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Review Article

Oculodermal melanocytosis: A comprehensive review

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Abstract

Oculodermal melanocytosis or Nevus of Ota refers to the condition of skin melanocytosis observed in relation to the ophthalmic as well as maxillary branches of the fifth cranial nerve. The condition is seen most commonly on the face and manifests itself to be a brownish, bluish, or grayish discolored area, with infrequently involving oral cavity. The present article is a detailed and comprehensive review of the condition and discusses several aspects including the etiology, clinical manifestations, differential diagnosis and management of Ota's nevus.

Keywords: Nevus of ota, Oculodermal melanocytosis, Oro-facial pigmentation, Trigeminal nerve, Unilateral.

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1. Introduction

Oculodermal melanocytosis, more commonly known as nevus of Ota, is a rare inherited hamartoma of dermal melanocytes leading to hyperpigmentation of the eye(s) and adjacent areas.[1] Pusey in 1916 first reported an incident of a student in China demonstrating both scleral and facial pigmentation. However, this melanocytic nevus was later identified as a definitive entity by Ota and Tanino, and was originally described as nevusfuscocaeruleus ophthalmomaxillaris in 1939.[2] The condition for the most part influences the regions that derive their nerve supply by the first (ophthalmic) and second (maxillary) divisions of the fifth cranial nerve, i.e. trigeminal nerve (**Figure 1**).[3]

2. Epidemiology and Prevalence

Nevus of Ota has been reportedly seen to be majorly prevalent in Japan, wherein its incidence is believed to be in the range of 0.2% to 1%.[3] Female predominance is typical, and the usual onset of the condition is either at birth in about 60% of cases or seen soon after birth. However, a few infrequent incidents of the acquired type have also been reported in the literature.[4] This type of melanocytosis has been frequently seen in Asian countries where it represents an average of 0.2–0.8% of the population. However, its incidence in India is relatively uncommon and only a limited number of cases have been reported till date.[5]

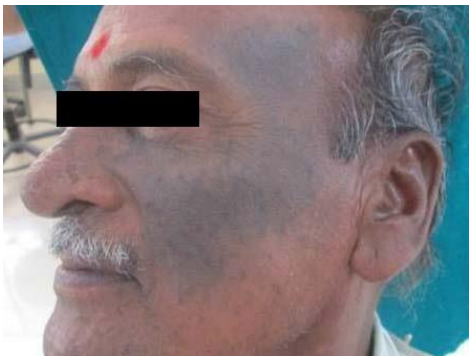

Figure 1: Bluish black hyper pigmented macule on the left mid face

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3. Classification

Many classification systems have been proposed till date for the condition. Tanino (1939) categorized it into four sorts according to the involved skin region as: [6]

1. Type I: Mild, the pigmentation macules area was limited, and it can be further subdivided into four categories:
2. Type Ia – Skin of upper and lower eyelid, orbit, as well as portion of the temporal area is affected by the pigmentation macules; commonly known as eye region type.
3. Type Ib – Skin of the lower eyelid, besides the zygomatic area is affected by macules; also called zygomatic region type.
4. Type Ic – Involves forehead.
5. Type Id – Nostril type.
6. Type II: Moderate, wherein skin of the eyelid, zygomatic area, orbit, temporal region, nasion, cheek, plus nose alae is affected.
7. Type III: Severe, discoloration involves scalp, forehead and auricle in addition to the moderate type are affected.
8. Type IV: Bilateral.

According to Mishima, Ota's nevi were divided into three categories based on the degree and distribution of pigmentation.[7] The classification has been summarized in **Table 1**.

Table 1: Mishima classification

Subtype	Intensity	Pigmentation	Area involved
Type I	Mild	Light brown	Upper and lower eyelids and zygomatic area
Type II	Moderate	Deep slate gray	Eyelids, zygomatic area, and base of nose
Type III	Intensive	Deep blue to brown	Affecting the first and second division of trigeminal neuralgia

Hirayama and Suzuki scrutinized histological verdicts and grouped the condition after studying 450 cases in accordance as per dermal melanocytic distribution as:[8]

1. Superficial (S): Dermal melanocytes positioned superficially in the dermis layer.
2. Deep (De): Melanocytes situated in the deeper dermis layer.
3. Diffuse (Di): Dermal melanocytes spread uniformly all through the dermis layer.
4. Superficial dominant (SD): Higher in number in the superficial layer, but widely spread otherwise.
5. Deep dominant (DD): Widespread dissemination of dermal melanocytes, however in large numbers deeply.

S, SD, Di, DD, and De were present in the ratio of 3:2:3:1:1, and the histologic classification, according to the authors, could be useful for predicting therapeutic outcomes.

