Renal Amyloidosis in Leprosy. Functional and Histopathologic Studies[1]

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Kidney involvement in leprosy is frequent. A wide spectrum of pathologic manifestation has been reported ([5, 8, 11, 12, 15, 21, 23, 24]). Chronic pyelonephritis is commonly observed ([5, 8, 23]). Secondary amyloidosis, however, occurs almost only associated with the lepromatous type of leprosy ([12, 15, 18, 19, 21, 24]), the kidney being the organ more often involved. The patients so affected die eventually in uremia.

The amyloid deposit may be found in glomeruli, tubules and vessels. Liver involvement with amyloidosis is frequent as is also the digestive tract. Tests to evaluate the function of these organs do not clarify the severity of the functional derangement nor the structures involved. The kidney, nevertheless, offers unique characteristics. A virtual functional dissection can be performed by means of different tests. Within certain limits the inulin clearance evaluates the glomerular function, the functional capacity of the tubules may be estimated by the ability to eliminate an acid overload, to concentrate and to dilute the urine, as well as by the maximal capacity to transport glucose and para-amino-hippuric acid, etc. The clearance of diodrast and *p*-amino-hippuric acid serves to measure effective renal plasma flow.

In those patients with evidence of renal damage disclosed by albuminuria and alterations of the urinary sediment the suspicion of the existence of amyloid deposits should be raised, especially if no other change responsible for the disorder is found.

The present investigation was initiated to evaluate the degree of functional disturbance of patients with lepromatous leprosy presumed to have renal amyloidosis because of albuminuria and abnormal urinary sediment and having no evidence of other disease process possibly responsible for these changes,[3] and to relate this disturbance to the different parts of the nephron. Twenty-one patients were investigated. Eleven refused to be biopsied and three did not have their diagnosis of renal amyloidosis confirmed by histologic criteria. Only the seven cases histologically proven to have amyloidosis are the object of this report.

PATIENTS, MATERIALS AND METHODS

Description of the patients. *Case 1.* White male, 51 years, with lepromatous leprosy of four years duration, whose first manifestation of the disease was an episode of *erythema nodosum leprosum* (ENL). He was admitted a number of times for reactive phases of ENL. Blood pressure was 120/80. Liver edge was felt one centimeter below the right costal margin. Urinalysis revealed proteinuria of 0.04 gm/l (+) and normal sediment. Total proteins 6.2 gm/100 ml (albumin 3.06, globulin 3.14 gm/100 ml). Blood creatinine 1 mg/100 ml, hemoglobin 12 gm/100 ml. Red blood cell count was 4,300,000/mm^3.

Case 2. Japanese farmworker from Okinawa, age 62 years, lepromatous leprosy for 19 years and blind for 15 years. He suffered numerous episodes of ENL and developed cardiac failure three years prior to these studies. A chest x-ray then revealed infiltration of the lower lobe of the left lung. Blood pressure was 140/90. Urinalysis showed proteinuria of 0.148 gm/l and hya-

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[3] Previous history of glomerulonephritis or evidence of active pyelonephritis revealed by a bacterial count of more than 100,000 per milliliter of urine, etc.

line, granular and waxy casts; blood creatinine 2.3 mg/100 ml.

Case 3. Negro male, 49 years old, farmworker, lepromatous leprosy for 16 years. For the previous six years he had a number of admissions for treatment of plantar ulcers. He never had any episode of ENL. Blood pressure was 200/110. Stool examinations revealed ova of *Ascaris lumbricoidis, Ancylostoma duodenale* and larvae of *Strongyloides stercoralis.* Total protein was 4.1 gm/100 ml (albumin 1.6, globulin 2.5); urinalysis revealed 5.2 gm/l, hemoglobin and waxy casts, blood creatinine was 9 mg/100 ml.

Case 4. Negro female, 64 years old, housewife, with lepromatous leprosy for 34 years; living in a sanatorium since 1944. She had frequent episodes of ENL and developed plantar ulcers. Blood pressure was 160/90. Urinalysis revealed proteinuria of 5.1 gm/l, granular, hyaline and waxy casts. Blood creatinine was 8.7 mg/100 ml. Total proteins 2.5 gm/100 ml. Red blood cell count was 3,700,000/mm^3, hemoglobin 9 gm/100 ml. She died about one month after the clearance studies.

