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THE IMMUNOTHERAPEUTIC EFFECT OF IL-22 VERSUS PRAZIQUANTEL TREATMENT AGAINST *S.MANSONI* – INDUCED LIVER FIBROSIS IN MICE

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Abstract

Background/Aim:

Praziquantel (PZQ), the primary medication for schistosomiasis treatment, exhibits a potential

resistance by the parasite. Therefore, the development of a new effective treatment is obligated. Interleukin-22 (IL-22) has been reported to have a hepatoprotective effect. The current study aimed to compare the effectiveness of IL-22 treatment versus PZQ against S. mansoni - induced liver fibrosis in mice.

Materials and Methods:

Forty male albino mice were divided into control, infected, IL-22 (0.36 µg/kg), and PZQ (a single dose of 600 mg/kg) groups. PZQ was administered alone or in combination with IL-22. Inflammatory indicators [tumor necrosis factor-α (TNF-α), interleukin-17 (IL-17), IL-22, and immunoglobulin E (IgE)], hepatic expressions of signal transducer and activator of transcription 3 (STAT3), β-catenin, and miR let-7a gene expressions, and liver granuloma index (GI) were estimated.

Results:

The present result revealed a significant (P <0.05) reduction in liver GI and the pro-inflammatory cytokine, TNF-α, after the treatment with IL-22. Moreover, the treatment enhanced significantly (P <0.05) let-7a miRNA and STAT3 gene expressions as well as downregulated (P <0.05) β-catenin mRNA, which in turn could reduce fibrosis resulting from S. mansoni infection. On the other hand, PZQ treatment alone or in combination with IL-22 reduced significantly (P <0.05) proinflammatory cytokines and IgE but failed to decrease GI or β-catenin gene expression, which might cause a negative impact on liver fibrosis.

Conclusion:

IL-22 could be a potential immunotherapeutic agent for S.mansoni-induced liver fibrosis, compared to PZQ, through activating STAT3 and let-7a downstream signalling pathways and inhibiting β-catenin pathway.

Keywords
IL-22, PZQ, Schistosomiasis, Liver Fibrosis, STAT3, B-Catenin, Let-7a.

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