Nevus of Ota was reclassified in the year 2001 which allowed for outcome likelihood of laser therapy by Chan HH *et al* into the following types:[9]

1. Nevus lesion lacking periorbital involvement, another birthmark and extra cutaneous association.
2. Ota nevus having only periorbital involvement.
3. Nevus deprived of extra cutaneous connection, but having another birthmark.
4. Extra cutaneous engrossment presents along with nevus of Ota.

In an observational study on 1079 patients, Huang WH et al. found that 213 patients i.e. nearly 20% were not in line with any type of Tanino's classification system. Hence, a different classification was put forward as per structures innervated by divisions of the trigeminal nerve. This system is more commonly known as "Peking Union Medical College Hospital (PUMCH)" classification (**Table 2** and **Figure 2**).[10]

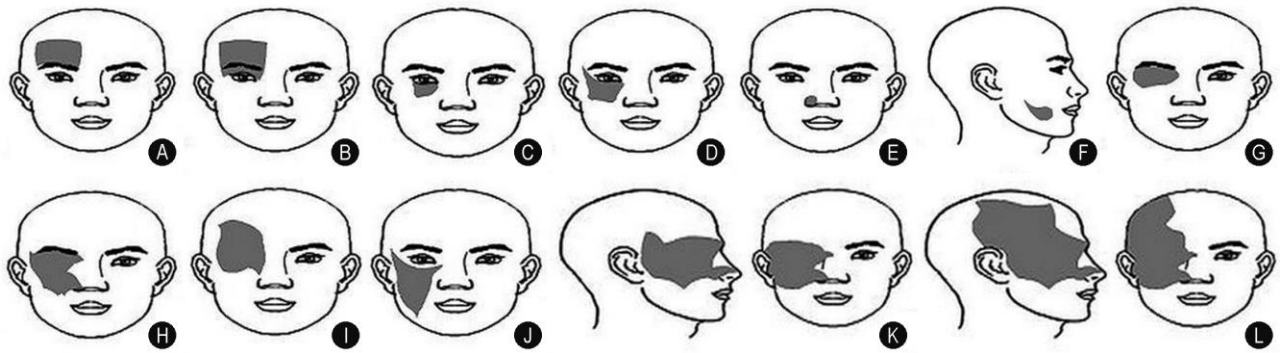

Figure 2: PUMCH classification of nevus of Ota. **A:** Type Ia1; **B:** Type Ia2; **C:** Type Ib1; **D:** Type Ib2; **E:** Type Ib3; **F:** Type Ic; **G:** Type IIa1; **H:** Type IIa2; **I:** Type IIa3; **J:** Type IIb; **K:** Type IIIa; **L:** Type IIIb.

Table 2: PUMCH classification

Type	Sub-type	Area Involved
Type I: Pigmentation macules involving one branch of the trigeminal nerve.	Type Ia: Pigmentation macules involving innervation area of first branch (ophthalmic nerve) of trigeminal nerve.	Type Ia1: Pigmentation macules involving forehead, i.e. innervation area of frontal nerve.
		Type Ia2: pigmentation patch involving forehead and eyelid, i.e. innervation area of frontal nerve and lacrimal nerve.
	Type Ib: Pigmentation macules involving innervation area of second branch (maxillary nerve, V2) of trigeminal nerve.	Type Ib1: Pigmentation macules involving lower eyelid and inferior area of lower eyelid, i.e. innervation area of infraorbital nerve. Type Ib2: Pigmentation macules involving lower eyelid, zygomatic region, temporal region, i.e. innervation area of infraorbital and zygomatic nerves. Type Ib3: Pigmentation macules involving skin of alae of nose, i.e. innervation area of infraorbital nerve.
	Type Ic: Pigmentation macules involving innervation area of third branch (mandibular nerve, V3) of trigeminal nerve.	
Type II: Pigmentation macules involving two branches of the trigeminal nerve.	Type IIa: Pigmentation macules involving the first and second branches (ophthalmic and maxillary nerves; V1 + V2) of the trigeminal nerve.	Type IIa1: Pigmentation macules involve orbit, i.e. innervation area of lacrimal and infraorbital nerves. Type IIa2: Pigmentation macules involving orbit, zygomatic region, temporal region, i.e. innervation area of infraorbital and zygomatic nerves. Type IIa3: Pigmentation macules involving forehead, orbit, zygomatic region, and temporal region, i.e. innervation area of lacrimal, infraorbital and zygomatic nerves.
	Type IIb: Pigmentation macules involving skin of the zygomatic region, temporal region, cheek, anterior auricle, i.e. innervation area (maxillary nerve and mandibular nerve; V2 + V3) of the second and third branch of trigeminal nerve.	
Type III: Pigmentation macules involving the first, second and third branches (ophthalmic, maxillary, and mandibular nerves; V1 + V2 + V3) of the trigeminal nerve.	Type IIIa: Pigmentation macules involving skin of orbit, zygomatic region, temporal region, cheek, anterior auricle, i.e. innervation area of the first, second and third branches (ophthalmic, maxillary, and mandibular nerves; V1 + V2 + V3) of the trigeminal nerve; Type IIIb: Pigmentation macules involving skin of forehead, orbit, zygomatic region, temporal region, cheek, anterior auricle i.e. innervation area of the first, second and third branch (ophthalmic, maxillary, and mandibular nerves; V1 + V2 + V3) of the trigeminal nerve.	
Type IV (bilateral type): Bilateral pigmentation maculeses respectively involve innervation areas of one or more branches of the trigeminal nerve.	Type IVa (symmetric): Pigmentation macules are symmetrical. Type IVb (asymmetric): Pigmentation macules are asymmetrical.	
Type V: Nevus of Ota combined complications.		

