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Tanruprubart Rapidly Improves Health-Related Quality of Life in Patients With Guillain-Barré Syndrome: A Randomised, Placebo-Controlled Phase 3 Study

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Introduction

- Guillain-Barré syndrome (GBS) is a rare, rapidly progressive neuromuscular emergency that can affect anyone at anytime, often requiring prolonged hospitalisation and intensive care, and in some cases can be fatal[1]
- GBS is often associated with life-long disability.[1-3] Patients with long-term residual effects often need to change their work and general lifestyle to adapt after acute recovery[4]
- Tanruprubart (ANX005), a monoclonal antibody, is a targeted immunotherapy that selectively binds to and inhibits C1q, the initiating molecule of the classical complement pathway, thus providing rapid inhibition of complement-mediated neuroinflammation and nerve damage[5,6]
- In GBS-02 (NCT04701164), a Phase 3, multicentre, double-blind, placebo-controlled trial of tanruprubart in patients with GBS, patients treated with tanruprubart 30 mg/kg had better outcomes (adjusted odds ratio [aOR] 2.4; 95% CI 1.3–4.5; p=0.0058) according to the GBS-Disability Score (GBS-DS) relative to placebo at Week 8[7]
- Tanruprubart was well tolerated, and most adverse events were mild to moderate in severity, attributed to GBS and not considered related to the study drug[7]
- This analysis presents patient-reported outcomes with tanruprubart 30 mg/kg vs placebo

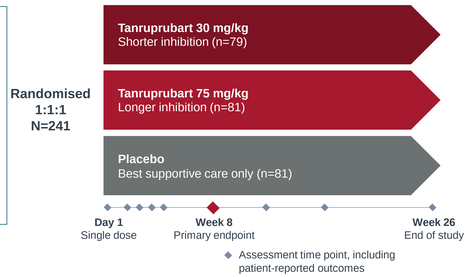
Methods

GBS-02 study design

Key inclusion/exclusion criteria:
- Aged ≥16 years
- With a GBS-DS of 3, 4, or 5
- ≤10 days from onset of weakness
- Did not have access to intravenous immunoglobulin or plasma exchange

Stratified by baseline prognostic factors: muscle strength and time from onset of weakness

Conducted at sites in Bangladesh and the Philippines



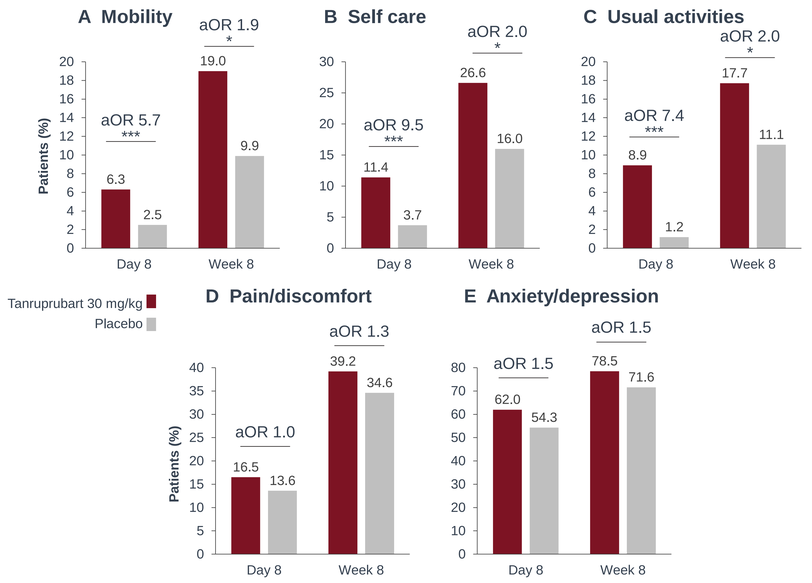
Patient reported outcomes:

- EuroQol 5-Dimension 5-Level tool (EQ-5D-5L)
- Rasch-built Modified Fatigue Severity Scale (rFSS)
- Patient Global Impression of Change (PGIC)
- Impact of treatment on health-related quality of life was evaluated using proportional odds logistic regression adjusted for baseline covariates

