Pathology #6

مجموعة التفريغ السريع

MSS

Subject: *Skin Tumors*

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Date: *Thu, Oct. 10 , 2013*

EHSAN إحسان

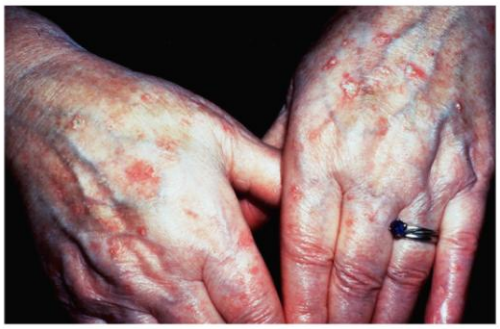
بسم الله الرحمن الرحيم

Actinic Keratosis

- **The other one seborrheic keratosis both are keratosis due to increase in keratin.**
- **Due to excessive, chronic exposure to sunlight so most cases are on the Face & hands of middle aged & elderly**
- **Considered as "premalignant" but not all of then become malegnant ,only some of them do.**
- **Typically seen as hyperkeratotic, scaly plaques**

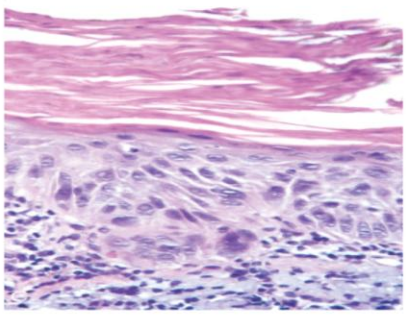
microscopically :

- **Parakeratosis (retintion of the nuclie in the keratin) & atypical keratinocytes (larger nucleus ,**
- **may show some mitosis , some loss of polarity) , may evolve to CA**
- **So in situ be Invasive Squamous Cell CA**



this is the scale patches on the hand , the skin underlying it is a bit atrophic , due to damage from sun , and this is what it shows .

and u can see there are nuclei here hyperkeratosis and parakeratosis



Hyperkeratosis
Parakeratosis

Dysplasia→ CA in Situ

Malignant Epidermal Skin Tumors

1: Basal cell carcinoma

2: Squamous cell carcinoma

BASAL CELL CARCINOMA

- **is the most common MALEGNAT skin tumor allover the world**
- **the majorty is presented with wxposure to sun exposed skin , however u can get it elsewhere.**
- **patients mostly over 40 years old , especially pale skin**
- **its never mucusal because the basal cell aren't available and its never inside the oral cavity for example , u can get squamous cell carcinoma in the moth but not basal ,,,**
- **Sporadic or familial and this is called Gorlin syndrome (in Gorlin syndrome you may have multiple basal cell carcinoma, and you may have other manifestation in the bone , CNS and different tissues)**
- **Infiltrative but NO METASTASES ! (may extends to the facial nerve and produce facial paralysis but NEVER to lymph nodes and only 100 cases have been reported worldwide that they have metastisaized)**
- **Pathogenesis:**

  1- Immunosuppression

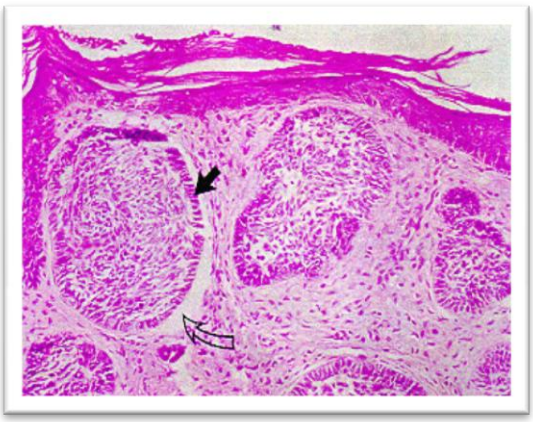
  2- PTCH gene mutation involved in signalling pathway

  3- P53 mutation... (p53 is a cancer supressor so when there is abnormality it wont supress the tumor espicall when u have immuno supression)

The picture :

