Study of Rare Hyperpigmented Lesions of the Skin in a Tertiary Care Centre in Western Maharashtra

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Authors’ contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Objectives: Hyperpigmented lesions are a common presentation in the dermatology outpatient department and causes significant burden on the dermatologist. Though they are harmless and asymptomatic in most cases they are a cause for concern because it can lead to cosmetic deformity which can cause psychological upset in the patients. Rare lesions are often misdiagnosed clinically because of overlapping clinical features. The aim of this study was to diagnose rare hyperpigmented lesions with the help of skin biopsies and correlate the findings clinically.

Methods: All skin biopsies coming to the department of Pathology were processed and stained by routine H&E stain. Special stains were applied whenever required (Ziehl-Nelson, periodic acid Schiff, Congo red).

Results: 56 cases were studied of which 27 rare lesions were diagnosed. All the cases were subjected to analysis according to age, sex, site of lesion, duration of lesion and clinicopathological correlation. 75% of the cases showed histopathological correlation in our study. 25% of the cases did not show concordance.

Conclusion: Hence to conclude histopathological examination of skin biopsies forms the gold standard for the diagnosis of hyperpigmented skin lesions more so in the case of rare lesions as most of them have overlapping clinical features which can be mistaken for common lesions.

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1. INTRODUCTION

Dermatology outpatient department witness a huge burden of hyperpigmented lesions. These lesions are mostly harmless and asymptomatic and cause concern because of their ability to cause cosmetic deformity which can cause psychological upset in the patients [1]. The skin and mucous membrane are usually affected by pigmentation which eventually leads to hyperpigmentation. Varied etiologies have been identified, which can be inflammatory, degenerative, endocrine, toxic and immunologic [1,2]. Hyperpigmented lesions can manifest either in the epidermal or dermal regions. Epidermal lesions give rise to brown pigmentation whereas dermal lesions manifest as blue pigmentation [2]. For the diagnosis of majority of the hyperpigmented lesions, history, clinical presentation, and histopathological examination are required. Skin biopsy forms the basis of differential diagnosis in clinically similar lesions, thereby yielding important information to the dermatologist and pathologist [3]. Histopathology remains the gold standard in the diagnosis of these lesions, and it helps in overcoming the diagnostic dilemmas faced by the dermatologist more so in the case of rare lesions [4].

2. MATERIALS AND METHODS

Ours was a prospective study conducted over a period of 2 years in the Department of Pathology of Dr. D. Y. Patil Medical College Hospital and Research Centre, Pimpri, Pune. Our study comprised of 56 cases presenting as hyperpigmented lesions to the Department of Dermatology. These cases were further subjected to histopathological study in the Department of Pathology. All the cases were analyzed after getting institutional ethics committee clearance. All the skin biopsies were processed and stained using haematoxylin and eosin and were further subjected to special stains as required. All inadequate skin biopsies and lesions other than hyperpigmented lesions were excluded from the study. Patient details along with clinical and histopathological features were collected and compiled.

3. RESULTS

Our current study was conducted to correlate histopathological and clinical findings of rare hyperpigmented skin lesions in a tertiary care centre in Western Maharashtra. 56 cases were studied under various parameters such as age and sex distribution, clinical pattern of presentation, histopathological findings and its correlation with clinical findings. In our study the lesions presented with a wide age distribution of 1 to 82 years. Majority of the patients were between 21-40 years of age (44.6% of the lesions), followed by those between 41-60 years (41.07%). Those above 60 years of age constituted 7.14% of the study subjects and those between 0-20 years were 7.14%. The mean age of the subjects was 32.8 years with median age being 31 years. The study showed a slight female predominance, with 58% of the subjects being females. The average duration of the lesion at the time of detection was found to be 1-3 months of appearance of lesion (46.4%) followed by 4 to 6 months (23.21%). Some patients had lesions of more than 7-12 months (14.2%) duration. 8.9% presented with duration less than 1 month, 1.7% presented with duration of 13-24 months. Majority of the patients had multiple lesions (60.7%) at the time of presentation. 39.2% of the patients presented with a single lesion. The commonest gross presentation was in the form of plaques which was seen in 41.07% patients followed by patches (33.9%) and papules (10.71%). Macules and nodules were noted in 7.41% of the patients. The most common site of involvement was of upper limb (33.9%), followed by trunk (21.42%), lower limbs and face (19.64%) and back involvement was seen in 3.5% of patients. The least common site was found to be the neck which comprised of 1.7% of the patients. Of all the rare lesions studies the most frequently encountered lesion was Becker's Nevus. Clinical diagnosis of Becker’s Nevus was done in 4 patients out of which 3 patients showed histopathological correlation (Table 1).

