

**Persistent pyrosis revealing generalized myasthenia gravis**

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**Abstract**

**Introduction:** Autoimmune myasthenia gravis is a condition associated with autoimmune disruption of neuromuscular transmission. Digestive manifestations are common, but lead to misdiagnosis.

**Observation :** An 18-year-old female with no specific pathological history was treated for gastroesophageal reflux disease (GERD) with persistent pyrosis and ulcerative epigastralgia, which prompted several consultations. An oesogastroduodenal transit revealed oesophageal dilatation with stage, and fibroscopy confirmed oesophageal dilatation and the presence of erythematous antral gastritis. In the internal medicine consultation, she presented with motor deficit in all 4 limbs and a waddling gait. Biological tests revealed positive anti-choline acetyl receptor antibodies. The electroneuromyogram suggested generalized myasthenia. We noted a good clinical course with symptomatic treatment of digestive disorders, pyridostigmine and conventional immunosuppressants.

**Conclusion:** Digestive disorders can complicate generalized myasthenia. They should prompt a holistic diagnostic approach for better patient management.

**Keywords:** digestive disorders; myasthenia; auto-immune.

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## Introduction

Autoimmune myasthenia gravis is a disorder caused by autoimmune disruption of neuromuscular transmission. It leads to muscular weakness, sometimes with systemic manifestations that can be life-threatening [1]. It is the most common autoimmune disease affecting neuromuscular transmission. Diagnosis is based on clinical and biological criteria, with specific antibody assays [2]. Digestive disorders are common in myasthenia but often lead to misdiagnosis, as they mimic other pathologies of the digestive tract [3].

We report the case of an 18-year-old female patient who was seen in consultation for epigastralgia and persistent pyrosis revealing generalized myasthenia.

## Observation

This 18-year-old patient with no specific pathological history was presented with a generalized motor deficit with speech disorders, dysarthria and dysphagia to solids and liquids, associated with gastro-oesophageal reflux syndrome with pyrosis, and epigastric pain with retrosternal irradiation and yaw sign. She had been referred to gastroenterology, where an oesogastro-oesophageal transit was performed, revealing dilatation of the oesophagus with cessation of contrast medium in the cardia. An oesogastroduodenal fibroscopy revealed a mega-oesophagus with a food stage and erythematous microerosive antral gastritis. Pathology revealed atrophic antral gastritis without metaplasia or dysplasia.

She had received several treatments based on gastric dressings and proton pump inhibitors without remission. She was referred to internal medicine, where the initial examination revealed: motor defi-

cit in the lower limbs with muscle strength at 2/5 in the upper limbs, a scarf sign, regular tachycardia without added noise; a waddling gait.

Biological tests revealed anti-Mi2A antibodies with a titre of 10 IU (0 - 7); anti-choline acetyl receptor with a titre of 100 IU (<10 IU). Blood count, creatine phosphokinase, tetraiodothyronine, blood calcium and creatinine were normal. Antinuclear antibodies were negative.

The electroneuromyogram revealed signs of generalized myasthenia gravis, with sharp osteotendinous reflexes in all 4 limbs and prolonged F waves.

Cerebral and thymus CT scans were normal.

Acute generalized myasthenia was ruled out and the patient was put on : Mestinon 60mg: 1 cp X 3/d and Immurel 50mg: 1 cp X 2/d, prednisone 1mg/kg/d with adjuvant means, pantoprazole 40mg: 1 cp x2/d, domperidone 10mg daily.

We noted a good clinical evolution with notable regression of the deficit in 3 weeks of treatment and improvement of the digestive signs after one month.

## Discussion

Myasthenia gravis is a well-known autoimmune disorder affecting the muscles. Its symptoms are muscular weakness with fluctuating physical asthenia, aggravated by exertion and relieved by rest, and may involve the ocular, limb, respiratory and bulbar muscles. It is a post-synaptic autoimmune disorder [2].

It occurs in young women (3 women vs. 1 man),

most often before the age of 40.

This diffuse muscular involvement is what makes the disease so serious, as it involves vital functions such as breathing and swallowing. The extent of muscular damage is assessed either by the 4-grade Osserman score (modified by Genkins) (Table I), or by the 100-point myasthenic score [4].

Table I: Osserman classification (modified by Genkins)

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| STAGE I: Ocular myasthenia   |
| STAGE II A: Generalized myasthenia without bulbar signs  |
| STAGE II B: Generalized myasthenia gravis with bulbar signs, but no false-routes                     |
| STAGE III: New-onset generalized myasthenia gravis, with rheumatic fever and respiratory involvement |
| STAGE IV: Old-onset generalized myasthenia gravis with bulbar signs and respiratory involvement      |

Digestive manifestations are polymorphous, occurring in 70% of cases of myasthenia gravis and dominated by swallowing disorders. Dysphagia is one of the most frequent digestive disorders. It may be mild or severe, leading to metabolic complications associated with undernutrition [3]. Other digestive disorders include gastroesophageal reflux disease, peptic ulcer disease and intestinal motility disorders. Esophageal manometry or videocapsule can be used to explore these digestive disorders [5, 6].

The diagnosis of myasthenia gravis is based on clinical, electrical and biological evidence. The discovery of anti-acetyl choline receptor and anti-

tyrosine kinase autoantibodies has improved diagnosis. As with most autoimmune processes, these may be absent in certain situations. A new autoantigen, low-density lipoprotein receptor-related protein 4 (LRP4), has been identified in variable proportions of otherwise seronegative patients. Recent data suggest that anti-LRP4 antibodies may define a new subtype of myasthenia gravis, supporting the concept that myasthenia gravis is not a single pathological entity and that different subtypes may differ in etiology [8].

Several publications have been made in Africa, Europe and the Middle East. Ocular manifestations are the most frequent of these. Quality of life is severely affected by this condition, hence the importance of good psychosocial care for these patients [9].

Treatment of myasthenia gravis involves immunosuppressive agents such as azathioprine, glucocorticoids, plasmapheresis, intravenous immunoglobulins and anticholinesterase agents and thymectomy [2]. Our patient progressed well on azathioprine 3mg/kg/d, pyridostigmine 60mg X2/d and prednisone 1mg/kg/j with additive means. This testifies to the efficacy of current treatment.

A number of biological therapies have been developed in recent years to help control forms of myasthenia refractory to conventional treatment. These include complement inhibitors, neonatal Fc receptor inhibitors, anti-B cell drugs and IL-6 receptor inhibitors [10].

**Conclusion:** Myasthenia gravis is an underdiagnosed condition. Its manifestations are polymorphous, and the digestive manifestations may be

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confused with several other conditions. Treatment is based on symptomatic means, classical immunosuppressive agents and the development of biotherapy.

**Conflict of interest:** we declare no conflict of interest

**Approved for publication** by the patient

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