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Ototoxicity of Acetic Acid: A Short Review

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Introduction

We previously published an animal study in this journal entitled "Differences in acetic acid ototoxicity in guinea pigs are dependent on maturity" [1]. Prior to that paper, we reported several other papers regarding animal studies on the ototoxicity of acetic acid. In this paper, we summarize the ototoxicity of acetic acid, as previously reported [2].

Various drugs, in the form of ear drops or topical irrigation, are used to treat chronic discharging ears. Recently, Burow's solution has gained popularity for its excellent antibacterial and antifungal activity. A specifically interesting attribute of this solution to clinicians is its efficacy in treating MRSA, i.e., Methicillin-Resistant Staphylococcus aureus as well as various other antibiotic-resistant bacteria and fungi. 13% aluminum acetate is the main component of this solution. The original Burow's solution 3.5 pH and, when it was applied in a guinea pig's middle ear cavity for 30 min or longer, it induced a curtailment in the compound action potential (CAP). But, a twofold diluted Burow's solution having higher pH of 4.4 led to no curtatilment in the CAP threshold [3].

Before our disquisition, there had been no consideration of the ototoxicity of acetic acid with various pH [2]. Compound action potentials (CAP) of the eighth nerve were calculated in guinea pigs prior to and after the application of acetic acid in the middle ear cavity. The different pH values of the acetic acid solutions were pH 3.0, 4.0 and 5.0, and their corresponding application time was 30 min, 24 h and 1 week. Acetic acid solution with a pH of 3.0 applied for 30 min led to no notable advancement of the CAP threshold at 4 kHz, but a remarkable advancement of the threshold was noted for 8 kHz and clicks. Acetic acid solution with a pH of 4.0 applied for 24 h led to notable advancement of the CAP threshold for 8 kHz, 4 kHz, and for clicks. Acetic acid of pH 5.0 applied for 24 h led to a notable advancement of the CAP threshold for 4 kHz, but not for 8 kHz or clicks, whereas acetic acid of pH 5.0 applied for a span of 1 week led to a little but notable advancement of the CAP threshold for 8 kHz as well as 4 kHz tone bursts, but no remarkable modification was observed for clicks. In brief, we explored a important poisonous action of acetic acid at pH of 5.0 or lower on eight-nerve CAP in guinea pigs. Distinctly, the stronger the acidity, the prolonged the exposure period, the higher advancement in the CAP threshold.

The poisonous action of acetic acid was also noted by Ikeda and Morizono [4] on the guinea pig cochlear when pertained in the middle ear cavity. A 2% acetic acid of pH 2.0 was applied to the round window in the guinea pig for 30 minutes. The authors found a reduction in the endolymphatic direct current potentials several minutes later. Jinn et al. identified damage to the outer hair cells with 2% acetic acid applied for 60 min [5]. The ototoxicity of Burow's solution in guinea pigs was also reported by Suzuki et al. [6]. Guinea pig's temporal bone was cultured for 7 days after irrigation of the middle ear cavity and destruction to the outer hair cells was delineated to initiate from the basal turn of the cochlea reaching towards the upper turns, showing that the drug dispersed from the circular aperture membrane.

Caution is needed when translating animal studies into clinical settings, since anatomical differences exist between humans and rodents. Guinea pig and chinchilla are the two most widely used laboratory animals for the studies of ototoxicity. Both the species have a circular aperture which is strongly revealed in the middle ear cavity, contrary to the circular aperture of human, which is enormously suspended in a straitened niche. More significantly, perhaps, there is a difference in the thickness of the circular aperture membrane between humans and rodents. The membrane of humans is about six times more profused and has a higher collagen density than the membrane of rodents. These anatomical differences could bestow a large hindrance to the passive diffusion of the drug from the middle ear to the inner ear, but it also shows that these rodent models could then be a more delicate sentinel models for ototoxicity. It is preferable to circumvent the merging of acidic solutions in the middle ear cavity or to let them remain in contact with the circular aperture for a long time. However, no adverse effect is expected in patients with an intact ear drum.

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