4. DISCUSSION

Skin is the largest organ in the human body, and it plays an important role in thermoregulation, fluid homeostasis and immune function. Skin is also target for diverse diseases. Pigmentation of the skin is a very common cause for dermatological consultation as well as histopathological examination. Hyperpigmentation is due to abnormality in the structure or functioning of the melanocytes [1,2]. Epidermal hyperpigmentation was found
Table 1. Clinicopathological correlation of various hyperpigmented lesions in our study

<table>
<thead>
<tr>
<th>Sr. No.</th>
<th>Name of lesion</th>
<th>Clinical diagnosis</th>
<th>HPE Diagnosis</th>
<th>Clinical correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Becker's Nevus</td>
<td>6</td>
<td>5</td>
<td>83.33%</td>
</tr>
<tr>
<td>2.</td>
<td>DLE</td>
<td>4</td>
<td>3</td>
<td>75%</td>
</tr>
<tr>
<td>3.</td>
<td>Morphea</td>
<td>3</td>
<td>3</td>
<td>100%</td>
</tr>
<tr>
<td>4.</td>
<td>Pityriasislichenoideschronica</td>
<td>2</td>
<td>2</td>
<td>100%</td>
</tr>
<tr>
<td>5.</td>
<td>erythema dyschromiumperstans</td>
<td>2</td>
<td>2</td>
<td>100%</td>
</tr>
<tr>
<td>6.</td>
<td>Lichen striatus</td>
<td>5</td>
<td>4</td>
<td>80%</td>
</tr>
<tr>
<td>7.</td>
<td>Prurigonodularis</td>
<td>4</td>
<td>4</td>
<td>100%</td>
</tr>
<tr>
<td>8.</td>
<td>Acropigmentation of kitamura</td>
<td>1</td>
<td>1</td>
<td>100%</td>
</tr>
<tr>
<td>9.</td>
<td>Amyloid deposits in skin</td>
<td>0</td>
<td>1</td>
<td>0%</td>
</tr>
<tr>
<td>10.</td>
<td>Chronic cutaneous erythematous</td>
<td>2</td>
<td>2</td>
<td>100%</td>
</tr>
<tr>
<td>11.</td>
<td>Destructive dermatitis</td>
<td>2</td>
<td>1</td>
<td>50%</td>
</tr>
<tr>
<td>12.</td>
<td>Erythema multiforme major</td>
<td>2</td>
<td>2</td>
<td>100%</td>
</tr>
<tr>
<td>13.</td>
<td>Erythema nodosummigrans</td>
<td>1</td>
<td>1</td>
<td>100%</td>
</tr>
<tr>
<td>14.</td>
<td>Lichen nitidus</td>
<td>2</td>
<td>2</td>
<td>100%</td>
</tr>
<tr>
<td>15.</td>
<td>Lichen planuspigmentosus</td>
<td>2</td>
<td>1</td>
<td>50%</td>
</tr>
<tr>
<td>16.</td>
<td>Lichen sclerosusatrophiucus</td>
<td>2</td>
<td>1</td>
<td>50%</td>
</tr>
<tr>
<td>17.</td>
<td>Linear epidermal nevus</td>
<td>1</td>
<td>1</td>
<td>100%</td>
</tr>
<tr>
<td>18.</td>
<td>Linear Lichen Planus</td>
<td>1</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>19.</td>
<td>Nevus of Ota</td>
<td>0</td>
<td>1</td>
<td>0%</td>
</tr>
<tr>
<td>20.</td>
<td>Pellagra</td>
<td>0</td>
<td>1</td>
<td>0%</td>
</tr>
<tr>
<td>21.</td>
<td>Polymorphous light eruption</td>
<td>2</td>
<td>1</td>
<td>50%</td>
</tr>
<tr>
<td>22.</td>
<td>SLE</td>
<td>3</td>
<td>2</td>
<td>66.66%</td>
</tr>
<tr>
<td>23.</td>
<td>Subacute chronic lupus erythematous</td>
<td>2</td>
<td>2</td>
<td>100%</td>
</tr>
<tr>
<td>24.</td>
<td>Verrucous epidermal nevus</td>
<td>1</td>
<td>1</td>
<td>100%</td>
</tr>
<tr>
<td>25.</td>
<td>Lichen morpheasclerotica</td>
<td>1</td>
<td>1</td>
<td>100%</td>
</tr>
<tr>
<td>26.</td>
<td>Lichenified eczema</td>
<td>1</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>27.</td>
<td>Lichen sclerosus</td>
<td>1</td>
<td>0</td>
<td>0%</td>
</tr>
</tbody>
</table>

