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Blink Reflex Value in the Early Diagnosis of Guillain-Barre Syndrome

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ABSTRACT

Introduction: Guillain Barre Syndrome (GBS) is an autoimmune-mediated demyelinating or axonal polyradiculoneuropathy characterized by progressive ascending weakness plus areflexia. Three major subtypes are demyelinating, Axonal, and Miller-Fisher Syndrome. The hallmark of the definitive diagnosis of GBS is an electrodiagnostic study. The current study is aimed to assess the value of the blink reflex, as part of an electrodiagnostic study, in the diagnosis of GBS. **Methods:** The current census cross-sectional study has been conducted on 55 patients with the definitive diagnosis of GBS based on clinical and electrodiagnostic manifestations from January 2018 to February 2020 in Isfahan, Iran. The patients were categorized into three groups of demyelinated, axonal, and Miller-Fisher variants and blink reflex entities, including R1, R2i, and R2c were assessed for them. All data were analyzed by independent T-test paired T-test and Chi-square tests. **Results:** R1 was abnormal in R1 was abnormal in 17 (30.90%), R2i in two patients (3.63%), and R2c in two patients (3.63%). Among the studied patients, five (8.47%) had abnormal latency in R1, five (8.47%) in R2i, and five (8.47%) in R2c. The comparison of abnormality in blink reflex entities among diverse subtypes, including axonal, demyelinating, and Miller-Fisher variants, revealed no significant differences (P-value>0.05). **Conclusion:** At least one of the blink reflex indices was abnormal in up to 30.90% of the patients, it seems that the blinking reflex cannot be considered a reliable early diagnostic test for GBS, but this fact cannot completely rule out this method and additional studies should be done with more patients in other populations.

Keywords: Axonal degeneration, Demyelination, Guillain-Barre Syndrome, Nerve conduction studies

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INTRODUCTION

Guillain Barre Syndrome (GBS) is an autoimmune-mediated demyelinating or axonal polyradiculoneuropathy with male predominance [1]. Progressive symmetrical ascending muscular weakness, particularly in the lower extremities than the upper ones and areflexia, are the major clinical manifestations of GBS, while sensory and brainstem abnormalities may be present or absent. Cranial nerve involvement in Guillain-Barré syndrome may present as a complaint in swallowing, eye movements, airway, and facial muscles [2].

This syndrome is classified into three major subtypes including Demyelinating Guillain–Barré: acute inflammatory demyelinating polyradiculoneuropathy (AIDIP), Axonal: acute motor axonal neuropathy (AMAN), Acute Motor and sensory axonal neuropathy (AMSAN) and Miller-Fisher syndrome [3]. The classification is based on nerve-conduction studies [4]. Also, there is a significant geographical distribution for each subtype. Demyelinating is mostly frequent in western countries [5] while Axonal and Miller-Fisher Syndrome are more common in Southeast and Eastern countries [6]. the frequency of the axonal GBS reported ranges from 30% to 65% and the frequency of Demyelinating GBS ranges from 22% to 46% [7-9].

The Miller Fisher syndrome was characterized by ophthalmoplegia, ataxia, and areflexia, In this subtype CSF of patients showed albumin cytologic dissociation [10]. Most patients with the Miller Fisher syndrome have evidence of infection 1 to 3 weeks before the development of symptoms (ophthalmoplegia or ataxia) in the most Miller Fisher Patients was reported [11] some studies reported, C. jejuni infection and Haemophilus influenzae infection in 20% and 8% of Miller fisher patients [12].

Electrodiagnostic studies have a critical role in the early diagnosis and differentiation of various neuropathies [13,14]. The significance of nerve conduction evaluations is that abnormalities can be detected within the initial days of the disease presentations, even in cases with improving clinical status [15,16]. The absence of or abnormality in the F waves accompanying low compound muscle action potentials and the concurrent normal sural response with the abnormality in the sensory action potentials of the upper extremities are the hallmarks found in early nerve conduction [14,17]. Despite the typical involvement of cranial nerves in GBS, the optic nerves as part of the central nervous system are usually spared [18]. Limited studies have reported abnormalities in the evoked potential assessments of optic nerves due to GBS [15,19].

The blink reflex, also known as the Trigeminofacial reflex, is an electrical analog of the corneal reflex in which the ophthalmic division of the trigeminal nerve acts as the afferent limb and the facial nerve is the efferent one. This reflex provides a condition to evaluate the facial nerve response, trigeminal nerve, and pons.

