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Spectrum of Neurological Disorders Related to Autoimmune Diseases in Brazzaville, Congo

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Abstract

Background: Autoimmune diseases, which are among the leading causes of morbidity and mortality in the world, are pathologies caused by a dysfunction of the immune system. They can affect the central nervous system, the peripheral nervous system or both nervous systems. **Objectives:** To describe the epidemiological, clinical, paraclinical, therapeutic and evolutive aspects of neurological disorders related to autoimmune diseases. **Methods:** This was a prospective cohort study. It was carried out from 1 January 2015 to 31 December 2019 (5 years). It focused on patients aged 15 years and above, who were hospitalized or followed as ambulatory patients for neurological disorders related to autoimmune diseases in the neurology department of the university teaching hospital in Brazzaville. **Results:** Among the 41 patients who fulfilled inclusion criteria, there were 29 (70.73%) women and 12 (29.27%) men. The average age of patients was 38.3 ± 13.8 years. An increase in the frequency of neurological disorders related to autoimmune diseases was observed every year. The main neurological disorders were neuromyelitis optica spectrum disorders (n = 14; 34.15%), acute polyradiculoneuropathies (n = 13; 31.71%), chronic polyradiculoneuropathies (n = 4; 9.75%) and acute disseminated encephalomyelitis (n = 3; 7.31%). The treatments administered, which consisted of corticosteroids and immunosuppressive drugs, had significantly improved the vital prognosis and functional status of patients (p = 0.025). **Conclusion:** In our study population, neurological disorders related to autoimmune diseases are rare. The neurological clinico-pathological entities diagnosed are similar to those reported in the literature. The therapeutic approaches used improve the quality of life of patients.

Keywords

Autoimmune Diseases, Neurological Disorders, Brazzaville

1. Introduction

Autoimmune diseases are pathologies that result from a dysfunction of the immune system, which is caused by genetic, hormonal and environmental factors [1] [2] [3] [4] [5].

Autoimmune diseases affect all ages [6]. However, they mainly affect young and adult populations [7] [8] [9] [10]. Worldwide, they are among the leading causes of morbidity and mortality [1] [11] [12] and their prevalence varies between 4% and 9% [3] [5].

Autoimmune diseases include single organ-specific autoimmune diseases and systemic autoimmune diseases [2] [5]. However, combinations of single organ or systemic autoimmune diseases may be encountered in patients; these are multiple autoimmune syndromes [5] [13] [14] [15].

Autoimmune diseases can affect the central nervous system, the peripheral nervous system or both nervous systems [4] [16]. The incidence of nervous system-specific autoimmune diseases is lower than for other organs-specific autoimmune diseases [4] [17] [18]. The prevalence of autoimmune diseases of the nervous system varies from continent to continent, but also from race to race [4] [17] [18].

If the landscape of autoimmune diseases of the nervous system is well explored in developed countries [4] [19] [20], this is not the case in low-income countries, such as those in Africa [21] [22].

In Congo, Lamini N'Soundat and N'Tsiba report that connectivitis and rheumatoid arthritis are predominant autoimmune and systemic diseases at the university hospital in Brazzaville, with frequencies of 42.2% of cases and 36.6% of cases, respectively [23]. Data on neurological disorders related to autoimmune diseases are not available. The objective of our study is to describe the epidemiological, clinical, paraclinical, therapeutic and evolutive aspects of these disorders in Brazzaville, Congo.

2. Methods

This was a prospective cohort study. It was carried out from 1 January 2015 to 31 December 2019 (5 years). It focused on patients hospitalized or followed as ambulatory patients for neurological disorders related to autoimmune diseases in the neurology department of the university teaching hospital in Brazzaville. It was approved by the National Health Sciences Research Ethics Committee.

Inclusion criteria were patients: aged 15 years and above; who gave consent to participate in the study; who had an autoimmune disease whose diagnosis was confirmed by findings of clinical examination, biological investigations and magnetic resonance imaging (MRI), in accordance with the recommendations for the

diagnosis of autoimmune diseases [24].

Patients with neurological disorder not related to autoimmune diseases were excluded from the study.

Findings of biological investigations were obtained through the following examinations: analysis of cerebrospinal fluid; blood tests for inflammation, blood cells count, muscle tests (creatine phosphokinase), immunological tests; histopathological examination; electroencephalography for epileptic abnormalities or pathological slowing of the background rhythm; electroneuromyography to search for types of peripheral disorder and to assess the electrical severity of disorder; visual evoked potentials for the search of a lengthening of latencies in agreement with retrobulbar optic neuritis.