Case 5. White male, 49 years old, farmworker, with lepromatous leprosy for 27 years. He suffered many episodes of ENL and presented plantar ulcers. Three years prior to this study the right lobe of the thyroid increased in size but there were no associated functional disturbances. Blood pressure was 190/110. Urinalysis disclosed specific gravity 1012, proteinuria of 2.3 gm/l, granular, hyaline and waxy casts, blood creatinine 7.8 mg/100 ml, red blood cell count was 3,400,000/mm^3 and hemoglobin 10 gm/100 ml, hematocrit 28%.

Case 6. White male, 69 years old, farmworker with lepromatous leprosy for 20 years. He suffered many episodes of ENL. His vision has been declining since the beginning of the illness. He never had plantar ulcers. Blood pressure was 100/60. The urine was positive for protein, and the sediment contained hyaline, granular and waxy casts. Blood creatinine was 1.8 mg/100 ml. He died of bronchopneumonia some time after these studies.

Case 7. White male, 49 years old, acquired lepromatous leprosy at the age of eight years. Parents, one brother and one sister also had the disease. He was considered cured in 1953. During his illness he had many episodes of ENL. Plantar ulcers occurred on many occasions; at the time of the renal studies they were healed. Blood pressure was 200/120. The urine was positive for protein and the sediment contained hyaline, granular and waxy casts. Blood creatinine was 2.3 mg/100 ml.

Renal studies. The clearance determinations for inulin and *p*-amino-hippuric acid (PAH) were done simultaneously. Two or three clearances were performed sequentially and their average reported as the result. The urine was collected for periods of 30 minutes for each clearance. The collections were begun after a steady plasma concentration of inulin and PAH acid had been reached, between 10 and 20 mg/100 ml for the former and between 1 and 2 mg for the latter. The bolus of the initial injection containing inulin and PAH acid and the concentration of these substances in saline infusions for maintaining these plasma levels were calculated on the basis of previous clearance of creatinine, taking this clearance as similar to the one of inulin. The clearance of PAH acid was estimated as being five times that of inulin. Treatment with sulfones was interrupted during the week preceding the day of the test in order to avoid false results in the determination of the concentration of PAH acid. Beginning two hours before and during the test, 200 ml of water was given every half hour. Special care was observed in collecting the urine which was obtained through a Foley catheter inserted into the bladder. The latter was fully emptied prior to the collection and washed with saline. This procedure was repeated at the end of each period of urine collection when air was also introduced into the bladder to assure full emptying. Blood was drawn, using heparin as anticoagulant, at the beginning, middle and end of each collection. Before the injection of inulin and PAH acid, enough plasma was obtained to serve as the blank and also to prepare the standard curves of these substances. This procedure was followed in all cases. The determination of the concentration of inulin and PAH acid followed the standard methods ([20, 22]).

The urine concentration test was done according to Fishberg ([7]) and the osmolarity was measured in the osmometer of Advanced Instruments, immediately after the samples

TABLE 1. *Clinical data and renal function studies.*

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7
Sex	Male	Male	Male	Female	Male	Male	Male
Age	51	62	49	64	49	69	49
Duration of illness (years)	4	19	16	34	27	20	41
ENL	4	many	none	several	many	many	?
BP (highest mm Hg)	120/80	140/90	210/110	160/90	190/110	140/80	200/120
Plantar ulcers	no	no	yes	yes	yes	no	yes
Blood creatinine (mg/100 ml)	1.0	2.3	9.0	8.7	7.8	1.8	2.3
Total protein (gm/100 ml)	6.2		4.0	2.5	2.3		
Albumin (gm/100 ml)	3.06	2.64	1.6				
Globulin (gm/100 ml)	3.14	1.70[a]	2.4				
Proteinuria (gm/l)	0.04	0.14	5.2	5.1	2.3	++++	0.16
Casts — Hyaline	absent	present	absent	present	present	present	present
Casts — Granular	absent	present	absent	present	present	present	present
Casts — Cereous	absent	present	present	present	present	present	present
Casts — Hematic	absent	absent	present	absent	absent	absent	absent
Inulin clearance (ml/min)[b]	98.73	42.15	8.89	8.13	11.61	38.88	38.02
PAH clearance (ml/min)	476.12	203.01	10.48	12.70	44.89	109.95	159.40
Filtration fraction	0.207	0.207	0.847	0.640	0.258	0.353	0.238
Maximal urinary concentration (mOsm/l)	921	not done	372	350	362	524	419

