REVIEW ARTICLE

Laugier–Hunziker syndrome

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ABSTRACT
Laugier–Hunziker syndrome is a rare acquired disorder characterized by diffuse hyperpigmentation of the oral mucosa and longitudinal melanonychia in adults. They appear as macular lesions less than 5 mm in diameter. Laugier–Hunziker syndrome is considered to be a benign disease with no systemic manifestation or malignant potential. Therefore, it is important to rule out other mucocutaneous pigmentary disorders that do require medical management. Prompt clinical recognition also averts the need for excessive and invasive procedures and treatments. In India, the reported cases of this syndrome are very few. We provide a review of literature on Laugier–Hunziker syndrome with its differential diagnosis.

Key words: Differential diagnosis, Laugier–Hunziker syndrome, mucocutaneous pigmentation, oral pigments

INTRODUCTION

Oral mucosal pigmentation may be physiologic or a sign of localized or systemic pathologic condition. A practical approach in a clinical situation is to examine whether the pigmentation presents as focal or as diffuse lesions. Sometimes the clinical behavior of focal oral pigmentation together with a thorough medical and family history, as well as history of onset, duration, and progression of the pigmentation, guides us to make a suggestive diagnosis. The clinical behavior of focal oral pigmented lesions ranges from benign, requiring no treatment, to highly malignant. Therefore, biopsy is usually required for accurate diagnosis of a focal pigmented lesion. Oral pigmentation caused by systemic diseases is usually diffuse and multifocal and has no specific histologic features. Although a solitary pigmented lesion may cause more suspicion, it should be kept in mind that diffuse oral pigmentation may be the first manifestation of an underlying systemic disease.[1] One such pigmented lesion which is of interest to the dentist is Laugier–Hunziker syndrome (LHS). It was first described in 1970 by Laugier and Hunziker who reported a series of five patients with acquired macular hyperpigmentation of the lips and oral mucosa, with hyperpigmentation of the fingernails in two of the patients.[1-4] Since then, the condition has also been detected in other areas with similar histology and the term "idiopathic lenticular mucocutaneous pigmentation" has been used.[4]

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LHS occurs predominantly among middle-aged adults with a mean onset at 50 years of age and occurrence is usually seen after puberty. It is more prevalent in women and most reported cases have been in Whites, particularly in French and Italians.[4,5] Reported cases have varied between second and ninth decades of life.[4-9] We made an attempt to review LHS which is less popular in the field of dental health professionals. A MEDLINE/PubMed search of the literature on LHS published in the last four decades has been made based on the key word Laugier–Hunziker syndrome, idiopathic lenticular mucocutaneous pigmentation. The articles pertaining to case reports, differential diagnosis, and treatment modalities in english language literature were evaluated and studied. The articles in different languages which provided english language abstract were also reviewed. Our search yielded 172 case reports published till date. Details pertaining to these case reports are methodically tabulated [Table 1].

CLINICAL FEATURES

LHS is considered as an acquired benign macular hyperpigmentation disorder with unknown (or definite) etiopathogenesis and is believed to have no associated somatic abnormalities. The pigmentary lesions carry no risk of malignant transformation. Cases have been reported in related family members. The condition has been variably reported as sporadic and inherited in an autosomal dominant fashion. Others have suggested that no genetic factors are associated with the syndrome.[4-9,30]

The pathogenesis is thought to be linked to a functional alteration of the melanocytes that induces increased synthesis of melanosomes and subsequent transport to the basal cell layers. The etiology of the same is unknown.[1]

LHS is characterized by a varying number of asymptomatic, lenticular (lens-shaped), or linear, brown to black mucocutaneous macules, usually less than 5 mm in diameter. They may be single or confluent. They may have well-defined or indistinct margins. The hyperpigmentation occurs spontaneously and gradually and it is considered permanent.[1,4] The lesions are most commonly located on the lips, buccal mucosa, and hard palate. The less frequently affected areas include the soft palate, tongue, gingiva and floor of the mouth. The oral pigmentation usually persists, whereas the cutaneous lesions often fade after puberty.[11]

