p=0.01) when compared to patients with both abnormal SGUS findings and LGS focal lymphocytic sialadenitis.

Conclusion: SGUS and LSG histopathology showed only a moderate correlation in patients with pSS. SGUS may be useful to identify pSS patients with a more severe inflammatory LSG infiltrates, ultimately contributing to promote non-invasive phenotyping of multiple subsets in pSS.

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THU0260

SYSTEMIC LUPUS ERYTHEMATOSUS AND CYTOPENIAS: THE KEY FINDINGS IN BONE MARROW

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Background: Cytopenias are common in systemic lupus erythematosus (SLE), and it is fundamental to determine their etiology in order to establish an adequate therapeutic strategy.

Objectives: To describe the findings in the bone marrow aspirations (BMA) and biopsies of patients with SLE and cytopenias, as well as clinical and laboratory features associated with the etiology of the hematological abnormalities.

Methods: We performed a retrospective study in a third-level hospital in Mexico City. We included patients who fulfilled ACR criteria for SLE, presented with cytopenias and had a BMA and biopsy performed between 2000 and 2016. We described the main aspirate and biopsy findings, and also analyzed the final diagnosis and its association with clinical, laboratory and serological features.

Results: We included 101 patients; median age was 32 years and 81.2% were women. Leukopenia (<3000 cells/μl) was found in 47.5% of patients, with 29.7% having moderate or severe neutropenia (<1000 cells/μl). Lymphopenia (<1000 cells/μl) was a common finding (71.3% of patients). Moderate-to-severe thrombocytopenia (<50 K/μl) was present in 28.7% of patients. Finally, 25.8% of patients presented with pancytopenia. Regarding bone marrow findings, there was erythroid dysplasia in 50.5% of patients, granulocytic dysplasia in 28.7% and megakaryocytic dysplasia in 16%. Myelofibrosis was found in 2.6%. An increase in plasma cells (≥5%) was found in 21.8%.

In 72.3% of patients, bone marrow interpretation was conclusive. The most common diagnoses were disease activity (24.8%) and drug-associated myelotoxicity (28.7%). When compared to other etiologies, in patients with cytopenias secondary to disease activity, it was more frequent for the bone marrow be hypercellular (56 vs 23%, p=0.006) and to have increased megakaryocytes (40 vs 17.4%, p=0.048). Conversely, granulocytic dysplasia was less common in this group of patients (17.4% vs 54.3%, p=0.036).

We analyzed factors associated with both activity and toxicity as final diagnoses (Table 1). After multivariate analysis, a neutrophil count <1000 cells/μl was a protective factor for disease activity (OR 0.021; 95% CI 0.001-0.428, p 0.012). On the other hand, a history of renal activity (OR 4.3; 95% CI 1.3-14.2, p=0.024) and neutrophils less than 1000 cells/μl (OR 4.05; 95% CI 1.15-14.19, p=0.029) were found to be independent risk factors for myelotoxicity.

Table 1.

Characteristic	Disease activity (N=25)	Drug-associated toxicity (N=28)	p
History of disease activity	80%	86%	0.719
- Mucocutaneous	100%	96.4%	1
- Hematologic	44%	78.6%	0.012
- Renal			
Neutrophils (cells/μl)	16%	17.9%	0.004
- Mild neutropenia (1000-1499)	4%	7.1%	
- Moderate neutropenia (500-999)	4%	39.3%	
- Severe neutropenia (<500)			
Lymphopenia (<1000 cells/μl)	68%	70%	1

Platelets (K/μl)	16%	10.7%	0.859
- Mild thrombocytopenia (50-99)	28%	32.1%	
- Severe thrombocytopenia (<50)			
SLEDAI score	6 (0-23)	6 (1-14)	0.370
SLICC damage index	0 (0-5)	0 (0-6)	0.791
Treatment	24%	53.6%	0.048
- Azathioprine	0%	14.3%	0.113
- Mofetil mycophenolate	4% 56%	35.7% 46.2%	0.006
- Hydroxychloroquine	16%	28.6%	0.553
- Prednisone	28%	25%	
o ≤7.5 mg			
o 7.6-30 mg			
o >30 mg			

Conclusion: The main causes of cytopenias in our SLE patients were disease activity and bone marrow toxicity. Our findings suggest that when patients present with less than 1000 neutrophils/μl, it is unlikely for the cytopenias to be secondary to disease activity. Our study reinforces the diagnostic utility of BMA and biopsy, and the associations we described may assist clinicians to determine the etiology of cytopenias in SLE patients, in order to make appropriate therapeutic decisions.

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THU0261

THE URINARY CELLULAR PROFILE AS A BIOMARKER FOR LUPUS NEPHRITIS

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Background: Proliferative lupus nephritis is one of the most common and serious manifestation of SLE and is a major cause of morbidity. A search for the ideal biomarker for LN is still underway, one that can be used for early detection, and correlate with the class & activity of LN. Urine is normally devoid of leucocytes, however it has been observed that macrophages and T-lymphocytes are routinely present in the urine of LN patients and those with other proliferative renal diseases.⁽¹⁾ This provided the idea for their potential use as biomarkers for proliferative LN.

Objectives: To study the urinary CD4+, CD8+ T Lymphocytes, and CD14 + monocytes in patients with proliferative lupus nephritis, and explore their use as a biomarker for LN.

Methods: Our subjects included 30 patients with biopsy proven proliferative LN and 30 SLE patients without clinical or lab evidence of LN as controls. Lab investigations included serum creatinine, urine analysis, protein creatinine ratio, anti-ds DNA Ab, C3 and C4. For the flowcytometric analysis 100 ml of freshly voided urine in a sterile container were obtained from patients and controls. All samples were processed within 2-4 hours of collection to ensure viability of the cells. Urine mononuclear cell count was done using a hemocytometer. The urine samples were centrifuged then washed twice by phosphate-buffered saline/bovine serum albumin (PBS/BSA) and resuspended in about 300 μl PBS. The cells were stained with anti CD8- FITC, anti CD4- PE, anti CD14-PERCP and anti- CD3-APC monoclonal antibodies. The flow cytometric analysis was done using Becton Dickinson, FACS Calibur multi-parameter flow cytometer equipped with BD CellQuest Pro software for data analysis.

Abstract THU0261 – Figure 1