Results: 712 (out of 33,793) transcripts were overexpressed in kidneys from diseased mice (age > 6 months). These transcripts were significantly enriched in the following pathways: response to type I interferons, antigen processing and presentation, activation and regulation of B cells, complement activation, and regulation of cytotoxicity mediated by T cells. Proportions of renal CD8 T cells with an effector phenotype were significantly increased in the kidneys from 6 months and older compared to younger mice, and compared to paired spleens. Proportions of renal effector CD8 T cells correlated significantly with transcripts involved in interferon signature, adaptive immune responses, cytotoxic T cells, chemokaxis, and correlated negatively with pathways associated with renal tubular cell functions.

Finally, we found a correlation between biological parameters of kidney function (plasma urea, albuminuria) and transcripts involved in pro-fibrotic pathways, chemokines and adaptive immune responses.

Conclusion: Our results confirm the link between the presence of activated immune effectors in the kidney and renal outcomes in a mouse model of SLE, similar to our previous observations in human LN, and warrant further functional studies on the role of kidney-infiltrating T and B cells in this model.

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CAN ARTIFICIAL INTELLIGENCE REPLACE MANUAL SEARCH FOR SYSTEMATIC LITERATURE REVIEW ON CUTANEOUS MANIFESTATIONS IN PRIMARY SJÖGREN’S SYNDROME?

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Background: Manual systematic literature reviews are becoming increasingly challenging due to the sharp rise in publications. The task is particularly daunting when the study topic is complex.

Objectives: The primary objective of this literature review was to compare manual and in-house computer software retrieval of publications on the cutaneous manifestations of primary Sjögren’s Syndrome (pSS). The secondary objective was to evaluate the prevalence of cutaneous manifestations in pSS.

Methods: We compared manual searching and searching with the in-house computer software BIBOT (1) designed for article retrieval and analysis. Both methods were used for a systematic literature review on a complex topic i.e., the cutaneous manifestations of pSS. Articles published in French or English between 1 January 1990 and 30 May 2018 were sought.

Results: The manual search retrieved 855 articles and BIBOT 1042 articles. In all, 202 articles were then selecting by applying exclusion criteria. Among them, 155 were retrieved by both methods, 33 by manual search only, and 14 by BIBOT only. Further selection was performed by reading the 202 articles, of which 54 were deemed relevant, including 23 providing data on the prevalence of one or more cutaneous signs in a cohort of patients with pSS. Cohort sizes and the nature and prevalence of cutaneous manifestations varied across publications. In all, 52 cutaneous manifestations were reported, of which the most common were cutaneous vasculitis (561 patients), xerosis (651 patients), and annular erythema (215 patients).

Conclusion: Agreement was good between the two methods. BIBOT was faster and automatically classified the articles in a chart. Combining the two methods retrieved the largest number of publications. The prevalence of cutaneous manifestations in patients with pSS varied considerably across studies. The advanced machine learning techniques used in artificial intelligence hold promise for literature reviews.

REFERENCES


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shown to enhance their inhibitory effect of CD4+ T cell pro-inflammatory function and replication. We have reported a decreased serum levels of sema3A in systemic lupus erythematosus (SLE) compared to healthy controls and in correlation with SLE disease activity [1]. Sema3A was found to be over expressed in podocytes and epithelial cells in in animal models with diabetic nephropathy. Increased urinary sema3A was also detected in diabetic patients with proteinuria and in contrast-induced acute renal injury [2-3].

Objectives: To assess urine sema3A secretion in SLE patients with and without renal involvement compared to rheumatoid arthritis patients as disease control and healthy controls.

Methods: 50 ml of fresh urine samples were collected, centrifuged and the supernatant was then concentrated up to 50 times the initial concentration and subjected to specific human Sema3A ELISA kit (MBS732622, San Diego, CA, USA).

Results: Thirty-eight lupus patients fulfilling the 2012 SLICC criteria were recruited, 33 (87%) of whom were women, at an average age of 35±12 years. Eighty patients had active nephritis (21%) and additional 5 had a history of nephritis but were in remission. APLA was diagnosed in 13 (34%) of patients. Disease activity was evaluated by the Systemic lupus erythematosus disease activity index 2000 (SLEDAI 2K) and was 8±2.7±7.

Sema3A was lower in lupus patients compared to rheumatoid arthritis and healthy controls, 4.9±3.9 ng/mL, 8.5±2.7 ng/mL, 9.85±1.7 ng/mL, p=0.0006. Lupus nephritis patients demonstrated lower urine sema3A concentration compared to lupus patients without renal involvement 4±3.4 ng/mL, 6.5±3.8 ng/mL, p=0.03. Sema 3A reversely correlated with proteinuria r=-0.43 p=0.006 and SLEDIA2K -0.3, p=0.04, but not with creatinine concentration, disease duration and complement concentration. There was no difference in urinary sema3A between SLE patients with or without APLA syndrome.

Conclusion: Urinary excretion of Sema 3A was found to be decreased in the SLE patients with renal disease, reversely correlates with disease activity and proteinuria. These findings are in line with previous reports of decreased serum level of Sema3A in SLE, that may result in reduced efficacy of regulatory T cells, driving autoimmunity and kidney damage. The discrepancy between low sema3A urinary excretion in SLE nephropathy and increased urinary secretion in other "non-auto immune" conditions with renal damage, suggests that sema3A is able to provide protection in autoimmune diseases and detrimental in "non-auto immune" conditions. This differential effect of sema3A may has to do with different populations of effector cells and different expression of sema3A receptors (nuropi-lin1). Further studies should evaluate semaphorin 3A role in lupus nephritis and its potential as a treatment option.

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