- **it could be superficial or nodular which is more downward (deeper)**
- **it could be an ulcer that don't heal ,, and u have to visit a Doctor when u have an ulcer which is not healing**
- **it could be very erythematous which is more pink-red , Or could be sclerosing (like a scar) and sometimes its pigmented.**

- **Gross: Papule, rodent ulcer, pigmented lesion**
- **Micro: nests of epithelial cells that resemble basal cells forming palisades separated from surrounding fibroblasts by a cleft like space.**



Now if u look at this , here you have an ulcerating scar-like lesion , this is a raised popular lesion

Squamous Cell Carcinoma

- **Commmon tumor but less common than BCC**
- **Develops in sun-exposed skin of fair patients**

Mucosa may be affected (oral) ... (specially with bad teeth dentuer. case arritate of mucosa and produce ulcers wich cant heal with contenuse prolifration of malegnant cells)

- **Etiology :**
  - **1- Exposure to UVB light & ionizing radiation**
  - **2- Arsenicals & industrial carcinogens**
  - **3- Actinic keratosis**
  - **4- Chronic scarring ulcers, burns…etc**
  - **5- Immunosuppression (HPV 16 & 18)**
  - **6- Xeroderma pigmentosum, P53 & RAS**

Sites :

dorsal surface of hands, face ,ears, mucosal surfaces

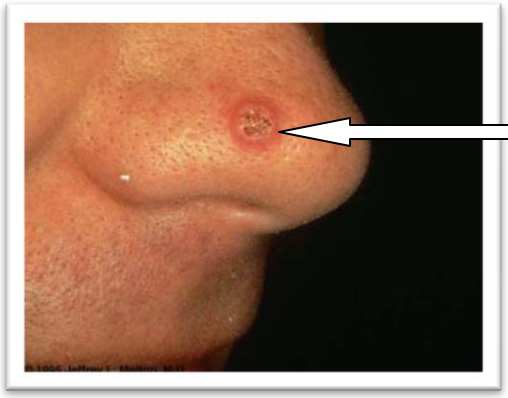
Gross features :

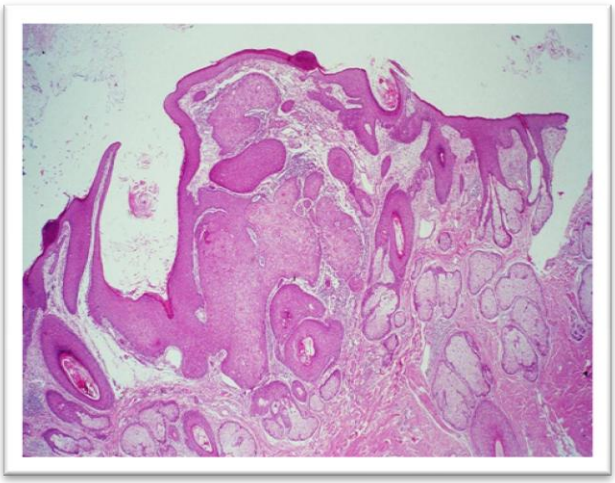
Small scaly lesion → ulceration later

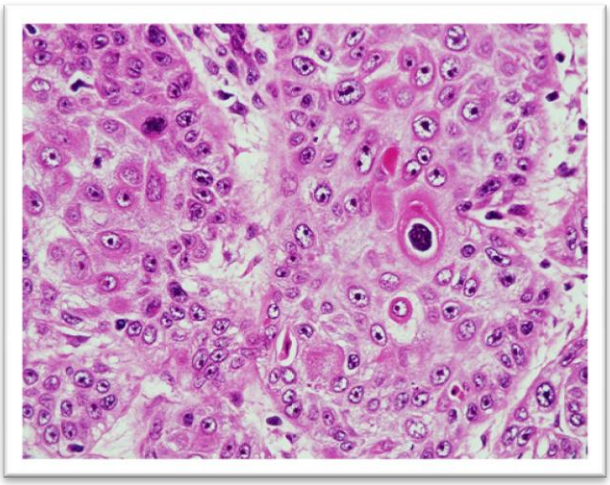
Microscopic :