The nails are affected in about 50–60% of the cases, and usually presents as single or double stripes or as homogeneous pigmentation on one-half of the nail or complete nail (melanonychia).[1,4,30] One or more of the fingernails and/or less often toenails are involved**,** which appear as a band without nail dystrophy. The cause of these pigmentary stripes in LHS is unknown, but is supposed to be similar to the involvement of the oral cavity.[10,28]

The pigmentation may spread from the proximal nail fold into the surrounding skin, which is known as Hutchinson's sign or pseudo-Hutchinson's sign. Hutchinson's sign is always present in the case of subungual melanomas; other than LHS, it is also seen in Peutz–Jeghers syndrome (PJS), subungual hematoma, racial pigmentation, and AIDS, in association with the use of certain drugs such as minocycline and zidovudine, and in Bowen's disease.[1,4,30] The other affected areas are hands, fingers, feet, neck, abdomen, thorax, genital and perioral area, vulva, conjunctiva, and in the region of the eyebrows.[1,4,5] The syndrome appears more common than the number of reported cases.[10]

HISTOLOGY

The mucosal macules of LHS have shown epithelial acanthosis with the pigmentation being localized to the basal layer of the epithelium. The pigmentation is thought to be due to accumulation of melanin in the basal keratinocytes.[1,5,30] Most reports have suggested that melanocytes are normal in number, morphology, and distribution, with three reports describing increased non-nested intra-epidermal melanocytes.[1,30] Epidermal and epithelial basal layer pigmentation is common in skin and mucosal lesions, respectively, with pigment-laden melanophages evident in the papillary dermis. Acanthosis of the epidermis is emphasized in several cases, saw tooth or elongated rete ridges are noted in few cases, whereas normal rete ridges are also described in some reports.[6] Reports have also noted that ultrastructurally the melanocytes appear larger than normal without increase in numbers.[2,30] Similarly even melanosomes are increased that vary in size and structure. Melanophages in the papillary dermis are also increased.[1,30]

DERMATOSCOPIC FEATURES

Dermoscopy is a noninvasive technique that has been used to make more accurate diagnoses of pigmented skin lesions. Ronger *et al.* were the first to describe the unique patterns for differentiation from nail melanoma in 12 patients with LHS, namely, thin longitudinal gray lines, homogeneous in thickness, color, and spacing, against a grayish background. However, medication-induced melanonychia, such as that induced by minocycline, zidovudine, and hydroxyurea, or ethnic-type melanonychia may have similar dermoscopic patterns.[31] Simionescu *et al.* reported one case of mucosal melanoma on the upper lip, associated with LHS. Pigmented nodular lesions have a blue-white veil with a globular pattern and unusual vasculature, which indicates the presence of a mucosal melanoma.[25] Tamiya *et al*. observed numerous brownish and gray-blue granules on a whitish-pink area with scattered linear and dotted vasculature on the lower lip, a parallel ridge pattern on the ventral aspect of the thumb, and thin and thick grayish longitudinal lines and bands on the nail plate.[31] Gencoglan *et al.* have reported parallel patterns on the patients' lips and vulva. On the lips, linear, streak-like brown pigmentation caused by the skin furrows and reliefs was associated with multiple brown dots of different sizes distributed regularly throughout the lesion. The parallel pattern seen on the vulva was partially linear and partially curvilinear, with light- to dark-brown streaks following the cutaneous profile. Homogeneous, brownish, regular band-like pigmentations with indistinct borders were seen on four toenails. The pigmented macules on the palms and on the sole showed a parallel furrow pattern.[23]

DIFFERENTIAL DIAGNOSIS

LHS is a diagnosis of exclusion. Solitary and multiple macules of the oral mucosa are associated with various causes. Solitary macules of oral mucosa are less common. Multiple labial macules are more likely to have association with a variety of syndromes.[1,33] Dentist, as a health care professional, should carefully rule out all the differential diagnoses associated with hyperpigmentation before diagnosing LHS [Table 2].

