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Letter to the Editor

Tarlatamab Likely does not Trigger Guillain-Barré Syndrome

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Letter to the Editor

We read with interest the article by Tokunaga *et al.* regarding a 68-year-old patient with end stage small-cell lung carcinoma who developed Guillain-Barré syndrome (GBS), specifically the acute inflammatory demyelinating polyneuropathy subtype, following the administration of tarlatamab as fourth-line therapy ^[1]. While the study is noteworthy, certain aspects warrant further discussion.

First, GBS cannot be attributed to tarlatamab until all alternative triggers have been adequately ruled out. Common triggers for GBS include infections (including sepsis), vaccinations, surgical procedures, immunological disorders, and malignancies ^[2, 3]. Since infections can also run a subclinical course, it is essential to systematically screen patients with GBS for clinical or subclinical viral, bacterial, fungal, protozoal, and parasitic infections. Were all potential alternative triggers for GBS thoroughly ruled out? Given that the patient suffered from end-stage small-cell lung carcinoma, the malignancy itself could have triggered the GBS.

Second, a search of various databases yielded no results linking tarlatamab to GBS, polyradiculitis, radiculitis, or neuropathy. The findings of this literature search provide further evidence suggesting that tarlatamab was likely not the trigger for the GBS in this patient.

The third point is that the patient had previously received neurotoxic chemotherapy (carboplatin, etoposide, atezolizumab, durvalumab, amrubicin, cisplatin, irinotecan). It is therefore conceivable that he was already suffering from polyneuropathy prior to the administration of tarlatamab and that tarlatamab was merely the "straw that broke the camel's back." Was he already examined using nerve conduction velocity before the GBS?

The fourth point is that no spinal cord imaging was performed to rule out spinal cord metastasis, myelitis, or spinal cord disease. The absence of tendon reflexes in a patient with chemotherapy-induced toxic neuropathy can lead to signs of central nervous system involvement, such as myelitis or spinal cord metastasis, being masked.

The fifth point is that the GBS was not confirmed via magnetic resonance imaging (MRI) with contrast enhancement to document swelling and contrast enhancement of the nerve roots in the lumbar spine. MRI of the nerve roots is a simple procedure for detecting polyradiculitis ^[4].

It is also remarkable that a patient with end-stage lung carcinoma appeared for the six-month follow-up.

Unless all alternative triggers for GBS have been conclusively ruled out, the GBS should not be attributed to tarlatamab.

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