Results

- **EQ-5D-5L:** By Day 8, significant improvements occurred in mobility, self-care and usual activities with tanruprubart 30 mg/kg vs placebo which continued to Week 8. Improvements in pain/discomfort and anxiety/depression numerically favoured tanruprubart (**Figure 1**)
- **rFSS:** Improvements in all domains favoured tanruprubart compared with placebo at Day 8 and Week 8 except for “exercise brings on my fatigue” (**Figure 2**)
- **PGIC:** By Day 8, 8 patients (10.1%) reported being “very much improved” with tanruprubart vs 3 patients (3.7%) with placebo (aOR 2.5; 95% CI: 1.4–4.6; p=0.0028). Improvements at Week 8 still favoured tanruprubart (aOR 1.4; 95% CI 0.8–2.6, p=0.2917; **Figure 3**)

Figure 1. Patients reporting no problems (0) across EQ-5D-5L domains at Day 8 and Week 8



***p<0.0001; **p<0.001; *p<0.05.

Figure 2. Patients who answered "disagree" to statements across rFSS domains at Day 8 and Week 8

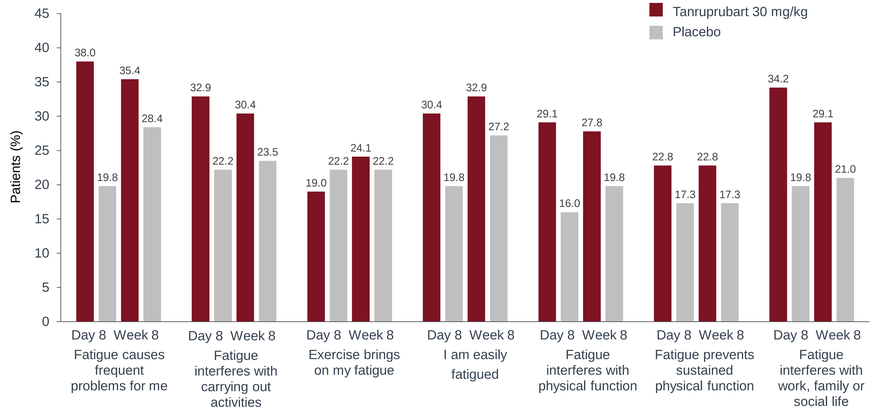
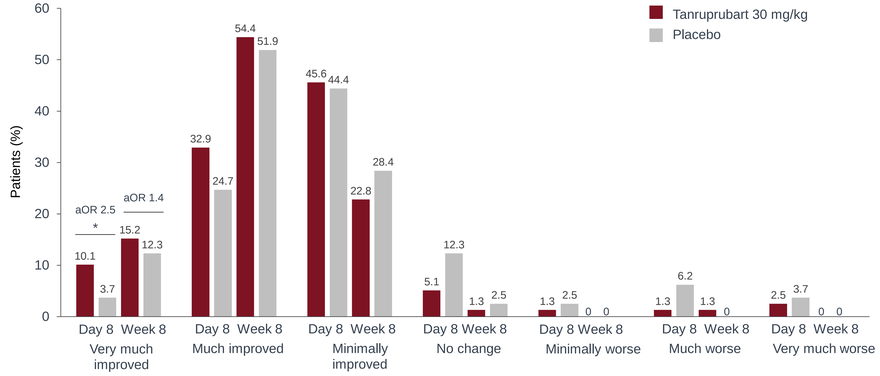


Figure 3. PGIC improvement at Day 8 and Week 8



*p<0.05.

CONCLUSIONS

- Tanruprubart demonstrated rapid and sustained improvements in health-related quality of life measures, consistent with observed objective clinical benefits
- These data further support the finding that classical complement inhibition with a single dose of tanruprubart rapidly reduces complement-mediated neuroinflammation and nerve damage leading to early stabilisation of patients during the acute progressive phase of GBS

References
1. Willison HJ, et al. *Lancet*. 2016;388:717–27. 2. van Doorn PA. *Presse Med*. 2013;42(6 Pt 2):e193–201. 3. van den Berg B, et al. *Nat Rev Neurol*. 2014;10:469–82. 4. Bernsen RA, et al. *Neurology*. 1999;53:409–10. 5. Lansita JA, et al. *Int J Toxicol*. 2017;36:449–62. 6. Suri P, et al. *Neurology*. 2022;98(18 Suppl):3867. 7. Kroon H-A, et al. Presented at the Neuromuscular Study Group Annual Scientific Meeting, September 20–22, 2022, Tarrytown, NY, USA.

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