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, led to no notable advancement of the CAP threshold at 4 kHz, but a twofold diluted Burow’s solution having higher pH of 4.4 led to no curtailment in the CAP potentials (CAP) of the eighth nerve were calculated in guinea pigs 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 4 kHz tone bursts for 60 min. A remarkable advancement of the threshold was noted for 8 kHz and for 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. 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 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 longer time. However, no adverse effect is expected in patients with an intact ear drum.

References


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