

Aureole and Corona - Halo Nevus

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Preface

Halo nevus is described as a pigmented melanocytic nevus with a circumscribing de-pigmented zone or a halo. The disorder also bears the nomenclature of Sutton's nevus or leukoderma acquisitum centrifugum. Halo nevus demonstrates a zone of hypo-pigmentation and cutaneous circumscription and is a frequently enunciated phenomenon of the adolescent population.

Halo nevus frequently depicts a characteristic inflammatory infiltrate with an infrequent erythema or centroidal crusting of the nevus and the variant is termed as inflammatory halo nevus. Non-inflammatory variant of halo nevi is devoid of an accompanying inflammatory infiltrate with a consequent absence of nevus involution. Halo nevus typically displays a pre-existing melanocytic nevus with a characteristic hypo-pigmented zone or a halo which generally dissipates with the resolution of the nevus (1,2).

Clinical Elucidation

Halo nevi present as pruritic, solitary or multiple, painless, black or brown, elliptical cutaneous nodules with superficial scaling and a magnitude varying from 10 millimeter's to 30 millimeters. Commonly implicated sites are the dorsal torso, face, cheeks and sites on the head

and neck. The lesions are firm with indurated, irregular borders and a circumscribing irregular, vascular zone of hypo-pigmentation. Superficial ulceration or a serous discharge from the lesion can be occasionally discerned. Multiple halo nevi can occur simultaneously or sequentially (1,2).

Disease Characteristics

An estimated 1% of adolescents or young population preceding adulthood can enunciate a halo nevus. The condition is devoid of racial or gender predisposition. Halo nevi can be exemplified in an estimated 18% of subjects with Turner syndrome. Halo nevi persist for extended periods and involution of the halo nevus progresses over several months. Zone of depigmentation is devoid of inflammation and although persistent, ultimately disappears. In the event of failure of nevus involution and irrespective of an accompanying inflammatory exudate, re-pigmentation of the halo zone can occur (2,3).

Emergence of multiple haloes in the absence of inflammation and nevus involution is infrequent and such halo nevi can surround nodules of neurofibromas. Subsequent to surgical excision of a malignant melanoma, multiple halo nevi can

ensue. Exceptionally, a halo can circumscribe a congenital nevus, Spitz nevus, blue nevus, malignant melanoma or metastatic malignant melanoma. "Halo nevus phenomenon" or "halo nevus without halo" refers to the presence of a nevus with accompanying characteristic inflammatory infiltrate on histology and an absence of a halo. Halo nevus mandates a classification and categorization contingent to the nevus cell population, as the halo phenomenon with zones of hypo-pigmentation can occur in diverse categories of nevi. Halo nevus could possibly represent an autoimmune reaction against typical or atypical melanocytes and the aforesaid attribute can be contemplated recapitulating the pathogenesis of a malignant melanoma. Thus, a preliminary and efficacious segregation of halo nevus from a malignant melanoma is crucial (2,3).

Histological Elucidation

Morphological enunciation of halo nevus demonstrates a nevus situated in the dermis with accompanying epidermal hyperkeratosis, vesicle formation, spongiosis and a perivascular lymphocytic infiltrate.

Halo nevi can appear as well encapsulated lesions with a focally hyperplastic, encompassing stratified squamous epithelium. Aggregates of spindle shaped, melanocytic or nevus cells with a lightly stained cytoplasm emerge at the dermo-epidermal junction. Minority of nevus cells are incorporated with melanin granules. Dense dermal infiltration of chronic inflammatory cells, predominantly lymphocytes and plasma cells is exemplified (3,4).

Halo nevus is typically described as a zone of non-pigmentation at the margin of a melanocytic nodule or tumour, akin to a vitiligo stain.

Halo nevus depicts the presence of residual melanocytes accompanied with an intense infiltration of lymphocytes and histiocytes which eradicate melanocytes containing the pigment.

Typical histology of halo nevus is

enunciated with a mononuclear inflammatory infiltrate surrounding nevoid cells which progressively undergo disintegration.