Comparative to be more common than dermal hyperpigmentation [2]. Due to common presentation of various local and systemic disease there can be lack of clarity about diagnosis of the lesion, in this scenario histopathological examination is needed for helping the dermatologist in overcoming challenges and in establishing the diagnosis. The biopsies taken from the skin helps both pathologist and dermatologist to come to a diagnosis [2-4].

Out of the 56 cases studied, maximum cases were in the age group of 21–30 years which was similar to studies conducted by other authors. Female predominance was noted in our study which matched with various other studies [2]. The average time of onset of lesions and their presentation to the dermatologist was noted to be 7 to 12 months but few of the lesions presented with less than one month duration and some had a clinical history of more than 24 months. 60.2% presented with multiple lesions and 39.2% presented as single lesion. Of the 56 cases studies 27 different lesions were identified which were Becker's Nevus, Discoid lupus erythematous, Morphea, Pityriasislichenoideschronica, erythema dyschromiumperstans, Lichen striatus, Prurigonodularis, Acropigmentation of Kitamura, Amyloid deposits in skin, Chronic cutaneous erythematous, Destructive dermatitis, Erythema multiforme major, Erythema nodosummigrans, Lichen nitidus, Lichen planuspigmentosus, Lichen sclerosusatrophiucus, Linear epidermal nevus, Linear Lichen Planus, Nevus of Ota, Pellagra, Polymorphous light eruption, Systemic Lupus Erythematous, Subacute chronic lupus erythematous, Verrucous epidermal nevus, Lichen morpheasclerotica, Lichenified eczema and Lichen sclerosus. Becker’s naevus was found to be the most common lesion in our study.

Becker in 1949 was the first to describe a melanocytic nevus with hypertrichosis. He described two cases one was a 17year old who at the age of 11 presented with a blotchy brown pigmentation over the right side of the shoulder.
after severe sunburn. The second case was of a 24-year-old male who also presented with prolonged history of sunburn. The central area showed hypertrichosis. Other than increased pigment in the basal portion of the epidermis no alteration of the epidermis or dermis was seen, large amounts of melanin was seen in the basal cells. Stratum corneum showed decreasing amounts of melanin. Nevus cells were absent in both epidermis and the dermis. In our study there were 6 patients, 4 were male and 2 were female. All the six patients came with the clinical diagnosis of Becker's naevus, out of which 5 patients showed Becker's naevus on histopathology and one patient was diagnosed as compound naevus. These presented as single patch over face, upper limb, and trunk respectively. Histopathology features included acanthosis and irregular elongation, flattening of the rete ridges with the tendency to fuse. The upper part of the dermis showed hyperpigmentation of the basal layer and melanophages (Fig. 1) [5].

The second most common lesion encountered in our study was lichen striatus. We studied 5 cases which came with the clinical diagnosis of lichen striatus. 3 were female patients and 2 males. Out of the 5 cases 4 cases showed histopathological corelation whereas one case turned out to be lichen planus. The histopathological findings encountered were perieccrine, superficial and deep perivascular and perifollicular infiltrate of lymphocytes and histocytes that extended to the acanthotic epidermis (Fig. 2).

Lichen nitidus was also seen in two female patients in the age group of 30-40yrs. Both had multiple lesions whereas one presented as plaque and the other as patch. The histopathological findings showed thin suprapapillary epidermis with vacuolar alteration of the basal layer, dense infiltrate of lymphocytes and histocytes in an expanded dermal papilla, and focal parakeratosis (Fig. 3) [6-8].

