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Corticosteroid Usage in Patients With Generalized Myasthenia Gravis Receiving Nipocalimab: Phase 3 Vivacity-MG3 Open-Label Extension

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Key Takeaways

- **During the VIVACITY-MG3 OLE phase, nearly half of patients receiving corticosteroids at baseline reduced or discontinued corticosteroids**
- **A substantial portion of patients who reached a low dose corticosteroid (≤10 or ≤5 mg/day prednisone equivalent) during the OLE achieved sustained MCI or MSE (≥8 weeks)**
- **These results support the corticosteroid-sparing effect of nipocalimab and concurrent meaningful clinical improvement and sustained disease control in patients with gMG**

Introduction

- Nipocalimab is a fully human monoclonal antibody that binds the neonatal Fc receptor (FcRn) with high specificity and affinity, reducing circulating immunoglobulin G (IgG), including pathogenic IgG autoantibodies[1,2]
- Nipocalimab is approved for the treatment of generalized myasthenia gravis (gMG) in adult and adolescent patients who are anti-acetylcholine receptor (AChR) and anti-muscle-specific tyrosine kinase (MuSK) antibody-positive[3,4]
- In the phase 3 Vivacity-MG3 study, nipocalimab demonstrated sustained disease control, with improvements in MG-Activities of Daily Living (MG-ADL) and Quantitative MG (QMG) total scores versus placebo during the 24-week double-blind phase and through Week 96 of the open-label extension (OLE) phase[5,6]
  - Corticosteroid dose adjustment was prohibited during the double-blind phase but permitted in the OLE, where all patients received nipocalimab

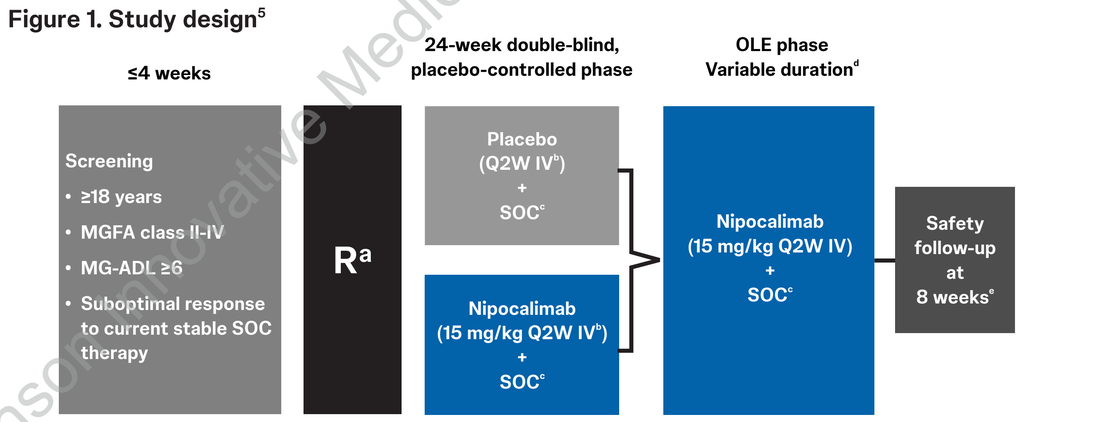
Objectives

To characterize corticosteroid usage, patterns of corticosteroid dose reduction/discontinuation, and symptom improvements among patients with gMG treated with nipocalimab during the OLE phase of the Vivacity-MG3 study

Methods

- Vivacity-MG3 (NCT04951622) is a double-blind, randomized, placebo-controlled, multicenter, phase 3 study evaluating efficacy and safety of nipocalimab in adults with gMG[1]
  - Tapering of concomitant gMG medications was allowed only in the OLE phase at every 4 weeks, if the disease has been stable in the past 4 weeks as reflected by MG-ADL scores and based on the investigator's discretion
- Analyses were conducted in patients who were seropositive (anti-AChR, anti-MuSK, or anti-low-density lipoprotein-related protein receptor 4 [LRP4])
- Corticosteroid doses (mg/day prednisone-equivalent) were evaluated during the OLE phase through data cut-off (August 2024)
- The following assessments were evaluated among patients who received corticosteroids at OLE baseline:
  - Mean steroid dose reduction
  - % of patients reaching ≤10mg/day or ≤5mg/day prednisone equivalent (eq), or discontinuing corticosteroid
  - Safety
- The following assessments were evaluated among patients who reached a low corticosteroid dose (≤10mg/day or ≤5mg/day) at data cutoff:
  - % of patients who achieved minimal clinical improvement (MCI) of ≥2 points from baseline in MG-ADL total score
  - % of patients who achieved sustained MCI (≥8 weeks)
  - % of patients who achieved minimum symptom expression (MSE; MG-ADL score =0/1)
  - % of patients who achieved sustained MSE (≥8 weeks)

