sults in failed regression of myelin-specific T cell reactivity and prolonged survival of activated T cells (13).

Repeated injections of a TNFα antagonist cause blockage of endogenous TNF, resulting in enhancement of T cell proliferative responses and cytokine production (12). Hence, it is believed that the prolonged administration of TNFα antagonists can enhance autoimmune responses by augmenting T cell receptor signaling and decreasing apoptosis of autoreactive T cells (14). TNFα antagonists are parentally administered and can gain access to the peripheral nervous system at the roots and motor nerve terminals in areas where the blood-nerve barrier is absent or relatively deficient. This will result in a deficiency of TNFα within the peripheral nervous system compartment which will in turn prolong the myelin specific T cell response and increase the risk of developing or prolonging an immune-mediated neuropathy (15).

In summary, it is postulated that TNFα antagonist therapy could promote the development of GBS by boosting the number of activated peripheral T cells or by disturbing the intrinsic balance of TNFα and its receptors in the peripheral nervous system compartment (16).

## CONCLUSIONS

In conclusion we have presented a case of etanercept biosimilar induced GBS which responded to standard therapy of IvIg. Such adverse reactions are rare but can result in high morbidity and are difficult to predict.

We reiterate that systematic and long-term monitoring while receiving biologics and biosimilars is the need of the millennium.

**Key message**

Biosimilars and biologics share similar adverse event risks and mandate vigilant monitoring.

**Ethical statement:** the study has received approval of the institutional ethics committee.

**Conflict of interest:** the authors declare no conflicts of interest.

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