SECONDARY AMYLOIDOSIS IN LEPROSY
(A case report)

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Summary

A case of amyloidosis secondary to lepromatous leprosy has been discussed. He had proteinuria, Congo red retention 64 per cent (first hour), hyperglobulinaemia, and renal biopsy revealed amyloid deposits. Factors responsible for amyloidosis are highlighted.

Amyloidosis secondary to leprosy though reported to be common in Western countries[1,2,3], is regarded rare in India and other tropical countries[4,5,6]. The factors responsible for such a varied incidence in different countries are not yet fully understood, although genetic predisposition, dietetic factors, or associcated venues of immune stimulation have been blamed from time to time. In the clinical course of a lepromatous leprosy patient no single factor can be blamed for the production of subesquent amyloidosis[2].

Case Report

40 years male patient suffering from lepromatous leprosy was admitted to Skin and V. D. Wards for investigations of proteinuria and possible secondary amyloidosis. He complained of extensive loss of sensations in all the extremities for 18 years, deformity of hands and feet, and multiple ulcers on the skin for 5 years.

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Examination revealed a moderately built patient having no fever, anaemia or oedema. Pulse was 80 per minute and regular. Blood pressure was 130/85 mmHg. General systemic examination revealed no significant abnormalities. Skin showed multiple ulcers on back, chest and limbs and multiple infiltrated patches on face, back, chest and limbs. These were symmetrical, dull red in colour and had ill-defined borders. They were not anesthetic. Extensive absorption defects were seen on hands and feet. All superficial sensory modalities were lost below elbows, on lower 1/3 of legs and feet as well as on chin and low back.

Ulnar and lateral popliteal nerves were thickened and slightly tender on both sides.

Dietetic History : Patient is a non-vegetarian, with a total caloric intake 2535/day.

—Carbohydrate	50.92%
—Proteins	15.08%
—Fats	34.00%
(Unsaturated	7%)
(Saturated	27%)

Investigations:

Lepromin test was Negative Haemoglobin 13.2 G%. Total leucocyte counts 7200/c. mm. with differential counts of Poly. 62%, Lym. 30%, Mono. 7%, Eos. 1%. Urine showed moderate proteinuria. Serum Proteins revealed total of 7.65 Gm%, Albumin was 39.88%, α_1 Globulin 3.66%, α_2 Globulin 6.48%, β Globulin 10.18%, γ Globulin 39.80%, Congo-red tissue retention 64% (1st hour) Blood urea 30 mg%. Plain X-ray abdomen NAD X-ray chest NAD.

Histopathology :

Skin biopsy — Suggestive of lepromatous leprosy.

Liver biopsy— H & E staining NAD. Ziehlneelson's staining No evidence of Lepra bacilli.

Methyl violet staining - No evidence of amyloid deposits.

Kidney biopsy — H & E staining-Amyloid deposits seen. Ziehlneelson's staining No evidence of Lepra bacilli.

Methyl violet staining -Amyloid deposits seen.

Discussion

The development of amyloidosis in leprosy is reported to occur only in lepromatous type of the disease[1,3,6,7]. The argument put forward for the confinement of this complication only to the lepromatous type of disease is that the reticulo-endothelial system gets directly stimulated by the entrapped and multiplying bacilli in lepromatous leprosy, whereas such reticuloendothelial stimulation and irritation by entrapped and multiplying bacilli does not occur in tuberculoid form of the disease[2,7]. It is also well known that fever due to ENL reactions is common in lepromatous leprosy and hyperglobulinaemia in initial stages is more pronounced in lepromatous than in tuberculoid form of the disease. These factors suggest presence of a more generalised and more intense immune stimulation in lepromatous than in tuberculoid type of disease which may, therefore, be contributory in the initiation and development of amyloid change in lepromatous leprosy. Williams et al[2] stated that extensive and multiple suppurative ulcers were also of importance in producing the amyloid state in patients with lepromatous leprosy. They further contemplated that a high intake of saturated fats predisposed to amyloid change.

The present case of lepromatous leprosy who showed evidence of amyloidosis on renal needle biopsy, had the disease for 18 years, had hyperglobulinaemia, and recurrent suppurative lesions in the form of multiple ulcers for five years. Patient was a non-vegetarian with a high caloric intake and diet containing nearly 34% of fats—out of which 27% was saturated and 7% unsaturated. The factors responsible for the production of amyloidosis in this case, therefore, are long duration of the disease, with frequent stimulation and irritation of reticulo-endothelial system by entrapped and multiplying bacilli and presence of secondary suppuration. The saturated fats consumed daily by this patient was only 27%. It is difficult to be sure if dietary factors played any role in the production of amyloidosis in this patient.

REFERENCES

1. Sh ttleworth JS and Ross H : Secondary amyloidosis in Leprosy. Ann Intern Med, 45 : 23, 1956.

2. Williams RC, Cathcart ES and Calkins E et al : Secondary amyloidosis in

lepromatous leprosy, Possible relationship of diet and environment. Ann Intern Med, 62: 1000, 1965.

3. Powell CS and Swan LL: Leprosy-Pathologic changes observed in fifty consecutive necropsies. Am J Path, 31 : 1131, 1965.
4. Kobayashi W: Uber di viscerale Lepra, monographia actorum dermatologicorum, Dermatologica, S Karger, AG Koyoto, A series No. 4, 1929.
5. Masanti JG: Secondary amyloidosis in Leprosy, Amyloidosis and Lepra reaction. Medicina (B Aires), 19: 1, 1959.
6. Krishnamurthy S and Job CK: Secondary amyloidosis in leprosy. Int J Lepr, 34: 155, 1966.
7. Sachdev JC, Puri D and Bansal S: Secondary amyloidosis in Leprosy, Lepr India, 41 : 73, 1969.

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