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MACHINE LEARNING MODEL OF THE ULTRASOUND INDEX OF MASEI ENTHESIS AND OTHER VARIABLES OF DISEASE ACTIVITY IN PATIENTS WITH SPONDYLOARTHRITIS.

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Background: Spondyloarthritis (SpA) are a group of chronic inflammatory diseases with affection, mainly of the axial skeleton, and also of peripheral joints. The enthesitis is one of the target organs, and its inflammation known as enthesitis could be unnoticed. Machine Learning is a branch of artificial intelligence that studies the construction of a function $y=f(x)$, from a finite set of observations $D=(x,y)$, where y is an endogenous variable and x are explanatory variables. The objective of this method is to obtain a model that best fit to the data without overfitting, that could be useful to make predictions.

Objectives: We try to find Machine Learning models that relate the MASEI index (Madrid Sonographic Enthesitis Index) in entheses depending on the activity of the disease (ASDAS, BASDAI and DAPSA) and other variables in patients with spondyloarthritis.

Methods: Observational, descriptive and cross-sectional study. We have analyzed 24 patients with SpA who underwent musculoskeletal ultrasound using the MASEI index and who were treated in our clinics from May 2021 to September 2021 and under the approval of the CEICm of our center. First, we have done a feature selection of the variables most related to MASEI. To do so, we compute mutual information and chi-square test, using the scikit-learn (python) library and Matlab, respectively. Using Matlab Regression Learner package, we obtain the best Machine Learning model with the lowest RMSE for 5 fold cross-validation, which turned out to be a linear regression.

Results: To obtain regressive models that explain TOTAL MASEI, the following variables have been chosen: Type of SpA, BASDAI-DAPSA-ASDAS activity, Arthritis, Enthesophytes, Corticosteroids and CRP because they present a high degree of mutual information with MASEI and a high chi-square index. (See Figures 1 and 2). With these variables we have obtained the model that presents a lower RSME error for validated data, which has turned out to be a linear regression, given by the formula:

$$\text{MASEI} = -9.29 - 2.29 * \text{type of SpA} + 10.42 * \text{ASDAS} + 4.08 * \text{Corticoids} + 8.2 * \text{Arthritis/sinov.} + 4.46 * \text{Enthesitis} - 8.6 * \text{CRP}$$

The basic statistical characteristics of the coefficients of this equation can be consulted in Figure 3. The characteristics of the model are specified in Figure 3. In Figure 4, we observe how the data fit the diagonal and in Figures 5 and 6, we have compute a prediction and confidence intervals of our model using ASDAS and MASEI as coordinate axes.

Conclusion: We have obtained a linear model, which explains the MASEI variable as an explicit linear combination of the variables: type of SpA, ASDAS, Corticoids, Arthritis/sinov, Enthesitis and CRP. Our model, not only is simple, but it is also optimal, in the sense that for 5-fold cross validation, it obtains the lowest RSME error, compared to other Machine Learning methods, such as: neural networks, SVM, Gaussian regression processes, etc. Our model is useful to build confidence intervals, make predictions and to understand the relation between the variables mentioned above.

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EVALUATION OF HEPATITIS E VIRUS INFECTION DURING JAK INHIBITOR THERAPY IN AUTOIMMUNE INFLAMMATORY RHEUMATIC DISEASES

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Background: Although impacting hundreds of thousands of people in Western nations each year, hepatitis E virus (HEV) is an under-reported health problem (1). HEV usually is a self-limiting disease, but especially immunocompromised individuals are at risk to develop a chronic course of infection, with rapid progression to fibrosis, cirrhosis or even the development of liver failure. Janus kinase (JAK) inhibitors are a novel drug class for the treatment of autoimmune inflammatory rheumatic disease (AIRD). As JAKs play a key role in innate immunity, viral infections and reactivation are frequently reported during JAK inhibitor treatment in AIRD patients (2).

Objectives: To characterize the influence of JAK inhibitors on HEV replication *ex vivo* and assess the risk for the development of symptomatic HEV infection during JAK Inhibitor therapy *in vivo*.

Methods: To determine the effect of JAK inhibitors on HEV replication we performed infection experiments with primary human hepatocytes (PHH) followed by immunofluorescence analysis and RNAseq. To evaluate the risk of HEV infection during JAK Inhibitor therapy, we monitored HEV RNA and HEV IgG/IgM of 111 AIRD patients, receiving JAK inhibitors. Moreover, we conducted a retrospective analysis of liver enzymes of patients which were anti-HEV IgG/IgM positive.

Results: Transcriptomic analysis of PHH revealed an upregulation of innate immunity components during HEV infection. This induction was perturbed in the presence of a JAK inhibitor, concomitant with strong elevation of HEV RNA levels. In line, infection experiments displayed an up to 50-fold increase of progeny virus production during JAK inhibitor treatment indicating that JAK signaling is critical to control HEV infection. Monitoring of seroprevalence identified 17 patients which were anti-HEV IgG and/or IgM positive, while no patient had detectable HEV RNA levels. Five patients had detectable anti-HEV IgM levels suggesting a recent HEV infection. Three of 17 had a period with elevated liver enzymes (f.e. GGT > 200 U/L) during time of retrospective analysis (ranging from 10 to 23 months).

Conclusion: Obtained *ex vivo* data suggest that JAK inhibition facilitates HEV life cycle progression. Considering that JAK inhibitors are routinely applied for the treatment of AIRD, these patients may be at higher risk for a symptomatic course and outcome of HEV infection. In addition to established protocols, screening for HEV seroprevalence and HEV RNA should be considered prior starting JAK inhibitor treatment and in case of elevated liver enzymes during JAK inhibitor therapy.

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TRADITIONAL AND NON-TRADITIONAL CARDIOVASCULAR RISK FACTORS IN THE DEVELOPMENT OF EARLY ATHEROSCLEROSIS IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS AND RHEUMATOID ARTHRITIS

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Background: It is known that patients with systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA) are characterized with a variability of traditional and non-traditional cardiovascular risk factors for the early development of atherosclerosis. The results of numerous studies, examining the significance of these factors for the development of atherosclerosis in patients with autoimmune rheumatic disease (ARD), are contradictory. Additionally the prevalence of these factors was compared between ARD patients and healthy persons. There are few data about comparison cardiovascular risk factors between patient with SLE, RA and ischemic heart disease (IHD).