1- **Full thickness epidermal dysplasia (CA in situ)**
2- **Invasive carcinoma**
3- **Variable degrees of keratinization**

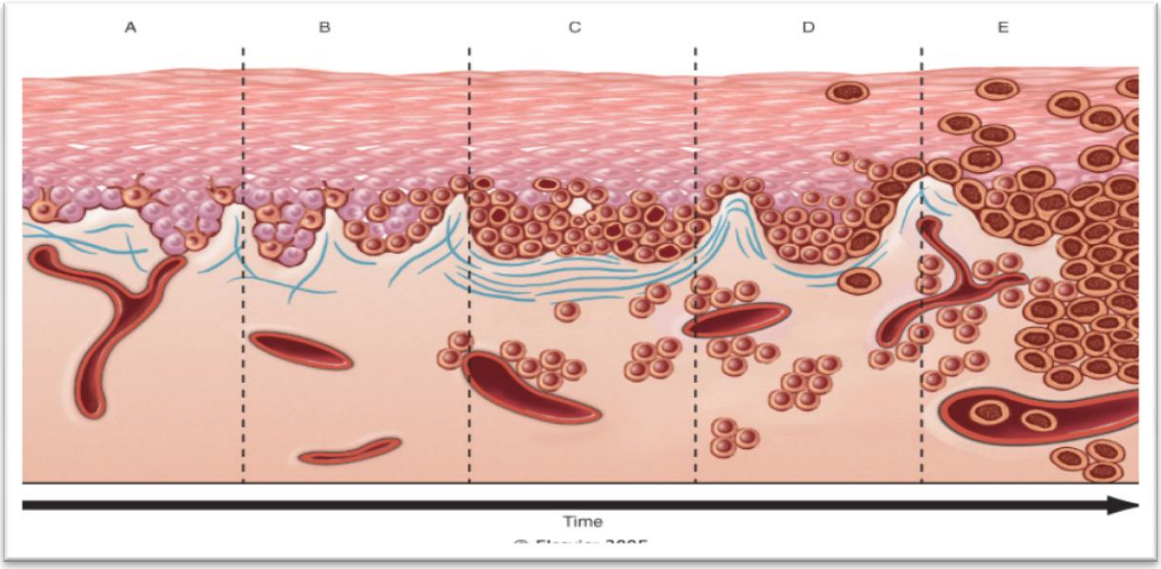
- **It has an increased tendency to infiltrate and metastasize locally to regional lymph nodes**







Melanocytic Tumors of the Skin



Development of melanocytic lesions

in the beginning there's normal skin then some melanocytes more deeply (not normal) later on in the last more and more melanocytes deeply become melanoma.

MELANOCYTIC NEVUS(mole/common nevus).

- **Commonest benign tumor in the body**
- **Derived from dendritic melanocytes present in basal layer of the epidermis**
- **Immature at junction, but mature as cells migrate down into dermis.(** they look like normal cell not dysplastic .)
- **Activating BRAF or RAS mutation identified**
- **There are several locations:**

  Junctional (Dermal epidermal junction), compound (dermal and epi dermal) & intradermal.

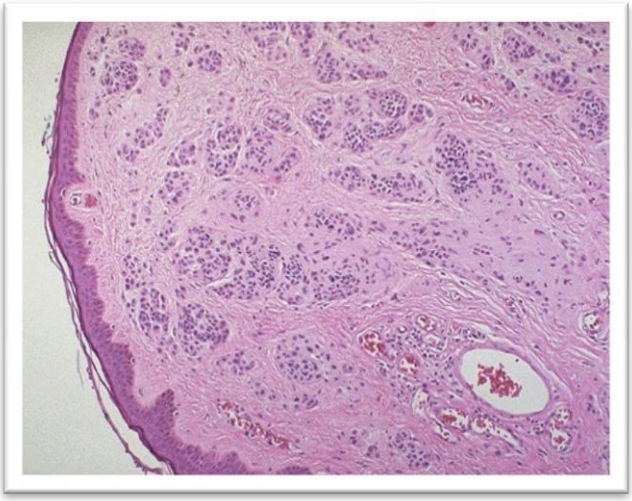
Gross:

Uniform tan/brown color with sharp delineation & tendency to be stable in size& shape.

