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Multiple Cranial Nerve Palsies in Otolaryngology Consultation: An Atypical Clinical Presentation Revealing Myasthenia Gravis

Adèle-Rose Ngo Nyeki[1,2*], Paul Cedric Mbonda[3,4], Léonel Atanga[1], Yannick Mossus[1], Roger Meva'a[1,2], Victor Sini[3], Mambo Olive Ngaba[1,2], David Mindja[1,2], Francois Djomou[1], Callixte Kuate[4], Richard Njock[1], Alexis Ndjolo[1]

[1]Department of Ophthalmology-ENT-Stomatology, Faculty of Medicine and Biomedical Sciences, University of Yaoundé I, Yaoundé, Cameroon
[2]Central Hospital of Yaoundé, Yaoundé, Cameroon
[3]Department of Neurology, General Hospital of Yaoundé, Yaoundé, Cameroon
[4]Department of Neurology, Faculty of Medicine and Biomedical Sciences, University of Yaoundé I, Yaoundé, Cameroon
Email: *adele-rose.ngonyeki@fmsb-uy1.cm

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Abstract

Introduction: Myasthenia gravis is a chronic autoimmune neuromuscular disease, presents with weakness and fatigability of striated skeletal muscles. It is a rare disease in Cameroon. We report an uncommon case of myasthenia gravis in a patient with feeding difficulties, notion of oronasal reflux and swallowing disorders as first complaints. **Observation:** We report the case of a 29-year-old woman consulted at our department of Otolaryngology and Cervico-Facial Surgery for dysphagia and swallowing disorders. She also presented with facial diplegia, oculomotor paralysis, nasal voice, and dysarthria which has been evolving for several years now. The clinical examination revealed multiple cranial nerve palsies. The complementary workup showed a decrement of more than 50% in the electroneuromyography and the presence of anti-acetylcholine receptor autoantibodies in the blood workup. A diagnosis of myasthenia confirms clinical presumption. We initially observed a worsening of neuromuscular disorders despite the pyridostigmine treatment and subsequently a clear improvement of the clinical features concerning swallowing and speech disorders, oculomotricity and facial diplegia under a treatment combining prednisone and azathioprine. **Conclusion:** Myasthenia gravis is a rare and potentially fatal autoimmune neuromuscular disease. We thus highlight the atypical clinical presentation and therapeutic itinerary of our patient and the importance to think about this clinical diagnosis in front of any multiple cranial nerve paralysis in otolaryngology consultation.

Keywords

Clinical Case, Dysphagia, Swallowing Disorders, Facial Diplegia, Nasal Voice, Myasthenia, Autoimmune, Paralysis, Cranial Nerves

1. Introduction

Myasthenia gravis (MG) is a chronic autoimmune disease, first described by Thomas Willis in 1672 [1], which manifests itself by extreme weakness and fatigability of the striated skeletal muscles, which can occur at any age. It is due to autoantibodies directed against acetylcholine receptors (AChR) and is characterized by a neuromuscular junction blockade [2]-[8]. Etymologically, the word "myasthenia" is composed of the contraction of: "myas" stands for "muscle", and "asthenia" stands for "fatigue" [9].

It is a rare disease with an estimated incidence rate of 5 to 30 cases per 1 million people per year and a prevalence of 10 to 20 cases per 100,000 [2] [4] [5].

In Africa, few studies have been carried out on this subject on the African continent [5] [10] [11] [12] [13] [14], all of the research shows the diagnostic difficulties and the delay between the first symptoms and the diagnosis. Myasthenia is a disease with a significant clinical polymorphism and amongst the revealing signs, there are symptoms of the otorhinolaryngological and cervical-facial sphere, in particular dysphonia, dysphagia and swallowing disorders or facial hypotonia.

The authors present an uncommon case of Myasthenia gravis in a patient with feeding difficulties, notion of oronasal reflux and swallowing disorders as first complaints. Our patient was seen by several doctors including ear-nose and throat (ENT) specialists who diagnosed asthma or isolated swallowing problem due to velar palsy for which surgery has been proposed. The patient categorically refused this option and for 6 years she endured all her symptoms until it turned out to be myasthenia gravis.

