Renal amyloidosis: A rare presenting manifestation of Hodgkin’s disease

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Summary Renal amyloidosis leading to nephrotic syndrome is very rare in Hodgkin’s disease as compared to minimal change disease. Amyloidosis usually develops insidiously after many years of active Hodgkin’s disease, and is often a late and irreversible complication. Concomitant presentation of Hodgkin’s disease and nephrotic syndrome are rarely reported in the literature.

We describe a 35-year-old female who presented simultaneously with Hodgkin’s disease and nephrotic syndrome, which was found to be secondary to renal amyloidosis on renal biopsy.

Introduction

Renal amyloidosis complicating Hodgkin’s disease (HD) is very rare. We describe simultaneous occurrence of HD and nephrotic syndrome with kidney biopsy evidence of renal amyloidosis in a female patient.

Case report

A 35-year-old woman had low grade fever, cervical lymphadenopathy for 1 year and gradually progressive anasarca of 2 months’ duration. Physical examination revealed anasarca, postural hypotension and non-tender, discrete left lower cervical lymphadenopathy. Laboratory investigations showed proteinuria of 9.39 g/24 hours, hemoglobin of 10 g/dL, normal white blood cell and platelet counts and serum electrolytes, serum urea of 96 mg/dL, creatinine of 3.0 mg/dL, cholesterol of 164 mg/dL, albumin of 2.4 g/dL, and globulin of 1.6 g/dL. Both kidneys were enlarged on ultrasonography. Immunological assay was negative for HIV, hepatitis B and C. No monoclonal protein was detected on serum electrophoresis. Mantoux test was negative. Renal biopsy showed focal irregular membranous thickening and deposition of hyaline material in the mesangium and blood vessels. Hyaline deposit in the mesangium and vessel wall...
was positive for Congo red and crystal violet stains, confirming the diagnosis of amyloidosis (Fig. 1). Lymph node biopsy revealed diffuse effacement of lymph nodes by a heterogeneous cellular infiltrate consisting of small lymphocytes, eosinophils, plasma cells and macrophages. Classical Reed-Steinberg cells were seen on hematoxylin and eosin staining, confirming the diagnosis of HD. Amyloid material was also demonstrated in lymph node tissue (Fig. 2). She was treated with chemotherapy using the ABVD regimen (doxorubicin, bleomycin, vinblastine, dacarbazine) along with dialysis. However, she died of sepsis and multi-organ failure.

Discussion

Renal amyloidosis as a presenting feature of HD is rarely reported. Only 40 cases of HD with amyloidosis have been reported in the literature from 1930 to 1986. Tuglular et al did not find a single case of HD causing renal amyloidosis among 287 cases of secondary amyloidosis. Similarly, no case of renal amyloidosis due to HD was observed in a series of 230 cases from India. However, simultaneous occurrence of HD and renal amyloidosis has been documented. Tüzün et al documented that the period between diagnosis of HD and development of renal amyloidosis were 1,

Figure 1  Renal biopsy shows: (A) hyaline deposits in the glomerular basement membrane and mesangium (hematoxylin & eosin, 400×); (B) orange red amyloid in the glomerular basement membrane and mesangium (Congo red, 400×).

Figure 2  Lymph node biopsy shows: (A) typical Reed-Steinberg cells (hematoxylin & eosin, 400×); (B) nodular collection of amyloid which was positive for Congo red; (C) numerous eosinophils (hematoxylin & eosin, 400×).
26 and 138 months in their three patients. Our patient had HD for 1 year before development of nephrotic syndrome due to renal amyloidosis. Probably in our patient, the HD that was undiagnosed for 1 year gave time for increased synthesis of interleukins and serum amyloid A, with subsequent development of renal amyloidosis. In conclusion, we need to be aware of renal amyloidosis, a rare complication of HD presenting as nephrotic syndrome.

References


