## SERUM SICKNESS FOLLOWING RITUXIMAB TREATMENT CHILDHOOD-ONSET SLE: A SINGLE CENTER EXPERIENCE

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**Background:** Childhood-onset SLE (cSLE) is an autoimmune disease characterized by multiorgan involvement often necessitating a variety of immunolytic and immunomodulatory therapies to achieve adequate disease control. One of these agents, is Rituximab, a chimeric monoclonal antibody that binds to CD20 expressed on B lymphocytes initiating B cell lysis and subsequent depletion of antibody burden. An uncommon adverse reaction is serum sickness (SS), an immune complex-mediated ("type III") hypersensitivity characterized as a triad of rash, fever and polyarthritis/arthralgias. Though not commonly documented, rituximab-induced SS has been noted in patients with underlying autoimmune diseases, and reported disorders including Sjogren's syndrome, chronic ITP, and rheumatoid arthritis.

**Materials/Methods:** A retrospective review of the electronic health records of patients with cSLE from July 2011 to May 2019 was performed. Patients who developed SS following Rituximab treatment were included. Descriptive analyses were done to ascertain similarities amongst the patients, possible risk factors for the development of SS, response to treatment and use of other anti-CD20 medication use.

Results: 210 cSLE patients were being actively followed, 20 patients had received Rituximab. 6 patients developed SS after Rituximab treatment, 1 was excluded due to incomplete record. Of the 5 patients, 4 were female. The mean age of patients was 16.6 years old. All 5 were Hispanic. Mean disease duration was 3.8 years (Range: 2 to 6 years). Mean SLEDAI score at time of Rituximab treatment was 6.6 (Range: 4 to 11); 2 patients had polyclonal hypergammaglobulinemia and cSLE treatment included steroids (4/5), MMF (2/5), hydroxychloroquine (5/5). 3 patients had history of prior rituximab treatment with a mean dose of 858 mg/dose (575mg/dose to 1000 mg/dose) and mean interval from previous to most recent dose of 17.3 months. Mean time for drug exposure to SS manifestation was 9.8 days (Range: 7-13 days). Manifestations of SS included fever (4/5), acute polyarthritis (4/5), and rash (5/5). Anti-rituximab antibody was not determined at time of SS diagnosis. All patients required increased dose of glucocorticoids. 2 required hospitalization. 2 patients received and tolerated alternative B-cell depletion (ofatumumab).

**Conclusions:** We described a small cohort of cSLE who developed SS following rituximab treatment. It is important to recognize this adverse reaction as SS manifestations mimic that of active SLE features