LUPUS MASTITIS: THE GREAT MIMMICKER

1Rita Cunha, 1Alice Pimentel, 1Bernardo Figueiredo Santos, 1Renata Aguiar, 1Joana Noronha, 1Anabela Barcelos, 2Rheumatology Dept., Centro Hospitalar do Baixo Vouga, Aveiro; 2General Surgery Dept., Centro Hospitalar do Baixo Vouga, Aveiro, Portugal

10.1136/lupus-2020-eurolupus.200

Background Lupus Mastitis is rare and can be easily confused with infection or breast cancer.

Methods We report two cases of lupus mastitis, in different clinical settings.

Results The first patient was a 34-year-old female with Systemic Lupus Erythematosus (SLE) with renal, hematological, musculoskeletal and cutaneous involvement, medicated with hydroxychloroquine (HCQ) 400 mg/day, mycophenolate mofetil 2000 mg/day and prednisolone 7.5 mg/day. She presented to the emergency department with painful right breast. On the examination, the breast was swollen, warm and painful to palpation. Breast ultrasound revealed a vascularized and lobulated mass at the upper external quadrant, with liquid inside, suggesting possible abscess, which was drained and biopsied.

Histopathological analysis demonstrated a fibroinflammatory process, with fibrotic and abscedated areas, ductitis, lobulitis and vasculitis, compatible with lupus mastitis.

Cultures from the aspirated fluid were negative.

The patient was medicated with antibiotic, NSAIDs and prednisolone dose was increased to 10 mg/day with significant improvement.

The second case refers to a 48-year-old female patient, with SLE with cutaneous, immunological and musculoskeletal involvement, who had withdrawn HCQ due to ocular toxicity. In the following months, the patient presented recurrent episodes of mastitis, on the same location. Breast ultrasound performed during one of the episodes revealed a hyperecogenic area of the fibroglandular tissue, with cystic areas. In spite of repeated treatment with antibiotics and NSAIDs, mastitis recurred, in a total of seven times. Because of worsening of the cutaneous lupus, the patient was medicated with methotrexate up to 15 mg/week. No more episodes of mastitis were recorded and biopsy of the breast, which had been considered, was not performed due to total recovery. The gynecology and rheumatology teams concluded that lupus was the etiology of the recurrent mastitis.

Conclusions Clinicians should be aware of this entity to avoid unnecessary invasive procedures, which may increase inflammation involved in lupus mastitis. Therapeutic approach usually demands increasing immunomodulation.

SIMILAR PROGRESSION OF CAROTID INTIMA-MEDIA THICKNESS IN 7-YEAR SURVEILLANCE OF PATIENTS WITH MILD SLE AND CONTROLS, BUT THIS PROGRESSION IN PATIENTS IS STILL PROMOTED BY DYSLIPIDEMIA, HYPERTENSION, HISTORY OF LUPUS NEPHRITIS AND A HIGHER PREDNISONE USAGE

1Sofia Ajejanova, 2Thomas Gustafsson, 2Linnea Lindberg, 1Ingiöld Hafström, 1Johan Frostegård. 1Dept. Medicine Huddinge, Karolinska Institutet, Stockholm, Sweden; 2Dept. Clinical Sciences, Vrije Universiteit Brussel, Brussels, Belgium; 2Dept. Laboratory Medicine, Karolinska Institutet, Stockholm; 3Dept. Environmental Medicine, Karolinska Institutet, Stockholm, Sweden

10.1136/lupus-2020-eurolupus.201

Background Effect of classical risk factors on progression of subclinical atherosclerosis in patients with SLE in comparison with population controls is not clear. We aimed to compare progression of carotid intima-media thickness (cIMT) and factors promoting it in patients with SLE and controls.

Methods Patients with SLE and matched population controls from the SLEVIC-cohort were assessed at inclusion and after seven years with standardized data collection and carotid ultrasound. Effect of risk factors on cIMT progression was examined with adjusted linear mixed models.

Results A total of 77 patients and 74 controls, 68% and 61% of the original cohort, completed follow-up. The patients were mean 47 years old, 90% females, controls were 51 years old, 92% females. Patients had disease duration of mean 11 years and mild disease activity. Baseline cIMT did not differ between the groups. An average absolute cIMT progression was 0.009 mm/year in patients and 0.011 mm/year in controls, intergroup difference p=0.9. Dyslipidemia and hypertension at both assessments and carotid plaque at inclusion were associated with cIMT progression in patients and controls. History of lupus nephritis and a higher average dose of prednisolone used since diagnosis were associated with cIMT progression in patients. Associations of risk factors with cIMT progression was stronger in presence of plaques.

Conclusions We observed similar progression of cIMT in SLE and controls over 7 years, which implies that progression of subclinical atherosclerosis in some patients with SLE could be normalized. Traditional CV risk factors, history of lupus nephritis and higher use of corticosteroids promote cIMT progression in SLE. Detection of carotid plaque may add to CV risk stratification.

RISK OF CV EVENTS AND MORTALITY IN SLE IS ASSOCIATED WITH ACCUMULATED DISEASE-DAMAGE, ANTI-PHOSPHOLIPID SYNDROME AND HIGHER CAROTID INTIMA-MEDIA THICKNESS

1Sofia Ajejanova, 1Ingiöld Hafström, 3Johan Frostegård. 1Dept. Medicine Huddinge, Karolinska Institutet, Stockholm, Sweden; 2Dept. Clinical Sciences, Vrije Universiteit Brussel, Brussels, Belgium; 2Dept. Environmental Medicine, Karolinska Institutet, Stockholm, Sweden

10.1136/lupus-2020-eurolupus.202

Background SLE is a strong risk factor for premature CVD and mortality. We investigated which factors could explain poor prognosis in SLE compared with controls.

