

Scenario of Histoid Hansen at a Tertiary Care Hospital in South India

R Subha¹, M Ananthi², V Suganthy³

Received : 09.09.2017 Accepted : 10.06.2018

Histoid Hansen's disease is a form of lepromatous leprosy with characteristic bacterial morphology and very high bacillary load. The occurrence of de novo Histoid leprosy in patients without other features of leprosy has been reported. We present our observations on eleven cases of Histoid Hansen's disease who had attended our Outpatient Department. A detailed clinical history and clinical examination was done. Slit skin smear for Bacteriological Index and skin biopsy were performed in all. All eleven cases were males. Cutaneous and subcutaneous nodules were the commonest skin lesions found. Slit skin smear revealed abundant organisms. Histopathology was consistent with Histoid Hansen. Seven were de novo cases. During treatment, one patient developed type II reaction. All patients except one showed clinical clearance of lesions and fragmented bacilli after one year of MB-MDT. The treatment was extended in this single case who did not respond well. Awareness about the clinical features, characteristic histology and early diagnosis of Histoid Hansen is of utmost importance. It will be important to determine the bacteriological and morphological indices at the end of the treatment for all Histoid Hansen disease patients. Continuation of treatment may be decided based upon the bacterial and morphological indices.

Keywords: Histoid Hansen, Bacteriological index, Erythema nodosum leprosum

Introduction

Hansen's disease is still a disease of public health importance although the goal of leprosy elimination as a public health problem was achieved in 2006 in India. Histoid Hansen, not a rare form of lepromatous leprosy, forms a potential reservoir of infection in this post leprosy elimination era (Palit & Inamdar 2007). It has been proved that trans-epidermal exit of the

M. leprae from Histoid Hansen patient is also possible (Ghorpade 2011). During the recent years, several case reports of Histoid Hansen have been reported from our country (Bhukya & Reddy 2016, Nair et al 2016, Meena et al 2017). As awareness about the overall profile of Histoid Hansen in Indian settings is important from the point of view of clinical management, we have analysed clinicopathological and bacteriological

¹ Dr R Subha MD(DVL), Assistant Professor

² Dr M Ananthi MD(DVL), Assistant Professor

³ Dr V Suganthy MD(DVL), Assistant Professor

Department of Dermatology, Govt. Villupuram Medical College, Villupuram, 605601, Tamil Nadu, India

Corresponding author: Dr. M. Ananthi, **Email id:** drananthi@yahoo.com

profile of 11 cases of Histoid Hansen who reported to our OPD over a period of six years.

Patients and Methods

Eleven cases of clinically diagnosed Histoid Hansen cases who had attended leprosy outpatient department during the period of six years (2011 to 2017) were studied. A detailed clinical history including age, sex, duration of disease, previous treatment taken, family history of contact and mode of development of the disease were noted. Clinical examination including a thorough general examination, detailed dermatological examination was done. All the particulars about the patches, trophic changes if any, were recorded. Palms, soles, hair, nail and mucous membrane were examined and findings recorded. Involvement of nerves and other systems were noted. Slit skin smear (SSS) for bacteriological and morphological Index and skin biopsy were performed in all. The clinical findings, SSS findings and histopathology findings were recorded, analyzed and correlated. All patients were treated with MB MDT for one year and followed up for 2 to 3 years.

Results

All eleven cases were males in the age group ranging from 22 to 40 years. Duration of the disease varied from 1 month to 5 years. The summary of the clinical, bacteriological and histological findings is presented in Table 1. Cutaneous and subcutaneous nodules were the commonest skin lesions found in 10 patients (91%). Papules in 8 patients (72%), plaques in 4 patients (36%) hypo pigmented patches in 2 patients (18%) and ulceration in 1 patient (9%) were seen (Figs 1 to 4).

No mucosal lesions were seen in any of our patients. The commonest anatomical site involved was arms (81%). Other areas of involvement were back (72%), chest (45%), thigh (45%), forearms



Fig 1 : Papules characteristic of histoid leprosy



Fig 2 : Multiple discrete, shiny, succulent histoid nodules on normal skin



Fig 3 : An ulcerated plaque studded with multiple papules over the elbow

Table 1 : Clinical, bacteriological and histological findings of patients studied

