Interferon Gamma Production by T-Lymphocytes in Patients with Chronic Viral Hepatitis B

Thesis

Submitted for partial fulfillment of

Master Degree

in

Internal Medicine

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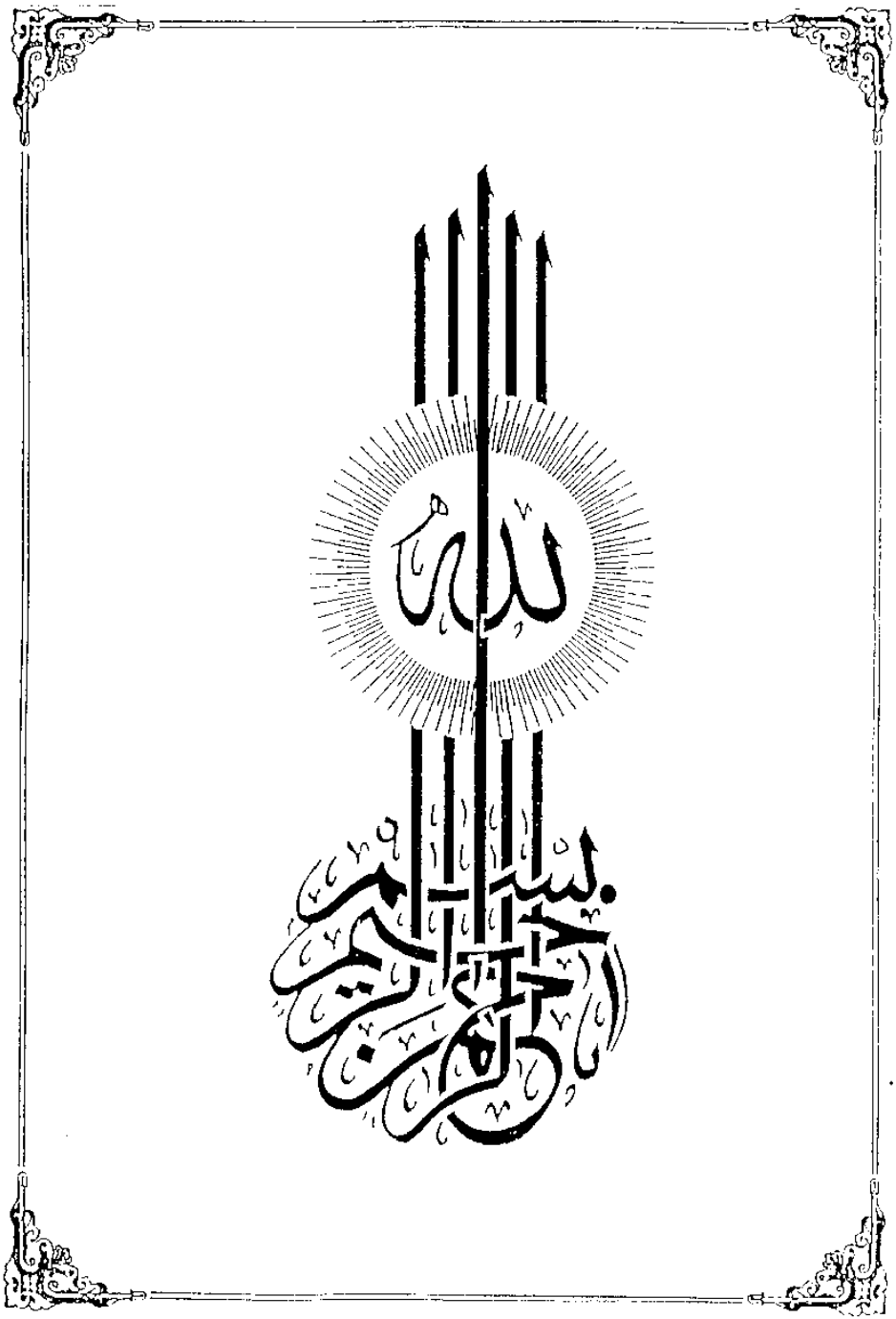

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ACKNOWLEDGMENT

First and foremost, thanks to GOD the Most Helpful and Beneficient.

I would like to express my deepest gratitude to Professor Dr. Omaima Esmail El-Lamie, Professor of Internal Medicine, Ain Shams University, for her indispensable guidance, kind care, continuous help and support.

My profound appreciation goes to Dr. Omnia Ahmad Fahmy, Assistant Professor of Internal Medicine, Ain Shams University, for her kind help throughout the whole work and for revising it.

I shall remain much obliged to Dr. Randa Mabrouk, Lecturer in Clinical Pathology, Faculty of Medicine, Ain Shams University, for her great effort and comprehensive technical help throughout the work.

Lastly, I would like to express my deep thanks to all who helped me finishing this work.

Wageeh Ahmad
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INTRODUCTION

It is generally accepted that liver cell damage does not depend on hepatitis viruses infection, but on the host's immune response to viral antigens in chronic viral hepatitis ***(Mondelli and Eddleston, 1984).***

Several cytokines are important mediators of the inflammatory response in various liver diseases. They are produced by the immune cells and circulate in abnormal concentrations inflicting tissue injury, inflammation, and pathophysiology.

Their secretions by the immune system is directly related to the factors that cause abnormalities in cell-cell interaction, proliferation, and function ***(Al-Wabel et al., 1993).***

Earlier cytokine levels were measured from serum samples from patients with various types of hepatitis ***(Bird et al., 1990; Sun et al., 1992).***

Many cytokines are not stable in serum because of the action of proteases and inhibitors and if serum is not

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protected the cytokines are degraded and might produce aberrant values.

Therefore measurement in-vitro production of cytokines by immune cells is a true indicator of cytokine involvement in the pathogenesis and pathophysiology of human disease ***(Al-Wabel et al., 1993).***

Several studies demonstrated that the various hepatic antigens were efficient immunogenes in terms of interferon gamma would be useful in determining the cellular immune response in chronic viral hepatitis ***(Wakita et al., 1992; Inoue et al., 1989).***

Al-Wabel et al., (1993) demonstrated an up-regulated production of interferon gamma in active viral hepatitis.

Kabumu et al., (1994) found out that the values of interferon gamma in cultures from blood were higher in acute hepatitis than in chronic hepatitis. They suggested that the different levels of interferon gamma may be due to changes in the proportion of lymphocyte subsets

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AIM OF THE WORK

The **aim of this study** is to measure in-vitro the interferon gamma production by stimulated T-lymphocytes in patients with chronic viral hepatitis B. The level of the interferon gamma will be correlated with the type of the underlying disease, and various clinical and laboratory parameters.

Interferon gamma in these patients will be also correlated to interferon gamma produced by stimulated T-lymphocytes in normal subjects to evaluate the cellular response in chronic viral hepatitis B.

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Review of Literature

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