Comparison Between Peripheral Facial Nerve Palsy in Man and Experimental Palsy in Animals

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Received for publication, February 25, 1972

Several reports concerning the histopathology of the facial nerve in BELL’s palsy and Hunt’s syndrome are found in literature. However, observation with an electronmicroscope has rarely been performed.

The results of electronmicroscopic studies on these diseases are presented in this paper, and in addition, a comparative study on facial nerve paralysis in rabbits induced by an injection of herpes simplex virus is reported.

REPORT OF CASES

Case 1. A 19-year-old male with BELL’s palsy in the right side. Paralysis developed on 14 Dec. 1968 without known cause. There were no history of fever, vertigo, hearing impairment nor earache. Physical examination was done on 22 April 1969 and otherwise no abnormality from the otolaryngologic point of view. Blood pressure 110 in systolic and 75 in diastolic. An electro-cardiogram was normal. Liver function test showed A/G ratio 1.7, serum total protein 7.4 gm/dl, Kunkel test 5.0, glutamic transaminase 26 SF units. In hematology, RBC was 5, 27 million and WBC was 4, 500 with normal differential counts. Audiogram and equilibrium tests were normal. Middle ear roentgen film was negative. Schirmer’s test showed 9 mm in the right and 11 mm in the left which were within normal limits. Taste sensation was almost absent qualitatively. An evoked electromyography on the peripheral muscles made no response to the maximum volume.

Operative findings (on 25 April 1969, 133rd day from the onset).

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The nerve tissue around the chorda tympani bifurcation region showed an atrophic change and no bleeding was seen by cutting the nerve sheath.

Electronmicroscopic study revealed that the lesion showed a marked reduction of nerve fibres and a replacement by collageneous fibres which were highly degenerative. In some sections, a few nerve fibres remained and regenerative fibres were surrounded by the hypertrophic myelin sheaths and in other sections there were non-myelinated fibres noticed. (Fig. 1)

Case 2. A 65-year-old male with HUNT’s syndrome in the right side.

A retroauricular pain developed on 2 Dec. 1969 and was followed by the paralysis of right side on next day. Small vesicles were noticed in the right auricle at that time, accompanied by marked pain. There were no vertigo, tinnitus nor hearing impairment. Physical examination (4, January 1970) revealed facial nerve paralysis in the right side. Crust formation was seen at the helix and the meatus area. Blood pressure was 128 in systolic and 84 in diastolic. RBC 5,09 million, WBC 6,800 with normal differential counts. GOT 24 SF units and GPT 18 SF units. Electro-cardiogram was normal. Roentgen film of the middle ear was negative. Audiogram showed a bilateral high tone loss due to neural deafness. The facial nerve examinations revealed through Schirmer’s test 4 mm right and 9 mm left, an almost total loss of taste sensation and no response of peripheral muscles to the maximum volume in evoked electromyographic test.

Operative findings on 27 January 1970, the 56th day from the onset.

There were no abnormalities seen in the vertical portion distal from the chorda tympani. However, the nerve tissue looked reddich and edematous in the horizontal portion, proximal from the stapes tendon, the tissue in some part came out of the decompression canal.

Electron microscopic findings: In this case there was marked reduction of the nerve fibres as in case 1. The nerve fibres were replaced by collageneous fibres. There were some regenerative nerve fibres seen in the section where nerve fibres remained and there was a hypertrophic sheath seen in other sections. (Fig. 2) In the degenerative lesion, the nerve fibre was damaged in such way that the myelin sheath got into axon which contained vacuoles of various size (Fig. 3) Schwann’s cell showed high electron density and were phagocytising degenerated substances.

COMMENTS

The histopatologic findings on BELL’s palsy and HUNT’s syndrome have been reported by MINKOWSKI, DEJERING, MILLS, ALEXANDER,
In general, the reporters describe a variety of findings. Some were normal and others had a high degree of degeneration which depended on the damage in the nerve tissue. Fowler alone noticed a high degree of degeneration with intraneural hemorrhage and made the speculation that one of the possible causes of the disease might be due to intravascular disturbance resulting in the formation of the microthrombi. On the other hand, as the etiology of Bell’s palsy, Blatt proposed an inflammatory process instead of an ischemic process.

