

A Case Report on Amikacin Induced Vestibular Ototoxicity

Jisha M Lucca^{1*}, Prathiba Pereira², Rinsa Cherian Amprayil³, Ashmy Jose³

¹College of Clinical Pharmacy, Imam Abdulrahman Bin Faisal University Dammam, KSA

²Department of Internal Medicine, JSS Hospital, Ramanuja Road, Mysore – 570004, Karnataka, INDIA.

³JSS College of Pharmacy, JSS University, SS Nagara, Mysore-570 015, Karnataka, INDIA

ABSTRACT

Amikacin, a semi-synthetic aminoglycoside, is the drug of choice for multidrug-resistant tuberculosis. It may adversely affect auditory, vestibular and renal functions. Reporting here is an interesting case of amikacin induced vestibular toxicity.

Key words: Amikacin, Vestibular toxicity, Ototoxicity.

INTRODUCTION

Aminoglycosides are still considered as crucial part in treatment of life threatening infections like septicaemia and multidrug-resistant tuberculosis.¹ Nephrotoxicity and ototoxicity are the most persistent and pertinent adverse reaction of aminoglycoside. Nephrotoxicity is reversible and clinically managed with hydration therapy, however ototoxicity is permanent and profoundly hinder the patient quality of life.¹⁻³ Aminoglycoside induced ototoxicity can be due to damage of vestibular system or auditory system or both. Amikacin, kanamycin and neomycin, are mainly cochleotoxic. Whereas to bramycin, streptomycin and netilmicin are vestibulotoxic. It is reported that both cochlear and vestibular damage can occurs with gentamicin.^{1,3} Symptoms of cochlear damage consist of long-lasting hearing loss and or tinnitus, whereas disequilibrium dizziness, ataxia and/or nystagmus are common with vestibular damage. The incidence of amikacin induced vestibular ototoxicity vary from 7- 28.5%.¹⁻⁵ Reporting here is an interesting case of amikacin induced vestibular ototoxicity.

CASE REPORT

A 47-year old, teacher from urban background visited to the medicine outpatient department of a university hospital. On presentation he complained of fever, burning micturition and abdomen pain which was sudden onset and persisted for last few days. On examination patient was febrile, dehydrated, complaints of burning micturition, suprapubic tenderness and three to five episode of loose stools and vomiting. Past medical history revealed that over the last three years patient was on insulin mixard for Type 2 diabetes mellitus. A provisional diagnosis of Type 2 diabetes mellitus with urinary tract infection was considered and patient was started with tablet paracetamol 650mg, Injection ondansetron 4mg three times day, racecadotril 100mg, amikacin 500 mg twice a day and insulin actrapid 15-15-0 and insulin mixtard 0-0-15. All his laboratory values were within the normal limit except a slight elevation of random blood sugar 218 mg/dl.

On the fourth day of hospitalization patient was complains of giddiness, vertigo and hearing difficulty. On examination he was drowsy and complaints of positional nystagmus. As these are the new symptoms

DOI: 10.5530/ijopp.12.1.10

Address for correspondence:
Dr. Jisha M. Lucca,
Assist. Professor
College of Clinical Pharmacy,
Imam Abdulrahman Bin Faisal
University, Dammam, KSA.
Phone no: +966 0558181537
Email Id: jmlucca@iau.edu.sa



www.ijopp.org

the physician requested for ophthalmic and neurologic consultation to rule out the other cause and was insignificant. But the audiometry report shows bilateral minimal hearing loss. Comprehensive patient's medical history and extensive literature review suggestive of suspected amikacin as the causal agent and discontinued. Ciprofloxacin 500 mg twice daily was started as alternative antibiotic choice for his UTI. The patient was put on symptomatic therapy of injection thiamine, betahistine 10 mg and cinnarazine 25mg OD. The symptoms like giddiness, vertigo, nystagmus were resolved within 42 h, however patients still complaints of minimal hearing loss. A casual association between vestibular ototoxicity and amikacin was assessed by World Health Organization probability scale and Naranjo's algorithm it showed "probable" association between the adverse drug reaction and Amikacin.

