

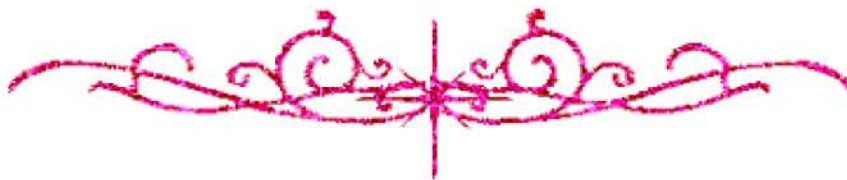
بسم الله الرحمن الرحيم



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شبكة المعلومات الجامعية التوثيق الالكتروني والميكرو فيلم



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جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
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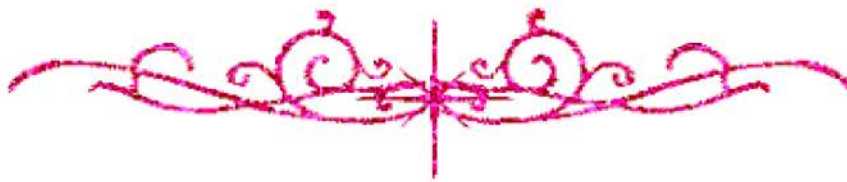
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لم ترد بالأصل



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The Role of Cytomegalovirus in Alopecia Areata: PCR and "Nested" PCR Study

Thesis

*Submitted to the Faculty of Medicine, Tanta
University, in Partial Fulfillment for the
Requirement of the Master Degree*

In
Dermatology and Venereology

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**Faculty of Medicine
Tanta University
2001**

This work is dedicated to
**The Memory of my Father and
Mother**
and
to My Wife and Children



بسم الله الرحمن الرحيم



Acknowledgments

A great deal of credit goes to *Professor Wafa'a Ramadan, M.D.*, Department of Dermatology, Faculty of Medicine, Tanta university, for her continuous guide and encouragement throughout this work.

I'd like to express my deep gratitude to *Professor Amani Kolkaila, M.D.*, Department of Dermatology, Tanta University, *Professor Raghda Talaat M.D.*, Department of Microbiology, Tanta University, for their patience, support and enthusiasm.

I am most grateful to *Osama Roshdy, M.D.*, Lecturer, Department of Dermatology, Tanta University, who cared to review the drafts of the manuscript and provided me helpful hints. I would also like to thank *Mohamed Sharaf-El-Deen, M.Sc.*, Assistant Lecturer, Microbiology Department, Ain Shams University, for his help in the laboratory part of this study.

This work could almost be impossible without the kind cooperation of the *patients* involved in this study. I am obliged to them.

I would also like to thank all the *Staff Members* of the Department of Dermatology, Tanta University. I am quite thankful for all of them.

List of Abbreviations

AA	Alopecia areata
ALL	Acute lymphoplastic leukemia
CMI	Cell mediated immunity
CML	Chronic myeloid leukemia
CMV	Cytomegalovirus
CTCL	Cutaneous T cell lymphoma
DEBR	Dundee experimental bald rat
EBV	Epstien Barr virus
GVHD	Graft-Versus-Host Disease
HF	Hair follicle
HLA	Human leukocytic antigen
HPV	Human papillomavirus
HSV	Herpes simplex virus
ICAM	Intercellular Adhesion Molecule
IL-1	Inter-leukin-1
KD	Kilo Dalton
LAH	Loose anagen hair
MHC	Major histocompatibility complex
MG	Myasthenia gravis
nDNA	Native DNA
nm	Nanometer
nPCR	Nested PCR
PA	Pili annulati
PAS	Periodic acid Schiff
Pb	pair base
PCR	Polymerase chain reaction
SCID	Severe combined immunodeficiency
TCR	T cell receptor

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INTRODUCTION & AIM OF THE WORK



Alopecia areata (AA) is one of the most common non-cicatricial alopecias. It is an inflammatory, usually reversible disorder, characterized by the spontaneous appearance of circumscribed patches of complete hair loss, which, when severe, can result in loss of all scalp (alopecia totalis) and body (alopecia universalis) hair (Bystryn and Tamesis, 1991)¹.

It is not possible to attribute all or indeed any case of AA to a single cause. Among the many factors that appear to be implicated, in at least a proportion of cases, are the patient's genetic constitution, non-specific immune and organ-specific autoimmune reactions and possibly emotional factors (Madani and Shapiro, 2000)².

Many different lines of therapy have been used for AA. However, the subsequent studies have done little more than reinforcing, the generally held belief, that both genetic predisposition and the atopic state influence prognosis but the triggering mechanisms remain obscure (Dawber et al., 1998)³.

Occasional epidemics of AA observed among subjects living in closed communities have suggested a viral etiology (Thiers and Galbraith, 1986)⁴. Support for this possibility comes from the report, by Grimes, of polymerase chain reaction (PCR) evidence of cytomegalovirus (CMV) in 12 of 13 cases of vitiligo, a closely related condition (Grimes, et al., 1996)⁵.

Skinner et al., (1995)^{6, 7} used PCR technique to detect CMV DNA in paraffin block sections of AA lesions. They suggested that hair root could be a site of latency for CMV, which would explain much of the behavior of AA with its tendency to intermittent remissions and relapses. It is known that if any virus is implied in the etiology of certain disease, it means that it must be found in a significant percentage of the skin lesions of this disease.

Hence, the aim of this work is to search for CMV in the lesional tissue in patients of AA by PCR and nested PCR in order to shed a light on the possibility of a role for CMV in this disease.



REVIEW OF LITERATURE