Lichen planuspigmentosus was seen in two female patients one case corelated clinically whereas the other turned out to be lichen planus on histopathology. As compared to the common presentation over the face, our study showed multiple hyperpigmented patches over the extremities. Though rare pigmentation can be seen over the extremities. The histopathology of Lichen PlanusPigmentosus forms a continuous spectrum with the earliest lesions being marked inflammation at the interface, which later subsides to leave behind the characteristic dermal pigmentation. The epidermis is atrophic as compared to acanthosis which is seen in Lichen Planus. The inflammatory phase is characterized by a dense band of lymphohistiocytic infiltrate in the upper dermis with prominent basal vacular degeneration. Melanin incontinence is seen with scattered dermal melanophages [9].

Figs. 1a & 1b. Becker’s naevus – 1a shows single hyperpigmented patch occupying large area over the chest with irregular borders. 1b shows H & E stain under 40x epidermis with slight acanthosis and irregular elongation and flattening with a tendency for fusion of the rete ridges. There is hyperpigmentation of the basal layer.
Fig. 2. Lichen striatus – 2a shows H & E stain under 40x showing perineural infiltrate. 2b shows H & E stain under 4x showing papillary dermis infiltrate, perieccrine, perifollicular, superficial, and deep perivascular infiltrate of lymphocytes and histocytes that extend to epidermis. 2c shows H & E stain under 40x showing perivascular infiltrate

Fig. 3. Lichen nitidus - H & E stain under 10x showing dense lymphohistiocytic infiltrate in papillary dermis, which is expanded, basal layer shows vacuolar alteration and thin suprapapillary epidermis
Lichen sclerosus et atrophicus is a chronic inflammatory dermatosis with anogenital and extragenital presentations. Exogenous lesions are more frequently found on the neck, shoulders, and upper trunk. Three cases with the clinical diagnosis of lichen sclerosis etatrophicus were subjected to histopathological examination in our study. The lesions presented on the trunk and lower limbs. Two cases were diagnosed histopathologically as lichen sclerosis etatrophicus whereas one of the cases turned out to be lichen simplex. The cases were mostly diagnosed in the 2nd to 3rd decade with an average duration of presentation of 6 months. Histopathological examination revealed follicular plugging, thinning of the epidermis, loss of the rete ridges, focal basal cell vacuolization, oedema, pigmenitary incontinence, and hyalinization of the papillary dermis. These features showed concordance with similar studies conducted by various authors (Fig. 4) [10].

Systemic lupus erythematosus is an autoimmune disease with multiorgan involvement with skin being the second most affected organ. Skin lesions in patients with lupus may be specific or may be non-specific. Acute cutaneous Lupus Erythematosus has a strong association with systemic disease and non-specific skin lesions always indicate disease activity for which patients seek treatment [11].

Discoid lupus erythematosus (DLE) is one of the most common form of chronic cutaneous lupus erythematosus. Classic DLE lesions starts as a red-purple macule, papule or small plaques and rapidly develops into a hyperkeratotic surface. DLE lesions occurred most frequently on the head and neck followed by the upper extremities and trunk. Most of the lesions in DLE evolved into peripheral hyperpigmentation with central atrophic scars, followed by post-inflammatory hyper & hypopigmentation and erythema. Histopathological examination of the skin biopsies shows perieccrine lymphocytic infiltrate and basal vacuolization. Superficial and deep perivascular homogeneous granular deposition along dermo epidermal junction was also seen [11-13]. In our study DLE was noted in 4 patients and two patients were clinically diagnosed as chronic cutaneous erythematosus. The LESIONS were multiple in number with facial and

![Fig. 4. Lichen sclerosis atrophicus - H & E stain under 10x showing hyperkeratosis with follicular plugging, basal cells showing hydropic degeneration, oedematous collagen with homogenization in the upper dermis](image_url)
upper limb involvement. These presented as itchy patches or plaques. Histopathological examination showed dermis containing periadnexal and perivascular lymphohistiocytic infiltrate under an interface dermatitis. The epidermal interface activity showed apoptotic keratinocytes and a marked thickening of the basement membrane and degeneration of the basal layer [12]. Our study was similar to the study done by Chandraprapaph et al (2021) with respect to clinical presentation showing plaques and histopathological features. The number of cases of DLE noted in our study was four out of which only three cases were diagnosed as DLE on histopathology (Fig. 5) [12,13].

