A case of guillain-barré syndrome (GBS) preceding systemic lupus erythematosus (SLE) diagnosis in a young woman

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SUMMARY

Guillain-Barré syndrome (GBS) is an autoimmune cause of demyelinating polyneuropathy often triggered by infections. Systemic lupus erythematosus (SLE) is an autoimmune disorder caused by genetic and environmental factors. Although they are distinct conditions, there is a well-documented relationship between SLE and GBS. **Case Presentation:** A 41-year-old woman presented with progressive bilateral lower limb weakness which is ascending in nature over one week after experiencing diarrhoea and fever. Examination showed lower motor neurone weakness, hypotonia, areflexia, and loss of sensation in a stocking pattern. Nerve conduction studies showed non-length-dependent motor predominant axonal polyneuropathy. Her lumbar puncture was normal. She was diagnosed with GBS - AMAN subtype and was given IVIG for five days. Despite treatment, she deteriorated with respiratory failure, requiring intubation. Simultaneously, vasculitic rashes developed on her palms and toes. Blood tests showed hypocomplementemia, elevated ESR levels and positive for ANA, anti-dsDNA and anti-Sm. She was diagnosed with SLE and received plasmapheresis, hydrocortisone and hydroxychloroquine. Despite treatment, she succumbed due to severe sepsis. SLE and GBS can co-exist, likely due to shared autoimmune mechanisms. In GBS, infections may trigger immune responses that attack and damage peripheral nerves due to the molecular mimicry mechanism. In SLE, autoantibodies, vasculitis and cytokine overproduction can contribute to nerve damage, which can manifest as length-dependent neuropathy, mononeuritis multiplex, or a GBS-like pattern. While GBS and SLE respond to IVIG and plasmapheresis, SLE-related neuropathy may require more aggressive treatments like cyclophosphamide or rituximab. Early recognition of SLE in acute peripheral neuropathy allows for timely therapy, reducing morbidity and mortality.