Methods Patients with SLE and age- and sex-matched controls were recruited for this prospective study. Carotid ultrasound was performed at inclusion. The outcome was
incident CV event and death. Event-free survival rates were compared using Kaplan-Meier curves. Relative hazard ratios (HRs, 95% CI) were used to estimate the risk of the outcome.

**Results**

The patients, n=99, 87% females, were mean (SD) 47 (13) years old, had disease duration of 12 (9) years. The controls, n=109, 91% females, were mean 49 (12) years old. Baseline carotid intima-media thickness (cIMT) did not differ between the groups. During 9.6 (1.5) years, 12 patients and 4 controls were documented with the outcome, p=0.022. Compared with the controls, the risk of the outcome in the patients was 3–4-fold increased at the same level of traditional CV risk factors and carotid measures. SLE-patients with poor outcomes had higher cIMT, SLICC, APS diagnosis and used prednisolone longer time than those without. Higher SLICC and APS diagnosis were associated with increased risk of the poor outcome, respective HRs 1.66 (1.20–2.28) and 9.08 (2.71–30.5), as well as with cIMT, HR 1.006 (1.002–1.01), independent of age and sex. The combination of SLICC and APS with cIMT significantly improved outcome prediction, p<0.001.

**Conclusions**

Patients with SLE compared with controls at the same level of CV risk factors and cIMT had increased long-term risk of clinical events. Applying accumulated disease-damage, APS and cIMT may improve risk stratification in SLE.

---

**Abstract P161 Table 1**

<table>
<thead>
<tr>
<th>Age</th>
<th>Manifestations</th>
<th>Photodermosensitivity</th>
<th>Skin lesion</th>
<th>Oral ulcers</th>
<th>Alopecia</th>
<th>Arthritis</th>
<th>Pericarditis</th>
<th>Leukopenia</th>
<th>Hemolytic anemia</th>
<th>Highly positive antinuclear antibodies</th>
<th>Highly positive anti-beta-2-glycoprotein 1 antibodies</th>
<th>Highly positive antibodies to dsDNA</th>
<th>Scleroderma</th>
<th>Hypocomplementemia</th>
<th>ANF, heparin-2 positivity</th>
<th>Diagnosis</th>
<th>Treatment tactics</th>
</tr>
</thead>
<tbody>
<tr>
<td>1996 (16 years old)</td>
<td>2000-2001 (22-23 years old)</td>
<td>2005-2010 (23-23 years old)</td>
<td>2011-2012 (33-34 years old)</td>
<td>2017 (39 years old)</td>
<td>July 2018 (40 years old)</td>
<td>September 2018 (40 years old)</td>
<td>2019 (41 years old)</td>
<td>False positive serological test for syphilis</td>
<td>Chest X-ray: detection of focal changes in the right lung</td>
<td>1st Pregnancy and childbirth</td>
<td>2nd Pregnancy and childbirth</td>
<td>Portal hypertension as a manifestation of microangiopathy of APS</td>
<td>PET-CT: Infiltrative changes in the upper lobe of the right lung</td>
<td>No reliable data was obtained for antiphospholipid syndrome (APS)</td>
<td>Antibacterial therapy (drugs and doses unknown)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>No SLE (Hematologic diseases were excluded)</td>
<td>Methylprednisolone 16 mg/day, Hydroxychloroquine 400 mg/day, Mycophenolate mofetil 2 g/day, Intravenous pulse methylprednisolone (1500 mg IV/2) (in May and August), Rivaroxaban 15 mg/day</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>SLE, APS</td>
<td>Methylprednisolone 18 mg/day, Hydroxychloroquine 400 mg/day, Mycophenolate mofetil 2 g/day, Intravenous pulse methylprednisolone (1500 mg IV/2) (in May and August), Rivaroxaban 15 mg/day</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>SLE, APS</td>
<td>Methylprednisolone 8 mg/day, Hydroxychloroquine 400 mg/day, Mycophenolate mofetil 2 g/day, Intravenous pulse methylprednisolone (1500 mg IV/2) (in May and August), Rivaroxaban 15 mg/day</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>SLE, APS</td>
<td>Methylprednisolone 16 mg/day, Hydroxychloroquine 400 mg/day, Mycophenolate mofetil 2 g/day, Intravenous pulse methylprednisolone (1500 mg IV/2) (in May and August), Rivaroxaban 15 mg/day</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>SLE, APS</td>
<td>Methylprednisolone 8 mg/day, Hydroxychloroquine 400 mg/day, Mycophenolate mofetil 2 g/day, Intravenous pulse methylprednisolone (1500 mg IV/2) (in May and August), Rivaroxaban 15 mg/day</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>SLE, APS</td>
<td>Methylprednisolone 16 mg/day, Hydroxychloroquine 400 mg/day, Mycophenolate mofetil 2 g/day, Intravenous pulse methylprednisolone (1500 mg IV/2) (in May and August), Rivaroxaban 15 mg/day</td>
<td></td>
</tr>
</tbody>
</table>

**Background**

Systemic lupus erythematosus (SLE) is an autoimmune disease characterized by multiorgan tissue damage, including skin, that is frequently treated with high doses of immunosuppressive drugs. Thus, patients with SLE are at increased risk for infections (for example, tuberculosis) and malignancy. SLE and tuberculosis may have similar presentations and mimic each other; also, prior tuberculous infection may precipitate SLE in genetically predisposed individual. An increased standardized incidence ratio for non-melanoma skin cancer has been reported, however, there is no evidence of correlation between SLE and melanoma.

**Methods**

We report a case of a patient with SLE and antiphospholipid syndrome (APS) with a moderate disease activity index (SLEDAI = 7) with infiltrative tuberculosis and melanoma.