[a] Gamma globulin.
[b] Not corrected for body surface.

were obtained. The highest result was taken as the maximal capacity of the kidney to concentrate the urine.

The biopsies were performed percutaneously. The specimens were kept in Zenker's solution until processed. The sections were stained with hematoxylin and eosin and Congo red. The latter preparations were examined under polarized light for determination of green birefringence which is the hallmark of amyloid deposit ([3]). The material of the only case coming to autopsy (No. 6) was preserved in 10% solution of neutral formaldehyde and similarly stained.

RESULTS

Table 1 presents the main clinical data and the results of the functional tests. Table 2 summarizes the main pathologic findings. The biopsy and single autopsy reports on the kidneys follow.

Case 1. Biopsy (Fig. 1). Normal renal architecture. Many glomeruli show a clear mesangial axis with slight increase in the number of cells, usually in small clusters in the middle of a dense acidophilic stroma. The capillary loops are permeable. An artery of small caliber shows discrete, subendothelial fibrous thickening. There is no abnormality in the interstitial tissue. The basal membrane of the medullary tubules is slightly positive for amyloid deposit.

Case 2. Biopsy (Fig. 2). Renal architecture fairly well preserved. There are focal areas of interstitial fibrosis and tubular atrophy. Generalized glomerular amyloidosis. Amyloid deposits are present in the mesangium and basal membrane of the capillary loops, diffusely distributed or concentrated in nodules. The affected glomeruli show hyperplasia of mesangial and endothelial cells. Some glomeruli are totally hya-

TABLE 2. *Major pathologic findings in the kidney.*

		Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7
Glomeruli	Amyloid deposit	–	++	+++	+++	+++	+++	++
	Endothelial cellularity	–	+++	–	+	++	++	–
	Mesangial cellularity	+	+++	–	+	++	++	+
	Neutrophilic exudate	–	–	–	–	–	–	–
	Mesangial sclerosis	–	++	–	–	–	–	+
	Hyalinization	–	++	+	–	+	+	++
	Capsular fibrosis	–	++	++	–	+	+	+
	Adhesions	–	–	–	+	–	–	–
Vessels	Amyloid deposit	–	++	+++	+++	+++	+++	++
	Arteriosclerosis	+	–	–	+	–	+	++
	Arteriolosclerosis	–	–	++	–	–	–	++
	Subendothelial concentric fibrosis	–	–	–	–	–	–	++
Cortical tubuli	Amyloid deposit	–	–	+	++	+++	+	+
	Atrophy	–	+	+++	+++	++	++	+++
Medullary tubuli	Amyloid deposit	+	+++	–	+	–	++	–
	Atrophy	–	+	–	++	–	+	–
Interstitium	Inflammatory infiltration	–	–	++	+	++	+	+++
	Fibrosis	–	+	+++	+++	++	++	++
Renal Architecture		Preserved	Altered (+)	Altered (+++)	Altered (+++)	Altered (++)	Altered (++)	Altered (++)

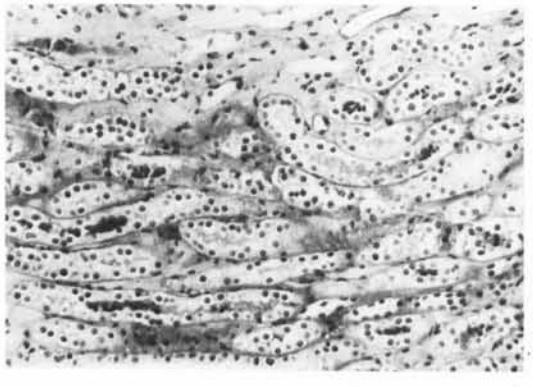

FIG. 1. Case 1. Discrete peritubular amyloidosis. The tubular architecture is well preserved. The amyloid deposit follows the contours of the tubules near the basal membrane. Congo red stain. Magnification ×63.

