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Secondary Amyloidosis in Leprosy

S. Krishnamurthy and C. K. Job²

Amyloidosis is rare in Oriental people, particularly in India (5,7). Even though there is a high incidence of chronic suppurative disease, secondary amyloidosis is not seen as often as in the West. Leprosy being a disease of many years' duration, insidious in nature and destructive to body tissues, one might expect a high incidence of amyloidosis to be associated with it, and indeed, studies in the West by Powell and Swan (⁶), and Shuttleworth and Ross (⁸) have proved this to be true. The present study has been undertaken to assess the incidence of secondary amyloidosis associated with leprosy in the autopsied cases in our hospital in South India.

MATERIALS AND METHODS

Among 2,848 consecutive autopsies performed from June 1953 to May 1964 at the Christian Medical College and Hospital, Vellore, India, there were 27 cases of leprosy. In two postmortem examinations the tissues showed extensive autolytic changes, and hence were not included in this study. Of the remaining 25 cases, selected for review, 17 were of the lepromatous type, 5 were nonlepromatous, and 3 were unclassified. The tissues examined were of kidney, liver, spleen and where available, the adrenal. These are the organs that show secondary amyloid deposits most frequently. Paraffin sections six microns thick were made and stained with the hematoxylin and eosin and congo red stains. The sections stained with congo red were also examined by polarized light microscopy. Amyloid material gives a greenish colored fluorescence when examined under polarized light after staining with congo red $(^{1})$. Tissues found positive for amyloid by the methods, cited above were stained with methyl violet to confirm the findings.

OBSERVATIONS

With these methods, specimens from two autopsies out of 25, showed the presence of amyloid, amounting to 8 per cent of autopsied cases of leprosy studied. A short history, and the relevant portion from the postmortem report of each of these cases, are given below:

P.M. 2505. The patient was a 36-yearold male, with clinically diagnosed lepromatous leprosy for 16 years. Associated with the lepromatous leprosy, the patient had a nephrotic syndrome with anasarca, albuminuria and proteinemia.

At autopsy the body was seen to be that of a fairly well built adult male. Pitting edema was present over the upper and lower extremities and the abdominal wall. Each hand had a claw-hand deformity. The ear lobes and skin of the face were markedly thickened.

The kidneys were slightly larger than normal. The external surface was pale and mottled. The cut surface showed poor demarcation between cortex and medulla. The cortex was pale yellow, with transverse red streaks. There was bilateral testicular fibrosis. Other organs, including the liver, spleen, and adrenals, were grossly normal.

Microscopic examination of sections of kidneys stained with hematoxylin and eosin, showed deposits of a pink-staining homogeneous hyaline-like material in the basement membrane of many of the glomeruli (Fig. 1) and in the walls of the capillaries. Similar material was seen in the external walls also. The spleen showed evidence of amyloid deposit in the walls of the small arteries. Extensive and diffuse amyloid infiltration of its pulp was also seen (Fig. 2). The liver showed amyloid change in the arterial walls in the portal triads and in the walls of the sinusoids

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²⁸S. Krishnamurthy, Candidate for M.B.B.S. and C. K. Job, M.D., M.C. Path., Professor of Pathology, Christian Medical College and Hospital, Vellore, S. India.



FIG. 1. Glomeruli showing extensive deposition of amyloid (P.M. 2505).

FIG. 2. Spleen with amyloid deposits in the pulp (P. M. 2505).

(Fig. 3). The adrenal cortex had amyloid deposits between the cords of cells of the zona glomerulosa, the zona fasciculata, and the zona reticularis (Fig. 4). In many areas the cortical cells showed evidence of pressure atrophy. The postmortem record was reviewed for evidence of amyloid elsewhere in the body. None was present. The cause of death was renal failure due to amyloid infiltration, secondary to lepromatous leprosy.

P.M. 3112. The patient, a 38-year-old male, an arrested case of lepromatous lep-

rosy, had been treated for generalized edema and albuminuria for one year.

At postmortem examination, the body was seen to be that of an adult male with edema of the extremities and face. There were bed sores on the hips. The left pleural cavity had 500 ml. and the right pleural cavity 800 ml. of clear yellow fluid. The kidneys were pale, but no significant gross lesion was found. The testes were small, fibrous and atrophic. All other organs showed no significant lesions grossly.

Histopathologic examination showed ex-



FIG. 3. Liver with amyloid deposits in the walls of the sinusoids (P.M. 2505).

FIG. 4. Amyloid deposits in the adrenal cortex (P.M. 2505).

tensive deposition of amyloid in most of the glomeruli of the kidneys. In some places it completely occluded the glomerular tuft. Some small arteries also showed amyloid material in the walls. There were a number of hyaline casts in the tubules. The liver showed amyloid deposit in the arteries of the portal triad. Deposition of amyloid in the walls of the sinusoids was also noted in patchy areas. The spleen showed diffuse amyloid deposition in the Malpighian corpuscles and in the pulp. All three layers of the adrenal cortex showed diffuse amyloid deposits between the parenchymal cells. Amyloid change, as noted from the postmortem records, was present also in the testes. The cause of death was renal failure due to amyloidosis, secondary to lepromatous leprosy.

DISCUSSION

Shuttleworth and Ross (⁸), in their autopsy study of 18 patients, found 10 cases of amyloidosis. Powell and Swan (⁶), who analyzed 50 consecutive necropsies on lepromatous leprosy patients, found secondary amyloid change in 45 per cent of cases. These reports show that secondary amyloid

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In a study of the first 100 consecutive autopsies performed in persons dying with leprosy in Bombay, however, no amyloid change was detected in any of them $(^4)$. Chitkara *et al.* $(^2)$, in their review of Indian literature, found 50 autopsied cases of amyloidosis, but none of them associated with leprosy. Cochrane $(^3)$ states that, except in Caucasian races, amyloidosis is extremely rare as a terminal complication in leprosy.

In the present study amyloid change was found extensively in two of 25 cases. Both were of the lepromatous type. It is interesting to note that among the other 23 cases, nine had tuberculous lesions as well, but no amyloid change was detected. On comparison of the figures with those of Western countries, it is seen that the incidence of secondary amyloidosis in leprosy in India is very low.

SUMMARY

In a review of 25 autopsied cases of leprosy two were found to have generalized secondary amyloidosis (8%). Both patients had lepromatous leprosy. It is concluded that amyloidosis may occur in Indian leprosy patients, but is not so common as in the Western countries.

RESUMEN

En la revisión de 25 casos autopsiados de lepra, fueron encontrados dos con amiloidosis secundaria generalizada (8%). Ambos pacientes tenían lepra lepromatosa. Se concluye que la amiloidosis puede ocurrir en pacientes leprosos Indios, pero que no es tan común como en los países occidentales.

RÉSUMÉ

Vingt cinq autopsies de cas de lèpre ayant été passées en revue, deux ont été trouvées atteints d'amyloïdose secondaire généralisée (8%). Les deux malades avaient été atteints de lèpre lépromateuse. Il en est conclu que l'amyloïdose peut survenir chez des Indiens souffrant de lèpre, mais qu'elle n'est pas aussi commune que dans les pays occidentaux.

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