The study variables were: demographic (age and gender); clinical (medical history, reasons for consultation/hospitalization, time to diagnosis, types of neurological disorder, and types of autoimmune disease); biological; radiological; therapeutic (types of medication); evolutive.

Physician Global Assessment (PGA) was used for evaluating treatment response [25]. It was based on the principle of a visual analogy scale of disease severity improvement. It was scored from 0 to 3: 0 = no evolution of the patient's functional status; 1 = weak evolution of the patient's functional status; 2 = moderate evolution of the patient's functional status; 3 = good evolution of the patient's functional status.

The patients were followed for a duration of 12 months. For each patient, follow-up was done at regular intervals of 1, 3, 6 and 12 months.

For each patient, data related to the study variables were collected through a survey record, which consisted of 5 parts. The first part was devoted to demographic data. The second part focused on clinical data. The third part was devoted to biological and radiological data. The fourth part was devoted to treatments. The fifth part focused on clinical evolution data.

The data collected was analyzed using CS Pro 7.4 software. Qualitative and quantitative variables were expressed as percentages and mean ± standard deviation, respectively. Fisher's test and Pearson's correlation were used to investigate the relationship between the clinical evolution and the treatment administered. The significance level was set at $p < 0.05$.

3. Results

3.1. Epidemiological Aspects

Out of a total of 4325 patients with a neurological pathology during the study period (5 years), 47 had neurological disorders related to autoimmune diseases, for an overall average frequency of 1.09% (95% CI = 0.71 - 1.30%).

Of the 47 patients, 6 (12.77%) did not consent to participate in the study. The latter had generalized myasthenia gravis with thymoma only. Thus, only 41 (87.23%) patients were included in the study.

Among the 41 patients, there were 29 (70.73%) women and 12 (29.27%) men. The average age of patients was 38.3 ± 13.8 years (range: 15 - 69 years).

Table 1 shows the frequency of neurological disorders related to autoimmune diseases by study year. There was an increase in frequency of: 33.33% in 2016, compared to 2015; 53.01% in 2017, compared to 2016; 14.43% in 2018, compared to 2017; 62.98% in 2019, compared to 2018.

3.2. Clinical and Paraclinical Aspects

Of the 41 patients with neurological disorders related to autoimmune diseases, 34 (82.93%) had a medical history. Table 2 presents the medical history of these 34 patients. The predominant medical histories were hypertension (35.29%) and infectious diseases (32.34%). No family history was reported.

A post-vaccination circumstance for the occurrence of neurological disorders related to autoimmune diseases was noted in 12 (29.27%) patients.

Table 3 presents the data related to the reasons for hospitalization/consultation of patients. Motor deficit (68.29%), sensory disorders (34.15%) and muscular weakness (29.27%) were the main reasons.

Table 1. Frequency distribution of neurological disorders related to autoimmune diseases by study year.

	Total number of patients with nervous system disease	Number of patients with neurological disorder related to autoimmune diseases	Frequency
	n	n	
2015	753	2	0.26%
2016	1026	4	0.39%
2017	845	7	0.83%
2018	824	8	0.97%
2019	877	23	2.62%

Table 2. Data related to the patients' medical history.

	n	%
Arterial hypertension	12	35.29
Infectious diseases		
Hepatitis C	4	11.76
HIV	3	8.82
Pulmonary tuberculosis	2	5.88
Hepatitis B	1	2.94
Lyme disease	1	2.94
Dry eye and mouth syndrome	6	17.65
Excessive alcohol consumption	5	14.71
Repeated miscarriages	4	11.76
Long-term use of medication	2	5.88
Thyroiditis	2	5.88
Type 1 diabetes mellitus	2	5.88

Table 3. Data related to the reasons for hospitalization/consultation of patients.

	n	%
Motor deficit	28	68.29
Sensory disorders	14	34.15
Muscular weakness	12	29.27
Headaches	8	19.51
Alteration of general status	7	17.07
Reduced visual acuity	4	9.76

The average time to diagnosis from the onset of symptoms was 106 ± 82 days (range: 5 - 740 days).

The autoimmune diseases found in our patients included nervous system-specific autoimmune diseases (n = 34; 82.93%) and systemic autoimmune diseases (n = 7; 17.07%).

