New presentation of lupus nephritis following treatment with interferon

**Abstract:**

**Introduction:**

Interferon is a cytokine with immune modulatory characteristics which has been utilised as immunotherapy for the treatment of multiple sclerosis. Interferon has been reported to induce a lupus-like syndrome however, it has never been reported to cause Class IV-G (A) lupus nephritis.

**Case Presentation:**

A 49 year old lady with multiple sclerosis, previously treated with β-interferon 1a, presented with acute kidney injury requiring haemodialysis. There were no prodromal symptoms, rashes, arthritis or other extra-renal symptoms. Her auto-antibody screen was negative but there was a low C3 level and a normal C4 level. A subsequent renal biopsy revealed Class IV-G (A) lupus nephritis. This was treated with intravenous corticosteroids and cyclophosphamide as per the Euro-Lupus protocol. The patient responded well to treatment and was discharged home with a near normal renal function tests. The patient was then seen in clinic after two weeks and the renal function and the C3 level had completely returned to normal.

**Discussion:**

Type I interferon (IFN) plays a central role in pathogenesis of systemic lupus erythematosus (SLE) with IFN-α the dominant mediator, as central to the pathogenesis of this disease and is usually noted to be elevated in patients with lupus. Administration of interferon has been recognized to cause a lupus-like syndrome. This is thought to be related to the immuno-modularity effect of the drug causing autoantibodies production.

In our case, the patient has been receiving this for 15 years and she developed lupus nephritis 18 months after stopping the drug. It is not clear from the literature how long the immunologic effect lasts but it appears this effect may be proportional to the duration of use, as evidenced by this case.

**Conclusion:**

To our knowledge, this is the first report of an interferon beta-1a-induced proliferative glomerulonephritis characterized by histologic, immune-histologic, and electron-microscopic features that resembled lupus nephritis. This occurred in a patient without evidence of systemic lupus erythematosus and with negative lupus serology. In this case, the onset was a significant period of time from the stoppage of interferon, which may put doubt on direct causality, but the glomerulonephritis responded extremely promptly to immunosuppression.