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 petite 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.

Conflict of interest statement. None declared.

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Reply

Sir,
The remark by Kazory is correct and we (the EBPG group on renal transplant) 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.

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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, Agel et 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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