Neurological disorders related to nervous system-specific autoimmune diseases included:

- Acute polyradiculoneuropathies, which were diagnosed on the basis of clinical manifestations and electroneuromyography in 13 patients. The clinical manifestations were: proximal motor deficit with tingling paresthesia of the lower limbs, ascending and symmetrical (69.23% of cases; n = 9); pure motor deficit (30.77% of cases; n = 4); multimodal hypoesthesia (100% of cases); abolition of the reflexes of the 4 limbs (100% of cases); absence of cranial neuropathy and dysautonomia symptoms (69.23% of cases; n = 9). The electrophysiological forms were: acute inflammatory demyelinating polyradiculoneuropathy (n = 3), acute motor axonal neuropathy (n = 4) and acute motor sensitive axonal neuropathy (n = 6). Acute polyradiculoneuropathies were all Guillain-Barre Syndrome. Cerebrospinal fluid was normal in 5 patients and abnormal in 8 patients. Cerebrospinal fluid analysis of these 8 patients showed albumino-cytological dissociation;
- Chronic polyradiculoneuropathies, which were diagnosed on the basis of clinical findings and electroneuromyography in 4 patients. The clinical findings were: asymmetrical sensory-motor deficit of the lower limbs (100% of cases); proximal motor impairment (100% of cases); distal burn-like paresthesia (100% of cases); tacto-algesic hypoesthesia in "glove and socking" type with proprioceptive ataxia associated with diminished pallesthesia (100% of cases); abolition of the reflexes of the 4 limbs (100% of cases); absence of cranial neuropathy and dysautonomia symptoms (100% of cases). There were 2 cases of Lewis-Sumner syndrome and 2 cases of chronic inflammatory demyelinating forms. Cerebrospinal fluid was normal in all patients;
- Acute disseminated encephalomyelitis, which was diagnosed on the basis of clinical features, electroneuromyography and cerebro-medullary MRI with gadolinium injection in 3 patients. The clinical features were: onset of disease after an infectious episode in 2 (66.7% of cases) patients and vaccination in 1

(33.3% of cases) patient; disturbance of consciousness (100% of cases); motor deficit (100% of cases); retrobulbar optic neuritis (66.66% of cases; n = 2); multimodal hypoesthesia of the lower limbs (33.33% of cases; n = 1). Cerebro-medullary MRI with gadolinium injection showed large (>2 cm), disseminated, poorly delineated and asymmetric multifocal lesions, predominantly in the grey matter in the subcortical regions and the spinal cord, hyperintense signal on T2-weighted sequences (see **Figure 1**). These lesions were of the same age. They were not enhanced by gadolinium injection. Electroneuromyography showed moderate sensory-motor axonal polyradiculoneuropathy in all patients. Electroencephalography showed a slowing of the background activity in 2 patients; however, it was normal in 1 patient. Cerebrospinal fluid was normal in 1 patient and abnormal in 2 patients. Cerebrospinal fluid analysis of these 2 patients showed moderate hyperproteinorachia, lymphocytes and an oligoclonal band;

- Neuromyelitis optica, which was diagnosed on the basis of clinical manifestations and cerebro-medullary MRI with gadolinium injection in 14 patients. The clinical manifestations were: retrobulbar optic neuritis (100% of cases); spinal cord syndrome (100% of cases). On MRI, spinal cord injuries were observed in the cervical region in 2 patients and in the dorsal region in 12 patients; these were extensive and contiguous spinal cord lesions on more than three vertebral segments. The typical features of retrobulbar optic neuritis were not visible on all MRI images. Visual evoked potentials performed in 6 patients showed a latency prolongation consistent with retrobulbar optic neuritis in only 4 patients. Cerebrospinal fluid analysis showed pleocytosis and lymphocytes in all patients; there was no oligoclonal band. The anti-aquaporin-4 antibody test was seropositive in 1 patient.