Figure 1. Study design[5]



*[a]Randomization was stratified by autoantibody status (anti-AChR+ and/or anti-MuSK+, anti-AChR negative and anti-MuSK negative), Day 1 MG-ADL total score (≤9, >9), and region (East Asia, US, rest of world). The analysis plan, however, grouped all seropositives together (AChR+, MuSK+ or LRP4+) vs triple seronegatives. [b]All patients received the loading dose of placebo or nipocalimab 30 mg/kg at Week 0 and then started placebo or nipocalimab 15 mg/kg Q2W IV from Week 2 to Week 24. [c]SOC includes acetylcholinesterase inhibitor, glucocorticosteroid, and/or immunosuppressant. [d]In the EU, the OLE phase will be up to 240 weeks. [e]Participants who withdraw or discontinue after receiving any amount of study intervention are required to complete a safety follow-up visit 8 weeks after their last dose. **AChR**=acetylcholine receptor antibody, **EU**=European Union, **gMG**=generalized myasthenia gravis, **IV**=intravenous, **LRP4**= low-density lipoprotein-related protein receptor 4, **MG-ADL**=Myasthenia Gravis-Activities of Daily Living, **MGFA**=Myasthenia Gravis Foundation of America, **MuSK**=muscle-specific tyrosine kinase antibody, **OLE**=open-label extension, **Q2W**=every 2 weeks, **R**=randomized, **SOC**=standard-of-care.*

Results

Patient demographics and baseline characteristics of patients receiving corticosteroids at OLE

- A total of 89 patients received corticosteroids at OLE (44 patients in the Placebo/Nipocalimab group and 45 patients in the Nipocalimab/Nipocalimab group)

Table 1. Demographics and baseline characteristics in patients receiving corticosteroids[a]

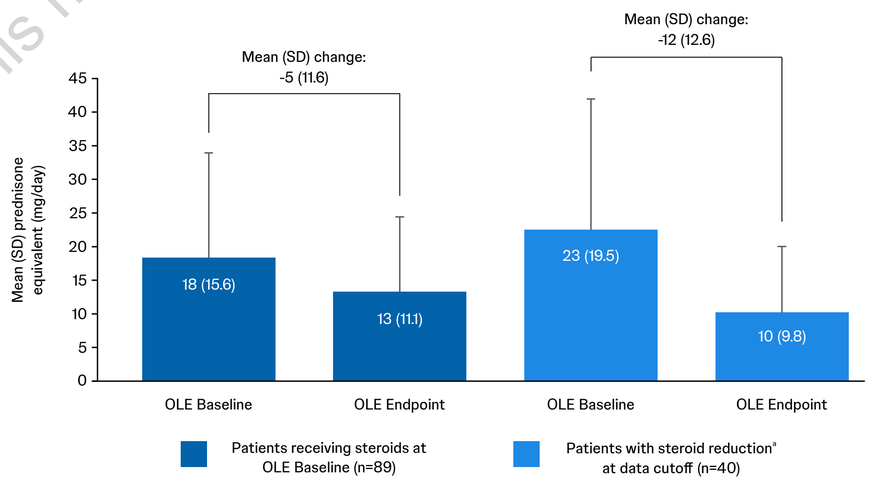
	Placebo/ Nipocalimab (n=44)	Nipocalimab/ Nipocalimab (n=45)	All Nipocalimab (n=89)
Age, mean (SD), years	51.9 (16.89)	49.7 (15.15)	50.8 (15.98)
Sex, female, n (%)	27 (61.40)	27 (60.00)	54 (60.70)
BMI, mean (SD), kg/m^2	28.3 (5.89)	27.7 (5.58)	28.0 (5.71)
Baseline MG-ADL total score, mean (SD)	9.1 (2.00)	9.4 (2.92)	9.2 (2.50)
Baseline QMG total score, mean (SD)	16.4 (4.94)	15.4 (5.06)	15.9 (5.00)
Antibody positive at screening, n (%)			
Anti-AChR+	39 (88.60)	35 (77.80)	74 (83.10)
Anti-MuSK+	4 (9.10)	9 (20.00)	13 (14.60)
Anti-LRP4+	1 (2.30)	1 (2.20)	2 (2.20)

