Physical examination revealed weight bearing pain of foot or ankle in all patients. No other sensory, vasomotor, trophic or neurological signs and symptoms were detected. Vitamin D deficiency was found in all cases (range 10 to 22 ng/ml). All patients were thus treated with adequate vitamin D daily intake. Pain relief was achieved with paracetamol, low dose ibuprofen, or a short course of oral prednisone. Rest from intense physical activity and physical therapy, avoiding detrimental feet and ankle immobilization were recommended. All children (100%) fully recovered in 3-month lag period.

Conclusion: BMO in children is a cause of disability and it is often misdiagnosed and incorrectly treated. Environmental factors, such as underestimated articular or bone microtraumas, as well as joint hypermobility, typical of paediatric age, in a bone turnover milieu of vitamin D deficiency could be the cause of the clinical conditions. Adequate vitamin D supplementation, associated with physical and analgesic therapy, are crucial in the management of BMO. We highlight the importance of supplementing vitamin D as recommended in the current guidelines, in order to avoid its deficiency and prevent invalidating clinical complaints.

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AB1061 SHORT TERM FOLLOW-UP RESULTS OF CHILDREN WITH FAMILIAL MEDITERRANEAN FEVER AFTER CESSATION OF COLCHICINE: IS IT POSSIBLE TO QUIT?

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Background Familial Mediterranean Fever (FMF) is considered as the prototype of autoinflammatory diseases, and colchicine is the main treatment option for FMF. However, the data on cessation of colchicine treatment in FMF patients is limited

Objectives To define the characteristics of children with familial Mediterranean fever (FMF) whose colchicine treatment was discontinued and then to compare these features of the patients whose colchicine was restarted with the ones not restarted.

Methods Sixty-four out of 1786 children with FMF whom colchicine was stopped by the physician or patients/parents own decision were enrolled. These patients were grouped into two as: group 1; children whose colchicine was re-started and group 2; children whose colchicine was not re-started. The demographic, clinical and genetic data were collected and compared between group 1 and group 2.

Results Colchicine was stopped in 59.4% (38/64) by the physician and 40.6% (26/64) of them had stopped colchicine by patients/parents will. Colchicine was ceased at a median of 10.6 (2.120.5) years of age, and attack- and inflammation-free periods of 18.2 (6-148) months. The median follow-up of 64 patients after colchicine cessation was 37.4 (6.4-154.7) months. It was re-started in seventeen patients due to attacks (n=11) or elevated acute phase reactants (n=6), while remaining 47 patients did not require colchicine. The age at cessation of the colchicine was lower (p= 0.04) and duration of colchicine treatment until its cessation was shorter (p= 0.007) in group 1 than group 2.

Conclusion Even though the results of our study are not satisfactory enough to endorse the hypothesis that colchicine may be discontinued by close follow-up; older age and long duration of colchicine treatment before cessation may be two important features that should be considered in the future studies.

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AB1062 INTER-LABORATORY COMPARISON OF TYPE I INTERFERON SIGNATURE ANALYSES: PAVING THE WAY TO SHARE RECOMMENDATIONS.

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Background Interferon (IFN) signature analysis is experimentally used to classify pathological conditions characterized by a type I IFN dysregulation (i.e. monogenic interferonopathies, dermatomyositis, systemic lupus erythematosus), and to formulate targeted therapy approaches. Indeed, IFN signature is used to differentiate patients with IFN-related inflammation from conditions predominantly mediated by other cytokines (e.g. JIA and periodic fevers), through the calculation of an IFN score (IS). However, at the moment, there is not a shared threshold able to discriminate among different inflammatory conditions.

Objectives To characterize the IS in different inflammatory diseases and evaluate the concordance of IFN signature results among the laboratories of the three Italian Hospitals.

Methods Assessment of the expression in peripheral blood of six IFN-induced genes (IFI27, IFI44L, IFIT1, ISG15, RSAD2, SIGLEC1) in Real Time PCR, referring to a calibrator sample (healthy controls). Calculation of the "interferon score" (IS) for each patient using the median fold change of the six relative quantifications.

50 patients with different inflammatory disorders considered not related to IFN (e.g. Crohn, JIA, hereditary periodic fevers) were compared with established type I IFN-related diseases (SLE, monogenic interferonopathies, dermatomyositis). We analysed the concordance of the clinical classification of different inflammatory diseases and the IS results. To assess the inter-laboratory variability, twenty patients with different degree of type I IFN inflammation were analysed at three different laboratories.

Results Despite the IFN signature showed a similar and comparable trend, the IS was not consistent between different laboratories, in particular for samples with higher IS score. Thus, the concordance between clinical classification and IS could be assessed only separately for each laboratory. Although in each series IFN-related disorders showed a IS significantly higher than other kind of inflammatory disorders, preliminary results suggest there is no clear threshold able to differentiate among these groups.

Conclusion Analysis of type I IFN activation in peripheral blood showed a relevant inter-laboratory variability between our three centers, limiting the possibility of identifying a shared defined threshold to differentiate inflammatory syndromes, and suggesting the need to determine reproducible calibrator samples. Cooperation between different institutes could facilitate the collection of clinical and laboratory data in a common database, to exchange expertise in type I IFN-mediated diseases, and provide practical advices for IFN signature assessment and interpretation in the clinical practice.

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AB1063 INTERSTITIAL LUNG DISEASE IN A NEWBORN AFFECTED BY MEVALONIC ACIDURIA

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Background: Mevalonic aciduria (MA) is the most severe phenotype of mevalonate-kinase deficiency (MKD), with early onset and poor prognosis. Given its rarity and its unspecific symptoms, MA diagnosis may be