attribute to increased risk of cancers can be genomic instability and impaired DNA repair.

Objectives: The aim of the study was to assess the processes of endogenous and exogenous DNA damage and its repair in patients with IIM as compared to healthy controls.

Methods: The study included 10 patients (9 men and 1 woman, mean age 46.6 ±16.5) with idiopathic inflammatory myopathies (dermatomyositis or polymyositis) as well as 7 healthy control individuals (4 men and 3 women, mean age 33.8±9.8). DNA damage and repair were investigated by the comet assay. To perform the comet assay human peripheral blood mononuclear cells (PBMCs) were isolated and incubated with tert-butyl hydroperoxide (t-BOOH) or bleomycin. Both compounds are common DNA damaging agents – t-BOOH induces oxidative DNA lesions whereas bleomycin induces also DNA double strand breaks (DSBs).

To test the DNA repair capability, PBMCs were allowed to recover for 2 hour. The level of endogenous DNA lesions was also investigated.

Results: The levels of endogenous DNA damage were not statistically significantly different between tested groups (IIM-3.3±3.6 % vs 3.2±3.8 % in control; p=0.648). The extent of the DNA damage induced by bleomycin (IIM-23.3 ±19.5 % vs 9.8±5.9 % in control) as well as oxidative stress (IIM-14.7±16.2% vs 10.4±7.4% in control) was significantly higher in PBMS derived from IIM patients than in healthy counterparts (p<0.001). Kinetic curves of DNA repair are different but the background mechanism underlying observed differences in the repair curve between healthy subjects and patients need to be evaluated further.

Conclusions: Understanding the etiology of this phenomena in these diseases may provide insight into disease pathogenesis and explain the increased susceptibility of patients to malignancies. Finding the patients with increased DNA instability could potentially serve as a biomarker and indicate the group of patients who should be carefully screened for neoplastic disorder.

Disclosure of Interest: None declared


AB0012

ALLELE AND GENOTYPE FREQUENCY OF SOME GENE POLYMORPHISMS DIFFERS IN VARIOUS JUVENILE IDIOPATHIC ARTHRITIS SUBTYPES

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Background: In spite of the fact that chronic arthritis has been the core of paediatric rheumatology, etiology and pathogenesis of juvenile idiopathic arthritis (JIA) are still unclear. It is important to ascertain genetic nature of such a heterogeneous disease.

Objectives: The aim of the study was to assess the role of some SNPs of six genes implicated in immune and inflammatory responses: TNF (rs1800629, rs361525), PTPN22 (rs2476601), MIF (rs755682, rs5844527), TRAF5 (rs3761847), CTLA4 (rs5742909, rs231775), STAT4 (rs7574868), as well as homozygous deletions of xenobiotic biotransformation genes GSTT1 and GSTM1.

Methods: 206 patients diagnosed with JIA (mean age 8.87±4.92), and 218 hospital controls with no signs of autoimmune or inflammatory diseases (mean age 13.8±2.72) were recruited for the study. The JIA patients were divided into subgroups according to JIA classification criteria; the majority of them (125 patients) had oligoarthritis, 42 children developed RF-negative polyarthritis and 27 patients were diagnosed with systemic arthritis. Genomic DNA was extracted from peripheral blood mononuclear cells and analyzed by PCR-RFLP, Real-Time PCR or fragmental analysis.

Results: The allele frequencies for all SNPs in the hospital control group were similar to those in European populations. The allele and genotype frequency distribution for all SNPs was identical in patients and controls. However, when comparing distinct subtypes of JIA, STAT4 polymorphism demonstrated higher frequencies of minor T allele (30.5% vs. 17.8%, p=0.01, OR=2.03, 95% CI [1.14–3.6]) and G/T genotype (46.3% vs. 25.6%, p=0.03, OR=2.51, 95% [1.25–5.24]) in RF-negative polyarthritis than in oligoarthritics. On the contrary, the GSTT1 appeared to prevail in oligoarthritics (23.7% vs. 7.9% in RF-negative polyarthritis, p=0.03, OR=3.65, 95% CI [1.03–12.7]). As to systemic arthritis, it was shown that minor G allele of TRAF5 was more frequent in comparison with oligoarthritics (p=0.037, OR=2.43, 95% CI [1.05–5.6]).

Conclusions: Our data show that the expression of salivary miRNAs 17, 18a and 146b may be altered in SS patients and associated with worse ultrasound and ESSPRI scores and anti-La/SSB positivity.

Disclosure of Interest: None declared


AB0013

EXPRESSION LEVELS OF MIR-124 IN THE PLASMA OF RHEUMATOID ARTHRITIS PATIENTS

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Background: Micro-ribonucleic acids (microRNAs) comprise a class of small non-coding RNAs that regulate gene expression on post transcriptional level. Levels of miR-124 have been found to be decreased in rheumatoid arthritis (RA) synovocytes and in vivo studies have shown that treatment with pre-miR-124 suppresses the progression of joint damage.1

Objectives: To evaluate the expression levels of miR-124–3p in plasma of RA patients and to determine its possible role as biomarker for diagnosis and disease activity.

Methods: 34 RA patients according to the 1987 ACR criteria were included in the study. Expression levels of miR-124–3p in the plasma were determined by PCR