Dear Sir,

After the publication of the article by Verresen et al. [1] and others [2], including our own report [3], the clinical intensity of anaphylactoid reactions described in the literature has been very diverse. The majority of the reactions described have been serious (anaphylactic shock, severe dyspnea, broncho-spasm) although minor reactions can occur as seen in our case. It would be interesting to correlate the clinical symptoms with the type and dosage of ACE inhibitor used. The results probably would indicate extreme variability between individuals, more in relation with the ACE inhibitors than with the type of membrane used, although most of the descriptions relate the AN69 membrane with this reactions.

From this point of view, the pathogenic hypothesis proposed by Tielemans et al. [2] is of interest. They propose an increase in the formation of bradykinins on the part of this membranes, also taking into consideration the inhibition of their degradation brought about by the ACE inhibitors [4]. On the other hand, regarding the pathogenic hypothesis in which bacterial products cross the dialysis membrane (AN69), Dinarello [5] indicates that this is in fact a clinical manifestations of bacterial shock and not an anaphylactoid reaction.

Even though most of the clinical observations described to date appear to implicate the AN69 membrane and the ACE inhibitors as the cause of the anaphylactic manifestations, it seems obvious that controlled studies are necessary (in vivo and in vitro) to adequately define the underlying pathogenic process; probably other mediators of the inflammatory process are involved in these manifestations. At the present time, we must be extremely cautious with the use of ACE inhibitors in hemodyaüzed patients with high-flux membranes (AN69).

References


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