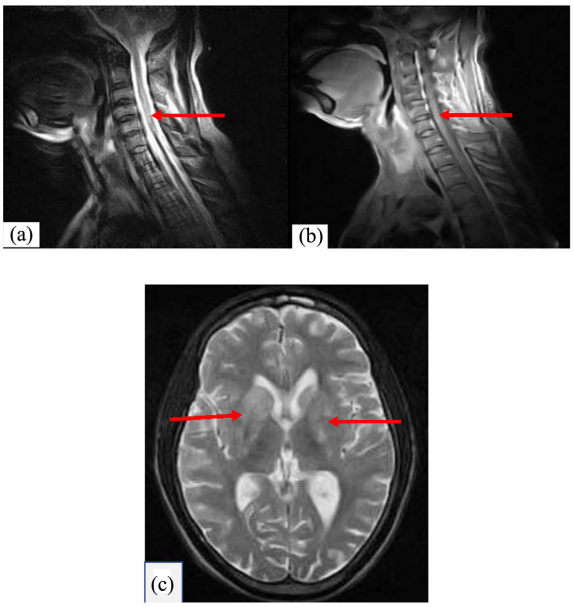

Figure 1. MRI Images related to acute disseminated encephalomyelitis: presence of intramedullary ((a) and (b)) and basal ganglia (c) hyperintense signals on T2-weighted sequences.

The following systemic autoimmune diseases caused neurological disorders: systemic lupus erythematosus; systemic scleroderma; systemic sarcoidosis; systemic vasculitis; Sjögren's syndrome.

In the context of systemic lupus erythematosus, autoimmune encephalitis was diagnosed on the basis of RMI and clinical manifestations in 1 patient. MRI showed multiple cerebral arterial infarctions caused by occlusion of the middle cerebral arteries. The clinical manifestations were: spastic tetraplegia without sensory disorders, grand mal seizures, butterfly wing rash, non-erosive elbow arthritis, lupus nephritis not histologically classified, and autoimmune hemolytic anemia with thrombocytopenia. Antinuclear antibody was found seropositive in the patient.

In the context of systemic scleroderma, myogenic damage was identified using electroneuromyography in 1 patient. The latter had the following clinical findings: muscle pain; muscle weakness; erythema on the face, neck, back of hands and thighs, accompanied by oedema. Serum creatine phosphokinase activity was elevated (356 IU/l), compared to the normal laboratory value (25 - 238 IU/l). The patient tested seropositive for anti-Jo1 antibody.

In the context of systemic sarcoidosis, neurosarcoidosis was diagnosed on the basis of clinical manifestations, electroneuromyography and MRI in 1 patient. The latter had the following clinical manifestations: confusion, impulsivity and disinhibition, generalized tonic-clonic seizures, flaccid paraparesis without sphincter disorders and bilateral Babinski, non-productive irritative cough and exertional dyspnea, ankle arthritis, fever, weight loss. Moderate sensory-motor axonal polyneuropathy was demonstrated by means of electroneuromyography in the patient. Brain MRI with gadolinium injection showed FLAIR hyperintense signals in the supratentorial and subtentorial white matter (see **Figure 2(a)**). Chest X-ray showed bilateral and symmetrical hilar adenopathies without parenchymal lesions (see **Figure 2(b)**). Cerebrospinal fluid analysis revealed lymphocytic meningitis (hyperproteinorachia with normoglycorachia) associated with intrathecal IgG synthesis.

In the context of systemic vasculitis, 2 patients had moderate distal sensory-motor axonal polyneuropathy demonstrated by means of electroneuromyography, and one of them also had cerebral artery infarction. Symptoms suggestive of cerebral artery infarction were: parieto-temporal headaches; dystonic-type abnormal movements; left hemiparesis. MRI showed that the affected arterial territory was the deep middle cerebral artery. Both patients had the following clinical manifestations: flaccid paraparesis; pain; painful paresthesia like burning or electric shocks; oedema of the lower limbs; Raynaud's syndrome; butterfly wing erythema; altered general status; oligoarthritis; hypertension. Cerebrospinal fluid analysis showed hyperproteinorachia and pleocytosis. Erythrocyte sedimentation rate was high. The complete blood count test revealed hyperleukocytosis, anemia and thrombocytopenia. Serologies were negative for hepatitis B and C, HIV, *Helicobacter pylori*. Both patients were seropositive for anti-neutrophil cytoplasmic antibody (ANCA).

