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Original Research Paper

Clinicopathological Correlation of Acquired Hypopigmentary Disorders

Medical Science

KEYWORDS : acquired hypopigmented disorders, histopathological correlation,vitiligo

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ABSTRACT

Hypopigmentation means decrease of melanin in skin which appears white or lighter than normal colour. The hypopimented disorders can be genetic or acquired. Acquired hypopigmentary disorders include various inflammatory, infectious disorders.

The aim of this study was to study histopathological findings in various acquired hypopigmented disorders using routine Haematoxylin and Eosin stain and to study correlation of clinical and histopathological findings of various acquired hypopigmented disorders. The present study was conducted for a period of three years. Skin biopsy from 50 patients which included the lesion and the perilesional adjacent normal looking area, were received in the Department of Pathology, Government Medical College, Amritsar.Then paraffin embedded tissue sections were stained with routine haemtoxylin and eosin and diagnosis was made on the basis of the histopathological findings. Maximum numbers of patients had presented with clinical diagnosis of vitiligo (20 cases) followed by leprosy (BT-10 cases and LL-5 cases). On histopathology 24 were typified as vitiligo,11 as leprosy (11 PB leprosy and 5 MB leprosy),6 out of 50 cases were diagnosed on histopathology as lichen sclerosis et atrophicus, 1 as morphea, pityriasis versicolor, post inflammatory dermatitis and idiopathic guttate hypomelanosis each. The overall level of concordance between clinical and histopathological diagnosis was noted in 80% cases. Adequate clinical data and workup in combination with pathological resources can help in elucidation of specific etiology and good clinicopathological correlation.

INTRODUCTION

Skin is a complex organ, the largest in the body in which precisely regulated cellular and molecular interactions govern many crucial responses to our environment[1].

Skin is divided into two separate, but functionally interdependent layers, epidermis and dermis composed of cells with various functions. Melanocytes within the epidermis appear as randomly dispersed cells within the basal cell layer and are responsible for the production of melanin, a brown pigment that protects against potentially injurious ultraviolet radiation (UV) in sunlight[1]. .Dysfunction or loss of these melanocytes result in a variety of hypopigmentation disorders.

The categorization of hypopigmentation disorders is generally based on their etiology (congenital or acquired), age of onset (childhood or adulthood) and extent of lesions (localized or generalized). Further differentiation can be made on the basis of clinical findings, such as degree of pigment loss, associated morphological signs, and sites of involvement[10].

Localized hypopigmentation disorders are a common presentation in adults. The commonest cause of depigmented lesions is vitiligo. Vitiligo is characterized clinically by depigmented macules and patches that correspond histologically to a decrease or absence of melanocytes in the epidermis and less often the hair follicles. In general, vitiligo can be categorized into localized, generalized and universal forms. Localized forms can be segmental, focal or mucosal. The generalized forms are more common, and can be acrofacial or the vulgaris type. Acrofacial vitiligo consists of depigmented patches around the fingers and periorificial regions whereas vitiligo vulgaris presents as widely scattered patches. Post-inflammatory hypopigmentation and chemical leucoderma are other common cause of localized hypopigmentation. However, there are some inflammatory diseases in which the lesions are hypopigmented from the onset and preceding inflammatory changes may be clinically absent.Examples include pityriasis versicolor, mycosis fungoides(MF),lichen sclerosus (LS),morphoea(localized sleroderma), generalized scleroderma (systemic sclerosis),tuberculoid leprosy, sarcoidosis and follicular mucinosis.[12] If the hypopigmented lesions are associated with an atrophic epidermal surface, the hypopigmented variant of MF, LS and morphea are to be considered. If induration is felt on palpation, it indicates the presence of dense collagen, which characterizes LS and morphoea[14]. If the hypopigmented lesions are associated with an infiltrated dermal consistency, the differential diagnosis includes tuberculoid leprosy, sarcoidosis and follicular mucinosis

Widespread hypopigmentation involves hypopigmented lesions occurring in a widespread manner. An example is progressive macular hypomelanosis (PMH), which is characterized by ill-defined, hypopigmented macules and patches on the trunk, often confluent in the midline and occasionally extending to the proximal limbs and the neck. The diagnosis of PMH is made clinically, and it should be differentiated from pityriasis versicolor, pityriasis alba and MF[16]. Idiopathic guttate hypomelanosis (IGH) is a common condition presenting in middle-aged and elderly people, and presents as sharply defined white macules distributed in a widespread manner, usually on sun-exposed areas of the limbs. The pathological hallmark of IGH seems to be an absolute reduction in the number of melanocytes.[11]

Acquired generalized hypopigmentation is rare in adults. It may be present in nutritional deficiencies and endocrinopathies, although these conditions are mainly associated with hyperpigmentation. In panhypopituitarism, generalized hypopigmentation may occur due to decreased adrenocorticotrophic and melanocyte stimulating hormones, which normally stimulate epidermal melanogenesis[17].

