

# CUTANEOUS TELANGIECTASIA

THESIS

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

" قالوا سبحانك لا علم لنا الا ما علمتنا  
انك انت العليم الحكيم "

صدق الله العظيم

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# **INTRODUCTION**

## INTRODUCTION

Telangiectasia is rather a common dermatologic sign. Literally speaking telangiectasia is a dilatation of the terminal vessels i.e. of the capillaries. However the term is used to describe dilated veins, capillaries or arterioles (Braverman, 1981). In general they are formed by dilated capillaries in the upper corium and mucous membranes (Mirrer et al., 1971).

If the distribution and morphology of telangiectasia were observed as carefully as the associated other lesions, the telangiectatic syndromes associated with systemic diseases would be easily recognized. Although such vascular changes can usually be detected without magnification, a hand lens is sometimes required (Braverman, 1981).

Telangiectasia is in itself a banal phenomenon and has diverse forms which may be linear, stellate or punctate. Although some are diagnostic markers for systemic diseases (e.g. the mat telangiectasia of scleroderma, the angiokeratoma of Fabry's disease, and the red puncta of hereditary hemorrhagic telangiectasia) most telangiectases are considered to be cosmetic problems without any medical significance (Braverman and Keh-Yen, 1983).

Usually telangiectasia can be divided into two basic categories, primary telangiectasia of unknown etiology which

includes the nevoid group and secondary telangiectasia which is associated with known disturbance as connective tissue disease, radiodermatitis and mastocytosis (Mirrer et al., 1971).

The aim of this review is to revise the importance of this sign in each of these disorders, the mechanism of its formation and the different methods of treatment of both the telangiectatic vessels and its associated disorders.



# **REVIEW OF LITERATURE**

### CUTANEOUS TELANGIECTASIA

Telangiectasia (literally 'end vessel dilatations') are permanently dilated small vessels (Champion, 1979). Oslen in 1985 defined telangiectasia as permanent dilatation of venules while Braverman (1981) stated that it consists of dilated venules, capillaries or arterioles in the skin.

Lookingbill (1985) explained that an individual lesion is a telangiectasis, multiple lesions are telangiectases and the adjectival term is telangiectatic and he added that telangiectases blanch completely with pressure and they may be of different shapes, including:

- (1) Linear, as in the periungual telangiectases seen in patients with collagen vascular diseases.
- (2) Punctate as in hereditary hemorrhagic telangiectasia and sometimes in scleroderma. or
- (3) Stellate as in spider telangiectases.

There are general agreement that cutaneous telangiectasia can be classified into a primary group of unknown etiology and a secondary group resulting from or associated with a known disease or syndrome (Oslen, 1985). The following table represents the classification of telangiectasia.

## **I. Primary**

### **A- *Nevus flammeus***

#### **1- *Localized***

#### **2- *Systemic involvement***

##### **(a) *(Sturge-Weber syndrome***

##### **(b) *Klippel-Trenaunay syndrome)***

### **B- *Idiopathic vascular spiders (spider nevus, nevus araneus).***

### **C- *Venous lakes.***

### **D- *Generalized essential telangiectasia (angioma serpiginosum, essential progressive telangiectasia)*** **- *Unilateral nevoid telangiectasia syndrome.***

### **E- *Hereditary hemorrhagic telangiectasia (Rendu-Osler-Weber disease).***

### **F- *Ataria telangiectasia.***

### **G- *Syndromes with telangiectatic erythema:***

#### **1- *Bloom's syndrome,***

#### **2- *Rothmund-Thomson syndrome,***

#### **3- *Hereditary sclerosing poikiloderma,***

#### **4- *Dyskeratosis congenita,***

#### **5- *Cockayne's syndrome.***

### **H- *Poikiloderma vasculare atrophicans (idiopathic).***

## **II- Secondary**

### **A- *Physical insults: X-rays, actinic, trauma, steroids***

### **B- *Tumors: Mastocytoma, basal cell carcinoma, metastatic carcinoma.***

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**C- Miscellaneous diseases:**

- 1- *Connective tissue disease: lupus, dermatomyositis, scleroderma, mixed connective tissue disease.*
- 2- *Rosacea.*
- 3- *Secondary poikilodermas: connective tissue disease, parapsoriasis en plaques, mycosis fungoides.*
- 4- *Mastocytosis.*
- 5- *Xeroderma pigmentosum.*
- 6- *Pseudoxanthoma elasticum.*
- 7- *Malignant atrophic papulosis (Degos' disease).*

\* *After, Oslen (1985).*

Another classification based on the clinical significance of telangiectasia in the skin was given by Shelley (1971).

The following table represents his classification:

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*Telangiectasia in the Skin*

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- I- *Component of*
  - Rosacea*
  - Varicose veins*
  - Actinic dermatitis*
  - Radiodermatitis*
  - xeroderma pigmentosum*
  
- II- *Associated with systemic disease*
  - Lupus erythematosus*
  - Dermatomyositis*
  - Scleroderma*
  - Mastocytosis* \*
  - Carcinoma telangiectaticum*
  
- III- *Hallmark of*
  - Basal cell epithelioma*
  - Necrobiosis lipoidica diabetorum*
  - Poikiloderma atrophicans vasculare*
  - Capillaritis* +
  
- IV- *Essential primary lesion in*
  - Vascular nevi*
  - Congenital neuro-angiopathies* ++
  - Hereditary hemorrhagic telangiectasia (Osler)*
  - Essential progressive telangiectasia* §

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\* *Telangiectasis macularis eruptiva perstans.*

+ *Purpura annularis telangiectodes.*

++ *Eg, ataxia telangiectasia.*

§ *Includes generalized telangiectasia and angioma serpiginosum.*

However, other causes of telangiectasia not mentioned in the previous two tables include: angiokeratoma (Imperial and Helwig, 1967a; Braverman and Keh-Yen, 1983; Caro and Bronstein, 1985) Morquio syndrome (Greaves and Inman, 1969) some patients with thyroid disease (Thomson and Mackie, 1973) in the skin of some workers in aluminium industry (Theriault et al., 1980), Cherry hemangioma (Braverman and Keh-Yen, 1983) and among the cutaneous manifestations of functioning carcinoid (Kierland, et al., 1958).

#### Cutaneous vasculature

In order to understand the subject of cutaneous telangiectasia, it is very important to describe shortly the normal cutaneous vasculature. The vascular architecture of the dermis consists of a superficial and a deep plexus of arterioles and venules interconnected by communicating blood vessels that are oriented perpendicular to the skin surface (Fig. 1) (Jakubovic and Ackerman, 1985). The deep plexus of arterioles and venules is situated in the lower reticular dermis, whereas the superficial plexus is situated in the upper reticular dermis, both of them parallel the skin surface. From the subpapillary plexus, an arcade of capillaries loops upward into each dermal papilla.