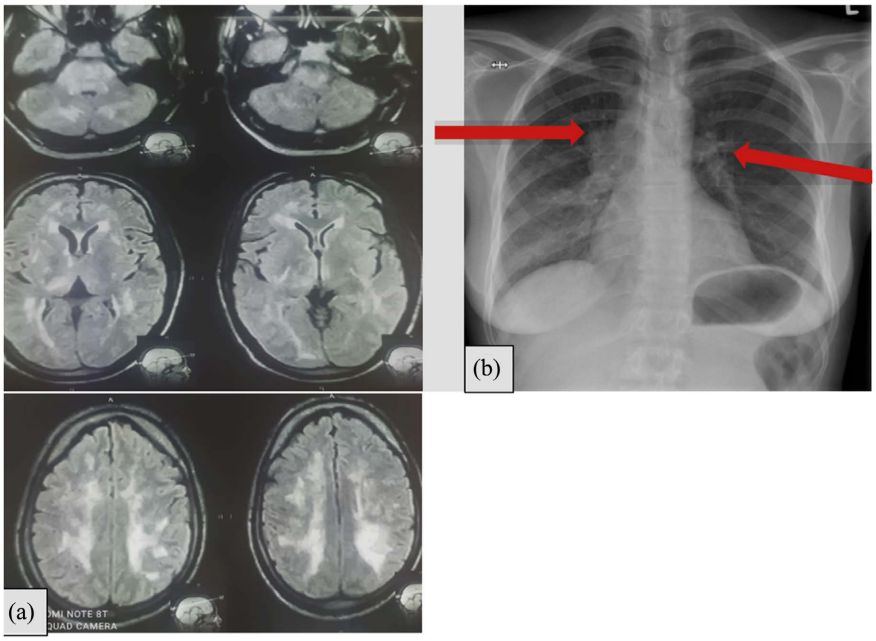

Figure 2. Images related to systemic sarcoidosis: presence of diffuse hyperintense signals on FLAIR sequences on brain MRI (a); presence of bilateral and symmetrical hilar adenopathies on chest X-ray (b).

In the context of Sjögren's syndrome, severe sensory-motor axonal polyneuropathy was diagnosed in 2 patients using electroneuromyography and based on the following clinical features: heaviness with sensory disorders such as tingling in all four limbs, ataxia on walking associated with abolition of osteotendinous reflexes. Extra neurological manifestations were: dry eye and mouth syndrome; Schirmer's test positivity in both eyes; presence of focal lymphocytic sialadenitis in the labial salivary gland biopsy with a focus score ≥ 1 in the 2 patients. The anti-SSA/Ro and anti-SSB autoantibodies tests were seropositive in the 2 patients. Both patients had no history of hepatitis C, HIV infection, sarcoidosis or radiotherapy.

Table 4 shows the distribution of neurological disorders related to autoimmune diseases by type of nervous system, which was as follows: 51.22% (n = 21) for the peripheral nervous system; 36.58% (n = 15) for the central nervous system; 12.20% (n = 5) for both nervous systems.

3.3. Therapeutic and Evolutive Aspects

Of the 41 patients, 32 (78.05%) had received drug treatment, 4 (9.75%) had died in the neurology department of the university hospital before the start of treatment, and 5 (12.20%) were not treated.

Of the 5 patients who did not receive treatment, 3 had acute inflammatory demyelinating polyradiculoneuritis and 2 had acute sensory-motor axonal neuropathy. The 3 patients with acute inflammatory demyelinating polyradiculoneuritis had not been treated because they could not afford the drugs. The 2 patients with acute sensory-motor axonal neuropathy refused treatment.

Table 4. Distribution of neurological disorders related to autoimmune diseases by type of nervous system.

	n	%
Central nervous system		
Neuromyelitis optica spectrum disorders	14	34.15
Cerebral arterial infarction in systemic lupus erythematosus	1	2.44
Peripheral nervous system		
Acute polyradiculoneuropathies	13	31.71
Chronic polyradiculoneuropathies	4	9.75
Neuropathy in systemic vasculitis	1	2.44
Neuropathy in Sjögren's syndrome	2	4.88
Myogenic damage in *systemic scleroderma*	1	2.44
Central and peripheral nervous systems		
Acute disseminated encephalomyelitis	3	7.31
Neuropathy and encephalitis in systemic sarcoidosis	1	2.44
Neuropathy and cerebral artery infarction in systemic vasculitis	1	2.44

The first-line treatment in all 32 patients was corticosteroid therapy. Methylprednisolone and prednisolone were administered intravenously in 30 (93.75%) patients and orally in 2 (6.25%) patients, respectively.

