INTRODUCTION

Traumatic facial nerve injury is a common clinical disorder with significant morbidity. It is the most frequent cause of facial paralysis during childhood and is the second most frequent in adults, followed by Bell's palsy (1). Besides its serious functional deficits, it can cause psychological trauma due to facial asymmetry.

We aimed to evaluate the effects of HBO, methylprednisolone (MP) and combined HBO-MP treatments on traumatic facial nerve regeneration by histopathological examination of the facial nerve in rats in this experimental study.

MATERIALS AND METHODS

Approval was obtained from Haydarpasa Numane Education and Research Hospital Ethical Committee. Twenty Sprague-Dawley rats weighing between 350-450 g were used throughout the experiment.

The procedures were carried out by two surgeons. The main trunk of the nerve before branching was compressed with a hemostat for 1 minute. Complete loss of the facial function on the right side was achieved confirming the crush injury in all rats. Animals were divided into four experimental groups based on the type of treatment: Group 1: HBO therapy for 60 minutes under 2.4 atm pressure/day for 5 days (twice a day only for first day) together with methylprednisolone (MP) 30 mg/kg Group 2: Control animals receiving no therapy Group 3: HBO therapy for 60 minutes under 2.4 atm pressure/day for 5 days (twice a day only for first day) together with methylprednisolone (MP) 30 mg/kg via intraperitoneal route Group 4: Methylprednisolone 30 mg/kg via intraperitoneal route

The animals were sacrificed with high dose general anesthesia one week after the trauma, followed by removal of 1 cm nerve segment from the crush site to distal end.

All statistical calculations were performed with NCSS (Number Cruncher Statistical System 2007) and PASS (Power Analysis and Sample Size) 2008 Statistical Software (Utah, USA). Besides standard descriptive statistical calculations (median, frequency and ratio), Kruskal Wallis test and Mann Whitney U were used in the assessment of parameters. The statistical significance level was established at p<0.05.

RESULTS

The evaluations of the axonal degeneration, vascular congestion, macrovacuolisation, the size of axon diameter and the thickness of myelin sheath according to the groups were presented in Table 1.

The median of control group was statistically lower than the medians of group 1, 2 and 4 regarding axonal degeneration (p<0.05). The medians of macrovacuolisation scores in group 3 was statistically lower than the median of control (group 2) (p<0.05). But the differences between the medians of the other groups were not statistically significant (p>0.05) (Figure 2). The medians of group 3 and 4 were statistically lower than the medians of group 1 and 2 according to vascular congestion (p<0.05) (Figure 1). The medians of vascular congestion scores in group 3 and 4 were statistically lower than the median of control (p<0.05; p<0.05).

There was a statistically significant difference between the groups according to axonal degeneration scores (p<0.05). The medians of macrovacuolisation scores in group 1 was statistically higher than the group 3 (p<0.05). But the differences between the medians of the other groups were not statistically significant (p>0.05). There was a statistically significant difference between the groups according to axonal diameter scores (p<0.05). The medians of axon diameter in group 3 and 4 were statistically higher than control (p<0.05; p<0.05). The myelin thickness score in group 3 was statistically higher than the group 4 (p<0.05). There was a statistically significant difference between the other groups according to myelin thickness (p<0.05).

Consequently, besides lesser axonal degeneration and vascular congestion, and larger diameter of axons were observed in group 3 (combined HBO-MP) compared to control group. Furthermore we observed thicker myelin and lesser axonal degeneration in group 3 in comparison to the group 4. HBO therapy was dropped in this study, there was no significant difference in the histopathological findings between group 1 (HBO) and the control group.

DISCUSSION

Various therapies were designed to enhance regeneration of the facial nerve. Electrical stimulation, gonadal steroids and the combination of the two, HBO and MP were studied after a crush injury [3, 4].

The effects of HBO on peripheral nerve regeneration were reported with conflicting results. In a study performed by Mohajerani et al. at the degree of nerve regeneration evaluated histologically was decreased in HBO treated compared with control animals (5). Lesser edema was detected in HBO treated animals in another study (6). But, Saroiu et al. found no influence of HBO on nerve regeneration (7). We observed statistically significant difference in HBO group other than control according to the evaluation of histopathological examination results.

MP acts directly to retard secondary neuronal degeneration. The mechanism of action of the steroids is by reducing edema and protecting the cell membrane against peroxidation. Steroids have ability to slow the anterograde degeneration of experimentally injured cat soleus motor nerves (8, 9). We used a single dose of 30 mg/kg MP in group 3 and group 4 as used in the study performed by Cayh et al at (9). We found no statistically significant difference in axonal degeneration and myelin thickness scores between the MP group and control. Vascular congestion was less and axon diameter was significantly larger in MP group with compared to control and HBO group. Group 3 (HBO-MP) had lower axonal degeneration and higher myelin thickness scores. Myelin thickness and axonal degeneration scores were not statistically different between group 1 (HBO), group 2 (control) and group 4 (MP).

This findings suggested better recovery in group 3 than group 1, group 4 and control. Neural lesions result in edema and increase in endothelial pressure which provokes ischemia and axonal degeneration (10). Facial nerve injury is thought as the result of the same ischemic processes. The combination therapy with MP and HBO may give us the benefit of treating both ischemia and edema occurring through the facial nerve injury cascade. According to the histopathological results of our study, the best results were obtained with combination treatment. In conclusion combination therapy may be a therapeutic alternative that might bring about better results.

REFERENCES