Inflammatory variant of a halo nevus in the preliminary phase demonstrates numerous nests and aggregates of nevus cells within the upper dermis or at the dermo-epidermal junction in a compound nevus, accompanied with a dense inflammatory infiltrate. Subsequently, innumerable, singularly scattered nevus cells are cogitated instead of nevus cells nests. Melanin containing nevus cells can exhibit deterioration of the nucleus and cytoplasm. Majority of the inflammatory cells comprising the dermal infiltrate are lymphoid cells in addition to a few melanin containing macrophages (3,4). Invasion of the inflammatory infiltrate occurs into the nevus cell aggregates, thus a categorical distinction amidst lymphoid infiltrate and morphologically identical type B nevus cells crowding the mid dermis can be challenging. Inflammatory infiltrate is clearly demarcated and expands into the inferior stratum of superimposed epidermis. The inflammatory infiltrate is typically dense and devoid of vascular dilatation or intercellular edema. Subsequent stages of halo nevus are devoid of or depict minimal quantities of nevus cells. Eventually, the lesions display an absence of nevus cells and the inflammation subsides (5,6).

Superimposed epidermis of the cutaneous halo demonstrates a decline in the cellular melanin content terminating in a complete absence of melanin, a feature enunciated in inflammatory as well as non-inflammatory variant of halo nevus. Melanocytes partially invade superior layers of superficial epidermis with eventual shedding and an ultimate absence of melanocytes with consequent depigmentation.

Melanin content of the skin covering the nevus persists for an extended period although disappears with nevus involution. Halo nevus lacks the presence of fibrosis, in contrast to lesions of malignant melanoma. Halo nevi can undergo spontaneous retrogression usually on account of cell mediated immunity or infrequently

incriminated humoral immunity or as an emergence of a granulomatous inflammation (5,6).

Figure (1) Halo nevus with dermal dissemination of melanophages, intermingled mononuclear inflammatory infiltrate and epidermal spongiosis (9).

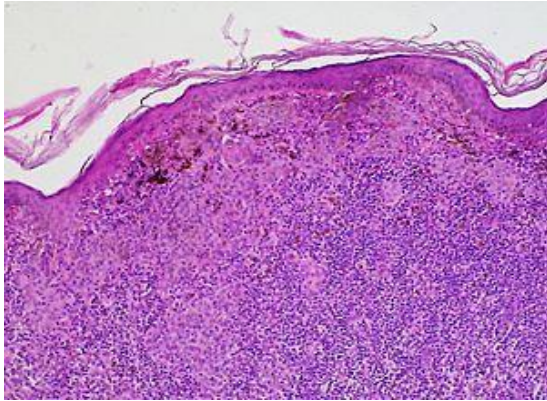


Figure (2) Halo nevus with an intense lymphocytic infiltrate surrounding the nevus cell clusters with epidermal hyperkeratosis (10).

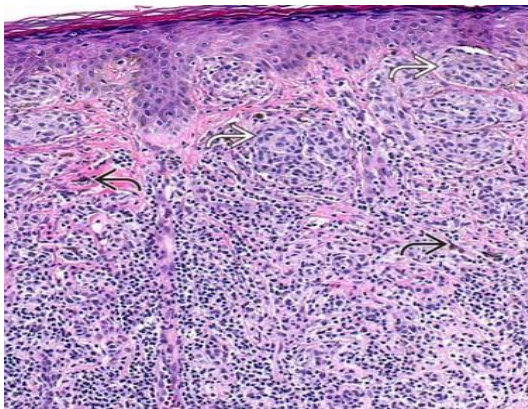


Figure (3) Halo nevus with nevus cell aggregates commingled with lymphocytes and melanophages (11).

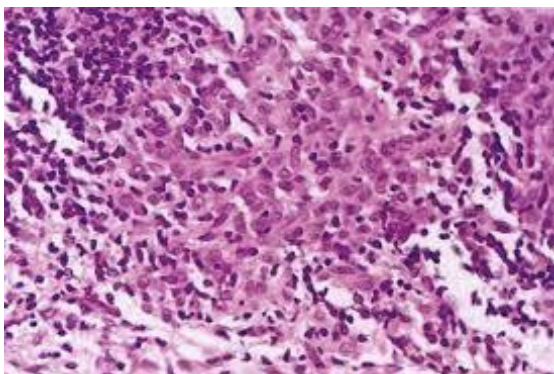


Figure (4) Halo nevus with melanin containing nevus cells, intermingled lymphocytes and macrophages with epidermal acanthosis (12)