*[a]Baseline for double-blind period. **AChR**=acetylcholine receptor, **BMI**=body mass index, **LRP4**=low-density lipoprotein receptor-related protein 4, **MG-ADL**=Myasthenia Gravis-Activities of Daily Living, **MuSK**=muscle-specific kinase, **OLE**=open-label extension, **QMG**=quantitative myasthenia gravis, **SD**=standard deviation.*

Corticosteroid use during OLE

- Of 89 patients receiving corticosteroids at OLE baseline, 40 (45%) reduced or discontinued corticosteroids by data cutoff
  - Among these patients, the mean (SD) steroid dose reduced from 23 to 10 mg/day prednisone-equivalent (**Figure 2**)

Figure 2. Mean corticosteroid dose at OLE baseline and OLE endpoint (data cutoff) among patients receiving corticosteroids at OLE baseline and those with steroid reduction at data cutoff



*The data cut-off date: August 24, 2024. [a]Reduction defined as a decreased dose or discontinuation of steroid. **OLE**=open-label extension, **SD**=standard deviation.*

Achievement of MCI and MSE among patients reaching a low corticosteroid dose

- Corticosteroid use decreased over time during the OLE phase (**Figure 3**)
- At data cutoff, 57.3% of patients reached ≤10 mg prednisone eq per day
- Sustained disease control was observed in patients who reached ≤10 mg/day or ≤5 mg/day prednisone eq doses at OLE data cutoff (**Figure 4**)

Figure 3. Proportion of patients in each corticosteroid dose group over time

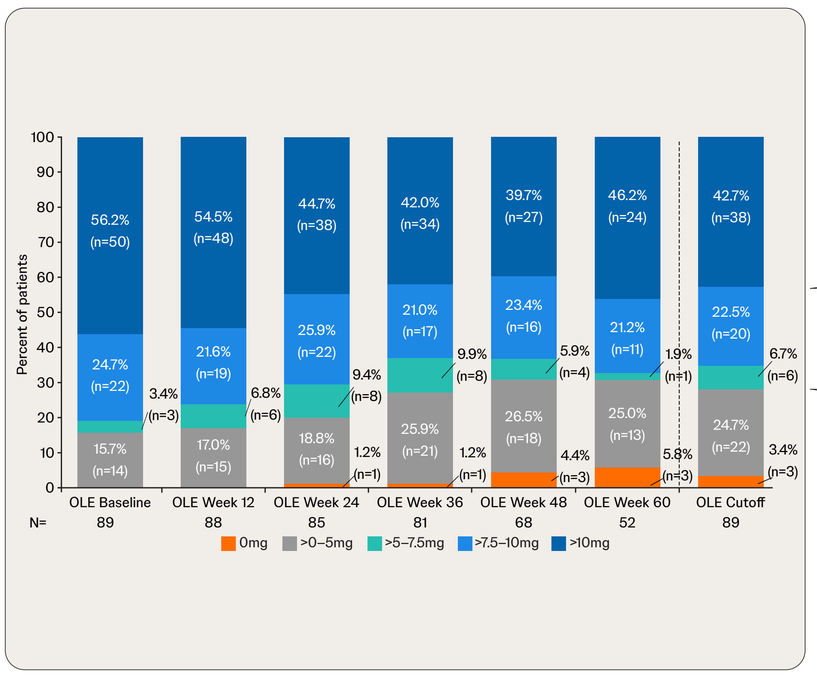
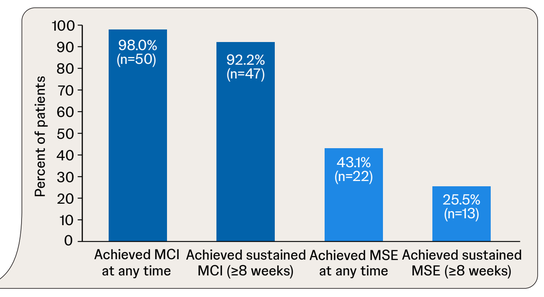
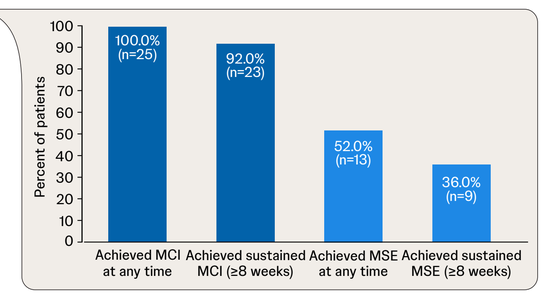