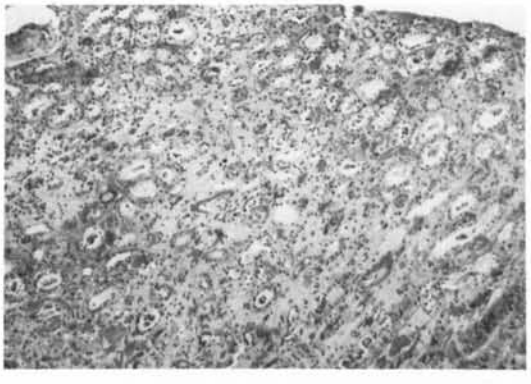

FIG. 2. Case 2. Marked peritubular amyloidosis. Renal medulla shows peritubular deposit deposition of amyloid material in thick and irregular hyaline bundles and areas of atrophy of the tubular epithelium reduced almost only to its contours marked by the amyloid deposit. Congo red stain. Magnification ×25.

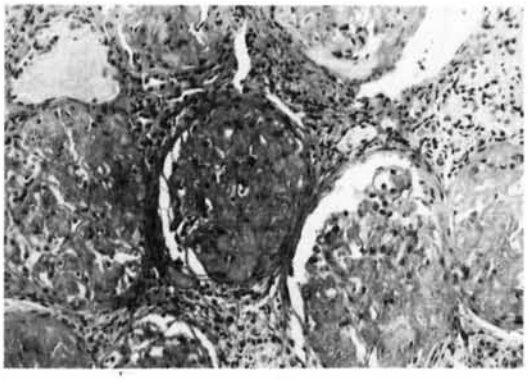

FIG. 3. Case 3. Intense glomerular amyloidosis. Group of glomeruli with diffuse deposit of the amyloid substance. The glomerular tuft is increased in size. The amyloid material located in the mesangial axis entirely blocks the lumen of the loops of the capillary vessels. Discrete capsular fibrosis and atrophy of the adjacent renal tubuli. Congo red stain. Magnification X63.

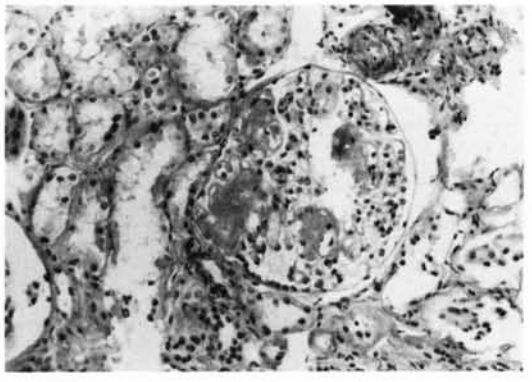

FIG. 4. Case 4. Glomerular and vascular amyloidosis. Glomerulus with focal amyloid deposition in the capillary subendothelium and along the mesangial axis. On top of the glomerulus a small vessel with the amyloid deposit obstructing partially its lumen is seen. Congo red stain. Magnification X63.

linized and present fibrous thickening of the capsules. There is moderate vascular amyloidosis and intense amyloid deposition in the basal membrane of the medullary tubules.

Case 3. Biopsy (Fig. 3). Renal tissue with altered architecture due to interstitial fibrosis and tubular atrophy. Only the cortex is seen in the biopsy fragment. Small foci of mononuclear inflammatory infiltrates are present in the interstitial tissue. There is generalized involvement of the enlarged glomeruli with amyloid deposits. The majority of the glomeruli show hyalinization of the tuft and fibrous thickening of the capsules. Amyloid deposit is also seen in the vascular subendothelium. Some arterioles present hyperplasia of the muscular layer and fibrous thickening of the intima. No amyloid deposit is observed in the tubules.