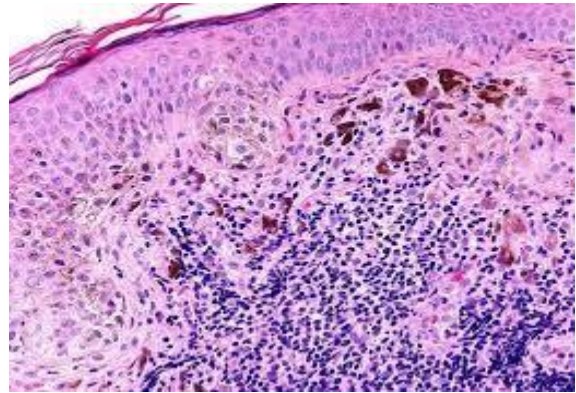


Figure (5) Halo nevus with nevus cell nests and incorporated melanin admixed with lymphocytes and macrophages (12).

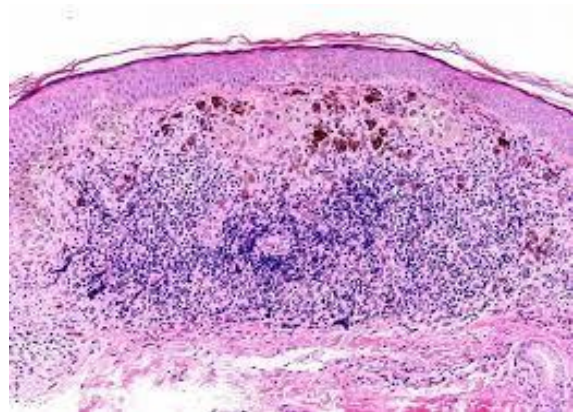


Figure (6) Halo nevus with epidermal acanthosis, hyperkeratosis and spongiosis with intense lymphocytic dissemination interrelated with nevus cell cluster (13).

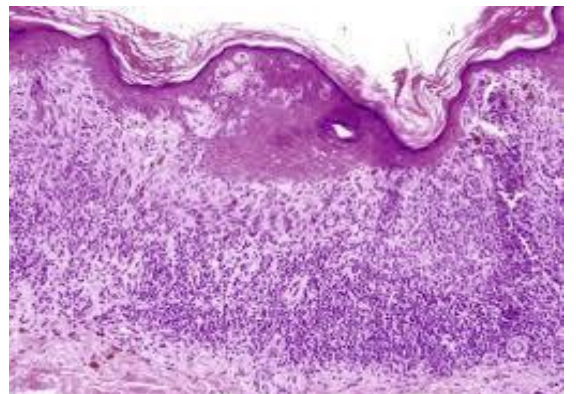


Figure (7) Halo nevus with dense lymphocytic infiltrate abutting aggregates of nevus cells and melanin containing macrophages (13).

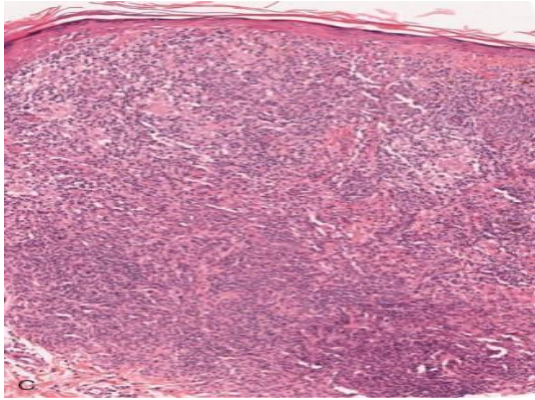


Figure (10) Halo nevus with a dense lymphocytic incursion within nevus cell clusters, few macrophages and migration within superimposed epidermis (16).