Case No.	Skin lesions	Nerve involvement B/L	Glove & stocking anaesthesia	Trophic change	Slit skin smear	Deformity	Histology	De novo/LL with Histoid
1	Nodules, papules	Ulnar, RCN, LPN, PTN	-	-	4+	-	Histoid	De novo
2	Papules, plaques, ulcer	Ulnar, RCN, LPN	+	-	4+	-	Histoid	De novo
3	Nodules, papules	Ulnar, RCN, median, LPN	-	-	4+	-	Histoid	De novo
4	Nodules, papules	RCN, LPN, DPN PTN	-	-	5+	-	Histoid	De novo
5	Hypopig. patches, nodules	GAN, ulnar, RCN, LPN, PTN, sural	-	-	5+	-	Diffuse granuloma/ Histoid	LL with histoid
6	Plaques, nodules, papules	GAN, ulnar, median, RCN, LPN PTN, sural	+	+	6+	-	Diffuse granuloma/ Histoid	LL with histoid
7	Nodule, papules, plaque	Ulnar, LPN, PTN,	-	-	5+	-	Histoid	De novo
8	Nodules, infiltrated skin	GAN, ulnar, RCN, LPN, PTN	-	+	6+	-	Diffuse granuloma/ Histoid	LL with histoid
9	Hypopig. Patches, papules, nodules	Ulnar, PTN	-	-	5+	-	Histoid	De novo
10	Nodules, papules	Ulnar, median, RCN, LPN	+	+	4+	-	Histoid	De novo
11	Nodules, plaques, infiltrated skin	Ulnar, median, RCN, LPN, PTN	-	-	5+	-	Histoid	LL with histoid

[GAN – Greater auricular nerve, RCN – Radial cutaneous nerve, LPN – Lateral popliteal nerve, PTN - Posterior tibial nerve]

(36%), abdomen (27%), face (27%), legs (27%), hands (18%) and elbow (9%) in descending order of frequency.

All our patients had thickening of more than two peripheral nerves. Ulnar nerve was the most commonly affected followed by lateral popliteal



Fig 4 : Hypopigmented patches with Histoid lesions

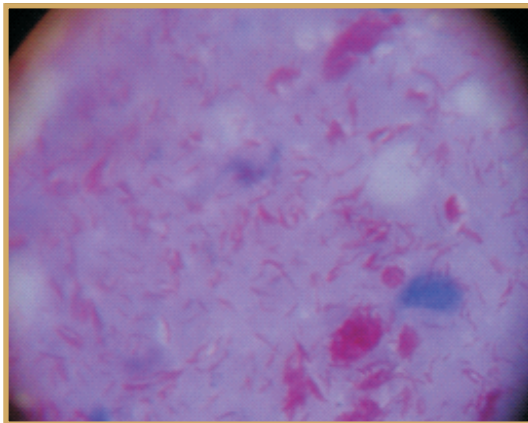


Fig 5 : Slit skin smear showing multiple elongated bacilli

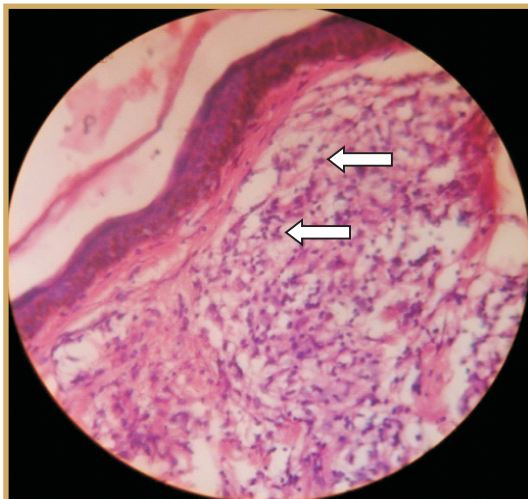


Fig 6 : HPE showing spindle shaped cells (H&E X 400)

nerve. Three patients had glove and stocking type of anesthesia and infiltration of skin.

In all patients, slit skin smear (by Ziehl Neelsen stain) revealed abundant uniformly stained organisms with tapering ends seen singly or in clusters (Fig 5).

Histopathology showed thinned out epidermis, Grenz zone and collection of spindle shaped histiocytes in a whorl like pattern (Fig 6).

Out of 11 patients, 7(63%) were denovo cases i.e. development of Histoid lesions without evidence of any type of Hansen's disease.

Only 3 patients (27%) had prior inadequate treatment with Dapsone for few months to 1 year. One patient took multi drug therapy few years back. One patient, who was diagnosed as borderline lepromatous leprosy, did not turn up for treatment and presented with histoid lesions two years later. It is noteworthy that his son developed borderline Tuberculoid Hansen. Seventy three % of our patients (8/11) developed Histoid Hansen without any history of treatment and 27% (3/11) developed histoid lesions after irregular treatment.

During treatment, one patient developed features of type II reaction (ENL), manifesting as erythematous and evanescent nodules, fever, joint pain and pedal edema after 5 months of treatment. He also had features of uveitis.

Repeat SSS after completing one year MB MDT showed long slender solid bacilli with tapering ends in one patient who had not responded clinically also. So MB MDT was continued for one more year for this patient. At the end of two years SSS showed fragmented bacilli and his skin lesions resolved.

Discussion

Histoid Hansen was first described by Wade in 1960 as a bacillary rich leproma (Wade 1963).

