Letters and Replies

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IgA Nephritis—ACE inhibitors, steroids etc?

Sir,

The review by Francesco Locatelli and colleagues [1] will surely become the basis for further therapeutic trials. However, I wish to raise three points, if you please. Firstly, the rationale for using ACEIs and Angiotensin Receptor Blocker (ARBs) is to control systemic and glomerular hypertension and proteinuria. Yet it is an uncomfortable undoubted fact, concerning which there is a deficiency of recording, that some 25% patients have to abandon ACEIs owing to their side-effects, and another ill-defined percentage of patients have to forsake ARBs. There are patients who cannot take either form of drug. One has to bring this matter into the open. I suspect that there could be racial/pharmacogenetic factors to be considered. The reality is that patients may instead have to take calcium channel blockers.

Secondly, many units still prescribe aspirin, and that is fine, for aspirin is an anti-thromboxane and even an immuno-proteasomal inhibitor [2]. I recently drew attention to the continued need for consideration of antithromboxanes [3]. The relevance of thromboxane to the pathophysiology of IgA nephritis has to be continually stressed [4], and each new generation of physicians should be reminded. It is a shame that the pharmaceutical industry did not see fit to continue the manufacture of those antithromboxanes, into whose development they had invested so much!

Thirdly, Locatelli et al. [1] mention patients with rapidly progressive IgA nephritis. In Nephron 1998;79, p. 221, I mentioned the use of melphalan in two patients, in whom a gratifying amelioration was achieved. Since I had previously worked in haematology, I knew how to cope with the leucopaenia for 3 weeks and the associated outbreak of herpes zoster in one patient. Melphalan is a viable alternative.

Conflict of interest statement. None declared.

London NW1 8JS

E. Nigel Wardle

Email: nigel@edwinwardle.freeserve.co.uk


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Reply

Sir,

I would like to thank Dr Wardle for his comments on our Review [1]. Actually ACEIs are generally very well-tolerated drugs and in our experience, very few patients have to abandon this therapy owing its side effects [2]. This is even more true for ARBs.

I agree with Dr Wardle, that a possible explanation of the difference among patients could be related to racial/pharmacogenetic factors. The message we wished to convey was to use inhibitors of renin–angiotensin II system in patients with IgA nephritis and clinically relevant proteinuria, before using steroids or immunosuppressants.

Moreover, considering that the control of blood pressure is of paramount importance, very often the addition of calcium channel blockers is required, in order to reach the blood pressure target values suggested by the Guidelines. We would like to underline the fact that just using inhibitors of rennin–angiotensin II system, without carefully considering the blood pressure values actually reached, is a very frequent wrong therapeutic approach.

We agree with the relevance of thromboxane to the pathophysiology of IgA nephritis and a possible prescription of a small dosage of aspirin, in patients who can tolerate it (unfortunately the number of these patients is much lower than the number of patients who can tolerate inhibitors of renin–angiothensin II system!).

In the case of rapidly progressive IgA nephritis, as we have already emphasized [2–3], we should be more aggressive, using cytotoxic drugs, possibly including mycophenolate mofetil, which seems much more manageable than Melphalan.

Department of Nephrology and Dialysis, A. Manzoni Hospital, Via Dell’Eremo 9/11, 23900 Lecco, Italy

E-mail: f.locatelli@ospedale.lecco.it


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