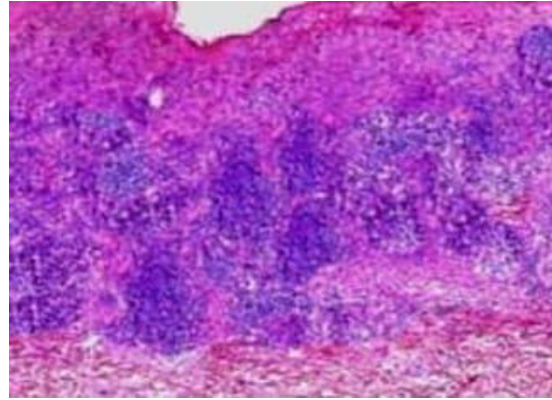


Figure (8) Halo nevus with melanin impacted nevus cell clusters with epidermal migration, commingled lymphocytes and macrophages (14).

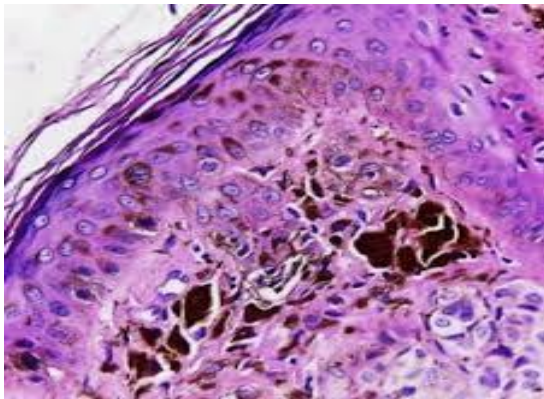


Figure (11) Halo nevus immune reactive to Melan A (17).

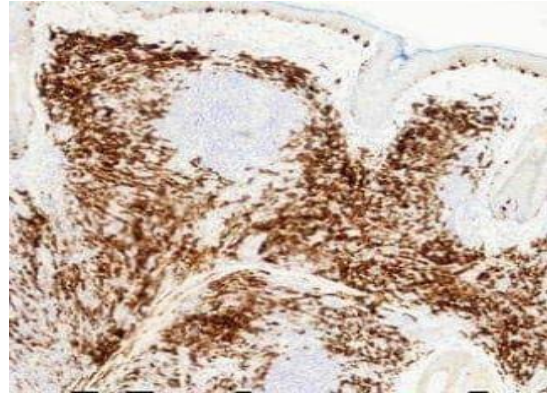


Figure (9) Halo nevus with intense lymphocytic ingress within mid-dermal nevus cell aggregates and upward migration of inflammatory and melanocytic component (15).

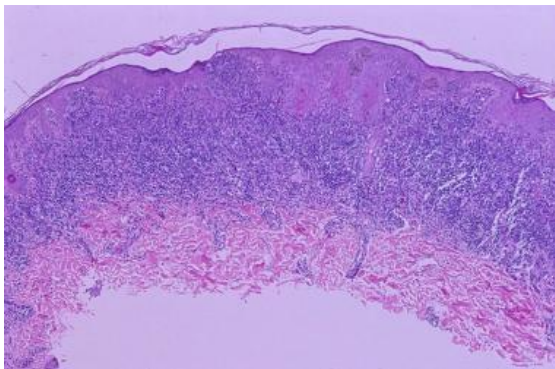
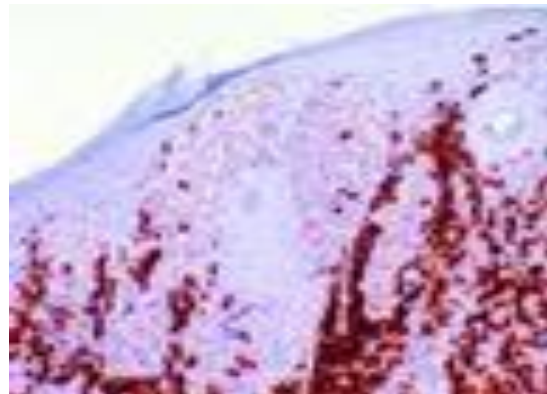


Figure (12) Halo nevus with aggregates of T lymphocytes immune reactive to CD8+ (17).



Immune Histochemical Elucidation

Lymphocytes cogitated in the inflammatory infiltrate of halo nevus are immune reactive to CD20+, thus exemplifying a proportion of B lymphocytes. Additionally, abundant quantities of CD3+ and CD8+ reactive T lymphocytes are delineated. Nevus cells are immune reactive to Melan A. On ultra-structural examination vacuolated nevus cells display miniature and minimal melanosomes whereas enlarged melanosomes appear within macrophages. De-pigmented halo is composed of deteriorating melanocytes with features such as vacuolization, cytoplasmic coagulation and auto-phagocytosis of melanosomes (2,3).

