Is prophylactic haemodialysis recommended to remove contrast medium immediately after angiography?

Sir,

We read with interest the recent article ‘Effect of haemodialysis after contrast medium administration in patients with renal insufficiency’ by Lehnert T et al. [1]. This is the first prospective study suggesting that haemodialysis eliminates contrast medium efficiently, but does not influence the incidence or outcome of radiocontrast-induced nephropathy (RCN), at least in patients with mild to moderate renal insufficiency.

The authors speculated that the failing efficiency of haemodialysis in preventing RCN is due to a very rapid onset of renal injury after administration of contrast medium, and is due to renal toxicity in much lower plasma levels. They concluded that increased elimination might not influence the final outcome of RCN, and therefore prophylactic haemodialysis appeared to be indicated for reversal of fluid overload only.

In National Cardiovascular Center Hospital of Japan, we have restricted the prophylactic haemodialysis immediate after angiography to cases with congestive heart failure who required body fluid volume control by haemodialysis. However, there are many hospitals where prophylactic haemodialysis was indicated routinely immediately after angiography in all patients with renal insufficiency. The present important report by Lehnert and colleagues gave the rationale for our limited indication for prophylactic use. We should recall here that acute renal failure requiring haemodialysis in this setting is more often due to renal atheroembolism, rather than to RCN [2]. Since it is well known that hydration with saline infusion before and during angiography is effective in preventing RCN [3], the important conclusion reached by Lehnert T et al. and our experience that indication of prophylactic haemodialysis should be restricted to reversal of fluid overload both seem reasonable.

On the other hand, it is not yet known how soon contrast medium must be eliminated after angiography in patients on maintenance haemodialysis, in whom contrast medium can be removed only by haemodialysis. In our hospital, we schedule angiography on an interdialytic day and contrast medium is removed by routine haemodialysis on the next day of angiography. Again, we restricted haemodialysis immediate after angiography to cases who require body fluid volume control. We have never experienced adverse toxic effects due to prolonged exposure of contrast medium left within body for a day and a half. However, safety of contrast medium must be clarified by prospective randomized studies in future.

Sir,

Vitamin K is a cofactor for carboxylation of osteocalcin and other bone matrix proteins. Several studies suggest that vitamin K deficiency in otherwise healthy adults is associated with increased urinary calcium loss [1], increased risk of hip fracture [2] and reduced bone mineral density [3]. Vitamin K supplementation may prevent reduced bone mineral density [4] and may normalize urinary calcium excretion [5]. Several years ago, a preliminary study showed a favourable effect of high dose vitamin K supplementation on bone mineral density in haemodialysis patients [6]. In a recent study in 68 haemodialysis patients, a low vitamin K concentration was associated with an increased fracture risk and a higher prevalence of hyperparathyroidism [7]. It has also been speculated that vitamin K may indirectly suppress parathyroid function.

The current study was planned to prospectively evaluate whether vitamin K1 supplementation had an effect on bone turnover and hyperparathyroidism of haemodialysis patients. Study parameters were plasma intact PTH (iPTH) and serum bone alkaline phosphatase (bAP). Thirty-four stable patients on maintenance haemodialysis (23 male, 11 female) were studied after informed consent. They received 2 mg vitamin K1 per day orally for 6 months. Inclusion criteria for the study were: (i) age between 18 and 75 years, mean age 59.5 ± 13.3 years; (ii) length of time on haemodialysis between 3 and 24 months, mean 13 ± 7 months; (iii) iPTH between 15 and 50 pmol/l, mean 26.3 ± 17.5; (iv) no previous renal transplantation.

Renal diseases were: diabetic nephropathy, n = 11; chronic glomerulonephritis, n = 7; vascular nephropathy, n = 5; polycystic kidney disease, n = 4; chronic interstitial disease, n = 3; unknown, n = 4. Any vitamin D medication was discontinued 4 weeks before initiation of the study. All patients received calcium-containing phosphate binders. None of the patients had been exposed to aluminium hydroxide. No other drugs known to influence calcium or vitamin K metabolism were taken by the study patients.

The vitamin K1 supplementation was well tolerated by all patients. Side effects were not observed. The results of blood measurements are summarized in Table 1. Mean serum vitamin K1 increased significantly during the study. After 3 months, serum vitamin K1 had risen approximately six-fold, after 6 months approximately four-fold. There was a large variation of serum vitamin K1 at all time points.

Mean serum calcium and phosphate were unchanged during the study. Ionized calcium could be measured in 14 patients. In those patients, ionized calcium did not change during the study period (data not shown). The phosphate binder dose also remained unchanged. Thirty-one patients received calcium acetate; three patients received calcium carbonate. Mean daily doses were 3.1 ± 1.0 g at study start, 3.2 ± 0.9 g after 3 months, and 3.3 ± 1.0 g after 6 months. Other routine blood tests were unaltered during the study (data not shown).

Both iPTH and bAP showed no statistically significant changes during the study. Mean intact PTH rose by 23%, this increase was not significant. Mean bAP stayed virtually constant during the 6-month investigation period. As expected, bAP and iPTH correlated significantly (r = 0.51, P < 0.02, Spearman’s rank correlation). There was no significant correlation between serum vitamin K1 and either iPTH or bAP.