Spontaneous Regression of a Congenital Melanocytic Nevus

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We report a case of a congenital, melanocytic nevus showing spontaneous regression. At birth, a pigmented lesion, 4×6.5cm, was already present on the scalp. At the age of 20 days, the center of the lesion became crusted with a purulent discharge. Following healing of the inflammed lesion, the nevus showed a tendency to regress, starting from the previously inflammed site. At the age of 11 months, the nevus showed marked regression, leaving an atrophic and depigmented center with pea-sized papules and a thin, pigmented rim. Skin sections taken from the central depigmented area showed marked fibrosis and scanty nevus cell nests in the dermis. Histological examination of the pea-sized papule in the center of the lesion revealed large epithelioid cells with abundant cytoplasm dispersed between the collagen fibers; it resembled a spindle cell and epithelioid cell nevus with epithelioid cells predominating. There were no clinical or histologically malignant changes. (Ann Dermatol 1:119–122, 1989)

Key Words: Congenital melanocytic nevus, Spontaneous regression

In general, most nevi appear in childhood, adolescence and early adulthood, and there is a progressive decrease in the number of melanocytic nevi with age.[1] The evolution and regression of melanocytic nevi correlate with their histologic appearance. With advancing age, junctional proliferation of nevus cells decrease whereas the proportion of intradermal nevi increases progressively.[2] The incidence of fibrosis, fatty degeneration, and neuroid changes increases with age.[3] Spontaneous regression has been reported in many cases of malignant melanoma with metastasis.[4-9] Nathanson's review[9] indicated that about 15 percent of primary melanomas regressed at least partially and nearly half of these entirely. However, in cases of congenital melanocytic nevus (CMN), spontaneous regression is a very rare event.[10-12]

The incidence of malignant melanoma developing in giant, congenital, pigmented nevi is approximately 15 percent.[13] Because of their lifetime risk of malignant change, prophylactic excision of CMN is very important. Excision of giant CMN should be considered as early as possible because melanoma may arise even in the first several years of life. But complete surgical excision is sometimes difficult because of extensive involvements with little normal skin available for graft donor sites and because of the risk of general anesthesia in infants and younger children. Thus, it would be most fortuitous that if some inflammatory reaction of the lesion induced artificially or occuring naturally could initiate the regression of CMN.

We report a case of the spontaneous regression of CMN without malignant melanoma following inflammatory changes.

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REPORT OF A CASE

A 20-day-old female infant was first seen at our outpatient clinic on March 8, 1988, because of a inflammed CMN on her scalp. The lesion was a black, 4×6.5cm, irregular, elevated plaque with thick crusts located at the center of the frontoparietal scalp (Fig. 1). She had no clinical evidence of vitiligo, halo or neurologic abnormalities. Fourteen days prior to her first visit, purulent discharge was observed at the center of the nevus by her parents. After systemic antibiotic therapy and wet dressing for 20 days, the lesion healed without scar formation. Since the age of 3 months,

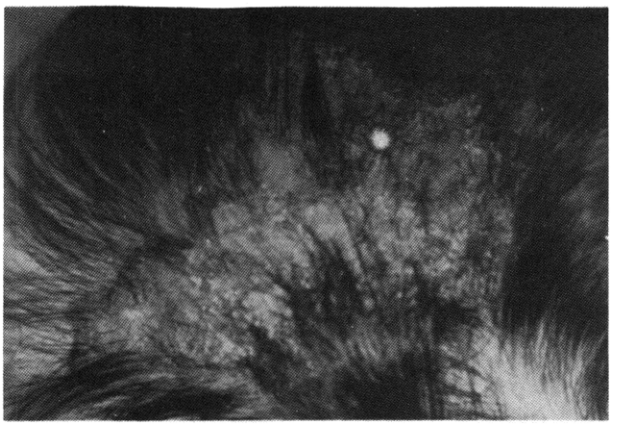

Fig. 1. At 20-days of age: Purulent discharge and thick crusts at the center of the CMN.

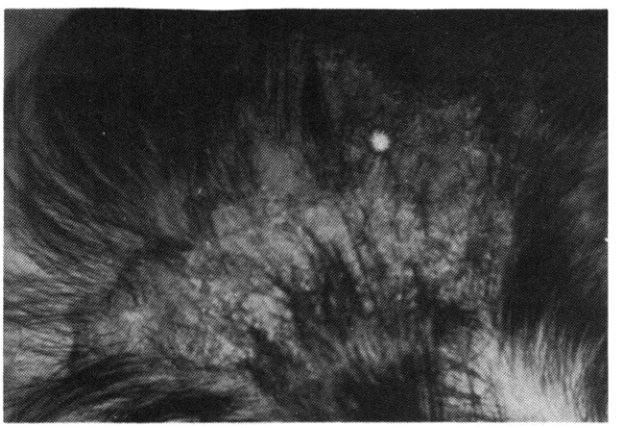

Fig. 2. At 11-months of age: An atrophic and depigmented scar and pea-sized papules centrally and a thin pigmented rim.