DISCUSSION

Ototoxic effects of aminoglycosides are well established, still they are widely used today. The patient's vulnerability to toxicity of aminoglycoside may increase with dose, frequency and duration of aminoglycoside therapy.^{4,6} Indeed, it is well documented that genetic predisposition as a risk factor for aminoglycoside induced ototoxicity.⁴ However in this case we fail to establish the association of hearing loss with these parameters.

Aminoglycoside ototoxicity is likely multifactorial, that damage the inner ear or the vestibulo-cochlear nerve.^{4,5} Cochleo-toxicity, can occur through damage to the cochlea or the cochlear branch of the vestibulocochlear nerve. Whereas Vestibular ototoxicity affects the balance organs or the vestibular branch of the vestibulo-cochlear nerve.^{2,6,7} The common symptoms of amikacin related vestibular toxicity includes dizziness, ataxia, nystagmus dysequilibrium and oscillopsia.^{1,7} A similar kind of presentation observed in our patients. Patient had sensorineural bilateral hearing impairment disturbing the higher frequencies, then developing to lower conversational-level frequencies. It is reliable with the pathophysiology of aminoglycoside-induced hearing Impairment.

Aminoglycosides enter the inner ear fluids of the organ of Corti and the sensory hair cells induce cell death by different cellular mechanisms. Mitochondrial protein production disruption, cellular membrane potentials changes, interact with the transition metals, free radicals formation, c-Jun N-terminal kinase (JNK) activation,

caspsases and nucleases are few reasonable pathological mechanisms.^{4,5,8} Ultimately, leads to perpetual hearing loss and balance dysfunction by the permanent loss of sensory hair cells in the cochlea and vestibular apparatus⁸

Onset or progression of aminoglycosides induced hearing loss may vary, it can happen even after the aminoglycoside treatment.⁶ The delayed onset of ototoxicity is probably owing to the slow clearance of aminoglycosides from inner ear fluids compared to serum.⁸ In our case, patient developed vestibular ototoxicity within three days with a total of 3gm covering up the majority of the symptoms. Family and personal history, medical and social history did not suggest any primary auditory diseases.

CONCLUSION

The undesirable effects of permanent hearing loss can have awful consequences in person's quality of life. Therefore, early detection, management and therapeutic approaches for prevention of hearing loss is crucial. All the patients on chronic treatment with aminoglycosides should be monitored up to 6 months after cessation of aminoglycoside treatment.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

ABBREVIATIONS

UTI: urinary tract Infection; **OD:** Once daily.

REFERENCES

- Selimoglu E. Aminoglycoside-Induced Ototoxicity. *Current Pharmaceutical Design*. 2007;13(1):119-26.
- Petersen L, Rogers C. Aminoglycoside-induced hearing deficits – a review of cochlear ototoxicity. *South African Family Practice*. 2015;57(2):77-82.
- Jerome A, John AR, Wayne LG. Aminoglycoside-induced ototoxicity. *CMAJ*. 2015;187(1):E52.
- Jing Xi, Andra ET, Jochen S. New developments in aminoglycoside therapy and ototoxicity. *Hear Res*. 2011;281(1-2):28-37.
- Rogers C, Petersen L. A Aminoglycoside-induced balance de cits: a review of vestibulotoxicity. *S Afr Fam Pract*. 2011;53(5):419-24.
- Charles AP, Shaun EB, Annette TN, Patricia MS, Marian C, Gwen AH, et al. Aminoglycoside Toxicity: Daily versus Thrice-Weekly Dosing for Treatment of Mycobacterial Diseases. *Clinical Infectious Diseases*. 2004;38(11):1538-44.
- Mohammad R, Javadi B, Abtahi K, Gholami B, Safari M, Payam T, et al. The Incidence of Amikacin Ototoxicity in Multidrug-Resistant Tuberculosis Patients. *Iranian Journal of Pharmaceutical Research*. 2011;10(4):905-11.
- Huth ME, Ricci AJ, Cheng AG. Mechanisms of Aminoglycoside Ototoxicity and Targets of Hair Cell Protection. *International Journal of Otolaryngology*. 2011;1-9.