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CASE PRESENTATION

Atypical presentation of Bruns-Garland Syndrome in a patient with metabolic comorbidities: a case report

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ABSTRACT

Introduction: Bruns-Garland syndrome, or diabetic lumbosacral radiculoplexopathy, is an uncommon neurological complication of diabetes mellitus, with an estimated prevalence of less than 1 %. It is characterized by pain and weakness in the lower limbs, typically of asymmetric and progressive onset, and may be misdiagnosed as musculoskeletal or compressive pathology.
Case presentation: We report the case of a 61-year-old male with a history of arterial hypertension and poorly controlled type 2 diabetes, who presented with one month of left thigh pain followed by progressive weakness in both lower limbs, without other neurological symptoms. Physical examination revealed decreased muscle strength and paresthesias. Magnetic resonance imaging ruled out radicular compression. Electromyography demonstrated motor axonal denervation consistent with lumbosacral radiculoplexopathy. Treatment with pregabalin, amitriptyline, cytidine monophosphate, and physical therapy was initiated, with favorable clinical response.
Conclusions: This case illustrates an atypical presentation of Bruns-Garland syndrome, without weight loss or autonomic symptoms, which delayed its initial recognition. Early diagnosis based on clinical suspicion and neurophysiological studies is essential to avoid unnecessary interventions and improve the patient's functional prognosis.

Keywords: Diabetic Neuropathies; Neuropathy; Diabetic Neuropathies.

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INTRODUCTION

Bruns-Garland syndrome, also known as diabetic lumbosacral radiculoplexus neuropathy, is a rare form of peripheral neuropathy associated with diabetes mellitus. It is characterized by involvement of the nerve roots, lumbosacral plexus, and peripheral nerves, likely secondary to a non-systemic microvasculitis causing focal ischemic nerve damage.[1]

Its estimated prevalence is below 1 % among diabetic patients, being more frequent in men over 50 years old with type 2 diabetes. Occasionally, it may represent the initial manifestation of metabolic disease.[2]

The clinical picture typically begins with acute pain in the thigh, hip, or lumbar region, followed by proximal muscle weakness and muscle wasting in the affected limb. Although usually unilateral, it may progress to bilateral involvement. Approximately half of patients experience significant weight loss, and some may present autonomic and sensory symptoms, such as paresthesias.[3]

Diagnosis is clinical, supported by electrophysiological studies. Electromyography reveals axonal denervation in proximal and distal muscles. Nerve conduction studies confirm axonal neuropathy.[3] Magnetic resonance imaging (MRI) is useful for ruling out compressive causes, and in some cases, cerebrospinal fluid shows elevated protein with normal cell count.[4]

Management is mainly symptomatic. Anticonvulsants such as pregabalin, tricyclic antidepressants, and physiotherapy are commonly used.[5] Strict glycemic control is essential. Immunomodulatory treatments such as corticosteroids or intravenous immunoglobulin have been used, although clinical evidence remains limited.[6]

CASE PRESENTATION

A 61-year-old male with a medical history of untreated hypertension and type 2 diabetes mellitus treated with metformin 850 mg daily and insulin degludec 20 IU before breakfast. He was under follow-up in the Traumatology Department for lumbosacral arthropathy, receiving pregabalin 75 mg every 12 hours.

The patient consulted the Neurology Department for a one-month history of moderate pain in the lower limbs, predominantly on the left side, localized to the posterior thigh, without irradiation. In the weeks prior to consultation, the pain was accompanied by progressive weakness in both lower limbs, causing a fall from standing height. He denied weight loss, autonomic symptoms, fever, severe sensory alteration, or other systemic symptoms.

On physical examination, he was conscious and alert, with a Glasgow score of 15/15, no meningeal signs or cortical focality. Facial symmetry was preserved. Muscle trophism was normal in the upper limbs but decreased in the lower limbs (Figure 1). Muscle strength was decreased in the left lower limb (4/5), with bilateral paresthesias in a non-dermatomal distribution. Deep tendon reflexes were present and symmetrical. Gait was impaired, with claudication on the left side.

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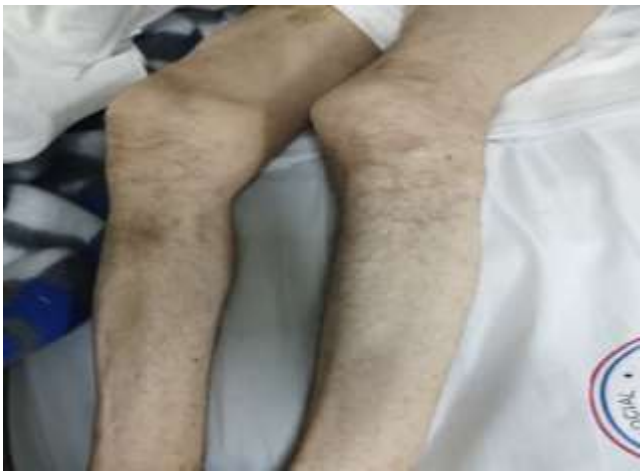

Fig 1. Muscle atrophy in the lower limbs.

Marked reduction in muscle volume was observed in both lower limbs, predominantly in the left quadriceps, consistent with the clinical picture of diabetic lumbosacral radiculoplexus neuropathy.

