Age at diagnosis (median $\pm$ IQR, y)	40 ($\pm$ 15)	41	WRS	0,54
Disease duration (median $\pm$ IQR, y)	11 ($\pm$ 12)	9	WRS	0,2
Number of events (median $\pm$ IQR, n)	1 ($\pm$ 1)	1	WRS	0,59
Arterial thrombosis, n (%)	6 (32)	22 (46)	Chi ²	0,4
Venous thrombosis, n (%)	12 (63)	27 (56)	Chi ²	0,7
Hydroxychloroquine therapy (n,%)	2 (10)	11 (23)	Chi ²	0,3
> 1 positivity (n,%)	7 (37)	33 (69)	Chi ²	0,026

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AB0483 A SYSTEMATIC LITERATURE REVIEW ON THE USE OF BIOLOGICS IN SJÖGREN'S SYNDROME

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Background: Primary Sjögren's syndrome (pSS) is a systemic chronic autoimmune disease characterized by dryness of the eyes and mouth. Current treatments provide modest benefit, leaving patients with limited therapeutic options. The pathophysiology of pSS involves hyperactivity of autoreactive lymphocytes. Biologic medications target mediators of the immune response. Modulation of autoimmunity in pSS with biologics has gained interest with open-label studies showing promising results.

Objectives: This review aims to conduct a systematic literature review of interventional studies investigating the efficacy of biologics in the treatment of pSS.

Methods: Literature was searched using the MEDLINE, EMBASE and PubMed databases as well as abstracts in EULAR, ACR and BSR.

Results: A total of twelve studies met the inclusion criteria. Infliximab and etanercept showed no significant improvements in fatigue and dryness in pSS compared to placebo. Anakinra showed improvement in fatigue after *post-hoc* analysis. Small trials in rituximab showed significant improvements in oral and ocular dryness but failed to replicate this in two larger randomised control trials. Belimumab significantly reduced overall disease activity which was driven by improvements in dryness and parotid gland swelling. Open-label studies in epratuzumab and abatacept showed significant reductions in fatigue with abatacept also improving salivary flow and pain in pSS.

Conclusion: Intervention with biologics in pSS has shown efficacy in alleviating pSS-associated fatigue and dryness in small RTCs and open-label trials. Larger randomised placebo-controlled trials have been inconsistent in replicating these results. This may be overcome by subgroup analysis, uptake of validated disease activity measuring tools, well-defined selection criteria to increase sample size and further understanding of pathophysiology in pSS. The small number of trials to date means the evidence base for biologics in pSS remains inconclusive.

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AB0484 EXPERIENCE WITH THE USE OF RITUXIMAB FOR THE TREATMENT OF SYSTEMIC AUTOIMMUNE DISEASES (SAD) IN A TERTIARY HOSPITAL

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Background: Rituximab is a chimeric anti-CD20 monoclonal antibody used in various clinical scenarios for the treatment of SAD, especially when immunocomplexes and/or lymphocyte B proliferation plays a central role.

Objectives: The objective of this study was to retrospectively assess the efficacy and safety of use of rituximab in a cohort of patients with SAD in a tertiary hospital within the last 12 years.

Methods: We carry out a retrospective study of 48 patients with a diagnosis of SAD in our Autoimmune and Minority Diseases Department using Google Sheets.

Results: Our cohort is mostly female (48 women and 8 men). The mean age is 40.9 $\pm$ 13.1 years (median 38 years). It is composed of different SAD: erythematous systemic lupus (22), granulomatosis with polyangiitis (6), dermatomyositis (6), eosinophilic granulomatosis with polyangiitis (3), rheumatoid arthritis (2), Takayasu's arteritis (1), Kikuchi-Fujimoto disease (1), mixed connective tissue disease (1), dermatomyositis with scleroderma overlap (1), systemic sclerosis (1) foliaceous pemphigus (1), gangrenous pyoderma, (1) polymyositis (1) and orbital pseudotumor (1). The mean time from diagnosis to rituximab was 7.7 $\pm$ 7.1 years. the efficacy of RTX in reducing **disease activity** after 6 month was 64.6% patients -according to the disease recommended scales-. Nevertheless, 39% maintained 1 year remission.

The indications for rituximab were: persistent activity despite improvement of classical immunosuppressive strategies (36), corticoid dependency (4), new relapses (3), corticoid resistance (3) and toxicity (others immunosuppressant) (2). The drugs used when rituximab was used: cyclophosphamide (24), hydroxychloroquine (21), mycophenolate mofetil or mycophenolic acid (20); cyclosporine (7), immunoglobulins (7), leflunomide (4), and methotrexate (6). 5 patients did not receive any immunosuppressant. 61.3% were taking at least 2 drugs. All of them were taking glucocorticoids.

In terms of security and side effects, we observed 11 (22.9%) adverse reactions related to the administration. We detect 3 infusion reactions (mild severity), 1 severe mucocutaneous reaction, 1 moderate herpes zoster, 1 fungic invasive infection and 5 severe bacterial infections (1 of them lead to rituximab suspension). We did not detect any hepatitis B virus reactivation, multifocal progressive leukoencephalopathy, cytomegalovirus infections nor herpes simplex infections.

We observed 65 different haematologic adverse events: lymphopenia (34), anemia (15), neutropenia (12) and thrombocytopenia (4). No one needed specific treatment.

Conclusion: Rituximab is a second line therapeutic alternative in patients with SAD in whom other treatments have failed. It is a effective drug that rescues a 65% of patients in whom other treatments have failed. Furthermore, rituximab is a safe immunosuppressant.

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