4. Etiology

The precise causative factor of the condition is under investigation not well understood till date. Nevertheless, a common perception is that Ota's nevus is seen due to persistent melanocytes who failed to relocate to the epidermis commencing from neural crest during development of the embryo. Also, hormones are believed to have a significant contribution in the development of nevus lesions owing to the dual pinnacle periods of onset in early infancy and adolescence.[3] It has also been observed that glycosaminoglycans filling the cell free area during the initial embryonic development, have a significant part in migration of neural crest cells. It has also been postulated that the migration process might be affected by alterations in the glycosaminoglycan concentration, resulting in dermal melanosis.[11] Additionally, infection, trauma, or exposure to ultraviolet light elicit nevus inception. Clinically visible nevus lesion is said to develop when these factors stimulate melanin production from amelanotic melanocytes.[12]

Once onset is over, nevus pigmentation enlarges and possesses a dark hue. Lesion appearance generally remains stable till adulthood. Personal as well as environmental factors like fatigue, menstruation, insomnia, besides change in the weather conditions also influence the color and perception of nevus of Ota. [8]

Nevus of Ota and nevus of Ito, that refers to a skin condition affecting the shoulder, often coexist. It is also linked to certain ocular and cutaneous conditions. Benign dermatological and leptomeningeal disorders allied with nevus of Ota include phakomatosis pigmentovasculari, nevus flammeus, Sturge-Weber syndrome, Takayasu disease, Klippel-Trenaunay syndrome, and neurofibromatosis.[13]

5. Clinical Features

Nevus of Ota occurs most commonly on the face and clinically appears as a brown, blue, or gray patch, which can either be inherited or acquired and lies within distribution of the trigeminal nerve's ophthalmic and maxillary divisions (**Figure 3**).[12] The condition is classically unilateral (90-95%), however, bilateral involvement has also been reported.[14] It usually does not present any symptoms but uncommon instances involving partial loss of sensation have been accounted for.[15] Involvement of extra cutaneous locations i.e. eyelids, along with adjoining skin regions, sclera, plus conjunctiva, have additionally been observed.4 In Ota nevi, involvement of the palatal mucosa is infrequent. Palatal pigmentation characteristically appears as an asymmetrical, vague, and frequently mottled area that amalgamates with the oral mucosa.[5]

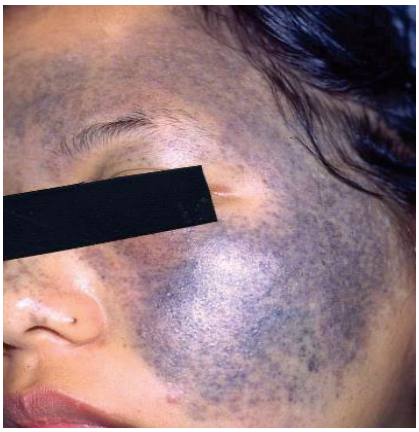

Figure 3: Presentation of Nevus of Ota

5.1. Ocular manifestations

Iris heterochromia is the most common presenting ocular change associated with this condition.[16] Iris mamillations (also known as iris nodules) are also present and may be linked with iris hyperpigmentation. These refer to dome-shaped protuberances on surface of the iris and comprise of clusters of plump, nevoid cells having mild pigmentation, intertwined with matured, thickly pigmented spindle-shaped uveal melanocytes.[17] An elevated iris mass should raise suspicion of iris melanoma, particularly if it exhibits characteristics for instance increased intraocular density, iris seeding, invasion of ciliary bodies, extraocular extension, or growth.[18] Other complications that are relatively infrequent are uveal, conjunctival, and cutaneous melanomas. However, there is an increased possibility of metastasis when uveal melanoma is present in an individual with nevus of Ota.[19]