Case 4. Biopsy (Fig. 4). Altered renal architecture. Diffuse interstitial fibrosis and atrophy of the tubules. There is diffuse but discrete mononuclear inflammatory infiltrate in the interstitial tissue. The enlarged glomeruli are generally filled with amyloid deposits. Foci of hypercellularity are present in the middle of the amyloid substance. Adhesions of the glomerular tuft are also observed. There is intense deposition of amyloid in the subendothelium but discrete deposition in the basal membrane of the cortical and medullary tubules. The small arteries show subendothelium fibrous thickening.

Case 5. Biopsy. Impaired renal architecture. Tubular atrophy and interstitial fibrosis are observed. The glomeruli are enlarged and there is pronounced amyloid deposition frequently associated with hypercellularity of the glomerular tuft, adhesions and proliferation of Bowman's capsule. Rare glomeruli show atrophy and hyalinization. There is intense deposition of amyloid in the vessels and in the cortical tubules. Foci of mononuclear inflammatory infiltrate are seen in the interstitial tissue. There is no medullary tissue in the fragment of the biopsy.

Case 6. Necropsy. The renal architecture is altered. Areas of interstitial fibrosis and tubular atrophy alternate with areas of dilated tubules. There is pronounced deposition of amyloid in the glomeruli, subendothelium of the vessels and in the basal membrane of the tubules, especially in those located in the medulla. Some glomeruli show hyalinization and atrophy with capsular fibrosis. Discrete arteriosclerosis is observed.

Case 7. Biopsy. Small fragment of renal tissue showing six glomeruli. There is marked alteration of the architecture. Interstitial fibrosis with atrophy of the tubules and areas of tubular dilation are observed. Diffuse mononuclear inflammatory infiltrate in the interstitial tissue is present. Three glomeruli are hyalinized showing residual

signs of hypercellularity and a central block of amyloid deposit. The remainder of the glomeruli present discrete mesangial hypercellularity with hyaline and acidophilic thickening of the mesangial axis. The walls of the arterioles are thick and hyalinized. Amyloid deposits are present in the subendothelium of the arteries in the middle of fibrous proliferation with decrease of the lumen of the vessels. Some tubules show discrete amyloid deposition in the basal membrane.

DISCUSSION

The seven lepromatous leprosy cases reported suffered in the past from *erythema nodosum leprosum*. None, however, was in reaction at the time of this study. Alterations of kidney function have been reported ([10, 15, 23]) during active ENL. Four patients, cases 3, 4, 5, and 7, presented or had a history of plantar ulcers of long duration. Cases 3, 4, and 5 were in an advanced phase of their illness and in frank uremia. In these circumstances, the inulin and the *p*-aminohippuric acid clearances do not reflect the glomerular filtration rate nor the effective renal plasma flow, especially the latter ([17]). In the case of the clearance of *p*-aminohippuric acid, the assumption that 90% of this substance should be extracted in its passage through the kidneys is not observed. The tubular mass seriously damaged is incapable of effectively excreting the *p*-aminohippuric acid. This is the main reason for the rise of the filtration fraction and not the diminution of the effective renal plasma flow. In these three patients the tubular functional involvement, measured by the capacity to concentrate the urine is very marked. The histologic study of these kidneys reveals extensive and uniform damage of the whole organ.

Case 1, however, showed excellent functional reserve; the glomerular filtration rate was normal, his ability to concentrate urinary solutes was good, but the filtration fraction was somewhat augmented, probably indicating a vascular involvement greater than that of the glomeruli, or a vascular involvement associated with deficient tubular excretion. Histologically, the vessels did not show amyloid deposit and the tubules showed only minor alterations.

The other three cases (2, 6, and 7) presented an intermediate type of involvement. The filtration fraction was slightly augmented suggesting greater vascular involvement, or the latter associated with tubular damage.

The incidence of amyloidosis in patients with lepromatous leprosy is variable; low in India and Mexico ([15, 18, 19, 24]), high in the U.S.A. ([21, 24]). Williams and co-workers ([24]), studying comparable groups in the United States (101 patients) and Mexico (119 patients), estimated the incidence of this complication as between 40% and 50% in the former group and 6% in the latter (diagnosis made by necropsy, gingival biopsy, tests of retention of Congo red). These authors observed a great discrepancy in the ingestion of animal fat, which in the American group was much greater than in the Mexican group, although the latter consumed much more calories. The frequency of this complication in Brazil is still undefined. Preliminary studies in the Hospital de Dermatologia Sanitária Lauro de Souza Lima, Bauru, Sao Paulo, however, suggest a high incidence ([14]).

