**CASE REPORT**

**Guillain Barré syndrome precipitated by the use of antilymphocyte globulin in the treatment of severe aplastic anaemia**

B Kaya, C E Davies, H E Oakervee, N C Silver, J Gawler, J D Cavenagh

This report describes the case of a 54 year old woman with very severe aplastic anaemia who was treated with antilymphocyte globulin (ALG) and developed Guillain Barré syndrome (GBS). No antecedent infective aetiology was identified. Although there are numerous reports of autoimmune disease after treatment with ALG in aplastic anaemia, and GBS after immunosuppressive treatment, there are none reporting GBS after the use of ALG for severe aplastic anaemia. The occurrence of autoimmune disease after immunosuppressive treatment, in particular ALG, is discussed, together with the possible mechanisms that result from T cell depression.

**DISCUSSION**

GBS is an acute demyelinating polyradiculoneuropathy, characterised by progressive, symmetrical weakness and areflexia, and in severe cases with respiratory muscle and autonomic involvement, although bladder involvement is unusual. It is believed to be caused by autoimmune mechanisms that are predominantly T cell mediated and is a rare but recognised event after autologous and allogeneic bone marrow transplantation, intensive conditioning regimens, and solid organ transplantation. GBS is also well documented in conditions where immune dysfunction is a feature, notably Hodgkin and non-Hodgkin lymphoma, HIV infection, and numerous autoimmune disorders such as chronic active hepatitis, hypothyroidism, sarcoidosis, Wegener’s granulomatosis, and ulcerative colitis. It is postulated that in these situations GBS may arise as a result of viral infection/reactivation or as a result of immune dysregulation, in particular iatrogenically suppressed T cell function in the case of transplant patients.

Immunosuppressive treatment in itself is not thought to be a risk factor for the development of demyelinating syndrome.

**Abbreviations:** ALG, antilymphocyte globulin; GBS, Guillain Barré syndrome; Hb, haemoglobin; IVIg, intravenous immunoglobulin; WCC, white cell count.

neuropathies. Supporting data for this include reports of GBS developing as patients were weaned off steroids and the fact that GBS is also successfully treated by immunosuppressive treatments, such as cyclosporin A. Steroids can also be beneficial in the management of chronic inflammatory demyelinating polyneuropathy. However, there are instances where GBS developed after other immunosuppressive regimens. FK506 treatment in liver transplant recipients, cyclosporin A in renal transplant recipients, and cyclosporin A in rodent experimental studies resulted in demyelinating neuropathies. Other immunosuppressive agents, such as Campath-1H, are also reported to induce autoimmune conditions—for example, a high incidence of Graves’ disease was reported after the use of Campath-1H in patients with multiple sclerosis. In some of these instances, an immune mechanism was suggested because symptomatic improvement was seen after plasma exchange or IVIg.

“Immunosuppressive treatment in itself is not thought to be a risk factor for the development of demyelinating neuropathies.”

Aplastic anaemia is thought to be mediated by a cellular immune reaction directed against haemopoetic stem or progenitor cells. ALG derived from horse or rabbit serum appears to act by reducing cytotoxic T cells and by releasing haemopoetic growth factors from certain T cells, thereby dampening down the cellular immune response. Reduced cytotoxic T cell responses that would normally restrict B cell activation but allow growth of haemopoetic progenitor cells may increase the likelihood of autoimmune disorders arising. Although there are no reports of GBS after treatment with ALG for aplastic anaemia, there are reports of other autoimmune disorders after ALG. Autoimmune thyroid disease, although rare, is described most frequently, and autoimmune haemolytic anaemia and fibrosing alveolitis have also been reported. In addition, pre-existing multiple sclerosis and fibrosing alveolitis were exacerbated after ALG, suggesting the potential of ALG to trigger and perpetuate autoimmune disease.

References

Guillain Barré syndrome precipitated by the use of antilymphocyte globulin in the treatment of severe aplastic anaemia

B Kaya, C E Davies, H E Oakervree, N C Silver, J Gawler and J D Cavenagh

J Clin Pathol 2005 58: 994-995
doi: 10.1136/jcp.2004.020354