Earlier it was reported to occur in patients who relapsed after Dapsone monotherapy or de novo also (Sehgal & Srivasatava 1985, Sehgal et al 2005). Histoid leprosy is considered as a well recognized expression of multibacillary leprosy, characterised by multiple discrete, shiny, smooth, succulent, skin coloured painless papules on normal appearing skin. Pathogenesis of Histoid Hansen's disease is yet to be resolved. Many factors like genetic factors, immune response and treatment determines the manifestations of Histoid leprosy (Kaur et al 2009).

Studies by Singh et al (1983) and Sehgal et al (1984) reported that 20 - 40 year age group was the most commonly affected. Similarly in our study, patients were of the same age group. Predominant male involvement is comparable with other studies. As per other studies, we also noted nodules and subcutaneous nodules to be the commonest presentation, followed by papules and plaques. The commonest sites (arms and back) of involvement and mucosal involvement (none) correlate with the study by Kaur et al (2009) but facial involvement was seen in only 27% of our cases.

Most of our patients were previously untreated and denovo cases. Only 3 out of 11 cases had taken anti-leprosy treatment earlier. Usually histoid leprosy is considered to develop after Dapsone monotherapy. Our study also shows that Histoid may occur in untreated as well as previously treated cases as reported by Sehgal et al (2005) and Kalla et al (2000).

Erythema Nodosum Leprosum is considered rare in histoid leprosy. There are only few published case reports of ENL in histoid leprosy (Sehgal & Srivastava 1987, Sharma et al 2002). We had one case of ENL reaction after starting MB MDT. Two years MB MDT was suggested by few studies (Sehgal et al 2005). In our study one patient who did not respond to the conventional one year MB

MDT resolved after one more year of MB MDT. This leads to the possibility of considering extended therapy for such unresponsive cases.

Conclusion

Histoid Hansen, a multibacillary infective form of leprosy, with male predominance, can occur de novo and in untreated Hansen's disease patients. Awareness about the clinical features, characteristic histology and early detection of Histoid Hansen is of utmost importance. It is essential to assess bacteriological and morphological indices at the end of the treatment in all Histoid Hansen cases. Continuation of treatment may be decided based upon the bacterial and morphological indices.

References

1. Bhukya AS, Reddy BSN (2016). De novo Histoid leprosy in a dwarf with lesions at unusual site. *Indian J Lepr.* **88**: 39-42.
2. Ghorpade AK (2011). Transepidermal elimination of *Mycobacterium leprae* in histoid leprosy: A case report suggesting possible participation of skin in leprosy transmission. *Indian J Dermatol Venereol Leprol.* **77**: 59-61.
3. Kalla G, Purohit S, Vyas MC (2000). Histoid, a clinical variant of multibacillary leprosy: report from so called endemic areas. *Int J Lepr Other Mycobact Dis.* **68**: 267-71.
4. Kaur L, Dogra S, De D Saikia UN (2009). Histoid leprosy: a retrospective study of 40 cases. *India. Br J Dermatol.* **160**: 305-310.
5. Meena DK, Sharma S and Chauhan P (2017). De-novo Histoid Leprosy Masquerading as Cryptococcosis: A Case Report. *Indian J Lepr.* **89**: 45-48.
6. Nair SP, Kumar GN and Mathew R (2016). Histoid leprosy presenting with keloid like lesions. *Indian J Lepr.* **88**: 117-121.
7. Palit A, Inamadar AC (2007). Histoid leprosy as reservoir of disease: a challenge to leprosy elimination. *Lepr Rev.* **78**: 47-9.
8. Sehgal VN, Ghorpade A, Saha K (1984). Urban leprosy an appraisal from Northern India. *Lepr Rev.* **55**: 159-66.

9. Sehgal VN, Srivastava G (1985). Histoid leprosy. *Int J Dermatol*. **24**: 286-92.
10. Sehgal VN, Srivastava G (1987). Status of histoid leprosy – a clinical, bacteriological, histopathological and immunological appraisal. *J Dermatol (Tokyo)*. **14**: 38-42.
11. Sehgal VN, Aggarwal A, Srivastava G et al (2005). Evolution of histoid leprosy (*de novo*) in lepromatous (multibacillary) leprosy. *Int J Dermatol*. **44**: 576-8.
12. Sharma SK, Rath N, Gautam R K et al (2002). Histoid leprosy with ENL reaction. *Indian J Dermatol Venereol Leprol*. **68**: 342-3.
13. Singh VV, Singh G, Kaur P, Pandey SS (1983). Histoid leprosy in Varanasi. *Indian J Dermatol Venereol Leprol*. **49**: 160-1.
14. Wade HW (1963). The histoid variety of lepromatous leprosy. *Int J Lepr*. **31**: 129-43.

How to cite this article : Subha R, Ananthi M and Suganthi V (2018). Scenario of Histoid Hansen at a Tertiary Care Hospital in South India. *Indian J Lepr*. **90** : 235-240.