The “Dysplastic” Nevus

Dermatopathology then and now –

Have we travelled?

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SAMs Questions

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Question 1:

In the long-term follow-up of patients with many clinical moles (putative nevi), which is true?

A. All of the moles should be removed to prevent melanoma from occurring.

B. If excised incompletely, the nevi are likely to develop into melanomas.

C. Melanomas will arise almost invariably in conjunction with nevi.

D. The morphological pattern of the nevi will determine if the patient has many moles.

E. They have an increased risk for developing melanoma.

Answer: E

Moley patients, patients with a history of melanoma, and patients with a family history of melanoma are at increased risk for developing melanoma. Melanocytic nevi of any pattern have a very low risk of developing melanoma within their substance, especially given the fact that millions of them occur in nature and only a small number are biopsied. Most melanomas arise *de novo*, unassociated with a melanocytic nevus. One cannot determine, from the pattern of a melanocytic nevus alone, whether a patient is moley or will develop melanoma.

References

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Question 2:

What is the critical point of disagreement between those who advocate in favor of the "dysplastic" melanocytic nevus and those who oppose it?

A. Different patterns of nevi can have zones of fibroplasia (lamellar and concentric).

B. "Dysplastic" nevi are morphologically and biologically intermediate between "common acquired" nevi and melanoma.

C. "Dysplastic" melanocytic nevi (or a host of other terms used for these melanocytic nevi) can occur in moley and non-moley individuals.

D. Melanomas usually arise *de novo*, not in conjunction with melanocytic nevi.

E. Melanomas, when they occur in conjunction with nevi, can occur with *any* pattern of melanocytic nevus.

Answer: B

To this day, there are two principal points of view. One is that "dysplastic" nevi are on a continuum morphologically and biologically, essentially "on their way," to becoming melanomas. The other rejects this and advocates that these "dysplastic" nevi are simply one pattern of many in melanocytic nevi (commonly referred to as lentiginous nevi and Clark's nevi) and are the most common pattern of all melanocytic nevi. All types of melanocytic nevi can harbor zones of fibroplasia; it is a non-specific finding. Most melanomas arise *de novo*; when they occur in conjunction with melanocytic nevi, they are separate and distinct from them, and melanomas can occur with any type of nevus. "Dysplastic" nevi can occur in moley and non-moley people.

References:

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Clark WH Jr, Ackerman AB. An exchange of views regarding the dysplastic nevus controversy. Semin Dermatol 1989 Dec;8(4):229-50. Review. PubMed PMID: 2701712.

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Question 3:

Historically, the concept of the dysplastic nevus syndrome (B-K mole syndrome) arose from observations on:

A. Patient studies of moley family members in the early 20th century.

B. Patients with *de novo* melanoma and no family history of multiple nevi.

C. Patients with few clinical melanocytic nevi.

D. Patients with melanoma with family members harboring melanoma.

E. Patients with well characterized melanocytic nevus patterns.

Answer: D

Clark et. al., in 1978, proposed the concept of B-K mole syndrome based on studies of 37 patients from 6 families prone to develop melanomas. They were the first to attempt to characterize in detail the histopathology of the melanocytic nevi in these patients, although a number of researchers in the latter half of the 20th century commented on moley patients with melanoma and family members with melanoma prior to the article by Clark, et. al. As far as is known currently, the article by Norris, in 1820, is the first documentation of such a family, although histologic techniques for tissue examination were unavailable.

References:

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Norris W. Case of fungoid disease. Edinburgh Med Surg J 1820; 16:562-565