66.67%) groups. All patients in invasive aspergillosis demonstrated significantly high incidence of pneumonia ($n=19$; 90.47%), concurrent infections ($n=17$; 80.95%), and high mortality ($n=14$; 66.67%). The coinfections with bacteremia ($n=8$; 38.10%), cytomegalovirus ($n=9$; 42.86%), and candida ($n=6$; 28.57%) were common in patients with invasive aspergillosis. Gram-negative bacilli (38 isolates; 69.09%), of which the most common isolates were carbapenem-resistant *Acinetobacter baumannii* (8 isolates; 21.05%). The most common Gram-positive isolates were methicillin-resistant *Staphylococcus aureus* (6 isolates; 35.29%), followed by Vancomycin-resistant *Enterococcus* (4 isolates; 23.52%). The daily glucocorticoid dose ≥ 20 mg ($p=0.007$, HR 2.000, 95% CI 1.185–3.377), rates of recent pulse glucocorticoid therapy ($p=0.022$, HR 2.800, 95% CI 1.387–5.654), rates of azathioprine use ($p=0.022$, HR 2.000, 95% CI 1.185–3.377), rates of rituximab within six months ($p=0.022$, HR 2.000, 95% CI 1.185–3.377), plasmapheresis during hospital course ($p=0.022$, HR 2.000, 95% CI 1.185–3.377), and acute respiratory distress syndrome ($p=0.022$, HR 2.000, 95% CI 1.185–3.377) among non-survivor group were statistically higher than the survivor group. Non-survivors had significantly higher rate of concurrent infections ($p=0.002$, HR 5.667, 95% CI 2.029–15.824) and cytomegalovirus (CMV) viremia ($p=0.040$, HR 1.750, 95% CI 1.112–2.775) than survivors. The sensitivity of Galactomannan assay was not good to exclude the diagnosis of invasive aspergillosis. Septic shock ($n=7$, 50% of non-survivor group) is the most common cause of in-hospital mortality in patients with invasive aspergillosis.

Conclusions: The daily glucocorticoid dose ≥ 20 mg, recent pulse glucocorticoid therapy, azathioprine, rituximab within six months, concurrent infections, and CMV viremia are risk factors of in-hospital mortality in invasive aspergillosis patients with Systemic Lupus Erythematosus.

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AB0518

EFFICACY OF BELIMUMAB FOR PRIMARY SJÖGREN SYNDROME: RESULTS OF A SYSTEMATIC REVIEW OF THE LITERATURE

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Background: Belimumab is a human monoclonal antibody that inhibits B-cell activating factor shown to be efficacious in systemic lupus erythematosus. In other B cell mediated autoimmune disease such as primary Sjögren's syndrome (pSS) the efficacy is unclear.

Objectives: To evaluate the efficacy of belimumab in patients with pSS.

Methods: A systematic literature review was performed (EMBASE, MEDLINE and Cochrane) as part of the Spanish Rheumatology Society's Recommendations for the Use of Biological Therapies in Primary Sjögren's Syndrome. Inclusion criteria was defined as: Population: patients with pSS according to American-European Consensus Criteria 2002 Intervention: belimumab; Control: synthetic or biologic DMARDs, corticosteroids, ursodesoxicolic acid or placebo; Outcome: efficacy in dryness, glandular and extraglandular manifestations. Studies with <10 patients or <3 months of follow up were excluded. Two reviewers independently selected the articles and evaluated the quality of the evidence following SIGN guidelines.

Results: 3 articles were included out of 135. All of them published results from the same study¹ at different timepoints. The study design was experimental but with a small sample size and no control group or randomization.

The article of Mariette et al 2015¹ evaluated the efficacy and safety of belimumab in 30 patients with systemic activity or early disease until week 28 (W28). There was a significant decrease in mean ESSDAI (8.8 to 6.3 $p=0.02$), ESSPRI (6.4 to 5.6 $p=0.02$) and VAS for dryness (7.8 to 6.2 $p=0.02$). Physician's VAS for disease systemic activity also decreased in 43% as well as parotid inflammation in 76.9% of patients. Also, B cell biomarkers decreased: IgG (21.2 to 18.2 g/L, $p=0.02$), IgA (3.7 to 3.3 g/L, $p=0.04$), IgM (1.6 to 1.3 g/L, $p<0.001$), RF (146.2 to 106.7 UI $p<0.001$) and number of B cells (187.2 to 65.1/mm, $p<0.001$).

The study of De Vita et al 2015² compared results at week 52 (W52) and W28 in 19 patients, of whom 15 had previously responded to treatment. Of these 15 patients 13 maintained response and 3 out of the 4 patients that did not respond achieved primary outcome. The improvement at W52 continued for ESSDAI, glandular inflammation, lymphadenopathy and joints. B cell biomarkers remained stable. Overall items contributing to ESSDAI decreased but only physician's VAS for disease systemic activity was statistically significant (3.2 W28 vs 2.5 W52; $p=0.04$).

Abstract AB0518 – Figure 1

The study of Quartuccio et al.³ compared clinical and lab variables of W52 with 12 months of follow up after interrupting belimumab. A significant decrease in ESSDAI in 9 out of 13 patients (3.5 ± 3.7 vs 7.0 ± 5.7 ; $p<0.01$) was observed as well as in RF (52 vs 69 UI; $p<0.01$), IgM (131.9 vs 165 mg/dl; $p=0.04$) and BLYS (1304 vs 2882 pg/ml; $p=0.01$).

Conclusions: Published evidence to determine the efficacy of belimumab in primary Sjögren is limited and poor. Belimumab seems to be effective to reduce systemic activity, parotid enlargement, dryness, lymphadenopathies, articular manifestation, fatigue and B cell biomarkers.

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