

EVALUATION OF HYPERPIGMENTED SKIN LESIONS

Thesis

Submitted for partial fulfillment of the Master Degree in
General Surgery

By

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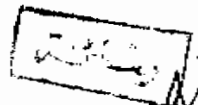
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1998

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

**قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا،
إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ**

صدق الله العظيم

سورة البقرة- الآية: ٣٢



**TO MY
Parents**

ACKNOWLEDGMENT

Thanks first and last to GOD as we owe him for his great care, support and guidance in every step in our life.

I would like to express my profound gratitude and cordial appreciation to my Prof. Dr. Ikram Ibrahim Safe, Professor of Plastic & Reconstructive Surgery, Faculty Of Medicine, Ain Shams University, for his moral support, valuable supervision and for enabling me to fulfill this work.. He is not only my Professor to whom I am very grateful, but he is also a human to whom I wish always the best of everything.

Indeed, words do fail me when I come to express my sincere indebtedness to Dr. Ahmed Salah El-Dein El-Badawy, Lecturer of Plastic & Reconstructive Surgery, Faculty Of Medicine, Ain Shams University, for his extraordinary honest effort and help.

Last but not least, my hearty thanks to my friends and colleagues in the department of Plastic & Reconstructive Surgery who provided me with their best knowledge, experience and effort until this work is completed.

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Introduction & Aim Of The Work

INTRODUCTION AND AIM OF THE WORK

Color variation of the human skin is dependent on melanin present inside the **melanosomes** (specialized epidermal melanin-bearing organelle) (*Quevedo et al. 1974*).

It is the presence of melanin inside these melanized melanosomes that imparts normal skin color.

Melanosomes are synthesized in melanocytes, transferred to keratinocytes, and retained inside these keratinocytes producing skin coloration.

The functional unit responsible for this orderly process is called the **epidermal melanin unit**, which is composed of a melanocyte and an associated cluster of keratinocytes (about 36 in number) (*Hadley et al. 1966*).

Clinical skin color depends upon the collective visual impact of many of such epidermal melanin units.

Hyperpigmented disorders may be **melanotic** where there is a normal number of melanocytes but melanin

pigment is increased (e.g. post-inflammatory hypermelanosis, and melasma) or **melanocytotic** where melanocytes are increased (e.g. oculodermal melanocytosis) or **nonmelanotic** hyperpigmentation (e.g. minocycline pigmentation, and tattoos).

Genetic factors play the primary role in determining the degree of pigmentation that is normal for the individual, i.e. **constitutive skin color** as well as the response to exposure to sunlight, i.e. **inducible skin color** (*Fitzpatrick 1988*).

Changes in pigmentation can arise in a number of ways and can be due to a variety of genetic and environmental factors. Abnormalities may involve:

1. Formation of melanosomes in melanocytes e.g. **Lentiginosis**
2. Melanization of melanosomes e.g. **Ephelides (freckles)**
3. Secretion of melanosomes into keratinocytes e.g. **Peutz-Jeghers syndrome**

4.Transport of melanosomes in keratinocytes with and without degradation in lysosome-like organelles e.g. Incontinentia pigmenti (**Bloch-Sulzberger syndrome**)

The present classification of hyper pigmentation is based on the color of the skin and on various causative factors.Hypermelanosis (increased melanin), our main concern in this thesis, may be due to genetic,and naevoid factors or it is acquired and due to a variety of factors.

Hypermelanosis is further classified according to the color change into brown color or gray (blue) color.

Detailed classification regarding various symptoms, signs, and diagnosis will be discussed later in this thesis.

Proper diagnosis of a hyperpigmented skin lesion is a difficult task due to the wide variety of causes and the different methods of diagnosis available *which depend on :*

1. Clinical aspects

History,Physical examination,and special clinical aids e.g. magnification , illumination , or epiluminiscent skin microscopy (*Sandhya et al. 1993*).

2. Histologic aspects

Staining of biopsies after preparation is done using Hematoxylin and Eosin, Dopa reaction, or Silver stains.

3. Specialized laboratory methods

Split dopa histochemical technique, Electron microscopy, Cell culture, newly developed physical methods (Spectrophotometry and Electron spin resonance (ESR) signal analysis) (*Takawaki et al. 1994*).

Reaching a proper diagnosis of hyperpigmented skin lesions would result in choosing the proper mode of interference, either medical or surgical and therefore increasing the rate of success of the treatment.

AIM OF THE WORK

The aim of this thesis is to establish a proper systematic approach to reach diagnosis of hyperpigmented skin lesions in order to facilitate the choice of treatment and so provide a better chance of reaching successful results.

Review Of Literature

CHAPTER 1

SURGICAL ANATOMY OF THE SKIN

An understanding of the normal histology of the skin is central to an understanding of all that is termed cutaneous pathology (*Murphy 1997*)

Obagi in 1990 stated that skin functions are:

1. Barrier between the organism and the environment
(absorption and secretion)
2. Protection of the organism from harmful external factors (sun, chemicals and physical)
3. Thermal regulatory function (vasculature and sweat)
4. Sensory function
5. Immune function (Langerhans cells)

The skin can be divided into three main components:

(Fig. 1.1)

1. Epidermis
2. Dermis and its appendages
3. Subcutaneous layer

Fig. 1.1a: Normal structure of the skin (fingertip).

Fig. 1.1b: Normal structure of the skin (palm).