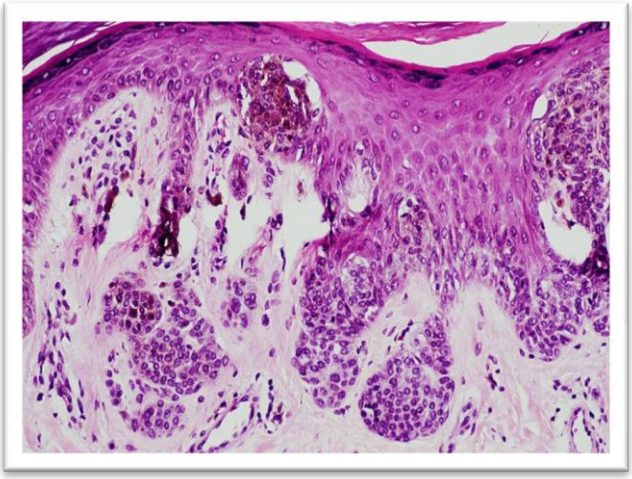
Microscopic picture :

Nests or cords of uniform nevus cells ± pigment

Malignant transformation is uncommon



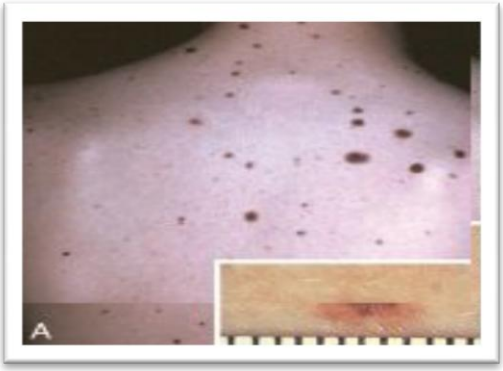
It is look like you can see the nevus cells which are in nests

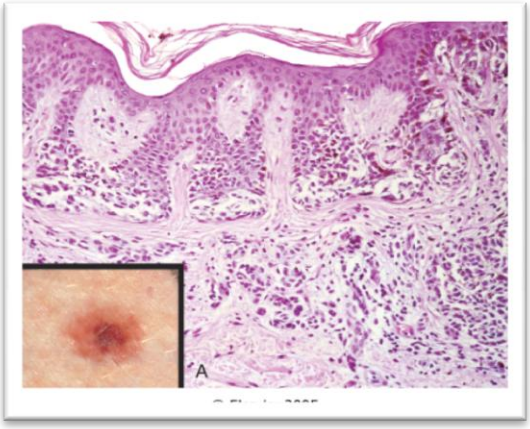


here in this case totally in derma not in epidermis and as you go

Dysplastic Nevus

- Sporadic low risk of transformation
- Familial autosomal dominant with melanoma risk up to 100%
- Considered a marker for future melanoma
- Usually large, multiple & on non-exposed skin
- Activating BRAF or RAS mutations
- Features :Compound nevus with increased melanocytes , cytological atypia, dermal fibrosis around proliferating cells





these cells are not uniform they are large small and irregular… there is some suggestion in packet on island>>>>> so this is dysplastic nevus.

MALIGNANT MELANOMA

- one of the most tumor in the body but recent advise in therapy some therapy is fairly successful and in some cases they have good immune response to the tumor and even have disappearance.
- It occur from light it expose white skin that's why more in **New Zealand & Australia why ? because they abcess O3 layer ok**
- **Intense intermittent exposure at early age**
- Again it can be sporadic or familial and only about 5-10 percent is familial
- **Sites : Skin, mucosa, eye …..etc**

Predisposing factors :

- **Pre-existing lesions : Dysplastic nevus.**
- **Exposure to carcinogens.**
- **Hereditary conditions :**
  - **Xeroderma Pigmentosum.**
  - **Retinoblastoma.**
  - **Familial melanoma.**
- **Many gene mutations (CDKN2A,BRAF,RAS).**
- **Stepwise accumulation.**