2. Case Presentation

A 29-year-old woman, mother of a 5-month-old child, came to our Otolaryngology (ENT) department for feeding difficulties with the notion of oronasal reflux and swallowing disorders. Her symptoms have been evolving for about 6 years and a sensation of facial numbness was about 2 weeks prior the medical consultation. She has been treated on several occasions 1 year after the beginning of feeding difficulties for respiratory disorders and apnea episodes identified as asthma attacks and treated with bronchodilators and oral and inhaled corticosteroids for several years to no avail.

Our patient was seen by several doctors including ear-nose and throat (ENT) specialists who diagnosed isolated swallowing problem due to velar palsy for which surgery has been proposed. The patient categorically refused this option and for 6 years she endured all her symptoms until it turned out to be myasthe-

nia gravis.

Her history included an asthmatic mother and an uncle with Alzheimer's disease who had been on treatment for 5 years.

Her clinical examination revealed: Cervico-facial level: multiple cranial nerve palsy, including facial nerve palsy manifested by total facial atony (**Figure 1**), erasure of forehead wrinkles, absence of blinking at rest and watery eyes. With voluntary movements, we noted: a facial diplegia with an impossibility to raise the eyebrows or to wrinkle the forehead. She had a bilateral Souques' sign with the possibility of locking the eyelids with maximum effort. She had no cochleo-vestibular complaints. We classified her as House and Brackman grade III.

In addition, she presented a common oculomotor nerve palsy with upper eyelids ptosis; an abducens nerve palsy (oculomotor paralysis and limitation of outward eye movements…); and also, a glossopharyngeal and pneumogastric nerves palsies (total velar paralysis, swallowing disorders to liquids and solids, phonation disorders with nasal voice, abolition of the velar and gag reflexes.

A neurological examination revealed: muscular weakness of the scapular and pelvic belts, right hemiparesis rated at 4/5, right hemi hypoesthesia, indifferent cutaneous-motor reflexes and conservation of the higher functions.

Complementary explorations showed no brain lesions on brain MRI. The electroneuromyography showed a decrement of up to 42.6% in the ulnar abductor pair (**Figure 2**) compatible with a generalized myasthenia. Motor and sensory neurography was normal. The acetylcholine receptor autoantibody (AChRAb) detected by RadioImmunoprecipitation Assay (RIA) was 31.7 nmol/l for a normal value of less than 0.2 nmol/l and the anti-muscle tyrosine kinase antibody (MUSK) also checked by RIA was normal (**Table 1**). The diagnosis of myasthenia gravis was retained and our patient was managed by our neurology colleagues. She was put on treatment: pyridostigmine 60 mg 4 times daily for 2 months; with improvement of asthenia and cranial nerves disorders, unfortunately followed by an aggravation of swallowing disorders. This treatment protocol was immediately replaced by a combination of intravenous corticosteroid therapy, methylpredinosolone 120 mg twice daily during 5 days then oral relay for 1 month at a dose of 1 mg/kg/day then 20 mg non-stop and azathioprine 50 mg twice a day.

There was a total regression of muscular fatigability and signs of cranial nerve damage, *i.e.* swallowing, speech, oculomotricity and signs of facial diplegia after 2 months of treatment.

Table 1. Table showing results of serological analysis of our 29-year-old patient revealing high level of AChRAb.

Test type	Normal values	Patient values	Interpretation
Acetylcholine receptor autoantibody (AChRAb) *RIA*	<0.2 nmol/L	**31.7 nmol/L**	Highly positive antibodies
Anti-muscle tyrosine kinase antibody (MUSK) *RIA*	<0.4 U/mL	<0.18 U/mL	No antibodies found

Figure 1. Picture showing our 29-year-old patient with total facial atonia.