AIMS AND OBJECTIVES

1. To study histopathological findings in various acquired hypopigmented disorders using routine Haematoxylin and Eosin stain.
2. To study correlation of clinical and histopathological findings of various acquired hypopigmented disorders.

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MATERIALS AND METHODS

In the present study, 50 cases were included. From each patient, skin biopsy, which included the lesion and the perilesional adjacent normal looking area, were studied once received in the Department of Pathology, Government Medical College, Amritsar. History and clinical examination of each patient was recorded on a self designed Performa.

After taking ethical committee approval, the patients were recruited in the study after informing them regarding the study in their vernacular language and taking written informed consent.

TECHNIQUE:

Paraffin embedded tissue sections were stained with routine haemtoxylin and eosin (H&E) and diagnosis was made on the basis of the histopathological findings

Methodology

Steps for Histopathological differentiation using H & E stain

1. Removal of wax from the paraffin embedded tissue sections with xylene. Sections were placed in xylene for 3-5 minutes.
2. Hydration: The section was removed from xylene, drained and transferred to absolute alcohol for 1-2 minutes, till it became opaque. The section was then rinsed in second bath of absolute alcohol.
3. Staining: Slides were immersed in Haematoxylin for about 12 minutes. Differentiation in acid alcohol for 3-10 quick dips was done.
4. Slides were then washed under tap water for about 10 minutes
5. Slides were then transfered to 1% aqueous stain for 2 minutes to counterstain them.
6. Washing of slides in running tap water was done for 2-3 minutes to differentiate the eosin.
7. Dehydration with alcohol- Slides were taken through 3 changes of absolute alcohol sequentially.
8. Clearing the slides in xylene and mounting was done.

OBSERVATIONS AND DISCUSSION:

The cases showed maximum incidence in the age group of 41-50 yrs comprising 14(28%) of cases .33 patients were male (66%) and 17 were females (34%). The most common site of biopsy in patients was upper limb constituting 15(30%) of cases. Maximum numbers of patients had presented with clinical diagnosis of vitiligo (20 cases) followed by leprosy (BT-10 cases and LL-5 cases).Other patients had presented as lichen sclerosis et atrophicus (6 cases), Morphea (2 cases), nevus depigmentosus (1 case), pityriasis versicolor(1 case),idiopathic guttate hypomelanosis(1 case), discoid lupus erythematosus(1 case),mycosis fungoides (1 case),incontinentia pigmenti (1 case), hypomelanosis of ito (1 case) (TABLE 1). Out of 20 cases who clinically presented with vitiligo, 19(95%) cases were confirmed on histopathology to be vitiligo and 1 case was confirmed to be Lichen sclerosis et atrophicus. Cases presenting as Discoid lupus erythematosus, Hypomelanosis of Ito and Nevus depigmentosus were established as Vitiligo on histopathology. Nevus depigmentosus, a congenital hypopigmented disorder, had presented in adult with short duration of history. On histopathology, it was confirmed to be vitiligo. 2 out of 6 cases that presented as Lichen sclerosis et atrophicus were typified as vitiligo. So on histopathology total of 24(48%) cases were found to be vitiligo(FIGURE 1). All 24 cases of vitiligo revealed loss of melanin pigment and melanocytes. Perivascular inflammatory infiltrate mainly lymphocytes was seen in 20 out of 24(83.3%) cases(FIGURE 2). There was 1 case with pigment incontinence and ectatic blood vessels each.

2 cases presenting as lichen sclerosis atrophicus found to be vitiligo. This was due to similar pattern of involvement of the site. Moreover, site of involvement of one of case was genital area.

There were 15 clinically diagnosed cases of leprosy[10 (66.6%) of Borderline Tuberculoid leprosy and 5 (33.3%) of Lepromatous leprosy] .On combined clinical and histopathological findings, 11 cases were found to be of Paucibacillary Leprosy(BT/TT) and 5 of Multibacillary Leprosy(BL/LL).So on histopathology all 10(100%) cases clinically presenting as BT leprosy turned out to be PB leprosy. These were characterized by epitheliod cell granulomas along neurovascular bundle, along with lymphocytes, multinucleated giant cells(FIGURE 3). Fite Faraco stain demonstrated no/very few bacilli in cases diagnosed as PB leprosy.

4 out of 5(80%) cases presenting as LL were typified as MB leprosy and 1 (20%) case presenting as LL was confirmed as PB leprosy on histopathology. 1 case clinically presenting as Mycosis fungoides was diagnosed as MB leprosy. These cases showed clear grenz zone, foamy macrophages collection and perivascular and periadnexal inflammatory infiltrate. Fite faraco stain revealed variable number of bacilli and bacterial index was done accordingly.