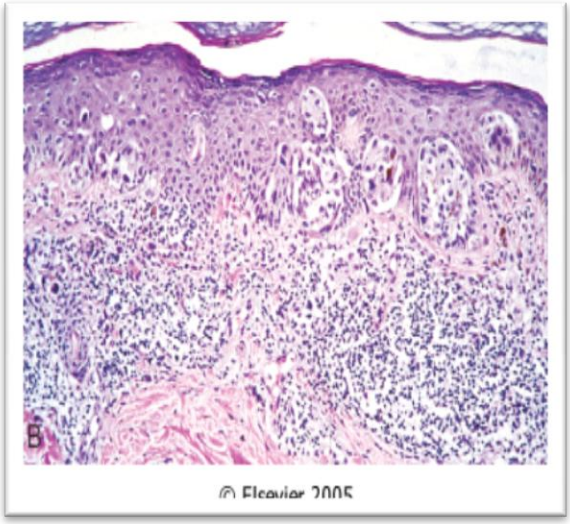
Type of Growth :

- **First Radial (Superficial)**
- **Later downgrowth (Nodular)**
- **Staging & prognosis depends on**

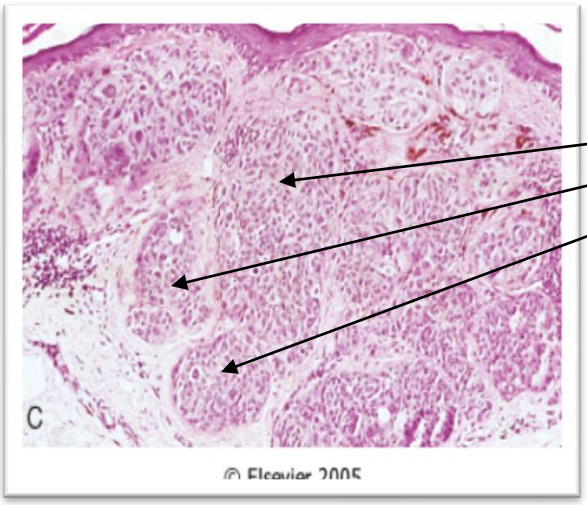
  DEPTH OF INVASION

  - **Breslow Staging: Depth of invasion in mm.**
  - **Clark's Staging: Depth of invasion by location**
- **---microscopic you actually** measure the depth in the Breslow you just measure it by the كانه عندك مسطرة وتقيس كم ملم in the **Clark's Staging by location (in the papillary dermis, deeper dermis 'or fat) --------but one is base on measurment ,one is base on location and --- is the most important corallation with prognosis and with metástasis which is extremely important**

- **Spread is by lymphatics & blood to any site (liver,lung , brain...etc)**



White bridge metastasis this is one of tumor that is the most metastasizing --- you can getting it in the lung , brain ,bone ,breast,--- in the body so it is widely metastasizing it is can spread lymphatic and blood to any site in the body.



nodule

Microscopic features :

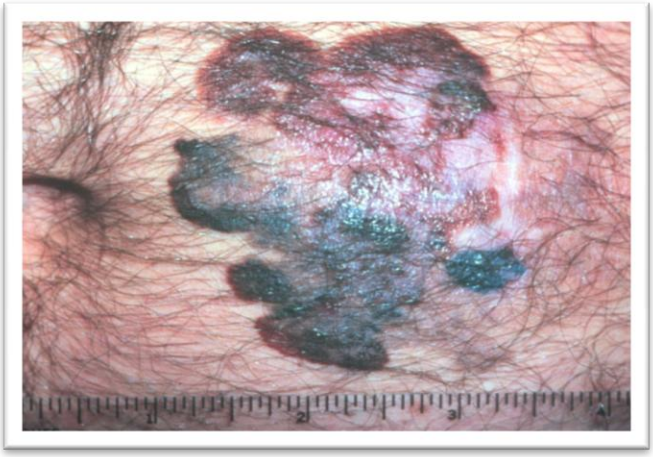
- **Neoplastic melanocytes are much larger**
- **Large nuclei with prominent nucleoli**
- **Tumor cells grow horizontally & vertically**
- **Loss of nesting pattern (sheets)**
- **Loss of maturation (it's very important because in the benign one when we go deeper the cells become smaller , but here all the cells in dysplasia)**
- **Prognosis is better in radial growth but bad in deeper vertical growth**

