Sudden Hearing Loss in a Cyclosporin-Treated Renal Transplantation Patient

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Sir,

Thromboembolic complications constitute an important risk in the postoperative course after renal transplantation. The incidence has been reported to be as high as 24% [1]. We present a case of sudden hearing loss due to a thromboembolic event in a renal transplant recipient on triple immunosuppressive treatment consisting of cyclosporin A (CsA), azathioprine (Aza) and prednisolone (Pred).

A 22-year-old Caucasian male with end-stage renal disease secondary to membranoproliferative glomerulonephritis underwent a living-related donor renal transplantation. The patient had been maintained on a hemodialysis program for 3 months prior to renal transplantation. Initial laboratory results showed that serum creatinine level and blood urea nitrogen were 397 $\mu\text{mol/l}$ and 18.3 mmol/l, respectively. The remainder of his laboratory results including prothrombin time (FT), partial thromboplastin time (PTT), chest X-ray and electrocardiogram were within normal limits.

Before the operation, CsA was given in a single dose of 4 mg/kg orally. Methylprednisolone 1,000 mg and Aza 150 mg were given intravenously during surgery. On the operation day, Pred 100 mg was given orally and tapered by 10 mg daily until the dose of 20 mg per day. He received CsA (4 mg/kg) b.i.d. and Aza (1.5 mg/kg/day) in a single oral dose. He tolerated the operation well and had good urine output postoperatively. On the 7th postoperative day, the patient complained of

right sudden hearing loss. On the same day, his blood CsA level was measured as 200 ng/ml, and serum creatinine level was 106 $\mu\text{mol/l}$. PT and PTT were within normal limits. Because of right sudden hearing loss, the patient was referred to the Otolaryngology Department. He had not any vestibular symptoms, otolaryngologic examination revealed no abnormality except for tuning fork tests. With the tuning fork at 512 cps, there was no response on the right ear. The patient localized the sound in the left ear on the Weber test. Investigations included a full range of

cochlear and vestibular tests that were done in order to find the etiology of sudden hearing loss. Cochlear function tests, pure-tone audiogram, speech discrimination score, loudness balance test, tympanometry and acoustic reflex decay were done. A flat type of sensori-neural hearing loss was found, and speech reception threshold was 80 dB on the right ear. Speech discrimination decreased to 12%. Radiological survey of temporal bone including tomography was done to exclude an 8th nerve tumor, and there was no abnormal finding. According to these findings, sudden hearing loss was considered to be related to vascular accident. Then, we put the patient on antiaggregan and vasodilator therapy. Bencyclone hydrogen fumarate 500 mg/day was given by the intravenous route for 7 days. He also received dipyridamole 75 mg/day and betapyridil carbonil 450 mg/day, orally. CsA dosage was reduced to 2 mg/kg/day. Seven days later, audiologic

examination was repeated. The patient's cochlear functions were within normal limits, his new blood CsA level was 50.8 ng/ml, and his serum creatinine level was 106 μ mol/l. Vasodilator therapy was stopped and antiaggregan therapy was continued. His cochlear and renal functions were within normal limits on his last control which was 3 months after the operation. Sudden loss of cochlear function has often been attributed to vascular origin [2]. Because of the difficulty in identifying inadequate vascular supply by conventional methods such as angiography, the exact incidence of sudden sensorineural deafness due to vascular insufficiency is unknown. The exact mechanism of vascular insufficiency in sudden sensorineural deafness is said to vary from thrombosis and embolism in the labyrinthine artery to spasm and sludging through vasomotor imbalance and disorder of coagulation such as polycythemia and macroglobulinemia [2].

The introduction of CsA has made a significant contribution to the immunosuppressive treatment of renal transplant recipients. Although graft and patient survival have improved, thromboembolic events and hemostatic changes have been reported with increased frequency in patients on CsA treatment [3]. The possible mechanisms through which CsA predisposes to thromboembolic events are increasing F VIII: C Ag, fibrinogen level and ADP-induced platelet aggregation and decreasing vascular prostacycline (PGI₂) level [3, 4]. Despite this, some authors reported that there were no significant differences in the incidence of thromboembolic events in the CsA-treated group [5, 6].

Our patient experienced sudden hearing loss possibly due to the vascular event. He responded well to antiaggregan and vasodilator therapy, reducing CsA dosages, and regained adequate cochlear function after 7 days.

We conclude that this sudden hearing loss was most likely secondary to the thromboembolic event, and CsA might be one of the responsible agents which cause thromboembolic events.

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