Prurigonodularis is a chronic condition which is characterised by papulonodular eruptions. The disease is of unknown aetiology. This condition was clinically diagnosed in 4 patients (3 female and 1 male) in our study. These patients presented as nodular or papular lesion with involvement of extremities. The lesions were multiple in number. Marked irregular acanthosis, compact orthokeratosis with parakeratosis and focal hypergranulosis was seen on histopathological evaluation. Our study corelated with the study conducted by Lee RM et al. (2005) with respect to clinical presentation and histopathological features [14]. All the four cases in our study showed clinicopathological corelation (Figs. 6 & 7).

Fig. 5a & 5b. Discoid lupus erythromatosis – 5a shows H & E stain under 4x with thinning of epidermis, degeneration of the basal layer, marked thickening of the basement membrane and apoptotic keratinocytes are seen. 5b shows H & E stain under 10x with dermis showing interface dermatitis contains periadnexal and perivascular lymphohistiocytic infiltrate

Fig. 6. Prurigonodularis: Multiple nodular lesions seen on the lower limbs. There are abrasions seen in the central area of the lesions due to excess itching, and surrounding area shows hyperpigmentation
Morphea is a rare autoimmune condition causing inflammation with sclerosis of the skin and underlying soft tissue. It is characterized by periods of activity (inflammation admixed with fibrosis), ultimately resulting in permanent damage (pigment change and tissue loss). Damage resulting from unchecked activity can lead to devastating, permanent cosmetic and functional sequelae including hair loss, bony atrophy, joint contractures, and growth restriction of the affected body site in children. Morphea is characterized by thickening of the skin resulting from the inflammation and deposition of collagen rich extracellular matrix [15,16].

Morpheas noted in 3 patients in our study. They presented as single and multiple lesions on the lower limb, neck, and face. All the patients had plaques. Histology included presence of interstitial lymphoplasmacytic infiltrate dispersed among deep dermal collagen bundles which are minimally swollen. lymphoplasmacytic infiltrate separate the collagen strands and in the deep dermis surround eccrine coils. There is also associated loss of adipocytes around the eccrine apparatus. Our study was correlating with the findings of Khalifa et al (2020). More number of subjects with diagnosis of morphea would have helped in further comparison since there were only three patients this was not possible (Fig. 8) [15,16].

Fig. 7. Prurigo nodularis - 7a shows H & E stain under 10x with marked irregular acanthosis. 7b shows H & E stain under 40x showing marked irregular acanthosis, focal hypergranulosis, and compact hyperkeratosis with orthokeratosis.

Fig. 8. Morphea - 8a shows single hyperpigmented plaque on the face with centrally depigmented sclerosed area surrounded by hyperpigmentation. 8b shows H & E stain under 10x with deep dermal collagen bundles with dispersed interstitial lymphoplasmacytic infiltrate. Swollen collagen bundles. Collagen strands are separated by lymphoplasmacytic inflammatory infiltrates separate such, eccrine coils in the deep dermis are also surrounded by inflammatory cells. There is loss of adipocytes around eccrine apparatus.
Sundhar J et al (2009) reported a case of erythema dyschromiumperstans with Sjogrens syndrome in a female subject who was 38 years old. The clinical features included patchy non-pruritic lesions on face, arms, chest, and neck. Biopsies and histopathological examination revealed lichenoid infiltrate of lymphocytes with vacuolar changes, melanophages scattered among dyskeratotic keratinocytes in the papillary and reticular dermis. Our study showed one male and one female patient with macules and patches over the upper limb and neck as presentation. The histological examination showed basal vacuolar degeneration, lymphocytic infiltrates, and significant percentage of melanophages. The presentation matched with the study conducted by Sundhar J et al in terms of upper limb involvement and patchy involvement and appearance around middle age, though one of the patients in our study was a 6yr old boy (Fig. 9) [17].

