عنوان مقاله:

Correlation of MicroRNA-۱۲۵b, Sirtuin, and Signal Transducer and Activator of Transcription ۳ with Biochemical Parameters and Risk Factors in Atherosclerosis Patients

محل انتشار:

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نویسندگان:

.Atefeh Mirderikvand – Department of Clinical Biochemistry, Faculty of Medicine, Lorestan University of Medical Sciences, Khorramabad, Iran

.Gholamreza Shahsavari – Department of Clinical Biochemistry, Faculty of Medicine, Lorestan University of Medical Sciences, Khorramabad, Iran

.Alireza Moayyed Kazemi – Department of Internal Medicine, Lorestan University of Medical Sciences, Khorramabad, Iran

Fatemeh Ahmadpour – Department of Medical Laboratory Sciences, School of Allied Medical Sciences, Lorestan University of Medical Sciences, .Khorramabad, Iran

Banafsheh Yalameha* – Student Research Committee, Tabriz University of Medical Sciences, Tabriz, Iran & Department of Biochemistry and Clinical .Laboratories, Faculty of Medicine, Tabriz University of Medical Sciences, Tabriz, Iran

خلاصه مقاله:

Background: Atherosclerosis (AS) is an inflammatory disease linked to vascular events, with dysregulation of microRNA (miR)-۱۲۵b, contributing to cardiovascular disease pathogenesis. Moreover, there is evidence of the involvement of signal transducer and activator of transcription ۳ (STAT۳) and sirtuin ۶ (SIRT۶) in AS. This study aimed to survey the expression levels of miR-۱۲۵b, STAT۳, and SIRT۶ in the peripheral blood mononuclear cells (PBMCs) of AS patients and controls, and to find their correlations with biochemical parameters and risk factors. Methods: This study included blood samples from ۴۵ controls and ۴۵ AS patients, with PBMCs isolated using Ficoll solution. Expression levels of miR-۱۲۵b, STAT۳, and SIRT۶ were determined via quantitative Real Time-PCR. Results: The findings revealed a significant increase in miR-۱۲۵b levels in patients compared to controls (P = ۰.۰۱۷). However, alterations in STAT۳ and SIRT۶ expression were not significant (P> ۰.۰۵). There was no substantial relationship between miR-۱۲۵b and STAT۳ (P = ۰.۵۲۲) or SIRT۶ (P = ۰.۸۸). miR-۱۲۵b showed a significant relationship with atherogenic indexes and creatinine (P<۰.۰۵), while the association of SIRT۶ with HDL and creatinine was significant (P<۰.۰۵). STAT۳ exhibited high diagnostic power for identifying individuals at risk of heart disease and hypertension (P<۰.۰۵). Conclusions: STAT۳ can serve as a valuable biomarker for detecting AS and AS-related risk factors. miR-۱۲۵b and SIRT۶ may be associated with AS lipid metabolism. However, further studies with larger sample sizes are recommended to mechanistically elucidate the .association of these genes. Keywords: Atherosclerosis, MicroRNA-۱۲۵b, STAT۳, SIRT۶, Leukocyte

کلمات کلیدی:

Atherosclerosis, microRNA-۱۲۵b, Signal transducer and activator of transcription ۳, Sirtuin ۶, Leukocyte