Laboratory studies revealed hemoglobin of 10,9 g/dL and hematocrit of 31,7 %, findings compatible with mild anemia. Glycated hemoglobin (HbA1c) was 7,9 %, indicating suboptimal glycemic control. The remaining parameters, including leukocyte count, renal function (urea and creatinine), electrolytes, liver profile, coagulation, and urinalysis, were within normal limits.

Lumbosacral MRI showed cervical and lumbar disc protrusions without signs of root compression or spinal canal compromise. Cauda equina syndrome or compressive myelopathy were ruled out. Electrophysiological study via electromyography revealed signs of motor axonal denervation, with abundant spontaneous activity (fibrillations and positive waves), especially in lower limb muscles, predominantly on the left, consistent with diabetic lumbosacral radiculoplexopathy.

Treatment was initiated with pregabalin 75 mg every 12 hours, amitriptyline 25 mg nightly, cytidine monophosphate once daily, and an intensive motor physiotherapy program. The patient showed favorable clinical evolution, with pain reduction and progressive improvement in muscle strength and gait over the following weeks.

DISCUSSION

Bruns-Garland syndrome, or diabetic lumbosacral radiculoplexus neuropathy (DLRPN), is an uncommon form of diabetic neuropathy characterized by simultaneous involvement of nerve roots, the lumbosacral plexus, and peripheral nerves. It presents with acute or subacute onset of pain and weakness in the lower limbs, typically in patients with long-standing or newly diagnosed type 2 diabetes.[1,2] The underlying pathophysiology involves non-systemic microvasculitis causing focal ischemia of the nerves, leading to motor denervation and muscle atrophy.[3]

Although rare—with a prevalence below 1% among diabetic patients—the true number may be underestimated due to frequent underdiagnosis or confusion with orthopedic or mechanical radiculopathies.[6] It is more common in men over 50 years old and may even represent the initial manifestation of type 2 diabetes mellitus in about one-third of cases.[7]

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The classic clinical presentation begins with sudden pain in the lumbar, hip, or thigh region, followed by weakness and muscle atrophy, usually unilateral and proximal, which may progress to bilateral involvement within weeks. Most patients experience significant weight loss (more than 10% of body weight), and a subset may develop autonomic manifestations such as orthostatic hypotension, bladder, or gastrointestinal dysfunction.[7,8]

The case described here partially aligns with this description: a middle-aged male with a previous diagnosis of diabetes who developed left thigh pain followed by progressive bilateral lower limb weakness. However, several features made this case atypical. First, the absence of weight loss and autonomic symptoms. Second, initially unilateral involvement progressing to bilateral weakness within a short period, without severe gait impairment or evident atrophy at onset. Finally, the history of lumbosacral arthropathy initially led to a musculoskeletal etiology, a frequent cause of diagnostic delay described in the literature.[9]

Diagnosis of DLRPN is primarily clinical, supported by electrophysiological studies confirming axonal neuropathy in a lumbosacral distribution with signs of active denervation (fibrillations, positive waves) and proximal involvement. In this case, electromyography showed a motor axonal radiculoplexopathy pattern, a typical finding of the disease.[3,6]

Lumbosacral MRI is useful to exclude structural causes such as disc compression or spinal canal tumors. In our patient, MRI ruled out significant compression, reinforcing the diagnosis of metabolic plexopathy. In some cases, cerebrospinal fluid analysis reveals elevated protein without pleocytosis, although this study was not necessary in this case.[10]

There is no specific approved treatment for Bruns-Garland syndrome. Management is based on symptomatic control of neuropathic pain (commonly with anticonvulsants such as pregabalin or gabapentin, and tricyclic antidepressants such as amitriptyline), intensive physical rehabilitation, and optimization of glycemic control.[1,4,10]

In selected cases, especially when active inflammatory components are suspected, corticosteroids and intravenous immunoglobulin have been tested, showing variable benefit; however, evidence remains limited and based on small series or open-label studies.[5] In this case, a favorable response was achieved with conservative management: pregabalin, amitriptyline, cytidine monophosphate, and progressive motor physiotherapy, without the need for immunomodulatory therapy.

The prognosis is generally favorable, although recovery is slow. Most patients improve within 6–18 months, although some degree of residual weakness or pain may persist. Bilateral progression, as in this case, does not necessarily imply a poorer prognosis but requires close and prolonged functional follow-up.[2,5,7]

CONCLUSIONS

Bruns-Garland syndrome is a rare diabetic neuropathy that causes neuropathic pain and asymmetric muscle weakness. Its symptoms can mimic other musculoskeletal disorders, complicating diagnosis. Early identification, combined with neuroimaging and electrophysiological findings, is essential for appropriate management. Although there is no specific cure, the use of neuromodulatory medications and physiotherapy can improve the patient's quality of life. This neuropathy should be considered in the differential diagnosis of diabetic patients presenting with lower limb weakness.

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Conflicts of Interest
The authors declare no conflicts of interest.

Author Contributions
MAMA: Conceptualization, Investigation, Original Draft Writing, Literature Review. **JGJ:** Investigation, Clinical Documentation, Image Acquisition, Review and Editing. **EGM:** Investigation, Clinical Documentation, Literature Review, Review and Editing. **JGM:** Supervision, Project Administration, Review and Editing.

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Ethical Standards
All ethical standards were respected. Informed consent was obtained from the patient's family (children) for publication of this report. The manuscript has not been simultaneously submitted to other publishers for consideration.

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