Case Report

EFFECT OF NEUROMUSCULAR REEDUCATION IN BILATERAL FACIAL PALSY ON PATIENT WITH GBS

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ABSTRACT

Background: Bilateral facial palsy is a rare entity and remains to be a challenging case to diagnose and manage which has the major impact on the physical and social aspect of the affected individual.

Objective: The aim of the report is to determine the role of neuromuscular reeducation in restoration of function in person with Guillain Barre Syndrome present with facial diplegia.

Case report: We report the case of 23 year male presenting with history of deviation of mouth to the right side, followed by bilateral facial involvement and latter distal symmetrical involvement of bilateral upper and lower limb. The facial diplegia was managed by PNF and Electrical stimulation.

Conclusions: Neuromuscular reeducation is an effective intervention for restoration of function after facial diplegia.

KEYWORDS: Guillain-Barre Syndrome (GBS), Bilateral facial palsy, Proprioceptive Neuromuscular Facilitation (PNF), Electrical stimulation, Neuromuscular Reeducation.

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BACKGROUND

The facial expressions of human convey both the strongest and gentlest emotions of the spirit and lower most bestial pleasures. Bilateral facial nerve palsy is a rare representation found in less than 2% of all the facial palsy cases, and has an incidence of 1 per 5,000,000 population.¹ Because of the problems occur at the very personal level it needs a special consideration.

In this case report, we present a case of GBS with bilateral facial palsy. Facial muscle as a manifestation in GBS is rare and few cases have been reported.² ³ In some cases of GBS, other cranial nerves may also involved with coexistence of dysphasia and dysarthria.⁴ Various methods have been used for facial rehabilitation such as neuromuscular re-education, meditation, soft tissue mobilization, and chemodenervation, which have shown signific-
There was no lymph node and thyroid enlargement. The patient was admitted and on the next day morning left side of the face was also involved and weakness of all the four limbs became more pronounced.

He underwent various investigation and report suggest following findings, Hemoglobin level was 17mg/dl, there was absence of WBC and RBC on the urine, CPK level was 392, HBSAg was negative, patients was non reactive to HCV and HIV. Lumbar puncture was performed, the cerebrospinal fluid (CSF) analysis showed the protein 82.9mg/dl, glucose 52mg/dl nucleated cell count was 4 cell/cm and CSF was colorless. There was no organism grown in culture, after 48 hours of incubation. Liver function test was performed which showed increased bilirubin (0.23gm/dl) and total protein was 8.3gm/dl, other readings were within the normal range. In mini renal profile except the decrease in urea level (11mg/dl) other reading were within range. Motor nerve conduction including elicitation of F-wave were performed on right median, ulnar, posterior tibial and common personal nerve which showed partial motor conduction block in right ulnar and common personal nerve. Distal CMAP amplitude was grossly reduced and distal motor latency were increased in all the nerve sampled. F- Wave was absent on all the nerve sampled. Sensory nerve conduction were performed on median, ulnar, superficial peronal and sural nerve which showed absent SNAP in ulnar nerve and reduced amplitude in other nerve sampled. SSR variation was elicitable in right hand and right foot and RR variation was normal during deep and quiet breathing as reported.

On examination the patient had grade 5 facial weaknesses on House Brackmann grading system. Total score of 10 on Yanagihara 5-point facial nerve grading system which is shown in table 1. Sunny Brook facial nerve grading system showed score of 8 on symmetry of contraction and 5 on synkynesias as shown in table 2. Bilateral facial MMT showed absent frontalis activity and weak functional activity of corrugator supercilli, orbicularis occuli, procerous, nasalis, zygomaticus major, orbicularis oris and mentalis. Resting symmetry on Sunny Brook grading system shows wide eye opening, less pronounced nasolabial fold and corner of mouth were not dropped. Patient had difficulty to initiate pursed lip. There was a bilateral symmetrical peripheral weakness of bilateral upper and lower limb. Most of the peripheral muscle had grade 3 on MMT. The reflexes were diminished bilaterally on the extremity. These investigation and compatibility of clinical presentation revealed the evidence of axonal demyelinating motor sensory neuropathy. The patient was under medical treatment as per the direction of medical consultant.

He was treated with the electrical stimulation applied to the appropriate motor point, rhythmic initiation and repeated stretch at the initial range to facilitate initiation of movement till 3rd days. Galvanic current was used to stimulate the facial muscles and faradic current for the facial nerve trunk. Thirty contractions of three set was used for each muscle. From 4th days after the patient can voluntarily initiate the movement electrical stimulation was combined with Facial PNF Technique which utilized rhythmic initiation and

Fig. 1: Eye closing before and after treatment sessions.
Combination of isotonic. Rhythmic initiation was progressively used from passive range to active range with or without manual resistance. Combination of isotonic was used when patient can initiate contraction voluntarily to achieve active control of motion, strength and coordination. Verbal command and resistance to reflex or voluntary movements was utilized throughout the sessions. Total seven days of treatment was given. Over the period, his facial asymmetry got reduced. House Brackmann grading was grade II on his both sides. The eye closing becomes narrow, nasolabial fold appears normal; patient was able to perform purse lip. Bilateral facial muscle became functional. Total score of on Yanagihara 5-point facial nerve grading system was 34 which is shown in Table 1. Sunny Brook facial nerve grading system showed score of 16 on symmetry of voluntary contraction and synkynesis was absent with all the facial movement as shown in Table 2.

