Customization of the electronic health record (EHRs) provides an opportunity to systematically collect disease activity scores in order to adequately implement treat-to-target approaches to RA treatment.

Objectives: To evaluate the overall impact of three health information technology (IT) initiatives at the point of care on the proportion of RA patient visits with disease activity scores recorded in an academic outpatient clinic.

Methods: We studied three initiatives designed to facilitate disease activity score documentation: 1. An EHR flowsheet, 2. Public reporting of physicians' performance, 3. An EHR SmartForm to facilitate the calculation and documentation of a validated RA measure, the Clinical disease activity index (CDAI). The initiatives were implemented at different time points over the study period. The study cohort included all adult RA patients with at least 2 face-to-face encounters in the outpatient rheumatology clinic at University of California, San Francisco between June 2012 – June 2017. Clinical data and covariates were retrieved from the EHR data warehouse.

To examine trends in CDAI documentation over time, we created a quality control chart (p-chart) (figure 1, where vertical lines indicate onset of each of the initiatives). For each initiative, we analysed our data using a two-pronged pre-post approach. First, we developed multiple logistic regression models in which the outcome was documentation of CDAI, controlling for patient age, sex, race/ethnicity, language and insurance category as well as physician sex and years in practice. Second, logistic mixed effect models were used to account for repeated visits by patients to the clinic and clustering by physicians.

Results: We included data from 7406 encounters from 978 unique patients. Mean (SD) age was 58.9 (16) years, 82% were female, 44% were racial/ethnic minorities, and 59% had public insurance. Over a 60 month period, overall documentation of CDAI scores per month in the clinic increased from 0% to 64% (figure 1). Results from mixed effect logistic modelling showed that Initiative 1 significantly increased CDAI recordings (OR=40.14 $p<0.001$); Initiative 2 further increased recordings (OR=2.86, $p<0.001$); Initiative 3 decreased the probability of CDAI being recorded (OR=0.61, $p<0.001$). No systematic differences were found across patient demographics or provider sex and years in practice.

Abstract OP0325 – Figure 1 proportion of visits per month with CDAI documentation during implementation of quality improvement initiatives

Conclusions: Introduction of a flowsheet and public reporting of physician performance within the practice significantly improved performance, but institution of the SmartForm did not further improve on these gains. However, gains were maintained through the end of the five-year study period. Future work should focus on whether improved CDAI documentation is associated with improved patient outcomes, such as lower disease activity and improved physical function.

Disclosure of Interest: None declared

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The stromal link to inflammation

OP0326 MODELLING THE INTERACTION BETWEEN DISEASE MICROENVIRONMENT AND MESENCHYMAL STEM CELLS IN SCLERODERMA

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Background: Mesenchymal stem cells (MSCs) are pluripotent bone marrow and tissue resident cells implicated in homeostasis and tissue repair. Systemic

sclerosis (scleroderma, SSc) is a severe connective tissue disease characterised by progressive fibrotic thickening of the dermis, accompanied by loss of subcutaneous fat and microvasculature. Aberrant activation of MSCs within the disease microenvironment may underlie the persistent fibrotic repair process, or account for the failure of adipogenesis and dysregulated vascular repair.

Objectives: We sought to: 1) determine whether activated MSCs are present within the SSc involved skin lesions, 2) test whether SSc suction blister fluid (BF) derived from involved forearm skin can induce phenotype changes in MSCs, 3) fully profile the altered gene expression in MSCs exposed to SSc BF, 4) investigate the role of key factors present at increased level in SSc BF (IL-31, lactate).

Methods: Novel post-fixation collagenase tissue dissociation techniques applied to 1 mm tissue sections, combined with Feulgen staining of DNA, were used to identify MSCs undergoing metakaryotic division within the involved skin of SSc patients. Fat derived MSCs from healthy controls were treated in tissue culture with blister fluid derived from the fibrotic skin lesions or from matched sites in healthy individuals, or exposed to key constituent factors, including cytokines (IL-31, 50 ng/ml), metabolites (lactate, 25 mM), and enhanced stiffness matrix (50 kPa gels). The responses of MSCs were studied by analysis of next generation sequencing (NGS) and phenotype changes.

Results: MSCs undergoing metakaryotic division were identified in SSc skin biopsy material but not in healthy control (HC) tissue (SSc vs HC, superficial dermis 0 vs 0, mid dermis 1.1 vs 0 $p<0.0001$, deep dermis 1.4 vs 0 $p<0.0001$ metakaryotic cells per x20 field). SSc BF (diluted 1:125 in media) induced disease-relevant phenotype changes in MSCs, such as α SMA expression ($p<0.05$), collagen gel contraction ($p<0.002$) and scratch wound repair ($p<0.016$), as well as loss of adipogenic potential, more than control BF or media alone, due in part to elevated IL-31 and lactate. NGS indicated that SSc blister fluid induced treatment-specific gene expression in MSCs (figure 1), more differentially than in normal dermal fibroblasts, consistent with activation of fibrosis, wound repair, migration, osteogenesis, connective tissue formation and loss of angiogenesis/vascular repair. Induction of α SMA in MSCs was dependent on the matrix stiffness in model systems.

Conclusions: Factors present at elevated levels in the disease microenvironment, including cytokines and metabolites, as well as the stiffened ECM, are capable of promoting the migration and differentiation of fat derived MSCs, towards tissue reparative cells implicated in the fibrotic process. Conversely, the adipogenic and vascular regenerative potential of these cells may be reduced by exposure to the SSc microenvironment.

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