Erythema multiforme major also known as Fuch’s Syndrome was initially described by the Germans, is mucositis associated with Mycoplasma pneumonia. It is characterized by targetoid lesions and occurs most frequently in young male adults. There are two variants, one with mucosal involvement which is called erythema multiforme major and the other without mucosal involvement, known as erythema multiforme minor. 2 cases both females in the second decade of life were seen in our study. They presented with multiple macules and patches over the trunk and lower limbs. Both the cases had a short history of presentation, and they were both diagnosed clinically as erythema multiforme major. 100% correlation was seen in our study. Histopathological examination revealed dense dermal lymphohistiocytic infiltrate, overlying epidermis shows degeneration with variable spongiosis (Fig. 10) [18].

Erythema nosophum migrans is a rare clinical variant of erythema nodosum. A single case of erythema nosophum migrans was seen in a 57yrs old female. She presented with multiple nodules over the face of 7 months duration. The clinical diagnosis of this patient was also erythema nosophum migrans. The histopathologic findings were mild lymphohistiocytic infiltrate with vascular proliferation in the superficial and deep dermis. Some of the lymphohistiocytic infiltrate extended to the periphery of the subcutaneous lobules. No neutrophilic infiltrate or well-formed granulomas were seen. The subcutaneous changes of septal panniculitis observed suggested a diagnosis of Erythema Nodosum Migrans (Fig. 11) [19].

![Fig. 9. Erythema dyschromiumperstans – H & E stain under 40x shows significant percentage of melanophages, lymphocytic infiltrates, and basal vacuolar degeneration](image-url)
Fig. 10. Erythema multiforme: 10a shows multiple well to ill-defined plaques on the lower limbs. The lesions show central pigmented area with peripheral hypopigmentation, the borders are raised and oedematous ("target lesions"). 10b shows H & E stain under 10x necrotic keratinocytes in the epidermis and eosinophils at all levels of epidermis. Superficial interface inflammatory infiltrate is also seen.

Fig. 11. Erthyemanodosum- H & E stain under 10x showing mixed cellular infiltrate of lymphocytes, histiocytes and occasional giant cells, eosinophils along with a characteristic absence of vasculitis. Septalpanniculitis is also seen.

Verma K et al. [20] studied a total of 100 cases of polymorphous light eruption. Papules and plaques were the common clinical presentation. Lesions over neck and ‘V’ area of chest were seen in 24% of the cases and lesions over extensor aspect of upper extremities were seen in 18%. On microscopic examination maximum cases showed spongiosis, upper and mid dermis showed dense perivascular lymphocytic infiltrate, oedema, and hyperkeratosis of epidermis [20]. Polymorphous light eruption was diagnosed clinically in one male patient and one female patient both were in the 3rd decade of life. They presented with multiple patches and plaques on
the upper limbs and upper back with a short history. On histopathology showed hyperkeratosis with mild spongiosis, perivascular and periappendageal lymphohistiocytic infiltrate in upper and middermis was noted. Only one case showed clinicopathological corelation whereas the other lesion showed features of dermatitis. The clinical presentation of multiple site involvement, presence of lesions in exposed part of the body and histopathological features were seen in both our study and the study done by Verma K et al. [20].

Pityriasis lichenoides is a papulosquamous disorder of unknown etiology with remissions and exacerbations. Histopathology helps greatly in the diagnosis of this condition. Pradeep S. Nair studied 51 cases of pityriasis lichenoides. The maximum number of cases, 14 (27.45%) were in their second decade of life. Pityriasis lichenoides chronica was diagnosed in 39 cases (76.47%). Histopathologically, basal cell vacuolation and perivascular infiltrate were seen in all the cases. Exocytosis was seen in 45.1% of the cases. In our study there were 2 female patients in the 4-6th decade of life who presented with multiple plaques and papules over the upper limbs. Both were clinically diagnosed as pityriasis lichenoides chronica. Prominent basal cell degeneration and perivascular infiltrate were the histological hallmarks seen in our study (Fig. 12) [21].

