RENAL INVOLVEMENT IN HANSENIASIS: A LONGSTANDING CONCERN

Envolvimento renal na hanseníase: uma preocupação de longa data

Vitorino Modesto dos Santos[1], Lister Arruda Modesto dos Santos[2]

ABSTRACT

The growing number of severe multibacillary cases of hanseniasis due to late diagnosis has contributed to the prevalent high burden of this disease in low-income and developing countries. Unfavorable courses have been associated with lesions of internal organs as the case of kidneys. Renal amyloidosis and chronic renal failure are among the main causes of poor outcomes. These comments aim to highlight the role of renal biopsy studies in patients with lepromatous hanseniasis.

Key-words: Hanseniasis, amyloidosis, renal failure

RESUMO

O crescente número de casos multibacilares graves da doença de Hansen, devido ao diagnóstico tardio, contribuiu para a alta carga prevalente dessa doença nos países em desenvolvimento e de baixa renda. Cursos desfavoráveis têm sido associados a lesões de órgãos internos, como o caso dos rins. Amiloidose renal e insuficiência renal crônica estão entre as principais causas de maus resultados. Esses comentários têm como objetivo destacar o papel dos estudos de biópsia renal em pacientes com hanseníase lepromatosa.

Palavras-chave: Hanseníase, amiloidose, insuficiência renal.

[1] Professor Adjunto I da Universidade Católica de Brasília (UCB) e Preceptor do Departamento de Medicina Interna do Hospital das Forças Armadas (HFA).
[2] General Surgery, State Workers Hospital, São Paulo-SP. Preceptor of General Surgery

To the Editor,

We read with interest the article done by Mushtaq *et al*. (2019) with the title "Trends and patterns of leprosy over a decade in a tertiary care hospital in Northern India: A retrospective analysis", published recently. They performed an epidemiological study about leprosy from 2005 to 20014 including 743 individuals and detected an increase in multibacillary cases and in grade 2 disability (20.1%) in comparison with the previous decade.[1] Male patients were 77.4% of the total; the age range was 5-90 years and the mean age was 36.43 ± 14.92 years. The mean duration of the disease was 16.38 ± 25.58 months, and the median of eight months. The distribution of the cases by frequency showed: multibacillary (627: 84.4%); borderline tuberculoid (34.3%), borderline lepromatous (25.6%), mid-borderline (14.0%), lepromatous (11.0%), pure neuritic (7.1%), histoid (3.6%), tuberculoid (2,0%), and indeterminate (1.5%).[1] The authors emphasized the current growing trend of lepromatous and multibacillary infections as well as of the associated higher grade of disability and the smear-positive cases. They highlighted the role of delayed diagnosis in the continuous transmission of the disease. This study contributes to better understand that leprosy is very far from being eliminated yet. Moreover, the crescent number of more severe cases is indicative of poorer outcomes related to the lesions of internal organs, in special the changes in the kidneys causing renal failure. Currently, one concern is about the scarce renal histological studies in lepromatous leprosy (LL).

Half a century ago, Brazilian authors reported a 63-year-old man with nephrotic syndrome, arterial hypertension, and LL treated 25 years before.[2] He had persistent elevated levels of urea, creatinine, and albuminuria. There were negative searches for Mycobacteria in the nasal mucus and skin slit smears from ear lobules, and biopsy sampling from the skin and the kidney. The skin histopathology showed intense adnexal atrophy, mild epidermal atrophy, subepidermal grenz zone (band of Unna), and dermal lesions rich in clear cells of hyperchromatic nuclei and vacuolar cytoplasm (Virchow cells). Worthy of note was the renal histopathology consistent with the diagnosis of secondary amyloidosis, a condition scarcely described in LL before 1969.[2] The stain methods were hematoxylin-eosin, Gomori reticulin, Congo red, periodic acid-Schiff, and cresyl violet. There were mesangial deposits of an amorphous eosinophilic and laminar substance causing variable occlusion of glomerular capillary vessels as well as of the afferent and interlobular arterioles; the renal tubules were markedly dilated and with hyaline cylinders.[2] He underwent dialysis, but his death occurred two months after hospital discharge. The authors called attention to albuminuria in patients with LL; which can constitute an early clinical sign indicative of secondary amyloidosis.

More recent studies about renal involvement in LL have described granulomas,[3] cortical cysts and an increased renal cortical echogenicity,[4] in

2238-5339 © Rev Med Saúde Brasília 2020; 9(2):334-336

addition to the presence of amyloidosis.[5] Mittal *et al*. (2019) reported a 47-years-old man with borderline tuberculoid leprosy and chronic interstitial nephritis plus chronic granulomatous inflammation. He had albuminuria (1.6 gm/ day) and renal failure, skin and renal biopsies showed epithelioid granulomas, giant cells, lack of caseation and negative Fite stain. There was a rapid clinical improvement with multidrug therapy.[3] The authors emphasized the rare occurrence of renal granulomas secondary to leprosy. Ozturk *et al*. (2017) evaluated 32 Turkish patients with LL and found a higher level of urea, creatinine, and leukocyturia than the control group of 35 individuals.4 Moreover, both the images of cortical cysts and of increased renal cortical echogenicity were significantly more frequent in the people with diagnosis of LL. The authors highlighted the role of renal function evaluation in the prevention of renal failure, which is a major potential cause of death among patients with LL. Sanz-Martin *et al*. (2016) reported a 50-year-old Colombian woman with arterial hypertension and nephrotic syndrome.[5] She had diagnosis of leprosy when she was six years old and was treated during 15 years. Renal biopsy data were consistent with interstitial fibrosis, tubular atrophy and the diagnosis of AA amyloidosis. Her renal function was maintained at normal levels without special support and the nephrotic syndrome improved spontaneously.[5] The authors emphasized the early effective therapy to avoid renal failure.

As a whole, the ancient and current commented works have contributed to clear the role of renal changes in leprosy. Advances in the field of molecular biology represent useful tools in the setting of early diagnosis and prompt adequate management of this challenging condition.

REFERENCES

1. Mushtaq S, Dogra N, Dogra D, Faizi N. Trends and patterns of leprosy over a decade in a tertiary care hospital in Northern India: A retrospective analysis. Indian J Dermatol Venereol Leprol 2019;86 (2):141-9. PMID: 31736465.

2. Mittal A, Barjatiya M, Gupta K, Jain M, Meena M. Renal granulomas in Hansen's disease. J Assoc Physicians India 2019;67(9):98-9.

3. Ozturk S, Ozturk T, Can I. Renal involvement in leprosy: evaluation of patients in Turkey. Adv Dermatol Allergol 2017; 34(3):240–4. PMID: 28670253.

4. Santino Filho F, dos Santos VM, Saldanha LF, Henrique S, Brasil MH. [Renal amyloidosis in lepromatous leprosy. Case report]. Hospital (Rio J) 1969;75(2):563-9.

5. Sanz-Martín N, Samillán-Sosa Kdel R, De Miguel J, Martínez-Miguel P. Renal amyloidosis in leprosy, an infrequent cause of nephrotic syndrome in Europe. BMJ Case Rep 2016;2016.