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*Editorial*

# Neutropenia develops after intravenous immunoglobulin administration in a patient with multifocal motor neuropathy and conduction block.

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## ABSTRACT

The preferred treatment for those with multifocal motor neuropathy with conduction block is intravenous immunoglobulin (IVIG) (MMNCB). Neutropenia following IVIG infusion has been identified as a rare hematologic adverse effect, despite the fact that IVIG is typically regarded as safe. Here, we present information about a patient with MMNCB who experienced significant neutropenia after receiving IVIG.

A 41-year-old lady who frequently experienced muscle cramps and growing weakening in her left lower leg presented. Right ulnar and right tibial nerves showed noticeable conduction blockages in a nerve conduction examination. Anti-GM1 serum IgM antibody was found. After receiving IVIG, the neutrophil count began to decline on day 2 and then began to increase two weeks later. Although it can be a significant clinical concern, neutropenia following IVIG infusion in MMNCB has never been separately discussed to our knowledge.

## INTRODUCTION

It is uncommon for multifocal motor neuropathy with conduction block (MMNCB), an immune-mediated polyneuropathy, to develop less than 1 per 100000 is known to exist [1]. Although uncommon, MMNCB is a curable illness if caught early and handled properly. Because MMNCB is resistant to corticosteroids and plasmapheresis, it should be recognised from other chronic acquired demyelinating polyneuropathies [2]. Due to its outstanding safety profile and clinical efficacy, intravenous immunoglobulin (IVIG) injection has been the cornerstone of MMNCB therapy to date [3,4].

Although hematologic toxicities were noted in a number of clinical applications, including idiopathic thrombocytopenia purpura, systemic lupus erythematosus, pemphigus vulgaris, Guillain-Barre syndrome (GBS), chronic inflammatory demyelinating polyradiculoneuropathy (CIDP), and polymyositis [6,] intravenous immunoglobulin is generally regarded as safe. As far as we are aware, leukopenia Although it can be a significant clinical concern, particularly for individuals with MMNCB, after IVIG therapy in MMNCB has never been separately discussed. We describe a patient with MMNCB who experienced leukopenia after receiving IVIG.

Neutropenia has been documented as a rare side effect since the debut of IVIG therapy for a variety of immunological disorders. Patients with idiopathic thrombocytopenia purpura were the first to exhibit neutropenia after IVIG [7,8]. A patient with active systemic lupus erythematosus who had two courses of IVIG infusion was described by Ben-Chetrit et al. [9] in their report. Following each IVIG treatment, marked neutropenia frequently emerged, confirming the link between IVIG infusion and neutropenic occurrence. In this instance, neutropenia began to manifest on day two after the infusion began and peaked on day four. Leukocyte levels of our patient returned to baseline after 2 weeks without any complications, which is consistent with earlier studies [8,10].

It is still unclear how neutropenia following IVIG occurs. Antineutrophil antibodies were found in a child with Guillain-Barre syndrome, according to Tam et al. [11], who also described the case. Atypical

antineutrophil cytoplasmic antibodies (ANCA) have been implicated in several studies [12, 13]. Some researchers hypothesised that IgG aggregates with immune-complex-like activity were to blame for transitory neutropenia [9,14]. Two components found on the surface of neutrophils were the focus of Matsuda et alstudy .'s [10]: the complement receptor CD11b and the Fc gamma receptor CD16. Only the expression of CD11b, one of the two surface antigens, was shown to be reduced after IVIG. The authors found that spreading IVIG ased a. The circulating syndrome, in which the authors identified the presence of antineutrophil antibodies, was concluded by the authors. Atypical antineutrophil cytoplasmic antibodies (ANCA) have been implicated in several studies [12, 13]. Some researchers hypothesised that IgG aggregates with immune-complex-like activity were to blame for transitory neutropenia [9,14]. Two components found on the surface of neutrophils were the focus of Matsuda et alstudy .'s [10]: the complement receptor CD11b and the Fc gamma receptor CD16. Only the expression of CD11b, one of the two surface antigens, was shown to be reduced after IVIG. The authors found that spreading When CD11b is present, neutrophils may stick to the vascular wall. Neutropenia was shown to be caused by the sialic acid-binding immunoglobulin-like lectin 9 (siglec-9) produced on neutrophils more recently by von Gunten et al. [15,16]. Natural siglec-9 autoantibodies may be present in IVIG preparations, and both caspase-dependent and caspase-independent mechanisms can be used to generate neutropenia by these autoantibodies.

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