Absence of Mesangial IgA in AIDS: A Postmortem Study

M.-C.	Marie-Christine	Béné ^a
P.	Philippe	Canton ^b
C.	Corinne	Amiel ^{a,b}
T.	Thierry	May ^b
G.	Gilbert	Faure ^a

^aLaboratoire d'Immunologie, et ^bDépartement des Maladies Infectieuses et Tropicales, Centre Hospitalier Régional Universitaire de Nancy-Braboïs, Vandœuvre, France

Marie-Christine Béné, Laboratoire d'Immunologie, Centre Hospitalier Régional Universitaire de Nancy-Braboïs, F-54500 Vandœuvre (France)

Sir,

A recent paper published in Nephron [5] reports the discovery of IgA nephropathy (IgAN) upon renal biopsy in 2 patients with AIDS-related complex. The authors stress the similarities that may be observed between these two diseases, namely high levels of serum IgA, IgAl, and IgA-containing immune complexes. Both patient populations also suffer from frequent respiratory or, at large, mucosal infections. These similarities have been noted for some time; therefore, the small number of reported AIDS patients with IgAN is quite surprising. It might be that the renal disease goes unnoticed in a population with major health impairment. AIDS patients also have an unfortunately short life expectancy, while renal failure often occurs in IgAN patients after several years of development [3].

Based on these assumptions, we undertook a systematic study of renal biopsies performed within 24 h post mortem in deceased AIDS patients, and snap-frozen in liquid nitrogen. IgA deposits have been shown to remain for up to 15 days in postmortem samples [7], and the protocol used was similar to that used in a previous study on liver cirrhosis patients [2]. Between January 1986 and May 1990, frozen-cut sections of 54 kidney samples, obtained from 48 men and 6 women (mean age 32 years) were thus examined in direct immunofluorescence using antisera to IgA and complement factor C3 (reagents from Behring, Marburg, FRG). There was no significant labelling in 53 of these biopsies. Granular endomembraneous deposits of both IgA and C3 were observed in 1 patient. This 40-year-old man was a hemophiliac with transfusional AIDS. He developed severe hepatitis associated with high levels of anti-HSV antibodies, and evidence of liver infection by HSV. The histologic examination of liver biopsies disclosed progressive cirrhosis. He had

portal hypertension and large volumes of ascitic fluid were repeatedly removed from his abdomen. Several urine analyses were performed, never disclosing hematuria. Serum IgA levels were assessed in February and May 1988 and were 2.22 and 3.21 g/l, respectively. He died in December 1988 from pulmonary complications. The immunofluorescent labelling observed in his kidney was very similar to the pattern previously reported in liver cirrhosis patients and differed from the strictly mesangial deposits of Berger's disease [1, 2].

Our data confirm the absence of correlation between HIV infection and IgAN in a larger series than the initial 15 cases reported by Jackson et al. [4]. These data, therefore, suggest that AIDS occurred in the 2 patients reported by Kenouch et al. [5] while they were already suffering from IgAN. The authors' second observation, i.e. a macroscopic hematuria episode following febrile pharyngitis, shows typical clinical features of IgAN patients. The microhematuria reported in the first patient on admission also reminds of a frequent mode of discovery of IgAN: upon routine urinalysis.

It has been widely demonstrated that the human immunodeficiency virus is more likely to induce AIDS-related complex in young males than in other populations. The same age incidence has been largely reported for IgAN. This suggests that the stakes are high for AIDS patients to also have IgAN, and that chance observations such as the two cases reported are likely to occur by the mere rule of statistics. What appears more puzzling, from a fundamental point of view, is the fact that patients with similarly high levels of serum IgA1 do not systematically develop IgAN. This feature favors the hypothesis suggesting that specific anomalies of the mucosal [1] and/or central [6] IgA immune systems are involved in the pathogenesis of IgAN.

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