Abdominal aortic aneurysm and autosomal-dominant polycystic kidney disease

To the Editor: In a recent issue of *Kidney International*, Gibbs et al [1] ascertained the risk of growth and rupture of intracranial aneurysms in autosomal-dominant polycystic kidney disease (ADPKD) patients discovered by presymptomatic screening. It is well known that ADPKD is associated with a variety of life-threatening cardiovascular diseases, such as intracranial and coronary aneurysms [2]. On the other hand, the coexistence of abdominal aortic aneurysm (AAA) has been also reported in some ADPKD patients [2–5]. Chapman and Hilson [3] have described three patients (9.7%) with AAA and polycystic renal failure out of a total 31 patients with adult polycystic disease who had been on their dialysis and transplantation program for some time. Roodvoets [4] has reported an AAA in 1 (5.0%) of 20 patients with polycystic kidney disease. Kato et al [2] have reported that the frequency of AAA was 7.1% in ADPKD patients. Ruptured AAA has been also described [3, 4], and the technical difficulty of the exposure of the aorta is increased by the presence of voluminous kidneys, causing delay in cross-clamping during emergency laparotomy for rupture [5]. A significant increase in the aortic diameters (2.7 ± 0.14 cm) was found in the ADPKD patients compared with those in the chronic glomerulonephritis (2.3 ± 0.03 cm) and diabetes mellitus (2.3 ± 0.05 cm) groups (P < 0.02), and a routine screening of the aortic size, using computed tomography or echography, is recommended to detect the presence of AAA in ADPKD patients [2]. Further studies of screening and follow-up of AAA in ADPKD patients are needed.

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Protective effect of vitamin C supplementation in dialysis patients: Not all that glitters...

To the Editor: An elegant study by Tarng et al [1] has demonstrated that intravenous vitamin C supplementation as 300 mg postdialysis three times a week reduces DNA-8-hydroxy-2′deoxyguanosine and reactive oxygen species in peripheral lymphocytes of dialysis patients. Its efficacy with serum ferritin < and > 500 μg/L speaks against the worried pro-oxidant effect of vitamin C on lymphocyte DNA oxidation in case of increased iron stores.

What would be the actual consequences of introducing this knowledge in the clinical practice? As in previous works on erythropoietin therapy, vitamin C as “adjuvant therapy” at dosages many times higher than that suggested for the general population became recommended in dialysis patients (Fig. 1), without putting evidence forward to demonstrate the safety in terms of oxalate metabolism [2–4].

Major concern for safety of vitamin C supplementation arises from its metabolism to oxalate, which, in uremic patients, may exceed the threshold of solubility of calcium oxalate, and result in tissue deposition of calcium oxalate crystals, eventually increasing cardiovascular risks and worsening renal function in predialysis patients.

The gap between theoretical knowledge and actual demonstration of benefits in major outcomes such as survival often being large, the ratio “known side effects/unknown clinical benefits” should be carefully evaluated.