Various differential diagnoses considered for LHS can be ruled out by the appearance and the etiology for hyperpigmentation. Drug-induced pigmentation will usually occur after months or years of chronic use of drugs and tends to resolve once the drug is discontinued. AIDS patients show hyperpigmentation secondary to the drugs taken [Table 2]. Smoking could result in oral mucosal pigmentation called smoker's melanosis and it is predominantly seen in anterior gingiva. In addition, smoking is not associated with pigmentation of the nails.

Table 1: LHS: Review of cases reported in the literature

Authors	Year	No. of cases	Mean age	Sex	Clinical findings	Atypical features
Laugier and Hunziker[10]	1970	5	60	2M/3F	Oral mucosa, lips, nails	*
Pellerat *et al*.[10]	1971	1	87	M	Oral mucosa, lips	*
Sartoris *et al*.[10]	1975	2	45	2M	Nails, lips	Spots on distal groove, cutis
Lougier *et al*.[10]	1977	2	53.5	2M	Oral mucosa, nails	*
Baran[10]	1979	9	42.5	3M/6F	Oral mucosa, lips nails	*
Bundino and Zima[10]	1979	1	45	F	Lips	*
Bertazzoni *et al*.[10]	1984	1	67	F	Lips, oral mucosa	*
Bisighini and Davalli[10]	1985	3	56	3F	Oral mucosa, lips	*
Beurey and Weber[10]	1986	1		F	*	Vulva
Baran and Barriere[3]	1986	2	62	M/F	Oral mucosa, nails	Spots on distal groove in one case
Kock *et al*.[10]	1987	1	32	F	Nails, lips	*
Offidani *et al*.[10]	1987	1	51	F	Oral mucosa, lips	Commissures
Sterling *et al*.[10]	1988	1	67	F	Nails, lips, oral mucosa	*
Borrello *et al*.[10]	1988	1	41	M	Nails, lips	Spots on distal groove
Dal Tio *et al*.[10]	1989	1	66	F	Lips, oral mucosa	*
Revuz and Clerici[10]	1989	5	26.5	M	*	Penis
Patrone *et al*.[10]	1989	3	46	1M/2F	Lips, oral mucosa	*
Satriano[10]	1989	1	50	F	Oral mucosa, lips	*
Golovinov[10]	1990	1	60	M	Oral mucosa	*
Kemmet *et al*.[10]	1990	6	53	1M/5F	Nails, lips, oral mucosa	Lupus erythematosus and psoriasis in one case
Dupre and Viraben[10]	1990	20			Oral mucosa	Penis, vulva, anus, perineum, fingers, and soles
Veraldi[10]	1991	4	48	2M/2F	Nails, lips, oral mucosa	*
Haneke *et al*.[10]	1991	15	50	10M/5F	Nails, oral mucosa	Onychomycosis in one case, melanoma in one case
Lamey *et al*.[2]	1991	2	46	M/F	Nails, oral mucosa	Lupus erythematosus in one case
Gerbig and Hunziger[11]	1996	1	50	M	Nails, lips, oral mucosa	Anal mucosa, dorsal and lateral aspects of fingers
Mowad *et al*.[10]	1997	1	59	F	Oral mucosa	*
Porneuf and Dandurand[10]	1997	1	47	F	Oral mucosa	Interdigital area
Seoaneher *et al*.[12]	1998	13	*	*	Oral mucosal, with or without nail pigmentation	*
Mignognam[10]	1999	12	50	1M/11F	Lips, oral mucosa, two cases showed nail pigmentation	*
Yamamoto *et al*.[13]	1999	1	62	F	Lips, palatoglossal arch, lingual margin, palm, mid-esophageal mucosa	*
Ferreira[14]	1999	1	49	F	Oral mucosa, hand, feet, nails	*
Kanwar *et al*.[15]	2001	2	*	*	Oral mucosa, buccal mucosa, lips, and nails	*
Papadavid and Walker[16]	2001	2	*	*	Lips	*
Lenane P *et al*.[17]	2001	1	62	F	Oral mucosa, nails	Vulval pigmentation
Lampe *et al*.[18]	2003	1	*	*	*	*
Makhoul *et al*.[8]	2003	3	50, 25, 21	F	Oral mucosa, buccal mucosa, tongue	One case showed conjunctival pigmentation
Siponem and Salo[1]	2003	1	57	F	Buccal mucosa, labial mucosa, lips	*