Those patients having lepromatous leprosy and albuminuria not explainable by other renal disease must be suspected of harboring amyloid deposits in the kidney. If a renal biopsy is not feasible, gingival or rectal biopsy ([2, 4, 6, 9, 13]), especially the latter, can confirm the diagnosis, and with almost absolute certainty it may then be concluded that the kidney is also involved. Functional evaluation should then be mandatory. For practical purposes, the clearances of inulin and *p*-amino-hippuric acid can be substituted by the clearances of creatinine and phenolsulfonphthalein and the determination of the specific density of the urine can be substituted for the measurement of the urinary osmolarity since these tests are much easier to perform in a clinical laboratory.

There is some evidence in the literature that the amyloid deposition is reversible with the amelioration of the primary illness ([1, 16]). This being so, the periodic performance of tests to evaluate the renal function would be a valuable means of following the evolution of amyloid deposition in the kidney. The follow-up of the cases reported in this paper and the periodic functional evaluation of other patients with renal amyloidosis secondary to lepromatous leprosy may confirm the validity of this hypothesis.

SUMMARY

Seven cases of renal amyloidosis secondary to lepromatous leprosy are reported. Six had the diagnosis confirmed by biopsy and one by necropsy. One patient had only mild tubular involvement, three were in a far advanced stage and the other three were moderately affected. Five had a previous history of repeated episodes of *erythema nodosum leprosum* (ENL), three of ENL and plantar ulcers, and one of plantar ulcers without episodes of ENL. None were in active phase of ENL during the renal studies.

Renal function was evaluated by the clearances of inulin (to measure the glomerular filtration rate), *p*-amino-hippuric acid (to measure the effective renal plasma flow) and by the maximal capacity of the tubules to raise the urine osmolarity after water deprivation.

The patient with only slight deposits of amyloid in the tubules showed excellent functional reserve. The three advanced cases presented serious impairment of the glomerular filtration rate, effective renal plasma flow and tubular capacity to concentrate the urine. The three cases with intermediate type involvement showed an increase of the filtration fraction suggesting a greater vascular involvement or this associated with a deficient capacity of the tubules to transport the dye.

The authors suggest that those patients with lepromatous leprosy and albuminuria not explainable by other illnesses should be suspected of harboring amyloid deposits in their kidneys. If renal biopsy cannot be performed a gingival or rectal biopsy would confirm the diagnosis of amyloidosis and kidney involvement could be assumed since this organ is affected in over 80% of the cases with systemic amyloidosis. Renal function evaluation should then be mandatory. The more practical tests of creatinine clearance, phenol red excretion and measurement of the specific gravity of the urine after water deprivation could be substituted for the clearances of inulin and *p*-amino-hippuric acid and measurement of urine osmolarity, respectively. The periodic performance of these tests should be a valuable means of following the amyloid deposits in the kidney, since there is evidence in the literature that these deposits may be reversible.

RESUMEN

Se presentan 7 casos de amiloidosis renal secundaria a la lepra lepromatosa. En 6 casos el diagnóstico se confirmó por biopsia y por necropsia en el caso restante. Uno de los pacientes tuvo sólo una afección tubular moderada, tres estuvieron en un estado muy avanzado y los otros tres mostraron una afección intermedia. Cinco pacientes tenían una historia previa de episodios repetidos de eritema nodoso leproso (ENL), tres de ENL y úlceras plantares y uno de úlceras plantares sin ENL. Ninguno estaba en fase aguda de ENL durante los estudios renales.

El funcionamiento renal se valoró por la depuración de inulina para medir la intensidad de filtración glomerular, de ácido p-aminohipúrico para medir el flujo plasmático renal efectivo y por la capacidad máxima de los túbulos para elevar la osmolaridad de la orina después de haber suspendido la ingestión de agua.