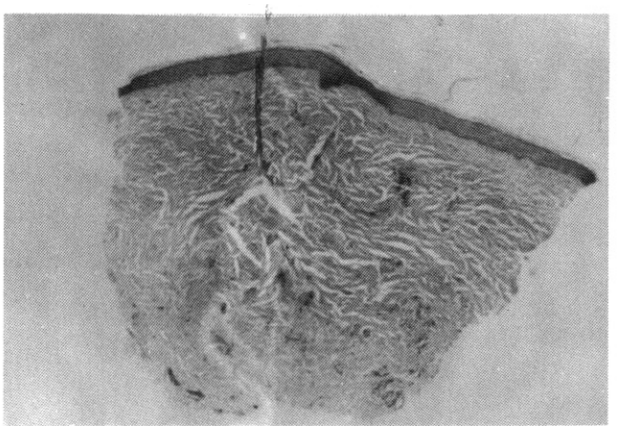

Fig. 3. Central depigmented area: Marked fibrosis and scanty nevus cells in the dermis (H & E stain, ×40).

the pigmented lesion showed a tendency to regress progressively from the site of the inflammatory change. At the age of 11 months, the nevus showed marked regression leaving an atrophic and depigmented scar with a thin pigmented rim (Fig. 2). Some pea-sized firm papules were found in the depigmented area. In monthly follow-up, there was no further regression in the depigmented area after 11 month of age.

Fig. 4. Pea-sized papule: Large cells with abundant cytoplasm and the small nevus cells in the dermis (H & E stain, ×100).

Fig. 5. Pigmented rim: Nevus cells throughout dermis (H & E stain, ×100).

Skin sections taken from the central depigmented area showed marked fibrosis and scanty nevus cell nests in the dermis (Fig. 3). Histologic examination of the pea-sized papule revealed a diffuse infiltration of large epithelioid cells with abundant cytoplasm and small nevus cells in the dermis (Fig. 4). Skin sections taken from the pigmented rim of the lesion showed the extension of the nevus cells into the deep dermis, distributed between collagen bundles singly or in double rows; those were largely in the vicinity of the cutaneous appendages and were without epithelioid cells (Fig. 5).

DISCUSSION

It is extremely rare for a CMN to regress spontaneously,[10-12] and such a spontaneous regression of CMN is different in pattern from the ordinary regression of pigmented nevi.

In general, pigmented nevi show complete regression in the old aged group. But the reported CMNs (summarized in Table 1) showed either complete[10] or incomplete[11, 12] regression during childhood. In our case, the regression was incomplete, but it may regress completely as she grows considering the present age of the patient.

Hasegawa et al [10] described a giant CMN with a malignant melanoma that developed during the spontaneous regression of the CMN. The tumor was excised immediately after its discovery and no recurrence was noted for the next 7 years until the CMN regressed totally.

The site of the CMN's regression was replaced with an atrophic scar in Ridley's case[12] as well as in our case. In other cases,[10, 11] there was no scar formation.

Fourteen days prior to her first visit, there were some signs of infection which included local tenderness, heat, and purulent discharge at the center of CMN in our patient. The infection subsided without scar formation after systemic antibiotic therapy and wet dressings for 20 days. About 50 days after healing, the nevus began to regress beginning at the site of the previous inflammation. In Ridley's case,[12] there were some superficial ulcerations in the center of the CMN at a few days of age. The ulceration healed rapidly with the avoidance of pressure on the lesion. At several months of age, the CMN was found to have become paler. Based upon our own experience and that of others, we postulated that the inflammation might be a triggering factor for the spontaneous regression of a CMN. Malkinson et al[14] made a comment that artificially induced inflammation might contribute to spontaneous regression of CMN. Berger et all[11] postulated that the autoimmune mechanism might contribute to spontaneous regression of CMN. Hasegawa et al [10] observed clearing of the nevus by casting off black crusts and scales from the surface without inflammation and suggested the transepidermal elimination of nevus cell as a contributing factor of

Table 1. Summary of the reported cases of CMN with spontaneous regression

Source	Present case	Hasegawa et al[10]	Ridley[12]	Berger & Voorhees
Site	scalp	trunk	trunk	trunk
Size	medium	giant	giant	giant
Age/Sex	11-month/f	13-year/f	6-year/m	53-year/f
Extent of regression	partial	total	partial	partial
Malignant melamoma	−	+	−	−
Regression site	scarring pea-sized papules	normal in color small neurofibroma -like nodules	scarring mole	N P
Inflammation	+ (local tenderness, warmth and purulent discharge)	−	+ (superficial ulceration)	+
Postulated pathomechanism	inflammation due to infection	transepidermal elimination	inflammation	autoimmune
Histopathologic findings of regression area	thick fibrosis without inflammatory cells	neurofibromatous change	fibrosis with melanophages	epithelioid nevus cells
Histopathologic findings of papule or mole	epithelioid nevus cells	neurofibromatous change	N P	N P

N P: Not Presented

spontaneous regression. Zack et al [15] observed pigmentary regression in a giant nevocellular nevus without the loss of nevus cells and suggested a simple cessation of pigment production by the nevus cells as a contributing factor of spontaneous regression.

The pea-sized papule which developed in the regressed atrophic lesion in our case was suspected clinically to be a malignant melanoma. The histopathologic findings were similar to spindle cell and epithelioid cell nevus with epithelioid cells predominating. The epithelioid cells were large, polygonal, and sharply demarcated. However, definite malignant changes were not found in any of the sections taken from three different sites of the lesion including the papule.

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