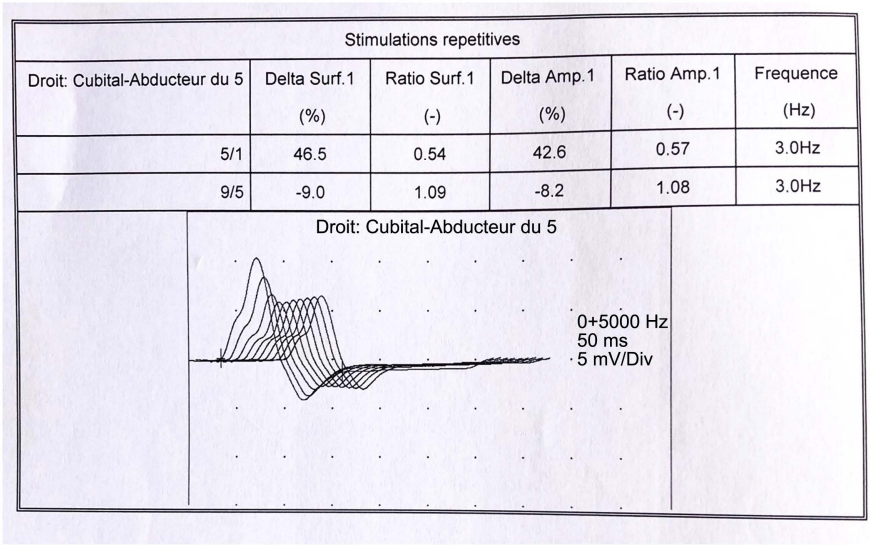

Figure 2. Results of electroneuromyography tests of our patient revealing an abnormal decrement up to 42.6% in amplitude, appreciated with 3 Hz repetitive nerve stimulations of the ulnar abductor pair, compatible with a generalized myasthenia.

3. Discussion

3.1. Epidemiology

We report an uncommon presentation of Myasthenia gravis in a patient with feeding difficulties, notion of oronasal reflux and swallowing disorders in first complaints.

MG is the most common disorder of the neuromuscular junction but remains a rare autoimmune disease in the world [6]. A few studies have been carried out on this subject on the African continent, especially in Tanzania [12], Morocco [6], Cameroon [10], Senegal [14], Madagascar [5] and South Africa [15].

The global incidence averages 10 cases per 1,000,000 people per year with a prevalence of 8 to 10 cases per 100,000 [16]. These numbers have steadily increased in recent decades [9]; for example, between 2006 and 2018, there was an increase ranging from 5 to 30 cases per 100,000 people [2] [17]. This rate is increasing due to improved diagnostic methods, management and increased life expectancy of the general population [4] [6] [9].

In Cameroon, this scarcity could be explained by diagnostic difficulties and by the lack of knowledge of this pathology by practitioners [10].

Although this pathology can be observed at any age, it is generally considered to be a pathology of the young adult, affecting women five times more than men [5] [6].

Our patient was diagnosed at the age of 29 years with an onset of symptoms at the age of 21 years and the authors agree on the existence of 2 peaks of frequency: a first peak between 20 and 40 years of age where women are preferentially affected and a second peak after 50 years of age where men are most often affected [2] [4] [6] [9].

3.2. Etiopathogeny

A correlation between autoimmune diseases and infections, especially viral infections, has been suggested, including hepatitis C, herpes simplex virus, Epstein Barr virus, cytomegalovirus, and more recently SARS-CoV 2, which trigger or exacerbate myasthenia. Recently, 3 cases of anti AChR positive myasthenia associated with COVID-19 infection have been described [18]. In addition, a multitude of toxins from animals, plants, or microorganisms are believed to be capable of disrupting the neuromuscular junction function [9].

3.3. Clinical Features

Because of the implications for its management, myasthenia gravis is classified according to the clinical presentation (ocular or generalized form), the severity of the disease, the type of antibodies found, whether or not it is associated with a thymic pathology, and also according to the response to treatment [4] [19].

The various diagnostic criteria for this disease are:

- Clinical signs: fluctuating muscular fatigability that can affect the ocular, orofacial, limb or respiratory muscles, fatigability that increases after physical effort or at the end of the day and seems to improve at rest.
- Electro physiological tests: repetitive nerve stimulation with a pathological decrement > 10% from the 4th/5th response.
- Serological tests: elevation of autoantibodies to acetylcholine receptors (AChR) or anti Muscle-specific Kinase (MusK) antibodies.
- Therapeutic tests by intravenous injection of cholinesterase inhibitors (edrophonium or neostigmine) [8].

Usually, the first complaints of MG are ocular signs (diplopia, ptosis…) in almost 80% of the patients [16] [20] [21]. Our patient presented several signs of

neuromuscular damages: oropharyngeal and facial muscular fatigability, ptosis and oculomotor paralysis. Electrophysiological examination showed a significant decrement of 42.6% and she also had a very high level of anti AChR at nearly 158 times.