Table 1 contd.....

Table 1 *(Contd...)*

Authors	Year	No. of cases	Mean age	Sex	Clinical findings	Atypical features
Aytekin and Alp[19]	2004	1			Oral mucosa	Associated with actinic lichen planus
Ayoub[7]	2004	2			Oral mucosa, lips, one case with nails	Ocular conjunctiva, penis
Moore *et al*.[6]	2004	1	64	M	Oral mucosa	*
Akcali *et al*.[20]	2004	1	*	*	*	*
Ozawa[21]	2005	1	63	F	Lips, oral mucosa	*
Sabesan *et al*.[9]	2006	1	80	F	Buccal mucosa, lower lips, nails	*
Sardana *et al*.[22]	2006	1	12	M	Perioral, intraoral, nails	*
Gencoglan[23]	2007	1	*	*	Oral mucosa, skin lesions, palmoplantar, lips	Genital mucosa, conjunctiva, vulva
Yago *et al*.[24]	2008	2	*	*	*	*
Simionescu *et al*.[25]	2008	1	*	*	Lips	Mucosal melanoma
Aliagaoglu *et al*.[26]	2008	1	*	*	Oral mucosa	Atypical location
Blossom[27]	2008	1	63	F	Thumb, index finger	*
Montebugnoli[28]	2010	1	43	F	Oral mucosa, toe nail	*
Sendagorta *et al*.[29]	2010	2	*	*	*	*
Jabbari *et al*.[5]	2010	1	45	F	Oral mucosa, lips, finger nails	*
Rangwala *et al*.[30]	2010	1	77	M	Lip, finger nails, and toe nails	*
Ko[31]	2011	1	58	F	Lips, buccal mucosa, finger nails	*
Zuo[32]	2010	22	*	*	*	*
Pereira[4]	2010	1	64	F	Oral mucosa, finger, and toe nails	*

* = Not available

Physiologic (racial) pigmentation of the oral mucosa is seen in Asians, Blacks, and other dark-skinned persons, resembling LHS. Racial pigmentation is most commonly demonstrated in the gingiva although it may be found in any location. Addison's disease is an endocrine disease marked by insufficient production of cortisol and aldosterone by the adrenal glands. It is characterized by hyperpigmentation of the skin and mucosal membranes, associated with increased level of circulating adrenocorticotropic hormone (ACTH).

Bandler syndrome is a rare genodermatosis that presents with hyperpigmented macules in the hands, nails, and oral mucosa during infancy, as well as intestinal vascular malformation that can cause significant gastrointestinal bleeding.

Mc Cunnie Albright syndrome exhibits labial and genital pigmentation, but it is often unilateral and does not involve the nails. The disease is also accompanied by precocious puberty in females and fibrous dysplasia.

Neurofibromatosis may display pigmented macules of the lips; there are multiple other skin findings and unknown nail changes.[1,30]

LAMB syndrome is characterized by pigmentation of the skin mucosa, atrial and mucocutaneous melanomas, and multiple blue nevi. LEOPARD syndrome is manifested by numerous lentigines, electrocardiographic abnormalities, occasional hypertelorism, pulmonic stenosis abnormalities of genitalia, retardation of growth, and deafness.[6]

Peutz Jeughers Syndrome (PJS) is an inherited autosomal dominant disease with a high degree of penetrance. It is characterized by intestinal polyposis and melanotic macules particularly of the face and mouth. Multiple melanotic macules resembling ephelides on the lips and periorally is a characteristic feature of the syndrome.[1,30]