So PB leprosy was the most common histopathological type encountered (68.75%). Clinicpathological correlation was maximum in cases of BT leprosy (100%) followed by LL (80%)

In case of Lichen Sclerosis et Atrophicus,4 out of 6(66.6%) clinically diagnosed cases were confirmed on histopathology as lichen sclerosis et atrophicus. 2 out of 6 (33.3%) cases presenting as LSA were found to be vitiligo.1 case presenting as vitiligo was found to be LSA and 1 case presenting as Morphea was found to be LSA. The diagnosed cases revealed thinned out/atrophied epidermis, orthohyperkeratosis, loss of rete ridges and dermis showed homogenized papillary dermis, ectatic blood vessels and perivascular inflammatory infiltrate. 2 cases were clinically diagnosed as morphea.1 out of 2 (50%) was confirmed to morphea and other as Lichen Sclerosus et atrophicus. On histopathology, morphea showed thick collagen bundles in the reticular dermis and perivascular lymphoplasmacytic infiltrate in superficial, mid and deep dermis.

In the present study, the overall level of concordance between clinical and histopathological diagnosis was noted in 80% cases. Parity for vitiligo cases was 95%, for individual types of leprosy BT(100%),LL 80%.Parity for other cases LSA(66.6%), Morphea (50%),for PV (100%),IGH (100%),Post inflammatory dermatitis (0%) was noted.

TABLE I
SHOWING CLINICAL DIAGNOSIS FOR THE SKIN DISEASES

CLINICAL DIAGNOSIS	NO.OF CASES	PERCENTAGE (%)
Vitiligo	20	40
BT	10	20
LL	5	10
Lichen Sclerosis et Atrophicus	6	12
Morphea	2	4

Nevus depigmentosus	1	2
Mycosis fungoides	1	2
Incontinentia pigmenti	1	2
Pityriasis Versicolor	1	2
Discoid lupus erythematosus	1	2
Idiopathic guttate hypomelanosis	1	2
Hypomelanosis of ito	1	2
Total	50	100

FIGURE 1
DIAGRAMATIC REPRESENTATION SHOWING PROPORTION OF VARIOUS HISTOLOGICAL DIAGNOSIS

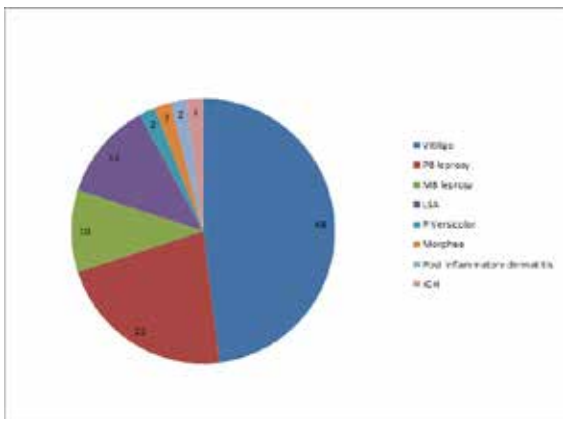
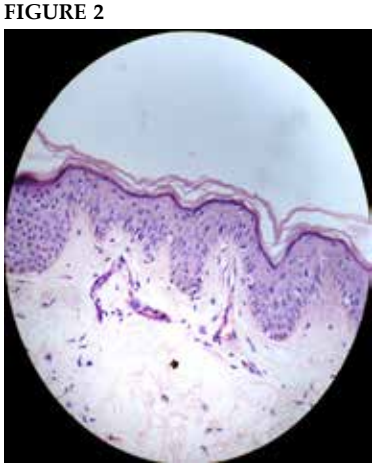
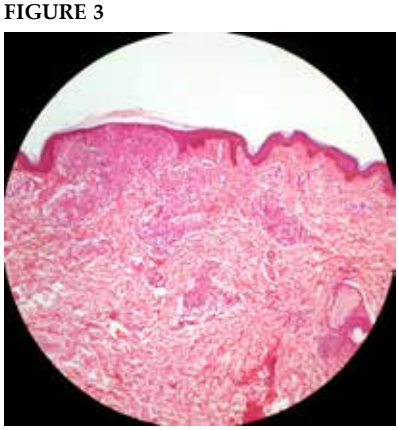


FIGURE 2



Photomicrograph showing loss of melanin and melanocytes in basal layer of epidermis(VITILIGO),H&E 400X

FIGURE 3



Photomicrograph showing granulomas invading the epidermis(PAUCIBACILLARY LEPROSY) H&E400X

CONCLUSION

There is a significant overlap in histopathological picture of different hypopigmentary disorders. Thus, morphology alone is seldom specific and can't be used as diagnostic tool for identification of specific diseases. Adequate clinical data and workup in combination with pathological resources can help in elucidation of specific etiology and good clinicopathological correlation.

Thus we conclude from our study that histopathological examination should be carried out in all cases of clinically diagnosed hypopigmented disease to arrive at a definite diagnosis.