Although Kimura and colleagues conducted a study in terms of assessing the blink reflex in diverse types of polyneuropathies, including GBS, Miller-Fisher syndrome, diabetic polyneuropathy, and chronic inflammatory polyneuropathy [20], the information about the values of the blink reflex in early diagnosis of GBS is limited and controversial [21]. Therefore, the current study is aimed at assessing the frequency and pattern of the blink reflex in the early stages of GBS diagnosis.

METHODS

Study population

This cross-sectional study has been conducted by census sampling on 55 patients who were referred to the Neurology Department of Alzahra Hospital (affiliated with Isfahan University of Medical Sciences) for electrophysiological studies within the first week of symptom presentations from January 2018 to February 2020. The Ethics Committee of Isfahan University of Medical Sciences approved the study protocol based on code IR.MUI.MED.REC.1399.196. The study process was entirely explained to the patients, and written consent was obtained. 67 patients ranging in age from 15 to 90 years who had GBS's clinical manifestations and electromyography-neuroconductive velocity (EMG-NCV) studies consistent with this diagnosis were selected as the study population. Of these, 55 patients whose access to medical record information and complete and accurate EMG-NCV results were available to the authors were selected as the study sample.

The primary diagnosis of GBS based on clinical presentations included progressive weakness of the legs and arms (sometimes initially only in the legs), ranging from minimal weakness of the legs to total paralysis of all four limbs, the trunk, bulbar and facial muscles, and external ophthalmoplegia plus areflexia, and progressive symmetrical ascending muscular weakness, as well as areflexia or decreased reflexes in weak limbs [22]. The diagnoses other than GBS achieved through EMG-NCV studies, and the incidence of a critical condition

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such as respiratory crisis or death were considered exclusion criteria. Due to the ill condition of patients with respiratory crises and moral and clinical considerations, these patients were excluded from the study.

Definitions

The assessed parameters for the diagnosis of GBS included:

- Motor conduction studies of median and ulnar nerves in the upper extremities and tibial and peroneal nerves in the lower extremities conductions, velocities, amplitude, and latencies;
- Sensory conduction studies of median and sural nerve conduction in upper and lower extremities, including velocities, amplitude, and latencies;
- F wave studies and latencies: The logic of F wave assessment was due to the reports regarding F waves slowing in GBS due to the demyelination of the proximal segments or even roots of nerves that cannot be detected by routine conduction assessments [23,24].

The primary outcome of this study was to evaluate the blink reflex status among the patients who met the diagnostic manifestations of GBS based on the mentioned evaluations and also for all of the patient's standard Electromyographic Studies to confirm the diagnosis was done.

Blink reflex: The patients were requested to position supine in a quiet room with their eyes open. The evaluations were performed simultaneously in both eyes. The recording electrodes were put over the orbicularis oris muscle on the lower eyelid, and the reference electrodes were embedded within 2-3 cm lateral to the recording ones, and eventually, the ground electrode was put on the chin.

The simulations were done by keeping the cathode electrodes over the supraorbital nerve on the supraorbital notch, and the anode was directed laterally.

The potentials that were recorded included R1, R2i, and R2c. R1 is a representative of ipsilateral muscle action potential controlled by the facial nerve of the stimulated side; R2i is the reflective response sensed by the trigeminal and facial nerve, both ipsilaterally and R2c is the reflective response made by trigeminal nerve input contralaterally [25]. Due to the unreliability of amplitude, this index has not been analyzed [26]. Abnormal R1, R2i and R2c were defined as >12 ms, >40 ms, and >0.45 ms respectively[27].

The technique of electrophysiological study

The tests were performed using a Medelec Synergy EMG system (Oxford Instruments, Oxford, United Kingdom) and the standard technique of supramaximal surface electrode stimulation and recording. Skin temperature was measured and maintained above 31°C over the palm and 30°C over the dorsum of the foot. The blink reflex was performed by stimulating the supraorbital nerves with cathodes over the nerve at the frontal notch on the forehead. Active electrodes were placed over the inferior orbicularis oculi on both sides. The reference electrodes were placed just lateral to the lateral canthus and the ground electrodes were over the chin [21].

Statistical analysis

The obtained data were entered into the Statistical Package for Social Sciences (SPSS) version 23. The descriptive data were presented in mean, standard deviation, absolute numbers, and percentages. For analytics, an independent T-test paired T-test, and Chi-square test was used. A P-value of less than 0.05 was considered a significant level.