The second-line treatment consisted of the following drugs: prednisolone (n = 24; 75% of cases); cyclophosphamide (n = 5; 15.62% of cases); azathioprine (n = 2; 6.25% of cases); hydroxychloroquine (n = 1; 3.13% of cases).

During the follow-up period of the treated patients, no deaths were recorded. At the sixth month of patient follow-up, 3 (8.82%) patients were lost to follow-up.

Figure 3 shows the evolution of the functional status of patients. At 1-month follow-up, among the 32 patients, 27 (82.4%) had no evolution of the functional status and 5 (17.6%) had weak evolution of the functional status.

At 3-month follow-up, and as compared to the 1-month follow-up data, there were a significant decrease in the rate of patients with no evolution of the functional status (12.5%; n = 4) (p = 0.000) and a significant increase in the rates of patients with weak evolution of the functional status (78.1%; n = 25) (p = 0.000) and patients with moderate evolution of the functional status (9.4%; n = 3) (p = 0.000).

At 6-month follow-up, and as compared to the 3-month follow-up data, there were a non-significant decrease in the rates of patients with no evolution of the functional status (9.4%; n = 3) and patients with weak evolution of the functional status (71.9%; n = 23), and a significant increase in the rate of patients with moderate evolution of the functional status (18.7%; n = 6) (p < 0.001).

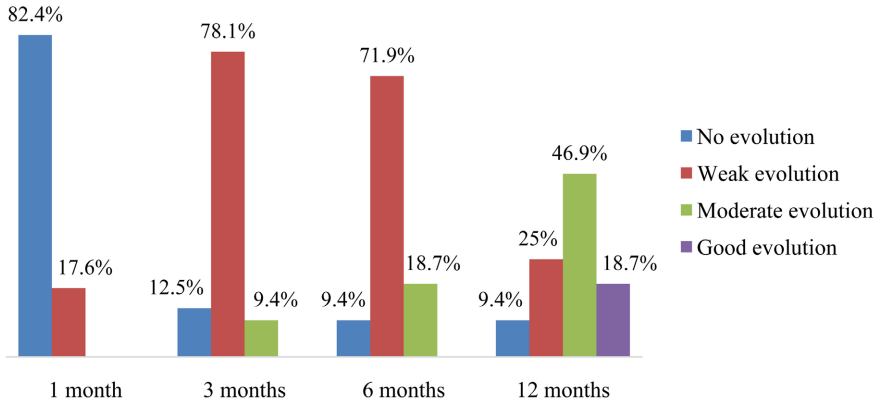

Figure 3. Evolution of the functional status of patients at 1 month, 3 months, 6 months and 12 months.

At 12-month follow-up, and as compared to the 6-month follow-up data, there were the same rate of patients with no evolution of the functional status (9.4%; n = 3), a significant decrease in the rate of patients with weak evolution of the functional status (25%; n = 8) (p = 0.000) and a significant increase in the rates of patients with moderate evolution of the functional status (46.9%; n = 15) (p = 0.000) and patients with good evolution of the functional status (18.7%; n = 6) (p = 0.000).

All these data indicate a positive impact of the treatment administered to the patients on their functional prognosis (p = 0.025).

4. Discussion

This study describes the epidemiological, clinical, paraclinical, therapeutic and evolutive aspects of neurological disorders related to autoimmune diseases.

The literature reports that the prevalence of neurological disorders related to autoimmune diseases varies by continent and race [4] [17] [18] [26] [27] [28] [29]. Worldwide, this prevalence is increasing every year [14]. An increase in the frequency of neurological disorders related to autoimmune diseases is observed in our study (see **Table 1**). It could be attributed to the factors mentioned in **Table 2** and to vaccinations. Indeed, the literature reports that the following factors are associated with autoimmune diseases: infections and vaccinations [19] [20] [22] [30] [31]; long-term use of drugs such as some antibiotics, some antiretrovirals, cardiovascular drugs, antiepileptic drugs, slow-acting anti-inflammatory drugs, antithyroid drugs, cholesterol-lowering drugs [19] [32] [33] [34] [35]; high blood pressure [36] [37]; stigmata of autoimmunity [38] [39]; stress, lifestyle, excessive alcohol consumption, smoking and change of dietary habits [4] [20] [40] [41].

Research reports that women have higher rates of neurological disorders related to autoimmune diseases than men [16] [21] [42] [43]. This feature is also seen in our series. The observed difference in gender could be attributed to complex interactions between genetic, hormonal and environmental factors [2] [3] [31] [44] [45].

