

The Anti-Inflammatory Effects of NaCl with KCl as a Potent Graphene Exfoliator in a Patient with Guillaine-Barré Syndrome and Facial Nerve Palsy

Chur Chin

Department of Emergency Medicine, New Life Hospital Bokhyun-Dong, Daegu, Korea
Email: gemcitabinecisplatin@nate.com

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Abstract

Guillain-Barré syndrome is a rare but fatal autoimmune disease of unknown origin. Infectious disease is the most common etiology of Guillain-Barré syndrome. We had a 75-year-old female patient with Guillain-Barré syndrome and a 90-year-old male patient with facial nerve palsy admitted to our hospital. Both patients experienced recovery from early Guillain-Barré syndrome and peripheral facial nerve palsy after receiving intravenous infusion of NaCl with KCl solution and taking vitamin C.

Keywords

Guillain-Barré Syndrome, Facial Nerve Palsy, Graphene Exfoliator, NaCl with KCl

1. Introduction

A Guillain-Barré syndrome (GBS) case

Guillain-Barré syndrome (GBS) is a common cause of acute flaccid quadriplegia and neuromuscular respiratory failure. There are several factors that contribute to respiratory compromise in GBS patients. Weakness in the pharyngeal and laryngeal muscles, as well as upper airway obstruction, can make it difficult to clear secretions and maintain a clear airway, increasing the risk of aspiration. Additionally, weakness in the respiratory muscles can lead to poor lung compliance, microatelectasis, hypoxemia, and a higher susceptibility to respiratory infections due to impaired coughing ability. Long-term follow-up has shown residual impairments in motor and sensory function and the occurrence of pain,

fatigue, decline in quality of life, and overall functioning in a large proportion of patients with GBS. Respiratory impairment in the acute phase is associated with long-lasting functional impairment. However, in a limited number of studies, lung function values improved steadily, leading to almost complete recovery and no residual detectable impairment after two years [1]. Blood pressure variability, a hallmark feature of GBS, may be closely related to transient increases in catecholamine levels and the dysregulation of baroreceptor reflexes. The demyelination of preganglionic sympathetic axons or axonal degeneration in postganglionic axons may alter feedback control or generate inappropriate ectopic discharges that account for the observed fluctuations. Elevated norepinephrine levels are associated with increased sympathetic outflow (Figure 1) [2]. In this report, we present the case of almost complete recovery of the blood pressure variability and dyspnea diagnosed with GBS through the intravenous infusion of NaCl with KCl solution.

A facial nerve palsy case

The ophthalmic clinical features of facial nerve palsy were mainly eyelid malposition. The facial nerve (the seventh cranial nerve) controls the muscles of facial expression, and dysfunction of facial expression or the blink response occurs with a damaged facial nerve. Blink dysfunction is induced by weakness of the orbicularis oculi and ectropion. Lid retraction may also be induced. Facial nerve palsy can be divided into central palsy and peripheral palsy. Central facial palsy is induced by a brain disorder, whereas peripheral facial palsy is induced by a disorder of the facial nerve pathway emanating from the brain. In this case, facial nerve damage occurs due to inflammation. The first treatment option for facial nerve palsy patients is nonsurgical in nature, defined by a conservative approach based on symptoms [3].

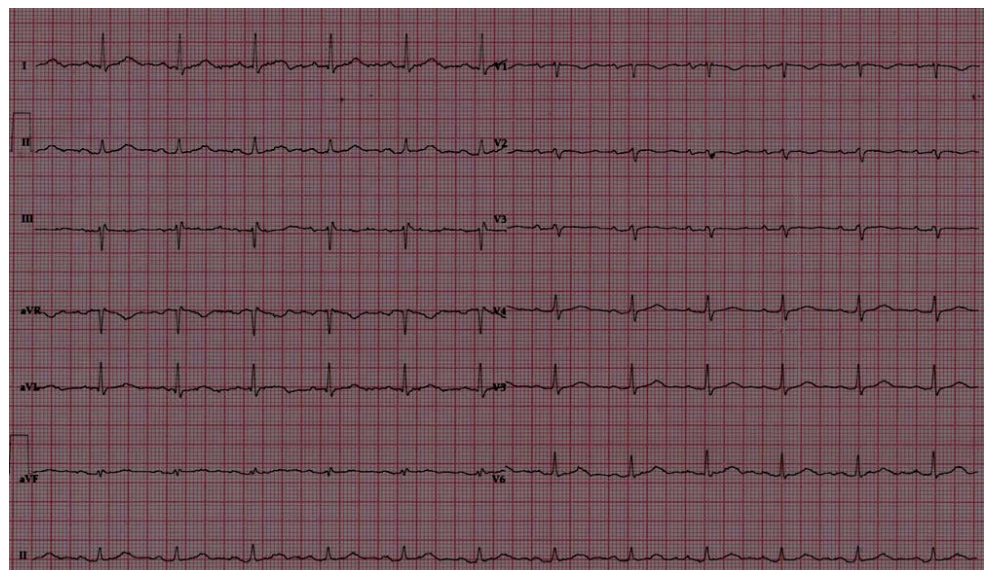


Figure 1. Initial electrocardiogram on admission showed left atrial enlargement. Axonal variant of Gullaine-Barré syndrome can develop dysautonomia and takotsubo cardimypathy.