RESULTS

In the current study, data on blinking reflex contributed to 55 patients with a definite diagnosis of GBS based on the electrophysiological studies were assessed. The studied population consisted of 30 (54.54%) males and 25 (45.46%) females with a mean age of 46.25±17.64 years old (range: 16-81 years). Due to our referral hospital, most of the patients (85%) were referred between the 4th day and the 7th day. And no patient entered after 7th days after starting symptoms. Table 1 demonstrates the detailed demographic information of the studied population.

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Table 1: The demographic characteristics of the studied population

Variables	
Gender*	
Male	30 (54.54)
Female	25 (45.46)
Age**	46.25±17.64
Height**	167.4±8.53
Weight**	70.69±14.46
* Number (%) ** Mean±standard deviation	

Among the studied population, R1 was abnormal in 17 (30.90%), R2i in two patients (3.63%), and R2c in 2 (3.63%) patients. The blinking reflex indices significantly differed by the comparison of the abnormal and normal eye reflexes. Based on Table 2, the mean duration of abnormal R1, R2i, and R2c in the left eyes were 15.36±2.04, 45.59±2.14, and 46.84±1.43, while for the right eyes were 15.88±1.98 and 43.81±0, respectively.

Table 2: The comparison of normal and abnormal blink reflex indices among patients with Guillain Barre Syndrome

	Left side blink reflex			**Right-side blink reflex**		
	R1	**R2i**	**R2c**	**R1**	**R2i**	**R2c**
Abnormal	15.36±2.04	45.59±2.14	46.84±1.43	15.88±1.98	43.81±0	-
Normal	10.18±1.45	30.98±4.65	31.07±5.58	10.37±1.49	30.40±4.80	31.07±5.62
P-value	<0.001	<0.001	<0.001	0.007	<0.001	-

Among the studied patients, five (8.47%) had abnormal latency in R1, five (8.47%) in R2i, and five (8.47%) in R2c. The mean latency of R1 was calculated as 4.02±2.44, R2i as 17.15±1.16, and R2c as 14.76±1.46. The comparison of the normal with abnormal latencies revealed significant differences (P-value<0.001) shown in Table 3.

Table 3: The assessment of latency in blink reflex among the patients with Guillain Barre Syndrome

Variable	**Normal**	**Abnormal**	***P*-value**
R1	0.34±0.30	4.02±2.44	<0.001
R2i	1.16±1.27	17.15±1.16	<0.001
R2c	1.39±1.34	14.76±1.46	<0.001

Table 4 shows the incidence rate of abnormal blink reflex among the studied patients based on the type of GBS. Based on this table, the highest rate of abnormality in blink reflex was found among patients with a demyelinated subtype of GBS followed by an axonal subtype; however, there was no statistical difference among the different variants (P-value>0.05).

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Table 4: The distribution of blink reflex status based on the variants of GBS

Side	Variables		Demyelinated N=24	Axonal N=27	Miller-Fisher variant N=4	*P*-value*
			Number (Frequency)	Number (Frequency)	Number (Frequency)	
Left side	R1	Normal	13 (54.2)	23 (85.2)	4 (100)	0.038
		Abnormal	11 (45.8)	4 (14.8)	0 (0)	
	R2i	Normal	23 (95.8)	26 (96.3)	4 (100)	0.99
		Abnormal	1 (4.2)	1 (3.7)	0 (0)	
	R2c	Normal	23 (95.8)	26 (96.3)	4 (100)	0.99
		Abnormal	1 (4.2)	1 (3.7)	0 (0)	
Right	R1	Normal	17 (70.8)	24 (88.9)	4 (100)	0.22
		Abnormal	7 (29.2)	3 (11.1)	0 (0)	
	R2i	Normal	23 (95.8)	27 (100)	4	0.50
		Abnormal	1 (4.2)	0 (0)	0 (0)	
	R2c	Normal	24 (100)	27 (100)	4 (100)	-
		Abnormal	0 (0)	0 (0)	0 (0)	
Right and left eye differences	R1	Normal	15 (62.5)	22 (81.5)	4 (100)	0.22
		Abnormal	9 (37.5)	5 (18.5)	0 (0)	
	R2i	Normal	23 (95.8)	24 (88.9)	4 (100)	0.69
		Abnormal	1 (4.2)	3 (11.1)	0 (0)	
	R2c	Normal	23 (95.8)	24 (88.9)	4 (100)	0.69
		Abnormal	1 (4.2)	3 (11.1)	0 (0)	

Among patients, 6 patients (10.9%) had an abnormal examination of the trigeminal nerve and 20 patients had an abnormal examination of the facial nerve (36.4%). As can be seen in Table 5, there was no significant relationship between blink reflex disorder and abnormal examination of nerves 5 and 7 (P <0.05).

