A Case of Sudden Deafness Probably Due to Antithymocyte Globulin Treatment

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Dear Sir,

Since we believe that sudden deafness due to antithymocyte globulin (ATG) administration has not been described before, we present the features of this unique case. A 26-year-old male patient who had end-stage chronic renal disease of unknown primary etiology underwent renal transplantation from a cadaveric donor. Acute tubular necrosis occurred after transplantation, and the serum creatinine level remained around 301 $\mu\text{mol/l}$ (3.4 mg/dl). An acute rejection attack occurred on the 38th day after transplantation, although ciclosporin, azathioprine, and prednisolone were used as immunosuppressive therapy. No response was observed to methylprednisolone (1 g/day i.v. for 5 days), and the serum creatinine level was elevated at 866 $\mu\text{mol/l}$ (9.8 mg/dl). Therapy with ciclosporin and azathioprine was terminated and ATG (Thymoglobulin; human antithymocyte rabbit immunoglobulin, Lot (No. G 0528) has begun 300 mg/day i.v. for 8 h), and hemodialysis was started.

On the 2nd day of ATG therapy, sudden and almost total hearing loss in both ears occurred after 20 min of infusion. The thresholds were determined by auditory brainstem response testing to be 78 dB in the right and 90 dB in the left ear. The stapedial reflex disappeared at 2,000 Hz, and Metz recruitment was present. According to these findings the hearing loss was considered to be of the sensorineural type. Prednisone and antihistaminic, vasodilating, and antiplatelet agents were added to the ATG therapy. Response to ATG therapy was seen, and the serum creatinine

level decreased to 265 $\mu\text{mol/l}$ (3.0 mg/dl) on the 7th day of ATG treatment. ATG administration was terminated, as was hemodialysis therapy which had been performed three times during the acute rejection period. Hearing began to improve on the 11th day after the end of the ATG therapy. The hearing thresholds improved at 38 dB in the right and 43 dB in the left ear. During the 6th month after therapy creatinemia was stable at 288 $\mu\text{mol/l}$ (3.2 mg/dl), and the hearing thresholds were 33 (right ear), and 37 dB (left ear).

Progressive and sensorineural hearing loss in chronic renal failure has already been reported, but the pathogenesis is still unclear [1, 2]. The effects of renal replacement therapy, especially hemodialysis, on hearing impairment are not enlightened, although some work has been done. Some authors point out that hemodialysis causes hearing loss, while others believe that it improves hearing, but some think that it has no effect on the hearing ability [1-6]. In our case the hearing loss had a sudden onset, was not progressive, and improved within

short time, so it seems that the hearing loss was not related to chronic renal failure or renal replacement therapy.

Sudden hearing loss due to ciclosporin-induced thromboembolic complications was reported in renal transplant patients [7]. In our case, ciclosporin therapy was terminated before sudden hearing loss occurred, so ciclosporin may be excluded as the causative agent. Onset of the sudden hearing loss in the present case was on the 2nd day of the ATG therapy and began to improve on the 11th day after

terminating ATG. Under this condition, sudden hearing loss should be kept in mind as a seldom complication of ATG therapy, and our patient is, to our knowledge, the 1st described in the literature.

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