**Table 1:** Yanagihara 5-point facial nerve grading system.

<table>
<thead>
<tr>
<th>Test items</th>
<th>Pre intervention score</th>
<th>Post intervention score</th>
</tr>
</thead>
<tbody>
<tr>
<td>At rest</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>wrinkle forehead</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>blink</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>slight closure of eye</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>slight closure of eye</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>closure of eye on involve side</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>wrinkle nose</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>whistle</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>grin</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Depress lower lips</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Total score</td>
<td>10</td>
<td>34</td>
</tr>
</tbody>
</table>

**Table 2:** Sunny Brook facial nerve grading system.

<table>
<thead>
<tr>
<th>Test item</th>
<th>Symmetry of movement</th>
<th>Synkynesis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td>Forehead wrinkle</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Gentle eye closure</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Open mouth smile</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Snarl</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Lip pucker</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

**DISCUSSION**

Facial diplegia is rare entity and occurs as a simultaneous onset which means the involvement of the opposite side occurs within 30 days of the onset of the first side. More often the bilateral facial nerve palsy is due to systemic causes compare to unilateral palsy, so wide differential diagnosis must to be considered. It is often associated with multiple idiopathic Cranial neuropathies, neurosarcoidosis, brain stem encephalitis, benign intracranial hypertension, Bacterial meningitis, leukemia, lyme disease, orofacial granulomatosis (Melkersson-Rosenthal syndrome), syphilis and GBS.

GBS is an inflammatory polyradiculoneuritis with uncertain etiology. Bilateral facial nerve palsy can occur in up to 50% of the fatal cases of GBS. The diagnosis is made on the basis of lumbar puncture which shows liquoric dissociation: elevated protein in the absence of raised in number of cell count with clinical findings of peripheral areflexia, and bilateral symmetrical involvement. In our patient lumbar puncture was carried out which showed increased CSF protein, NCV suggest demylinating axonal polyneuropathy and had peripheral weakness of bilateral upper and lower limb with diminished reflex. Hence it was diagnosed as a case of GBS.

Facial palsy in GBS is usually bilateral, but it may present as a symmetrical and rarely as unilateral facial involvement. The facial nerve palsy in GBS is secondary to direct attack of antibodies either due to the demyelination or axonal degeneration which depends on the type of antibody involved. The mechanism for abnormal manifestation of cranial nerve in GBS is still not clearly understood. However, due to autonomic disturbances hypertension may be seen in GBS which may also contribute to facial paralysis. Facial involvement due to hypertension may because of the edema or hemorrhage within the facial canal cause the neural compression.

There is also evidence suggesting disruption of blood brain barrier by the inflammatory infiltrate cells and gadonium production results from enlarge perineural structure due to surrounding inflammation as a cause of involvement of cranial nerve.
Facial palsy in GBS usually follow the limb weakness. In our patient also, the presentation of clinical symptoms were unilateral facial palsy followed by bilateral facial involvement and subsequent limb weakness.

Facial neuromuscular reeducation methods are used for recovery from facial paralysis. Electrical stimulation causes the better and faster recovery with increase tolerance to muscle fatigue and prevent atrophy. PNF is used to improve aesthetics of facial expression by initiating the voluntary effort through facilitation of the activity of neuromuscular mechanism via stimulation of proprioceptor. Facilitatory training involved stretching, strengthening, traction and approximation. As the PNF techniques have the effect of facilitation, so it has also effect on neuromuscular reeducation. Resisted movements of PNF stimulate the proprioceptor (i.e., muscle and tendon spindle) which facilitate muscle contraction. Facial muscle lacks the fascia and act mutuallly, PNF affect the cutaneous muscle distributed over the face and contributes to early improvement.

**CONCLUSION**

The GBS is manifested as a various peripheral-nerve disorders and also with several clinical variants that are characterized by the weakness of limb muscle or muscle innervated by cranial nerve. Muscle reeducation helps in early recovery and better prognosis.

**ABBREVIATIONS:**

GBS- Guillain-Barre Syndrome
PNF- Proprioceptive Neuromuscular Facilitation
CMAP- Compound Motor action potential
SNAP- Sensory nerve action potential

**Conflicts of interest:** None

**REFERENCES**

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