The onset age of autoimmune diseases varies according to the clinico-pathological entity. It is influenced by genetic and environmental factors [8] [46] [47] [48]. However, adult age has often been found in the literature [8]. The average age of our patients (38.3 years) is within the age range of 20 - 50 years reported in many African studies focusing on autoimmune diseases of the nervous system [21] [49]-[58].

The reasons for consultation/hospitalization of patients mentioned in our study (**Table 3**) have also been reported in other studies [20] [59].

The data related to the medical history of our patients (**Table 2**) corroborate those reported by other researchers [19] [20] [22] [30]-[39].

The literature reports that the clinical manifestations of autoimmune diseases of the nervous system are polymorphous. These clinical manifestations can vary from patient to patient [20] [60] [61]. This is also seen in our study.

In our series, a delay in diagnosis from the onset of symptoms is observed. Many African studies focusing on autoimmune diseases also report a delay in diagnosis [21] [23] [55] [57] [62] [63] [64] [65]. This could be explained by, among other things, the wide spectrum of clinical presentations of autoimmune diseases, the difficulty in recognizing symptoms at the onset of these diseases, self-medication, and therapeutic itinerary [55] [57] [60] [64] [65].

Research reports that neuromyelitis optica is associated with optic neuritis in some patients [66] [67] [68]. This has also been found in our study. The absence of the typical features of optic neuritis on MRI images in our patients could be attributed to the delay in diagnosis. Indeed, lesions related to optic neuritis can be detected on MRI images within 6 weeks of visual field loss in 92% of cases [69].

Studies of visual evoked potentials in patients with optic neuritis indicate that more than 90% of patients show a prolonged latency within 8 weeks of diagnosis. Above the acute phase, prolonged latency is detected in less than 50% of patients [70]. In addition, prolonged latency is often transient; it normalizes over time [71] [72] [73]. In our study, the lack of prolonged latency in some patients with optic neuritis could be explained among other things by the delay in diagnosis.

Studies on neurological disorders related to autoimmune diseases are rare in Africa [21] [22]. The neurological clinico-pathological entities found in our series have also been reported in other studies conducted in sub-Saharan Africa [21] [22] [30] [42] [49] [50] [52] [54] [59].

Based on the literature, the recommended first-line treatment for acute polyradiculoneuropathy is plasmapheresis or intravenous immunoglobulin injection [20] [74]. Because of the unavailability of specific drugs, our patients with acute polyradiculoneuropathies were treated with high-dose intravenous corticosteroids based on the results of previous studies [75] [76]. The administration of corticosteroids as first-line treatment for the other neurological clinico-pathological entities was in accordance with the literature [74] [77] [78] [79]. The second-line treatments used in our study are those reported in the literature [20] [74] [77]

[78] [79].

The therapeutic approaches used have beneficial effects on the quality of life of patients. Indeed, a significant clinical improvement in patients is noted from the sixth month of follow-up (**Figure 3**). The favorable evolution from the sixth month of follow-up has also been reported in other African studies focusing on autoimmune diseases [51] [55] [80] [81].

Our study has some limitations. Indeed, on account of the limited financial resources of patients and the insufficiency of material resources in medical biology laboratories, we were not able to carry out histopathological investigation in the patient with dermatomyositis, nor to detect antibodies in patients with either Guillain-Barre syndrome (antibodies targeting gangliosides on peripheral nerves: GD1a, GD1b, GM1, GQ1b, GalNAcGD1aGM1), acute disseminated encephalomyelitis (antibodies targeting myelin oligodendrocyte glycoprotein (MOG)), autoimmune encephalitis (N-methyl-D-Aspartate receptor (NMDA-R) and MOG antibodies) or neuromyelitis optica spectrum disorders (aquaporin-4 and MOG antibodies) [20] [31]. However, the present study has provided a better understanding of the neurological disorders related to autoimmune diseases in our context.

5. Conclusion Patients' Limited Financial Resources

In our study population, neurological disorders result more from autoimmune diseases of the nervous system than from systemic autoimmune diseases. The neurological clinico-pathological entities diagnosed are rare. They are similar to those reported in the literature. They affect more the peripheral nervous system, followed by the central nervous system, then both nervous systems. The therapeutic approaches used improve the vital prognosis and functional status of patients.

Conflicts of Interest

The authors declare no conflicts of interest.

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