El paciente que sólo mostró un ligero depósito de amiloide en los túbulos tuvo una excelente reserva funcional. Los 3 casos avanzados presentaron serias alteraciones en sus pruebas funcionales renales. Los 3 casos con afección intermedia presentaron un aumento en su capacidad de filtración sugiriendo una mayor afección vascular o una afección vascular asociada con una deficiente capacidad de los túbulos para transportar el colorante.

Se sugiere que aquellos pacientes con lepra lepromatosa y albuminuria no explicable por otras enfermedades deberían considerarse como sospechosos de tener depósitos de amiloide en sus riñones. Si no se puede hacer una biopsia renal, una biopsia rectal o gingival podrían confirmar el diagnóstico de amiloidosis y entónces podría suponerse una afección renal ya que este órgano se afecta en más del 80% de los casos con amiloidosis sistémica. Por lo anterior, el estudio del funcionamiento renal resulta ser de gran importancia. Otras pruebas más prácticas como la depuración de creatinina, la excreción del rojo de fenol y la medición de la gravedad específica de la orina después de suspender la ingestión de agua podrían substituír a las pruebas de depuración de inulina, del ácido p-aminohipúrico y a la medición de la osomolaridad de la orina. La ejecución periódica de estas pruebas constituye un valioso medio para seguir el depósito de amiloide en el riñón sobre todo porque hay datos en la literatura de que el depósito de amiloide puede ser reversible.

RÉSUMÉ

On relate ici sept cas d'amyloidose rénale secondaire à la lèpre lépromateuse. Six de ces malades ont eu leur diagnostic confirmé par biopsie. Chez le dernier, le diagnostic a été posé lors

de l'autopsie. Un de ces malades présentait seulement une atteinte tubulaire légère; trois étaient dans un état beaucoup plus avancé; les trois derniers n'étaient que modérément affectés. Cinq de ces malades présentaient une histoire antérieure d'épisodes répétés d'érythème noueux lépreux (ENL); trois d'ENL et d'ulcères plantaires, et un d'ulcère plantaire sans épisodes d'ENL. Aucun de ces malades n'était atteint de la phase aigue de l'érythème noueux durant le cours de cette étude.

La fonction rénale a été évaluée par le clearance de l'inuline, afin de mesurer le taux de filtration glomérulaire, la mesure de l'acide p-aminohippurique pour évaluer de débit du plasma rénal effectif, et la capacité maximale tubulaire pour l'élévation de l'osmolarité urinaire après privation d'eau.

Le malade qui ne présentait que de faibles dépôts d'amyloide dans le système tubulaire présentait une réserve fonctionnelle excellente. Les trois malades au stade avancé présentaient une détérioration grave, tant en ce concerne le taux de filtration glomérulaire, que le débit plasmatique rénal effectif ou/et la capacité tubulaire à concentrer l'urine. Les trois cas présentant une atteinte de type intermédiaire, montraient une augmentation de la fraction de filtration qui suggérait une plus grande atteinte vasculaire, ou bien une association avec une capacité déficiente des tubules à transporter le colorant.

Les auteurs suggèrent que ces malades, atteints à la fois de lèpre lépromateuse et d'albuminurie ne pouvant être expliquée par d'autres affections, devraient être soupçonnés de présenter des dépôts amyloides dans les reins. S'il n'est pas possible de pratiquer une biopsie rénale, il faudrait procéder à une biopsie gingivale ou rectale qui pourrait confirmer le diagnostic d'amyloidose; on pourrait dès lors présumer que le rein est également atteint, car cet organe est affecté dans plus de 80 pour cent des cas présentant une amyloidose systémique. Il devrait être impératif de procéder à une évaluation de la fonction rénale. Les épreuves plus pratiques de clairance pour la créatinine, d'excrétion du rouge phénol, de même que la mesure de la gravité spécifique de l'urine après privation d'eau, pourraient remplacer les clairances de l'inuline et de l'acide aminohippurique, de même que la mesure de l'osmolarité de l'urine. La répétition périodique de ces épreuves pourrait constituer une méthode fort utile pour suivre les dépôts de substance amyloide dans le rein, puisqu'il existe dans la littérature des données que montrent que ces dépôts peuvent être réversibles.

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