The first signs concern the oculomotor muscles where it can remain localized in 10% to 15% of the cases but most often the myasthenia extends in the 2 years which follow to other muscle groups. Other initial localizations, notably by bulbar involvement (dysphagia, dysarthria, mastication disorders) are less frequent and are observed in 15% of patients. Phonation disorders become evident during conversation, the voice becomes nasal and unintelligible. Chewing disorders and dysphagia increase during meals and can lead to false routes. The initial involvement of the limbs is less frequent and predominates in the roots [3] [6] [7].

In our patient, the first symptoms were bulbar involvement with dysphagia, oronasal reflux and false routes, but unfortunately these were not recognized as signs suggestive of her disease. Almost 1 year after the beginning of these symptoms, she has been treated on several occasions for respiratory disorders and apnea episodes identified as asthma attacks and treated with bronchodilators and oral and inhaled corticosteroids without remission.

Mandalyia *et al.* propose that myasthenia Gravis should be considered in patients with unexplained dyspnea [22].

In Africa, one of the major problems is the long delay between the onset of symptoms and the time of diagnosis; for Matuja *et al.* in Tanzania this delay ranged from 3 months to 6 years [12], in our case, there were 7 years between the first symptoms of our patient and the diagnosis while in developed countries, the diagnosis is made a few hours to a few days after the onset of symptoms.

The average age of discovery of the disease in women for Matuja, was 21 years as in our case with an average of 26.5 years in Western countries; for men it occurred 10 years later.

Autoimmune myasthenia is a condition that is often confusing because of its unclear symptomatology and its evolution, which may explain the long delay in diagnosis, especially since it is a pathology that is unknown to many practitioners.

Myasthenia perfectly mimics otolaryngological pathologies because of its bulbar presentations [2]. ENT specialists are the first doctors to whom patients are referred at the onset of the disease because the symptoms include functional disorders of the cervicofacial sphere as described since 1979 by Carpenter *et al.* [1], in this case, dysphagia [23] and dysphonia due to the pathological fatigability of the striated muscles of the pharynx and esophagus [2]. The dysarthria found by several authors as for our patient who presented an open rhinolalia or nasalization and a slowed vocal flow is due to an additional attack of the muscles of the tongue and the palate [2].

Associated with this were signs of weakness when chewing, facial hypotonia, muscular fatigability of the cervical region and even dyspnea can be found [1] as in the case of our patient.

3.4. Paraclinical Work-Up

- At the biological level, the positive diagnosis of myasthenia includes elevated levels of autoantibodies such as anti AChR (anti-acetylcholine receptor antibodies), anti MuSK (Muscle streptoKinase) antibodies and the presence of lipoprotein receptor-related protein 4 (Lrp4) [24]. Seropositive myasthenia is thus the most frequent form of autoimmune myasthenia. Anti-AChR antibodies are usually found in 80% - 90% of generalized myasthenias and in 50% - 60% of ocular myasthenias [6].

The determination of these antibodies is considered the gold standard for diagnosis [2] [25].

Other biological tests are necessary to search for an associated autoimmune disease: TSH, T3, T4, antithyroid antibodies, anti-DNA antibodies and rheumatoid factor [6].

- At the electrophysiological level, the usual electrical examination is the search for the decrement of the motor potential under the effect of repetitive supramaximal stimulations at low frequency (2 to 5 Hz). The maximum decrement occurs between the second and fifth potentials and must be greater than or equal to 10%. It clearly demonstrates a post-synaptic deficit in neuromuscular transmission [6] [7] [18] [25].
- Pharmacological tests are often neglected in countries where access to serological tests and electrodiagnosis is easy, but they remain very useful when access to anticholinesterases is possible and the availability of serological or electro physiological tests is limited [5] [10] [25]. Thus, an injection of pyridostigmine, an indirect parasympathomimetic with an acetylcholinesterase inhibitory effect, almost instantly improves the clinical presentation of patients by increasing the intensity and rhythm of muscle contractility and thus makes the diagnosis of the disease.
- Radiologically, a thoracic MRI is requested to exclude a thymoma [6] because thymoma induced mysathenia is reversible with the surgical removal of this tumor.

