Objectives: This study aimed to analyse the development of damage and death in an inception cohort of SLE patients recruited within 1 year of diagnosis with up to 13 years of follow-up.

Methods: This was a prospective multi-centre longitudinal study in the UK of SLE patients recruited within 12 months of achieving 1997 ACR revised criteria for SLE. Data were collected on BILAG-2004, BILAG2004-Pregnancy Index (during pregnancy), SLICC/ACR DI (SDI), cumulative drug exposure and death at every visit. Information on cardiovascular risk factors and antiphospholipid syndrome status were also collected. This study ran from 1st January 2005 to 31st December 2017. Mortality and development of damage were analysed.

Results: There were 273 patients recruited (91.2% female, 59.3% Caucasian, 17.2% African/Caribbean, 17.2% South Asian) with mean age at recruitment of 38.5 years (SD 14.8). 97.8% had no damage at recruitment (2.2% had SDI score of 1). Median follow-up was 73.4 months (range: 1.8, 153.8) with total follow-up of 1767 patient-years.

There were 13 deaths (4.8%): 76.9% female, 84.6% Caucasian, 15.4% South Asian, mean age 62.6 years ($\pm$ SD 15.8) and mean disease duration 3 years ($\pm$ SD 1.8). Causes of death were cancer in 5 (38.5%), infection in 3 (23.1%), ischaemic heart disease in 1 (7.7%) and unknown in 4 (30.8%).

114 new damage items in 83 patients occurred during follow-up. The distribution of damage was musculoskeletal (21, 18.4%), ophthalmic (18, 15.8%), neuropsychiatric (18, 15.8%), renal (14, 12.3%), malignancy (12, 10.5%), cutaneous (7, 6.1%), GIT (7, 6.1%), cardiac (6, 5.3%), pulmonary (4, 3.5%), diabetes mellitus (4, 3.5%) and vascular (3, 2.6%). The rate of development of damage appears to be higher in the first 3 years which subsequently stabilised (Table 1).

Table 1. Incidence rate of development of damage over period of follow-up at 3 yearly intervals

Period of follow-up (year)	Person-years at risk	Number of new items of damage	Incidence rate, per 1000 person-years (95% CI)
0 – 3	753.4	60	79.6 (61.8, 102.6)
3 – 6	534.0	31	58.1 (40.8, 82.6)
6 – 9	321.2	12	37.4 (21.2, 35.8)
9 – 12	152.5	5	32.8 (13.6, 78.7)
> 12	5.9	0	-

Conclusion: Mortality is uncommon during the first 12 years of follow-up for newly diagnosed SLE patients. However, development of damage appears to be higher in the first 3 years before stabilizing to a lower rate subsequently.

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Translating vasculitis

POS0112 CD8+ T-CELL INFILTRATION IS ASSOCIATED WITH LESIONAL GM-CSF OVEREXPRESSION IN GCA

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Background: Giant cell arteritis (GCA) is a large-vessel vasculitis characterized by granulomatous T cell- and macrophage infiltration. Granulocyte-macrophage colony stimulating factor (GM-CSF) is present in GCA-affected vessels where it may be involved in skewing of tissue destructive macrophages (1). Furthermore, a recent phase 2 clinical trial demonstrated that GM-CSF receptor blockade was superior to placebo at 26 weeks in GCA. However, the cellular source of GM-CSF in GCA is still unclear.

Objectives: As T cells have been identified as a major source of GM-CSF in other immune-mediated diseases such as multiple sclerosis, we investigated whether T cells are GM-CSF producers in GCA patients, both systemically and locally.

Methods: GM-CSF production capacity by PMA-stimulated PBMCs from newly diagnosed, untreated GCA patients and HCs (N=10 each) was assessed using flow cytometry. Temporal artery biopsies (TABs, N=5) were immunohistochemically stained for CD3, CD8 and GM-CSF. Colocalization of these markers was assessed with immunofluorescence.

Results: Proportions of CD4+ (median GCA=3.1%; HCs=2.76%) and CD8+ (median GCA=5.8%; HCs=5.8%) T cells produced GM-CSF after *in vitro* stimulation, but no significant differences were found between the groups. Immunofluorescence staining confirmed that CD3+ T cells (both CD8+ and CD8-) were positive for GM-CSF in TABs. Interestingly, GM-CSF positivity in TABs correlated strongly with the extent of CD8+ T-cell infiltration ($r=0.74$, $p<0.01$), but not with CD3+ T-cell infiltration ($r=0.38$, $p=0.16$).

Conclusion: Our data imply that T cells are an important source of GM-CSF in GCA lesions. The correlation of the extent of CD8+ T-cell infiltration with GM-CSF positivity suggests that CD8+ T cells are the major source of local GM-CSF overexpression in tissue.

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POS0113 BAFF-APRIL-BAFFR PATHWAY ON THE PATHOGENESIS OF IMMUNOGLOBULIN-A VASCULITIS

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Background: BAFF, APRIL and BAFFR are genes that encode cytokines with a key role in the development and survival of B-lymphocytes [1-4]. The B cell-activating factor (BAFF, also known as BlyS), a proliferation-inducing ligand (APRIL) and BAFF receptor (BAFFR), respectively. Previous genetic studies have revealed that the BAFF-APRIL-BAFFR pathway is implicated in the genetic predisposition to several immune-mediated diseases [5].

Objectives: To determine whether the BAFF-APRIL-BAFFR pathway represents a novel genetic risk factor for the pathogenesis of Immunoglobulin-A vasculitis (IgAV), an inflammatory disease in which IgA deposits and B-lymphocytes are crucial [6, 7].

Methods: A functional BAFF polymorphism (rs374039502) and two tag variants within APRIL (rs11552708 and rs6608) and BAFFR (rs7290134 and rs77874543)