIG) and corticosteroids, resulting in symptom stabilization or improvement in 5 of 7 cases. Patients with metabolic disorders were managed with appropriate supplementation and glycemic control, showing improvement in 4 of 5 cases. Idiopathic cases were managed symptomatically, with varying responses.

IV. Discussion

The present study provides a comprehensive overview of the epidemiological, clinical, electroneuromyographic, and etiological characteristics of ataxic neuropathies in a cohort of 18 patients. Our

findings highlight the complex and heterogeneous nature of this condition, underscoring the diagnostic challenges and the need for a systematic approach to evaluation and management.

Clinical Features:

The predominant clinical feature in our cohort was progressive gait ataxia, observed in 83.3% of patients. This aligns with previous studies that identify gait instability as a hallmark of ataxic neuropathy. Sensory disturbances, including numbness and tingling, were reported by 77.8% of patients, emphasizing the sensory component of the neuropathy. Additionally, limb weakness, particularly in the lower extremities, was noted in 55.6% of cases, indicating that motor involvement, although less common, is a significant aspect of the disease. These clinical features are consistent with the literature, which describes ataxic neuropathy as a condition with both sensory and motor deficits[1].

Electroneuromyographic Findings:

Electroneuromyographic studies revealed that 88.9% of our patients had axonal neuropathy, characterized by reduced nerve conduction velocities and absent sensory nerve action potentials. This finding is critical, as it helps distinguish axonal from demyelinating neuropathies, which have different etiologies and treatment approaches. The 11.1% of patients with demyelinating features also underscores the heterogeneity of ataxic neuropathy, suggesting that clinicians should not rule out demyelinating processes in patients presenting with ataxia[3].

Etiological Insights:

Our etiological assessment identified autoimmune causes in 38.9% of patients, with conditions such as chronic inflammatory demyelinating polyneuropathy (CIDP) and paraneoplastic syndromes being prominent[4]. This is in line with existing research that highlights autoimmune mechanisms as significant contributors to ataxic neuropathy. Metabolic disorders, including diabetes mellitus and vitamin B12 deficiency, accounted for 27.8% of cases, reinforcing the importance of metabolic evaluation in patients with ataxic symptoms[5]. Notably, 33.3% of our cohort had idiopathic neuropathy, reflecting the current gaps in understanding and the need for further research to uncover unknown etiologies[4,6,7].

V. Treatment And Outcomes:

The treatment outcomes in our study underscore the importance of etiological diagnosis. Patients with autoimmune neuropathies responded favorably to immunomodulatory therapies, such as intravenous immunoglobulins (IVIG) and corticosteroids, with 71.4% showing symptom stabilization or improvement. This highlights the effectiveness of targeted immunotherapy in autoimmune ataxic neuropathies . Metabolic neuropathies, managed with appropriate supplementation and glycemic control, also demonstrated significant improvement, indicating that early and accurate diagnosis of metabolic causes can lead to effective treatment. The varied responses in idiopathic cases suggest a need for more individualized and possibly experimental treatment approaches[1,8].

VI. Challenges And Recommendations:

One of the significant challenges in diagnosing ataxic neuropathy is the variability in clinical presentation and the overlap with other neurological disorders. Our study emphasizes the need for heightened clinical awareness and a high index of suspicion in patients presenting with ataxia, especially when initial systemic evaluations are unremarkable. Electroneuromyography remains a cornerstone in the diagnostic process, and its use should be complemented by a thorough etiological workup[1].

Future research should focus on identifying biomarkers and genetic factors that may contribute to the idiopathic forms of ataxic neuropathy. Additionally, longitudinal studies are needed to better understand the long-term outcomes and response to treatment in different etiological subgroups.

VII. Conclusion:

Ataxic neuropathies represent a diverse and diagnostically challenging group of disorders. This study highlights the importance of a comprehensive and systematic approach to the diagnosis and management of ataxic neuropathy. By enhancing our understanding of its epidemiological, clinical, electroneuromyographic, and etiological profiles, we can improve diagnostic accuracy, tailor treatments more effectively, and ultimately enhance patient outcomes. Continued research and education are essential to address the gaps in knowledge and improve care for patients with this debilitating condition.

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