Figure 4. Achievement of MCI or MSE among:

A. Patients on ≤10 mg/day prednisone eq at OLE data cutoff (n=51)



B. Patients on ≤5mg/day prednisone eq at OLE data cutoff (n=25)



*The data cut-off date: August 24, 2024. **MCI**=minimal clinical improvement, **MSE**=minimum symptom expression, **OLE**=open-label extension.*

Safety

- The safety profile of nipocalimab in patients receiving corticosteroids was consistent with that in the overall population (**Table 2**)

Table 2. Summary of TEAEs during the OLE: patients receiving corticosteroids at OLE baseline

	Placebo/Nipocalimab (n=44)	Nipocalimab/ Nipocalimab (n=45)	All Nipocalimab (n=89)
Average duration of follow-up, weeks	68.46	67.75	68.10
All AEs, n (%)	42 (95.5)	38 (84.4)	80 (89.9)
Treatment-related	20 (45.5)	15 (33.3)	35 (39.3)
Serious AEs, n (%)	9 (20.5)	12 (26.7)	21 (23.6)
Treatment-related[a]	4 (9.1)	1 (2.2)	5 (5.6)
AEs leading to treatment discontinuation, n (%)	6 (13.6)	3 (6.7)	9 (10.1)
Infections and Infestations, n (%)	32 (72.7)	23 (51.1)	55 (61.8)
Infusion-related reactions, n (%)	0	0	0
Adjudicated MACE, n (%)			
Fatal	1 (2.3)[b]	1 (2.2)[c]	2 (2.2)
Non-fatal	1 (2.3)	0	1 (1.1)

*The data cut-off date: August 24, 2024. [a]Included cellulitis (n=1), pneumonia (n=1), hemophagocytic lymphohistiocytosis (n=1), and weight increased (n=1) in the Placebo/Nipocalimab group, and Epstein-Barr virus infection (n=1) in the Nipocalimab/Nipocalimab group. [b]Myocardial infarction, deemed unrelated to the study drug. [c]Unrelated to the study drug. **AE**=adverse event, **MACE**=major adverse cardiovascular event, **OLE**=open-label extension, **TEAE**=treatment-emergent adverse event.*

PRESENTED AT: International Congress on Neuromuscular Diseases (ICNMD); July 7–11, 2026; Florence, Italy. **REFERENCES: 1.** Leu JH, et al. *Front Neurosci.* 2024;18:1302714. **2.** Ling LE, et al. *Clin Pharmacol Ther.* 2019;105(4):1031–1039. **3.** IMAAVY™ (nipocalimab-aahu) injection for intravenous use [Package Insert] Horsham, PA; Janssen Pharmaceutical Companies, 2025. **4.** European Medicines Agency. Imaavy™. European Medicines Agency; 2026 June 1. https://www.ema.europa.eu/en/medicines/human/EPAR/imaavy. **5.** Antozzi C, et al. *Lancet Neurol.* 2025;24:105-116. **6.** Antozzi C, et al. Presented at: American Academy of Neurology (AAN) Annual Meeting; April 5–9, 2025; San Diego, CA, USA. **DISCLOSURES: Yuebing Li:** has consulted for advisory board meeting by argenx, Amgen Annexon, and Johnson & Johnson. **Kavita Gandhi, Wisam Karmous, Marie Fitzgibbon, Ibrahim Turkoz, I-Ching Tsai, Zia Choudhry, Wim Noel,** and **Louis Jackson:** are employees of Johnson & Johnson and may hold stock or stock options in Johnson & Johnson. **Hiroyuki Murai:** Has served as a consultant for Alexion, argenx, and UCB, and has received speaker honoraria from Novartis, Johnson & Johnson, Merck, and Chugai Pharmaceutical, and research support from the Ministry of Health, Labour and Welfare, Japan. **ACKNOWLEDGMENTS:** The authors thank the study patients and investigators for their participation. Medical writing support was provided by Rajani Thakur, PhD, of SIRO Medical Writing Pvt. Ltd., India. Namiko Abe, PhD, of Johnson and Johnson, provided additional editorial support. Layout design support was provided by Mugdha Rokade of SIRO Medical Writing Pvt. Ltd., India, (funded by Johnson & Johnson). **FUNDING:** This work was funded by Johnson & Johnson.