The most frequent ocular manifestation in this condition is the collection of melanocytic cells at scleral and episcleral levels, and is particularly seen in the lower medial and superior temporal regions. Other tissues that might be unintentionally impacted as a result of pigment infiltration comprise the conjunctiva, cornea, and anterior surface of the crystalline lens. (**Figure 4**).[20]

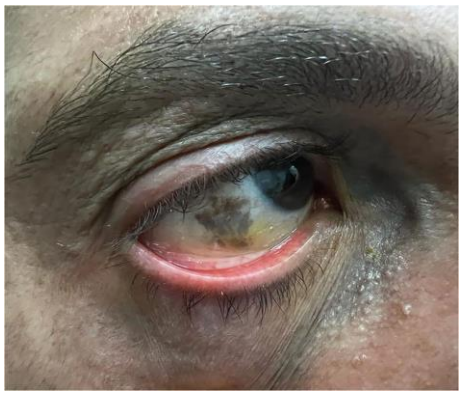

Figure 4: Periocular gray-blue pigmentation in the sclera

Due to pigment accretion in the trabecular meshwork and Schlemm's canal or abnormalities in the irido-corneal angle, open-angle glaucoma may develop thus leading to hindrance

of the aqueous humour outflow. Ten-percent of ODM patients develop glaucoma, which is typically ipsilateral to the pigmentation and regular follow-up is highly recommended in such cases. Glaucoma can likewise occur in instances wherein ODM is connected by means of further neuro-oculocutaneous illnesses, like Sturge-Weber & Klippel-Trenáunay syndrome, and phakomatosis pigmentovascularis.[20]

5.2. Dermoscopic features

Elmas F. and Kilitçi A. defined dermoscopic verdicts of brown and gray structure less areas having a patchy distribution and dispersed brown-gray dots, as occurring in Ota lesions on the forehead.[21] In the investigation of Zinoune et al, dermoscopic elements of an instance reported with Ota nevus was portrayed to be somewhat blue to slate gray uniform pigmentation.[22] Exogenous ochronosis can also manifest as nevus with a clinical features similar to that of an Ota with scattered grayish-brown pigments. A similar observation was seen when dermoscopic elements of exogenous ochronosis were accounted for as “dark brown balls, lengthened and curvilinear-worm like creatures.[23] Although orbital and cerebral melanomas are thought to be the most fatal complications, nevus of Ota has rarely been associated with them.[12]

6. **Differential Diagnosis**

Nevus of Ota lacks any conclusive diagnosis. In instances where clinical variations in the skin, ocular/mucosal tissues involved suggest malignant transformation, skin biopsies maybe necessary.[3]

Mongolian spot, melasma, blue nevus, and drug-induced hyperpigmentation are among the clinical differential diagnoses for Ota skin lesions. The mongolian spot is characterized by large, poorly defined blue-to-grey patches that typically disappear on their own between the ages of 3 and 6 and are more common in the lumbosacral region than the face. Melasma is clinically seen to have a well-to-poorly defined and unevenly outlined brown to gray brown spots and is characteristically associated with pregnancy. Melasma usually is bilateral without any palatal involvement.[4]

Additionally, it is possible for an oral melanotic macule to be mistakenly diagnosed as an Ota nevus as it is likewise to be present in the palate, yet differentiated from nevus of Ota as it is lesser in dimension and lacks scleral association. Also, it can happen lone or else be associated with Peutz-Jeghers disorder, Panther disorder, and Laugier-Hanziker syndrome.[4]

The most common etiologic factor causing drug-induced hyperpigmentation is the consumption of medications namely minocycline, amiodarone, or gold. Nevus of Ota can be distinguished from blue nevus as the latter has a plaque or papule like appearance and can be seen anywhere on skin, whereas nevus of Ota possesses a macular presentation. Acquired bilateral Ota alike macules (ABNOM) or Sun's nevus do not involve the sclera or oral mucosa.[4]

First described by Minor Ota in 1954, Nevus of Ito is another condition that could mimic Ota nevus. These two illnesses have comparable pathophysiology, however, their distribution territories are different. In nevus of Ito, ocular problems are not present.[24] Its anatomical location matches the branching of the cutaneous brachial and posterior supraclavicular nerves.[25]

7. **Treatment**

The condition has been managed using a variety of treatment options which include:

7.1. Camouflage

Camouflage has been used for centuries to hide facial flaws. Materials used as camouflage are made to adhere to slick, nonabsorbent scar tissue, are waterproof, and opaque. Color changes can be fixed with camouflage therapy. However, as skin irregularities tend to be exaggerated when scars are camouflaged, the outcome is dependent on the texture of the scar.[12]

7.2. Cryotherapy and dermabrasion

By destroying dermal melanocytes and suppressing the function of epidermal melanocytes, cryotherapy lightened Ota's nevus. Dermal melanocytes underwent direct cryonecrosis and activated lysosomal enzymes led to further damage.[12]

The removal of epidermal and superficial dermal melanin by dermabrasion may improve clinical appearance.[12] In one study, dermabrasion and carbon dioxide snow cryotherapy were combined to produce good clinical outcomes in 22 of 24 patients.[26] However, both these techniques have resulted in scarring and are no longer used.

7.3. Laser therapy

Advancements in the Q-Switched Nd:YAG laser (QSYL) and Q-switched ruby laser (QSRL) has made it possible to achieve comprehensive and scar less elimination of pigmentation.[3] The Q-switched laser therapy is built on Anderson and Parish’s hypothesis of selective photothermolysis. The laser light should possess a frequency that is absorbed by the objective chromophore and not the encompassing structures, in order to confine the heat to the target and prevent collateral thermal damage, the pulse duration ought to be not exactly or equivalent to the target's thermal relaxation time; and finally, adequate fluencies ought to be utilized to deliver the ideal outcome. As a result, shock waves are produced ultimately leading to target explosion.[27]

An examination of 114 Ota patients whose nevi lesions were treated with the QS ruby laser demonstrated its clinical viability. A good to excellent degree of lightening was

attained with insignificant side effects following three or more sessions.[28]

Nevertheless, there are growing concerns about the reappearance of Ota nevi. A 26-year-old male patient suffering from the condition presented to Lee HS et al nine years post a successful laser treatment.[29] It has been demonstrated that multipotent dermal stem cells (DSCs) can develop into functional melanocytes, which may be the etiologic factor contributing to pigmentary disorders.[30] Nestin, octamer-binding transcription factor 4 (OCT4), and nerve growth factor receptor (NGFRp75) markers were present in these DSCs but not in melanocytes, indicating that they came from the neural crest.[31]

7.4. Leech therapy

A new treatment modality was adopted by Rastogi S and Chaudhari P who in their study used leech therapy for management of nevus of Ota. Leeches were applied on the site according to the standard technique for application depicted in Ayurveda. The nevus lesion's hyperpigmentation significantly decreased after five consecutive two-month sessions of local leech therapy.[32] However, more clinical studies are needed to completely validate this modality in managing Ota nevus.

7.5. Surgery

Surgical modality is usually indicated in cases of ocular manifestations involving the sclera. A few strategies have been utilized to diminish these lesions, which include superficial sclrectomy,[33] flipped scleral flap,[34] sclera allograft[35] and grabbing method.[36]

In the preoperative planning of a patient with an Ota nevus, Mularoni A et al. used anterior segment optical coherence tomography (AS OCT). To ascertain the size of the sclerectomy, AS OCT pictures and an estimation of the profundity of scleral melanocytosis were utilized to quantify the preoperative depth of scleral inclusion. This made it possible to determine the amount of scleral tissue that needed to be removed in order to get rid of the pigmented area without harming the sclera. During the follow-up, AS OCT images revealed scleral tissue thinning, decreased pigmentation, and progressive healing.[37]

8. Dental Considerations

Pigmented lesions are frequently encountered by dentists. Dental professionals should explain the condition to the patient and be ready to spot any unusual change in size, variety, or morphology. Due to the fact that patients will initially visit a dermatologist for facial deformation, a superior relationship between dental specialists and dermatologists is anticipated. Since the patient will be alluded to the dental specialist for an intraoral assessment, it is essential for a dental specialist to know the differential diagnosis of the oral manifestations of Ota nevus. Because there will be no regular follow-up, a lack of mindfulness can result in a misdiagnosis and, as a result, an obscure risk of malignancy.[4]

9. Conclusion

Typically affecting females, nevus of Ota is a unilateral dermal melanocytic hamartoma. In order to avoid misdiagnosis and, as a result, the risk of malignant transformation, clinical professionals ought to have comprehensive knowledge of the condition and its differential diagnosis. Patients with nevus of Ota should have a multidisciplinary screening done by a dermatologist, an ophthalmologist, and a dentist.

10. Source of Funding

None.

11. Conflict of Interest

None.

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