The evolution of myasthenia gravis is capricious and is interspersed with attacks that can threaten the vital prognosis due to severe damage to the respiratory muscles and muscles of swallowing [6] [26]. This damage to the respiratory muscles may occur in up to 40% of myasthenic patients, leading to severe dyspnea on exertion or orthopnea.

In general, 15% - 20% of patients will experience a myasthenic crisis with respiratory failure requiring either non-invasive ventilatory or mechanical methods until clinical improvement is achieved [4] [5].

3.5. Treatment

The diversity and clinical polymorphism of this disease, as well as its severity due to its bulbar and respiratory involvement, require a rapid and adapted diagnostic and therapeutic management; an individual therapeutic plan must be

adjusted according to the clinical responses of each patient [14] [26].

Anticholinesterase drugs are the basis of the symptomatic treatment of the disease by improving neuromuscular transmission [5] [6] [14]. Pyridostigmine bromide is the most commonly used in the treatment of myasthenia. The daily dose should generally not exceed 600 mg. It is suitable for long-term treatment in patients with a moderate or slowly progressive form of the disease and as adjuvant therapy in severe forms where immunosuppressive therapy such as aziathropine has been instituted [26]. Two oral anticholinesterase drugs are available: pyridostigmine bromide and ambenonium chloride [6].

In Cameroon, as in most countries, the first-line treatment includes anticholinesterase drugs and corticosteroid therapy [5] [10] [23]. Our patient was put on this treatment based on pyridostigmine, which was replaced following a worsening of the signs by injectable corticosteroid therapy associated with azathioprine, a treatment on which the evolution was favorable. The usual initial dose of azathioprine is 1 mg/kg per day, improving 70% to 90% of myasthenic patients. It is the first immunosuppressant with a proven cortisone-sparing effect.

Corticosteroids are available in all countries, are reliable, and have a rapid onset of action (2 to 4 weeks on average) on immunomodulation in myasthenic disease, with maximum benefit achieved in an average of 5 to 6 months [6] [24] [27]. However, their long-term use exposes patients to the known adverse effects of these molecules. Prednisolone or prednisone are the molecules used in the first line [16] [21]. The doses are usually high at the initiation of treatment for generalized forms, but in localized ocular forms, the doses are lower and for a limited period of time, *i.e.*, about 20 mg for 2 to 4 weeks, with satisfactory results [21] [26].

Other immunosuppressive drugs such as cyclophosphamide in doses of 500 to 1000 mg/m^2, administered every 4 to 12 weeks, are occasionally used for refractory forms of the disease. Methotrexate is an alternative to azathioprine. It is an anti-metabolite that has been used for decades in cancer therapy, and in small doses methotrexate is a generally safe and well-tolerated drug in the treatment of certain autoimmune diseases [26].

New treatment options have emerged with monoclonal antibodies such as rituximab that will significantly change the management of myasthenia gravis in the coming years [23] [26].

During severe myasthenic attacks, certain therapeutic modalities are used, such as plasmapheresis or plasma exchange, which acts by transiently purifying circulating antibodies; it is performed 3 to 5 times in the first few days and then adapted according to the patient's clinical response; immunoadsorption or intravenous administration of immunoglobulins [26].

In the case of thymoma demonstrated or suspected by thoracic imaging, the theoretical indication for thymectomy is formal [5] [19]; it will be completed by radiotherapy, or even chemotherapy, in the case of extra capsular tumor extension [6].

In view of all this therapeutic development around myasthenia, nowadays the mortality rate of myasthenic attacks in developed countries is less than 5% and is generally due to complications of hospitalization or the treatment itself [4].

4. Conclusion

Myasthenia gravis is a rare and poorly understood disease. It is potentially fatal and presents a clinical polymorphism and an asymmetrical staged evolution that often leads to numerous diagnostic mistakes. Usually, the first complaints are ocular signs but given the frequency of significant functional disorders because of multiple cranial nerve paralysis, the ENT specialist is often in the front line and must be able to quickly suspect the diagnosis for an immediate treatment. Collaboration with neurology teams is essential for a better management of these patients.

Consent

This article is published with the consent of the patient.

Conflicts of Interest

The authors declare that there is no conflict of interest.

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