Effects of Edaravone on Ischemia-induced Facial Palsy

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[Introduction]

Although acute peripheral facial palsy (APFP) is initiated by reactivation of herpes group virus, nerve edema plays an important role in developing facial palsy. Since the intratemporal portion of the facial nerve is surrounded by a narrow bony canal, nerve edema results in an increase in facial nerve pressure within the fallopian canal. When the nerve pressure exceeds a certain limit, nerve conduction and axonal transport are blocked and regional ischemia occurs.

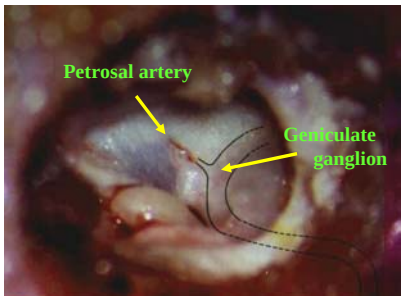
Reactive oxidative species (ROS) are formed under physiological condition in the human body and removed by cellular antioxidant defense system. However, this delicately balanced situation, gets easily tipped off in case of oxidative stress, e.g. ischemia, leading to increased levels of ROS. Harmful ROS is well known to result in tissue damage.

In APFP, ROS is easily deduced to increase in the facial nerve, consequently leading to nerve damage. If this is the case, an inhibition of ROS is likely to prevent the nerve damage in APFP.

Edaravone is an extremely potent scavenger of hydroxyl radical inhibiting not only hydroxyl radicals but iron-induced peroxidative injuries. In the present study, effects of edaravone on ischemia -induced facial palsy are studied experimentally.

[Materials and Methods]

1.Animal model of ischemia-induced facial palsy

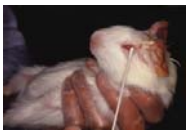


The interruption of the petrosal artery produces facial palsy in guinea pigs.

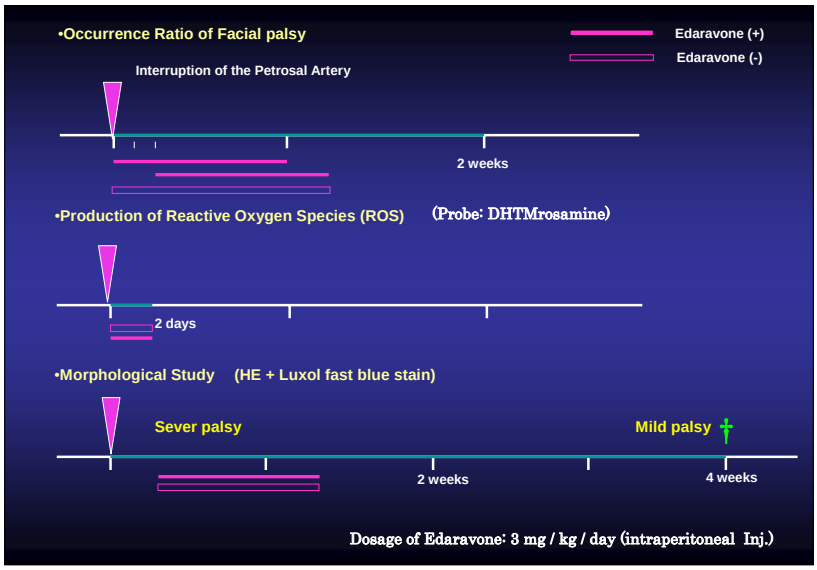
2. Grading of behavioral facial palsy

Severity of the facial palsy was graded by blink reflex.

Grade of Facial Palsy	
Grade 3	complete loss of reflex
Grade 2	unable to close eye completely
Grade 1	delayed reflex
Grade 0	normal

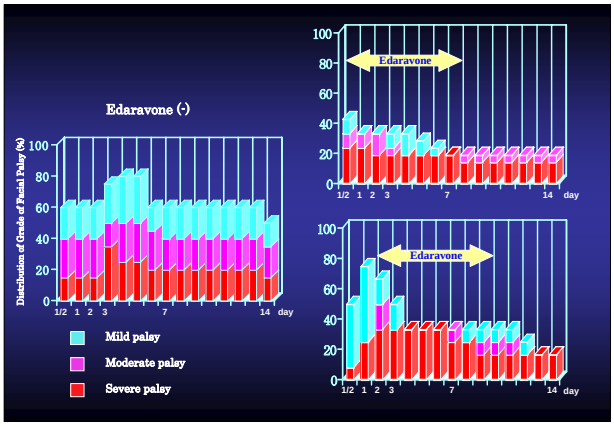


3. Study Design

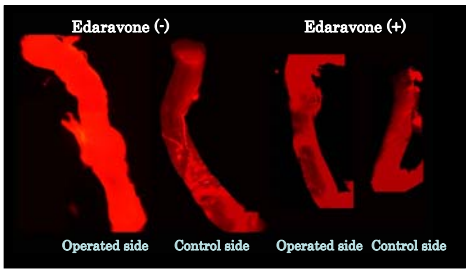


[Results]

1.Distribution of Grades of Facial Palsy and Edaravone

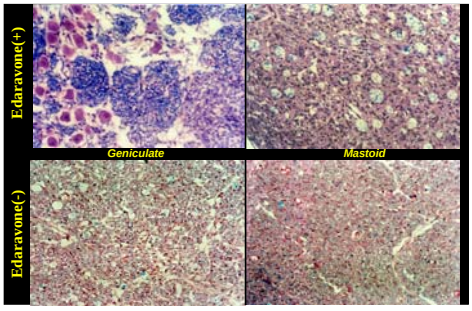


2. Production of ROS



Edaravone attenuated the product of ROS remarkably.

3. Morphological Study



Degenerative changes of the facial nerve was mild in the edaravone-treated animals.

[Conclusion]

1. Edaravone, an inhibitor of ROS, attenuated the development of ischemia-induced facial palsy in guinea pigs.
2. Edaravone prevented the production of ROS remarkably.
3. Edaravone tend to prevent the degenerative changes of the facial nerve caused by ischemia.