2. Case Presentation

2.1. Case 1

A 75-year-old woman with Parkinsonism visited the emergency department due to a progressively worsening weakness and numbness in her lower limbs, along with tingling sensations in the lower extremities that had been persisting for the past 10 days. She had no associated symptoms such as fever, rash, headache, backache or blurred vision. Upon arriving at the clinic, right lower lung field consolidation was observed on chest radiography. She was afebrile with dyspnea (O₂ saturation 96%) combined with nasal cannula oxygen therapy and her vital signs were as follows: blood pressure: 100/60 mm Hg; respiratory rate: 24 breaths/min; heart rate: 76 bpm. A neurological examination revealed no facial asymmetry and the cranial nerves were intact. A motor system examination of the lower limbs revealed the following: power of the knee: grade 1/5 below the knees; power of the hips: grade 1/5 of the hip flexors and extensors bilaterally; tone and reflexes of the legs: bilateral hypotonia (**Table 1**); joint position and vibration sense: reduced joint position and vibration as well as swallowing difficulty with L-tube insertion for feeding. The major clinical findings from the objective assessment were bradykinesia, deconditioning, decreased right-hand strength, resting tremors in the left hand, kyphotic posture, and decreased range of motion (shoulders, hips and ankles). The patient experienced blood pressure fluctuations (systolic/diastolic blood pressure 83/47 → 79/48 → 68/44 → 150/90). Laboratory investigations revealed the following: hemoglobin 11g/dl; a hematocrit of 32.8%; total leukocyte count of 4200/cmm; and erythrocyte sedimentation rate of 29 mm in the first hour using Westergren method. Cerebrospinal fluid examination revealed clear fluid and normal opening pressure with no abnormal findings,

Table 1. The electromyogram finding of the patient: Positive sharp wave and fibrillations with reduced recruitment patterns are noted in all examined muscles.

Muscle	Fibrillation	Positive sharp wave	Insertional activity	Bizzar potential	Normal motor unit	Large motor unit	Long duration polyphagic potential	Short duration polyphagic potential	Interference pattern
Lumbar paraspinalis, both	1	2	Incre		+				
Vastus medialis, both	1	2	incre		+		+		Prolonged conduction
Tibialis anterior, both	2	3	Incre		+	+	+		Prolonged conduction
Peroneus longus, both	2	3	Incre		+	+	+		Prolonged conduction
Tensor fascia lata, both	2	3	Incre		+	+	+		Prolonged conduction
Gastocnemius (medial head), both	2	3	Incre		+	+	+		Prolonged conduction

and while a confirmatory electrophysiological study revealed demyelinating neuropathy consistent with prolonged tibial latency of the somatosensory and motor evoked potential. The intravenous infusion of a solution consisting of 250 mL normal saline with 5 cc of potassium chloride (KCl) over 6 h with vitamin C (1000 mg/day) intake resulted in near-complete recovery.

2.2. Case 2

A 90-year-old man, who had previously experienced a cerebral infarction in the right hemisphere in 2008 and an infarction in the posterior limb of the left basal ganglia internal capsule in March 2023, arrived at the emergency department complaining of lid retraction and facial muscle weakness that had started just one day ago. He had a bilateral, relatively symmetric and elevated eyelid position. A neurological examination revealed no facial asymmetry and peripheral type seventh cranial nerve palsy. The intravenous infusion of a solution consisting of 250 mL of normal saline with 5 cc of potassium chloride (KCl) over 6 h with vitamin C (1000 mg/day) intake resulted in near-complete recovery.

3. Discussion

Cytokine storm caused by graphene oxide (GO) induces the production of pro-inflammatory cytokines and chemokines such as interleukin (IL)-1 β , IL-6, IL-18, tumor necrosis factor- α and macrophage inflammatory proteins, ultimately leading to respiratory failure and death due to multi-organ failure [4].

GO nanoparticles have attracted much attention due to their many applications. These applications include batteries, supercapacitors, drug delivery and biosensing. However, few studies have investigated the effects of these nanoparticles on the immune system. Several studies have demonstrated that GO is readily consumed by macrophages. Some investigated the potential inflammatory effects of GO in macrophages. GO elicited stronger nuclear factor-Kappa B (NF- κ B) activation and pro-inflammatory cytokine expression than sulfonated-GO *in vitro* and *in vivo*. Higher concentrations of GO also appear to induce nuclear factor-NF- κ B signaling via TLR4 and can induce IL-1 β release from lipopolysaccharides-primed macrophages.

Eyelid retraction is commonly thought to accompany facial nerve palsy, as it is related to unopposed levator function. The patients with facial nerve palsy and palsy of another cranial nerve presented severe upper eyelid asymmetry and ptosis. There were confounding effects caused by the paralytic ptosis of patients with third cranial nerve palsy. In this case, the patient had a bilateral, relatively symmetric eyelid retraction.

Both anions and cations are inserted in the space between the conjugated graphite layers during intercalation. Anions can enhance the salt-intercalation exfoliation by expanding the interlayer spacing. Compared to sodium chloride (NaCl) alone, a solution containing potassium chloride (KCl) and NaCl can significantly enhance the exfoliation yields of graphene. Specifically, optimization of the cation and anion species can improve graphene yield because co-intercalation of anions

and cations occurs during intercalation in an inorganic salt solution [5] [6].

4. Conclusion

In summary, based on our data, the administration of NaCl + KCl solution does not appear to have a significantly negative impact on the prognosis of Guillain-Barré syndrome (GBS) and peripheral-type facial nerve palsy, which could be attributed to the three-dimensional structure of graphene. This observation may be linked to the exfoliation effect of NaCl + KCl on graphene. Moreover, the intake of vitamin C may help prevent recurrence of these conditions months or even years after the initial diagnosis

Disclosure

Sancta Maria, Mater Dei, ora pro nobis peccatoribus, nunc et in hora mortis nostrae. Amen.

Statement of Ethics

This is an observational study. The requirement for written informed consent was waived because the study was a chart review.

Data Availability Statement

Data will be made available on reasonable request.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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