Post-transplant erythrocytosis and thromboembolic events: an error

Sir,
European guidelines for long-term management of the transplant patients were published in your journal in 2002. Section IV.9.3 of these guidelines discusses post-transplant erythrocytosis [1]. It refers to the paper of Wickre et al. [2] and claims that: 'One study in 53 polycythaemic transplant patients failed to find any increased incidence of thromboembolic events'. In fact, upon reviewing the original article of Wickre et al., one finds that the results of this study and the conclusion of the authors are exactly the opposite of the above-mentioned statement. They studied a series of 53 renal transplant patients with erythrocytosis, and compared them with a matched control group of 49 recipients. Over a follow-up period of 3.5 years, 11 thromboembolic events occurred in 10 of the 53 erythrocytosis patients, but none in the control group (P < 0.001). They then concluded that the incidence of thromboembolic events is significantly increased in transplant recipients with erythrocytosis. This finding was later confirmed by other studies [3].

I believe that this petit erreur merits correction even 1 year after publication, because it appeared in the ‘guidelines’, which are often read and used for a long time by many readers.

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Reply

Sir,
The remark by Kazory is correct and we (the EBPG group on renal transplantation) apologize for this mistake. This will be corrected in the update of these guidelines on the website (in process). However, this does not change the guideline in bold.

Conflict of interest statement. None declared.

Atherosclerotic renal artery stenosis in patients starting dialysis: an emperor with no clothes

Sir,
Van Ampting et al. [1] add more evidence to the literature regarding the common co-existence of atheromatous renal artery stenosis (ARAS) and renal failure. The fact that, in most cases, the ARAS was unilateral has only one explanation—the cause of nephron loss in these patients is not due to a stenosis of the renal artery.

Atherosclerotic hypertensive smokers are at risk of progressive renal failure and developing stenoses of any artery. This co-existence has been wrongly used to support the hypothesis that ARAS itself leads to progressive nephron loss. There is little evidence in favour of this, and lots against. Progressive renal failure is very unusual in the face of haemodynamically significant stenosis due to fibromuscular disease [2]. Individual kidney glomerular filtration rate studies in patients with chronic renal failure and unilateral ARAS show the renal impairment to be just as bad in the non-stenosed kidney [3]. Several studies have looked for and demonstrated ARAS in patients undergoing coronary angiography, for example, most recently, Agent al. found that 28% of patients had clinically silent unilateral ARAS and 10% had bilateral ARAS [4]. In other words ARAS is often present without renal impairment. The failure to achieve significant benefits with regard to renal function despite successful intervention [5] is explained most easily if the ARAS is not causing the renal impairment.

This latest study confirms that, although it is easy to find ARAS in elderly end-stage renal failure patients, in most cases it is not the cause of their renal failure. There are undoubtedly a small group of clinical problems that are caused by ARAS that can be relieved by revascularization, but not enough evidence to suggest that progressive renal failure is one of them.

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**Reply**

Sir,

We thank Main and Wroe for their interest in our paper [1]. They argue that the high prevalence of atherosclerotic renal artery stenosis (ARAS) in patients starting dialysis is no proof of its participation in the cause of terminal renal failure. Of course we agree with them: our study was purely observational. However, you could see this differently. Reversing the reasoning, the (hypothetical) finding of a low prevalence of ARAS in these patients would have ruled out its importance as a cause of terminal renal failure. We showed, for the first time with adequate techniques, that this was not the case. The next step, i.e. to connect this ARAS with the origin of terminal renal failure, is based on indirect arguments, but the step is not that big. First, the percentage of ARAS in patients with a known cause for renal failure was 22% (7/31), whereas it was 72% (13/18) in those in whom the cause was hypertension, renovascular disease or unknown. If ARAS is just some innocent bypassing event, these percentages should have been similar. Secondly, we found previously (as did others) that revascularization procedures such as stenting can retard progression of renal dysfunction [2,3]. This had also occurred in some patients in our report. Of course we agree that unilateral ARAS is by itself insufficient as a cause of terminal renal failure. This does not mean, however, that it cannot contribute to renal failure if there is also some other parenchymal renal problem. Also, we know very well that atherosclerotic disease is not limited to the renal arteries, but is a complex problem that will also involve the renal parenchyma and intrarenal vessels. Perhaps the term atherosclerotic renovascular renal failure is better to cover the problem than renal artery stenosis.

Indeed, we need more evidence about the contribution (and how serious this is) of atherosclerotic renovascular disease to the development of terminal renal failure and, specifically, if it is helpful to attack the extrarenal stenosis (apart from antihypertensive and metabolic treatments). Prospective, controlled trials such as the ASTRAL [4] and STAR [5] studies will hopefully give answers. Meanwhile, there is little reason for negligence. The problem is too serious for that.

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