Clinical Diagnosis :

- **Change in color or size of an existing nevus or lesion as lighter or bigger ...etc it may be : itching, ulceration**
- **New pigmented lesion in an adult**
- **Main signs summarized by :**

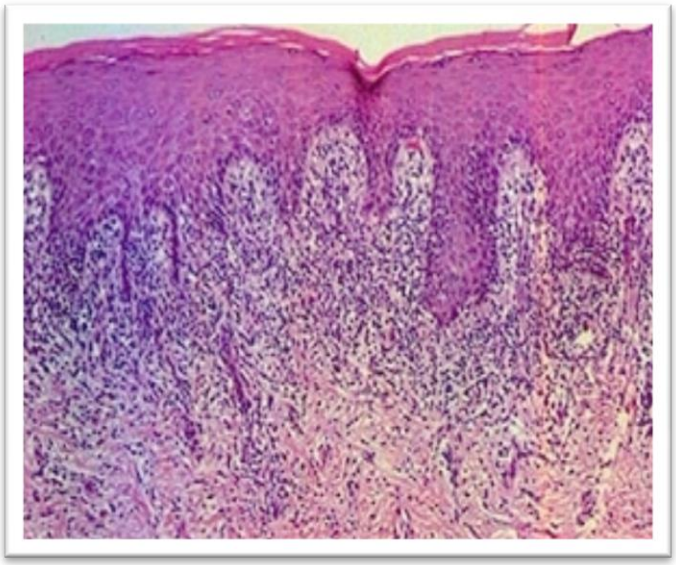
A,B,C,D,E

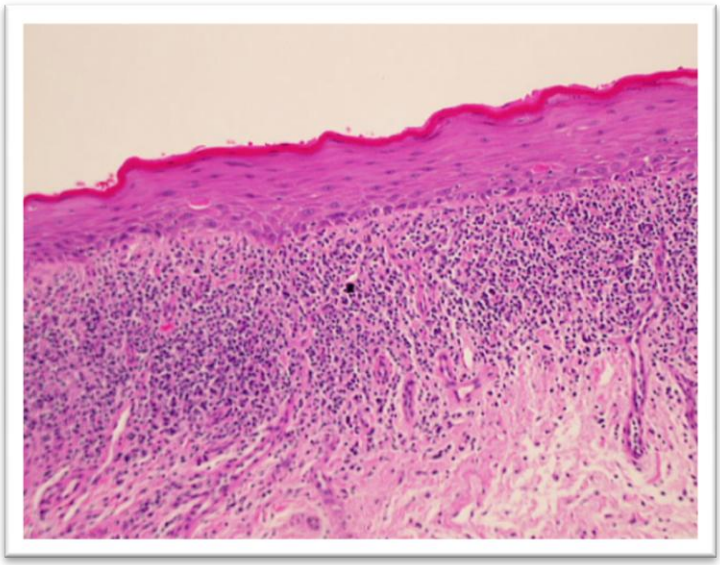
A. Asymmetry of shape
B. Border is irregular
C. Color is uneven
D. Diameter is enlarged
E. Evolution



This is an established melanoma , it's irregular , the colour in it dark and pale .. the centre is pale according to the surroundings which are darker .

At the end the doctor remembered us by the lichen's picture which we were promised to see …





THE END

سامحونا على أي خطأ ورد في هذا العمل ...

إعداد " المحاربون القدامى " :

1 -حسن نايل الرحابنه
2 -زيد إبراهيم نخلة
3 -عمار هايل السرحان
4 -عمار محمد عناقرة
5 -عدي عبد الوحش

وفي النهاية نتوجه بالشكر الجزيل لزميلنا " إسماعيل أبو الشعر" على مجهوده الجبار ونرجو من الجميع الدعاء لوالده بالشفاء العاجل وطول العمر بصحة وهناء