

Change Of Video Head Impulse Test Parameters After Intratympanic Gentamicin Injection In Intractable Meniere's Disease

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Objectives: This study was to determine changing pattern of vestibule-ocular reflex (VOR) gain of each semicircular canal in Head Impulse Paradigm (HIMPs) and saccades in Suppression Head Impulse Paradigm (SHIMPs) for intractable Meniere's disease patients after intratympanic gentamicin injection (ITGM).

Background: ITGM is a well-accepted means to treat intractable Meniere's disease because of feasibility and long-term results. ITGM affect to VOR gain of each semicircular canal and it can be measured by HIMPs. And SHIMPs evaluate anti compensatory saccades that imply residual vestibular function. But, there are a few studies about results of HIMPs and SHIMPs after ITGM in Meniere's disease. We hypothesized that VOR gain or saccades could be one of the prognostic factors in Meniere's disease after ITGM.

Methods: 16 patients suffering from definite Meniere's disease treated by ITGM were retrospectively reviewed. We conducted HIMPs, caloric test and pure tone audiometry (PTA) in all patients and SHIMPs in 5 patients each before ITGM and 1 month after ITGM. Patients were followed up 1, 3, 6 months after ITGM. We defined VOR gain difference as an amount of decreased gain during 1 month after ITGM. Patients were classified into two groups: single injection vs multiple injections. Multiple injections group had poor vertigo control in 6 months that required second or third ITGM later in follow up period.

Results: The patients mean age was 59.44 ± 14.79 (min: 40 years, max: 83 years). 6 patients were included in multiple injections group and 10 patients were included in single injection group. In 16 subjects, six didn't have canal paresis on the affected ear before ITGM. VOR gains of each semicircular canal in the treated side were decreased in all patients ($p < 0.05$). Mean VOR gain differences of the treated side were 0.32 ± 0.21 , 0.18 ± 0.22 and 0.26 ± 0.19 in each horizontal, anterior and posterior canal plane. And VOR gain difference of horizontal canal plane were higher than those of anterior canal plane ($p < 0.05$). Change of canal paresis value was correlated with VOR gain difference of horizontal canal plane ($P < 0.05$) but not of vertical canal plane. Subjects increased threshold more than 10dB in PTA were two and they were all multiple injections group.

Between two groups, mean VOR gain differences of each canal plane were lower in multiple injections group than single injection group, but it was not statistically significant ($p > 0.05$). In 5 patients who were conducted SHIMPs, the amplitudes of anti-compensatory saccade were decreased in 3 of 5 patients including one subject in multiple injections group and two subjects in single injections group. And other two subjects in single injections group showed increased anti-compensatory saccade after ITGM.

Conclusions: Our results suggest that ITGM caused a significant decrease of VOR gain in all patients treated with ITGM. However, ITGM appears to cause a differential loss of function across the three canals in the injected labyrinth. Although it was not statistically significant, if VOR gain difference is relatively low after initial ITGM, patients might have poor vertigo control and be required another ITGM.