Renal Artery Thromboembolism and Immunosuppressive Therapy

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Dear Sir,

Lo et al. [1] reported a case of recurrent allograft kidney infarction secondary to embolization from a thrombus of the hypogastric artery as extremely rare complication. We observed in 1 out of 244 renal transplant recipients a thromboembolic complication of the allograft renal artery 22 months after transplantation. The patient was a 42-year-old man with chronic pyelonephritis as cause for the renal insufficiency. Immunosuppressive therapy consisted of ciclosporin A (CsA) and prednisone (P) since transplantation.

Recently we investigated the occurrence of thromboembolic complications during the 1st year following renal transplantation in 244 patients treated either with a combination of CsA and P (CsA+P) (n = 118) or with a triple-drug therapy (CsA+P+azathioprine) (n = 126) [2]. A total of 41 thromboembolic complications occurred in 33 patients, most being thromboses of the leg veins, myocardial infarctions, or cerebrovascular accidents. The patients treated with the CsA+P therapy showed a significantly higher incidence of thromboembolic complications as compared with patients treated with the triple-drug therapy (19.5 vs. 7.9%; p < 0.01). During the preceding months, patients with thromboembolic complications have had significantly higher CsA whole-blood levels than patients without complications (CsA+P 117 ± 21 vs. 80 ± 13 ng/ml, p = 0.005; triple-drug treatment 102 ± 28 vs. 59 ± 11 ng/ml, p = 0.001 – monoclonal radioimmunoassay). The patient reported above had mean CsA blood levels of 475 ng/ml (polyclonal radioimmunoassay) during the preceding 6 months which was much higher as in patients without complications (287 ± 51 ng/ml; polyclonal radioimmunoassay).

CsA causes a vasoconstriction by an enhanced release of thromboxane A₂ [3] and a decreased production of prostacyclin [4]. Furthermore, CsA is a direct cause of pronounced vascular endothelial damage [5], endothelin (one of the most potent vasoconstrictors) release [6], and increased thrombocyte aggregation. Together these conditions are highly thrombogenic. Lo et al. [1] reported CsA trough level of 176 ng/ml in their case report which is also clearly higher than in our patients with thromboembolic complications.

With the introduction of triple-drug therapy and the ensuing dose reduction for the individual drugs, a decline in thromboembolic complications was anticipated which was demonstrated in our study [2]. This favours the use of triple-drug instead of CsA+P therapy for the prevention or reduction of thromboembolic complications.

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