No recurrence of vasculitis was detected within 7 months after correction of therapy.

PO.3.71 A UNIQUE CASE OF A DELAYED DIAGNOSIS OF STRONGYLOIDIASIS HYPERINFECTION IN A PATIENT WITH SYSTEMIC LUPUS ERYTHEMATOSUS, LYMPHOPLASMACYTIC LYMPHOMA, AND KIDNEY INJURY

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10.1136/lupus-2022-elm2022.100

Purpose To report a unique case of a delayed diagnosis of stronglyoidasis hyperinfection in a patient with systemic lupus erythematotus (SLE), lymphoplasmatic lymphoma and proteinuria with microhaematuria. Stronglyoidasis stercoralis is a rare nematode endemic to the tropical and sutbtropic regions and immunocompromised patients are at risk of infection. Stronglyoidasis hyperinfection has been described in SLE cases previously, yet not also co-occurring with lymphoplasmacytic lymphoma.

Methods A 67-year-old female with a fifteen year history of SLE was referred to the immunology outpatient clinic due to malaise and ataxia progressing over several months. Before the referral, neurological, radiological, and hematological workup was performed, which objectified the clinical symptoms, showed CNS affection, and confirmed remission of a lymphoplasmacytic lymphoma treated using an B-R protocol over the previous year. Physical examination findings were consistent with previous chronic SLE sequelae, yet a decrease in body weight was noted, as well as a mild anemia, proteinuria (175 mg/dU) with microhaematuria, and increased eosinophile count. Workup was broadened, including kidney biopsy, bone marrow biopsy, parasite serology, and stool ova and parasite test.

Results Follow-up laboratory showed a further increase in eosinophile count (25%), positive Stronglyoides stercoralis serology (immunoglobulin G ELISA), and strongyloides larvae in stool samples. Bone marrow biopsy showed no abnormalities and kidney biopsy analysis ruled out lupus nephritis as a cause of proteinuria and haematuria. Single oral dose of ivermectin (200 µg/kg) was administered. One month after treatment the eosinophile count normalized and follow-up stool sample was free of larvae. Proteinuria and haematuria had resolved and other chronic morbidity remains under remission.

Conclusion Stronglyoidasis stercoralis hyperinfection should be considered when differentiating between possible causes of general deterioration in multi-morbid SLE cases, presenting even in areas not considered endemic. Paraneoplastic syndrome remains a possible etiology of the observed kidney injury.

PO.3.72 EXTRACELLULAR VESICLES OPSONIZED BY MONOMERIC C-REACTIVE PROTEIN ARE ACCESSIBLE AS AUTOANTIGENS IN SYSTEMIC LUPUS ERYTHEMATOSUS

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10.1136/lupus-2022-elm2022.101

Purpose Under certain conditions, the pentameric (p) structure of C-reactive protein (CRP) can dissociate into the more proinflammatory isoform monomeric (m)CRP. Autoantibodies against mCRP have been reported by us and others in systemic lupus erythematosus (SLE), and their levels appear to associate with renal involvement and increased disease activity. The origin of this autoreactivity thus calls for further investigation. Extracellular vesicles (EVs) have been proposed as important mediators of lupus pathogenesis by exposing nuclear antigens on their surface, thereby serving as adjuvant for autoantibody generation and subsequent immune complex-formation. Herein, we investigated pCRP and mCRP on EVs in plasma from patients with SLE and potential associations with manifestations, disease activity and organ damage.

Methods A flow cytometry protocol was established to detect pCRP and mCRP on EVs in 67 well-characterized patients from a regional Swedish lupus registry, and 60 age- and sex-matched controls. Other proteins, such as surface-bound complement protein (C)3, 4, and 4d, were also measured. Clinical data was available for the patients and plasma levels of C3 and C4 were measured by nephelometry. SLE disease activity was assessed by SLEDAI-2K and organ damage evaluated by SLICC/ACR damage index (SDI).

Results The levels of mCRP and pCRP on EVs were higher in patients than in controls (median with (interquartile range): 6.6×10–4 (3.4×10–4 – 1.2×10–3) vs 9.3×10–5 (6.8×10–5 – 4.7×10–4) for mCRP, and 5.7×10–4 (3.5×10–4 – 1.1×10–3) vs 1.1×10–4 (6.0×10–5 – 4.1×10–4) for pCRP, respectively; $p<0.001$). mCRP was more abundant on EVs in patients with active SLE (SLEDAI-2K $\geq$ 5) compared to those with quiescent disease ($p<0.01$). Furthermore, mCRP surface levels correlated weakly with SLEDAI-2K (rho = 0.25, $p<0.05$), and even stronger with modified SLEDAI-2K (rho = 0.41, $p<0.001$), but levels were significantly lower in patients with established organ damage. EV-bound mCRP correlated with SDI (rho = -0.30, $p<0.05$), a correlation which was stronger among patients with lupus nephritis (rho = -0.61, $p<0.01$). Furthermore, soluble plasma complement protein 4 (C4) correlated significantly with C4d on microparticles (rho = 0.27, $p<0.05$), particularly in those with active SLE (rho = 0.73, $p<0.05$).

Conclusions Our results suggest that EV-bound CRP, especially mCRP, could indeed be of relevance for disease progression, and that opsonized EVs might provide a potential autoantigen source.