The author made electron microscopic study on a patient with Hunt’s syndrome whose nerve tissue of the horizontal portion was reddish and swollen. The nerve fibres became less in quantity and degenerative. This appearance was very similar to the experimental facial nerve paralysis in rabbits induced by an injection of herpes simplex virus. In this experiment, the tissue in early stage—7 days after injection—presented marked dissolution with an infiltration of erythrocytes and in later stage—13 days—Schwann’s cells and myelin sheaths were equally damaged and the sheaths were absorbed into the interior of the axon cylinders which were also highly degenerated and showed vacuolization of various sizes. The observation was continued until 223rd day. The regenerative process in the nerve fibres was much slower than in a case of experimental surgical division of the facial nerve, and the final results varied. Some of them showed a hypermyelinated regeneration and others were surrounded by growing collagenous fibres. Virus particles were not seen in the case of Hunt’s palsy. This may be due to the fact that the specimen was not taken from the geniculate ganglion. In experiment, however, viral particles could be seen until 50 days but not later that. Then it is likely that the viral particles disappear in the prolonged case.

The examination of case 1. was made after prolonged interval from the onset (133 days) and resulted in a poor regeneration, accompanied by a replacement of collagenous fibre and of connective tissue growth around the regenerative nerve fibres. These findings were similar to a case of long-term observation in experimental facial nerve paralysis which was induced by an injection of herpes simplex virus.

It is impossible to explain an ischemic condition from histopathologic specimens only, since from the author’s experience with Bell’s palsy which were so degenerative as to be unable to respond to the evoked electromuography, there were no abnormal vascular findings at the 49th day examination from the onset. (Fig. 4) However, it is interesting to see that both the experimental case induced by herpes simplex virus and Bell’s palsy case presented similar findings about 49th day from the onset, showing heavily degenerative change with phagocytosis by Schwann’s cells. (Fig. 5, 6)

It is well known that main region of damage in Bell’s palsy is in
the area close to the chorda tympani. The author had experience with a child who had aseptic meningitis which was said to be due to virus disease, complicated by facial nerve paralysis. Cerebrospinal fluid showed 54/mm³ lymphocytes and was negative in culture. The patient had normal taste sensation. Another case was a 12-year-old patient with chronic nephritis showing elevation of blood pressure, 220 systolic and 140 diastolic, complicated by bilateral renal retinopathy and left facial nerve paralysis. The patient also had normal taste sensation.

Accordingly, whichever virus originated case and vascular damaged case, the nerve damage was thought to be located in the infra-chordal portion. The vertical portion in which the chorda tympani bifurcate is the area complicated anatomically with narrow nerve canal and arteriovenous anastomosis nearby. MULLER14) mentioned that in the area of bifurcation of the chorda tympani vascular accidents occur very easily.

In order to stop blood circulation of the facial nerve author removed the epineurium including the vessels and cut off the stylomastoid artery at the stylomastoid foramen but facial nerve palsy did not develop. On the other hand, when in the experimental study herpes simplex virus was injected into the facial nerve canal, facial nerve palsy developed but did not develop in the cases of injection into the extra-temporal facial nerve. Furthermore, the axon flow in the nerve of the facial nerve canal which was examined by injection of fluorescent substance into the extra-temporal portion of the nerve, was slower than that of the extra-temporal portion.

Accordingly, injected viruses would continue to proliferate or cause circulus vitiosus in a case of vascular damage in the nerve of the facial nerve canal. In order to clear up the etiology of BELL'S palsy, further study must be done on a culture of virus from the facial nerve or by observation of the facial nerve by means of the immunofluorescent technique.

SYNOPSIS-ABSTRACT

1. HUNT'S paralysis was similar to experimental study in which paralysis was induced by the injection of herpes simplex virus into the stylomastoid foramen. Also this experimental facial nerve paralysis was similar to Bell's palsy in the case of severe damage histopathologically.

2. Axon flow was slower in the nerve of FALLOPIAN canal than that of the extra-temporal facial nerve. Accordingly, viruses would continue to proliferate or cause circulus vitiosus in the case of vascular damage in FALLOPIAN canal.
REFERENCISE


Fig 1. Case 1. Electron micrograph showing marked hypertrophic myelin sheaths and surrounded collagen fibres.

× 7.900
Fig 2. Case 2. Electron micrograph showing regenerated nerve fibres (Arrows) are extremely small with marked hypertrophic myelin sheaths. $\times 3,950$
Fig 3. Case 2. Electron micrograph showing the myelin sheath being absorbed into the interior of the axon which shows vacuolization. $\times 3,050$
Fig 4. Another case of Bell's palsy, 49th day from the onset. Photomicrograph showing no abnormal blood vessels. (hematoxylin and eosin) × 400
Fig 5. Experimental case induced by herpes simplex virus. Photomicrograph showing heavily degenerative change with phagocytosis by Schwann's cell. (hematoxylin and eosin) × 1,000
Fig 6. Another case of Bell's palsy, 49th day from the onset. Degenerative change shows to be similar in finding of experimental case. (hematoxylin and eosin) x 1,000