Overlapping clinical features in both LHS and PJS may cause diagnostic problems, especially when PJS occurs with mucosal and cutaneous macules without intestinal polyposis. On the other hand, in the absence of both family and personal history of intestinal polyposis, a definitive diagnosis of PJS can hardly be made. The pigmented lesions of the LHS are usually confined to the oral mucosa, lips, and nails, whereas PJS is often seen on the hands and feet. However, there have been reports of patients with additional pigmentation on the fingers, palms, feet, and face. Lamey *et al.* suggested that LHS can be confidently diagnosed only when both oral and nail pigmentation is present. Additionally, the cutaneous hyperpigmentation fades with puberty, whereas the oral pigmentation persists in LHS.[1,2,30]

Table 2: Various conditions associated with hyperpigmentation

Focal[1,30]	Diffuse[1,28,30]	Syndrome[4,5,10,28,30,33]
1) Amalgam tattoo	1) Addison's disease	1) Albright
2) Graphite tattoo	2) Drugs	2) Blander
3) Hemangioma	Amiodarone	3) Carney
4) Idiopathic melanoplakia and melanonychia	AZT (zidovudine)	4) Cronkhite–Canada
	Bleomycin	5) LAMB
4) Melanoacanthoma	Busulfan	6) Laugier–Hunziker
5) Melanoma	Chloroquine	7) Lentiginosis profusa
6) Melanotic macule	Chemotherapeutic agents	8) LEOPARD syndrome
7) Nevus	Ketoconazole	9) Peutz–Jeghers
	Oral contraceptive	
	Phenothiazine	
	Azidothymidine	
	3) Inflammatory disorders of the oral mucosalichen planus	
	4) Systemic exposures to heavy metals	
	Silver	
	Gold	
	Bismuth	
	Mercury	
	Lead	
	5) AIDS patients present with mucocutaneous and ungula pigmentation (secondary to systemic drugs)	
	6) Adrenal insufficiency	
	7) Physiologic (racial)	
	8) Smoking	

Negative evidence of systemic symptoms such as fatigue, weight loss, cardiovascular, or gastrointestinal disorders, normal plasma levels of cortisol and ACTH, negative drug history, and negative findings in upper gastrointestinal endoscopy and colonoscopy will aid in the diagnosis of LHS. Hence, detailed history taking and through clinical examination of a patient presenting with oral pigmentation is of paramount importance.[31]

ASSOCIATIONS

Case reports have described LHS with oesophageal melanocytosis, actinic lichen planus, hypocellular marrow, and thrombocytopenia. The relationships with these conditions have not been well established.[5]

TREATMENT

No treatment is required for this condition since it is not associated with systemic diseases or complications. There have been no reports of malignant transformation.[4] Patients may choose to have the associated pigmentation removed because of cosmetic disfigurement. A few case studies have demonstrated that cryosurgery, Nd–YAG lasers, and the Q-switched alexandrite laser may be safe and effective options for patients.[5,30] Recurrence may occur after treatment, but this may be reduced by avoiding exposure to sunlight.[30]

CONCLUSION

Although the condition has been recognized and several case reports have been published, it is still unknown to many oral pathologists, general dentists, and other oral health care providers. Most text books on oral pathology and oral medicine do not include LHS as a cause of oral pigmentation probably because of its benign nature and innocuous course. As Mignogna *et al.* pointed out, it is likely that the syndrome is more common than the reported cases. The appearance of new areas of pigmentation on mucus membrane in a middle-aged patient may be considered quite alarming. Therefore, it is important that clinicians be made aware of this benign condition as a cause of multifocal pigmentation of the oral mucosa and lips.

The importance of recognizing LHS is to avoid unnecessary investigations and treatment. LHS should be added to the list of conditions in which mucosal and ungual hyperpigmentation is seen without a significant risk of malignant development.

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