Some of the other conditions which were encountered in our study were linear lichen planus, naevus of ota, pellagra, verrucous epidermal naevus (Fig. 13), lichen morphea sclerotica and amyloid. Two cases of epidermal naevus were diagnosed in our study. The histopathological diagnosis of linear verrucous epidermal naevus was confirmed by presence of hyperkeratosis with focal parakeratosis, acanthosis with focal exocytosis and spongiosis in the epidermis. Agranulosis and parakeratosis alternating with hypergranulosis and orthokeratosis were seen. Dermis showed lymphocytic infiltrate. A single case clinically diagnosed as lichen planus turned out to be amyloid on skin biopsy. Special stain such as congo red was done to confirm the presence of amyloid in the deep dermis. Our study showed one 11yrs old female patient with multiple papular lesions which were itchy in presentation on the abdomen. Histopathological features included deposition of pink amorphous material within the papillary dermis. The basal layer showed increased pigmentation and scattered melanophages were seen in papillary dermis (Figs. 14 &15) [22].

Fig. 12. Pityriasis lichenoides - H & E stain under 10x showing lichenoid inflammatory pattern extravasated erythrocytes and infiltrate of lymphocytes. Acanthosis, pallor of upper layers of the epidermis, spongiosis and necrotic keratinocytes is also seen
Fig. 13. Epidermal nevus: Single linear hyperpigmented plaque on the forehead with irregular borders

Fig. 14. Lichen amyloidosis: Multiple papular lesions seen on the lower limbs. They are all the same size and heavily pigmented

Fig. 15. Amyloid deposition in the skin – 15a shows H & E stain under 10x showing deposition of pink amorphous material within the papillary dermis. 15b - Congo red stain under 10x shows amyloid deposition which stained red in colour
Garg G et al (2011) studied cases of pellagra with a female preponderance at age of 20-30 years. He described the same in a case report series where 2 female patients had developed pellagra reaction. In both the cases there was involvement of extremities. The histopathology study showed hyperplastic epidermis and perivascular lymphocytic and neutrophilic infiltrate. They noted this to be common on feet and hands [23]. Pellagra was seen in one female patient aged 45 years old who presented clinically as melocytic naevus. A single patch of 6 months duration was noted over the upper limb. Histologically epidermal hyperplasia was noted with psoriasiform pattern, parakeratosis, hyperkeratosis, and lymphocytic perivascular inflammatory cell infiltrate was noted. Our study correlated with the study done by Garg G et al. 2011. In above mentioned study there was predominant extremity involvement however our patient had additional truncal involvement to lower limb involvement [23].

Other rare causes of hyperpigmentation include systemic diseases such as reaction to antimalarial agents, chemotherapeutic drugs, fixed drug eruptions, Addison’s disease hyperthyroidism, pregnancy, neoplastic diseases, renal failure etc. However, none of these were observed in our study population considering the small sample size. The clinical and histopathological correlation was found to be 75% in our study. 25% did not show correlation. The lack of concordance could be due to challenges in clinical diagnosis without supplementation with histopathological examination, varied aetiology, and association of systemic disease. Various investigative techniques such as dermoscopy and gene testing can be used as an adjunct to support the diagnostic dilemma [24]. Limitations of our study was that the sample size is small to extrapolate regional and national level trends. The underlying risk factors contributing to the hyperpigmented lesions were not studied. The follow up treatment and interventions were also not discussed. Long term follow-up and conversion to any malignant lesions was not done. Any underlying systemic diseases which are co-existing, and which would have played a contributory role in development of the hyperpigmented lesions were not studied.

5. CONCLUSION

From our study conducted on 56 patients with hyperpigmented lesions we can conclude that hyperpigmented lesions are a common occurrence but with a widely different aetiology. Rare causes of hyperpigmentation do present to the clinician who should have a knowledge of these lesions along with the clinical presentations. The smaller number of consultations for hyperpigmented skin lesions when compared to the other lesions in an Indian scenario could be due to negligence and lack of awareness regarding treatment facilities available. The diagnostic dilemma at the level of dermatologist due to the wide range of clinical presentation can be solved with the help of histopathological examination. A detailed and relevant clinical history and an adequate biopsy can ease the work of the pathologist in diagnosis. Histopathological examination helps the dermatologist in diagnosing the lesion and helps them to plan the treatment accordingly. Various advances have been made in the field of dermatology in the diagnosis of hyperpigmented lesions like gene testing to rule out genetic aetiologies, dermoscopy and use of fluorescent microscopes. In all histopathology remains the gold standard for the diagnosis of these lesions in case of diagnostic dilemma.

DISCLAIMER

The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

CONSENT

As per international standard or university standard, patients’ written consent has been collected and preserved by the author(s).

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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