Addenda and Manifesto

Myerson Phenomenon is an infrequent clinical manifestation of obscure aetiology, typically displaying an eczematous halo which circumscribes a pre-existing melanocytic nevus and is associated with several clinical conditions. Therefore, malignant transformation of halo nevi and clinical disorders enunciating Myerson phenomenon require exclusion and distinction(3). Myerson phenomenon is also cogitated as halo dermatitis and is defined by an inflammatory reaction evoked by and circumscribing a preceding melanocytic nevus or adjunctive lesions. Initially scripted by Myerson in 1971, the phenomenon is typically elucidated in the trunk and proximal extremities. The manifestation is discerned predominately in males and young adults. The specified eczematous halo is distinctive, symmetrically encompasses a pruritic preceding nevus with superficial scaling and is cogitated in an estimated two thirds (65%) of instances (3). Multiple nevi can be incriminated with Myerson phenomenon either sequentially or simultaneously. On histological

examination, halo dermatitis demonstrates prominent spongiosis, cellular infiltrate of lymphocytes and eosinophils, irregular acanthosis, Para keratosis and an accompaniment of an unaltered nevus. Myerson phenomenon as a condition of unknown pathogenesis is cogitated as an outcome of allergic contact dermatitis or a hypersensitivity reaction or is contingent to sun exposure or can be contemplated as a specific reaction to administration of certain medications. Halo dermatitis is designated as an immune mediated reaction principally demonstrating an inflammatory infiltrate of CD4+ lymphocytes (3).

Apart from a benign nevus, Myerson phenomenon is enunciated in atypical nevi and can be denominated in non-melanocytic lesions such as seborrheic keratosis, molluscum contagiosum, dermatofibroma, stucco keratosis, lentigo, keloid, insect bites, basal cell carcinoma and squamous cell carcinoma (3).

Differential Diagnosis

Antecedent lesions of inflammatory halo nevus require a segregation from malignant melanoma as the lesions depict a dense inflammatory infiltrate. Nevus cells configuring halo nevi can appear atypical on account of invasion from cellular inflammatory infiltrate.

Misinterpretation betwixt melanoma and halo nevus is maximal in halo nevi without a halo, thereby enunciating a halo nevus phenomenon. Inflammatory infiltrate of inflammatory halo nevi is extensive, exceeds the infiltrate delineated in malignant melanoma and diffusely permeates the lesion, in contrast to inflammatory aggregates situated at the periphery of cellular aggregates of malignant melanoma (3,4).

Halo nevus also depicts melanophages interspersed within the dense inflammatory infiltrate and an absence of melanin within the superimposed epidermis. A distinction from conditions such as chronic dermatitis, cutaneous lymphoma and malignant melanoma is mandated. Myerson phenomenon necessitates a demarcation from malignant melanoma and halo nevus or Sutton's nevus. Halo

nevus commences as a benign nevus with progression towards a zone of depigmentation and subsequent retrogression of the nevus. Appropriate histological delineation can aid the distinction as halo nevus depicts a dense inflammatory infiltrate comprised chiefly of CD8+ lymphocytes. Cogent monitoring is necessitated as benign lesions with an inflammatory component can evolve with malignant transformation (2,4).

Therapeutic Options

Halo nevus can be appropriately managed with simple observation. Additionally, surgical excision of the nevus can be adopted for superior cosmetic outcomes. Surgical extermination of the nevus with a narrow perimeter of up to 2 millimetres of uninvolved tissue is recommended. Comprehensive resolution of eruptive eczema and zone of hypopigmentation ensues. Sequential follow up exhibits a lack of reoccurrence(7,8). Laser therapy can be employed for facial lesions in order to induce adequate repigmentation of the nevus. Dermatitis accompanying a halo nevus can regress spontaneously or may disappear secondary to surgical extermination in the absence of nevus involution. Topical application of potent steroids can eradicate inflammatory dermatitis (7,8).

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16. Image 10 Courtesy: Histopathology.india.net
17. Image 11 and 12 Courtesy: Pathology outlines