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Table 5: Evaluation of the relationship between blink reflex and abnormality in the trigeminal and facial cranial nerves in Guillain-Barre patients

Variable name (Percentage) Number		Trigeminal nerve		*P*-Value	Facial nerve		*P*-Value
		Normal Number = 49	Abnormal Number = 6		Normal Number = 35	Abnormal Number = 20	
R1	Normal	37 (77.1)	3 (50.0)	0.17	25 (73.5)	15 (75.0)	0.99
	Abnormal	11 (22.9)	3 (50.0)		9 (26.5)	5 (25.0)	
R2i	Normal	46 (93.9)	5 (83.3)	0.37	33 (94.3)	18 (90.0)	0.61
	Abnormal	3 (6.1)	1 (16.7)		2 (5.7)	2 (10.0)	
R2c	Normal	45 (93.8)	5 (83.3)	0.38	32 (94.1)	18 (90.0)	0.62
	Abnormal	3 (6.3)	2 (16.7)		2 (5.9))	2 (10.0)	

DISCUSSION

The questions in terms of relying on blink reflex status to make the definite diagnosis of GBS remained unknown. To the best of our knowledge, we are one of the first groups of researchers assessing the values of blink reflex evaluations in the early stages in a large population of patients with the definitive diagnosis of GBS. By the assessment of the blink reflex in GBS patients, we found an abnormality in R1 as the determinant of facial nerve response among 16-25% of our patients. The involvement of the left side was more prominent than the right side. The other entities of the blink reflex, including the afferent pathway by the trigeminal nerve (R2i) or the efferent pathway by the facial nerve (R2c), were abnormal to less extent of 4%. Evaluation of latencies revealed an abnormality in 8.47% of the GBS patients. In addition, the comparison of normal R1, R2i, and R2c with their abnormal levels among our patients revealed remarkable differences, a fact that shows a steep increase in blink reflex indices among those GBS patients with involved Trigeminofacial reflexes.

Of strength point of the current study was to assess and compare blink reflex status among different subtypes of GBS, which revealed demyelinated and axonal subtypes as the most common ones, a finding that is in line with the studies in the literature [17]. Further evaluation revealed that the highest rate of abnormal blink reflex was among the patients with a demyelinated subtype of GBS, followed by an axonal subtype; however, the comparison of different GBS variants showed insignificant differences. Small groups of studied populations in the other subtype than axonal and demyelinating may be responsible for these insignificant outcomes.

Chan et al. proposed a study in which they evaluated the blink reflex in five patients with GBS, repeatedly. The initial neurophysiological study of the patients revealed normal blink reflexes, as well as the Neuroconductive studies. Further evaluations within the next seven days revealed latencies in the blink reflex of four patients out of the five, among which three GBS patients represented latencies in the demyelinating range. Similar to our study, the highest rate of GBS subtypes belonged to demyelinating one. Surprisingly, two of the patients had unilateral facial palsy due to GBS, and the other two represented bifacial palsy [21].

Sharma conducted another study in which five patients clinically diagnosed with GBS underwent an electrophysiological study within a week after the acute phase of neurological presentations. Despite the controversy in the neuroconductive findings among the studied patients, latencies in R1, R2i, and R2c were detected in all patients. Similar to our findings, right or left-sided involvements were not presented equally [28].

A characteristic that absorbs the scientists' attention to blink reflex rather than NCV for the early detection of GBS, on hand is its ability to concurrently assess both trigeminal and facial nerves, the major sensory and motor roots of cranial nerves innervating the face, and on the other hand, to evaluate the proximal part of the facial nerve. At the same time, NCV can only test the facial nerve distal segments to the stylomastoid foramen which innervates the nasalis. The afferent fibers of the blink reflex are derived from the trigeminal nerve and the efferent ones from the facial nerve. The latency in the R1 response represents short latency as the indicator of

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involvement in a pontine oligosynaptic circuit. R2 latency consists of ipsilateral and contralateral side responses as the determinants of long-latency responses due to the involvement in spinal polysynaptic circuits [29]. Mentioned factors are the mainstay features of blink reflex for GBS diagnosis as the other pathological conditions may involve different parts of the facial nerve. For instance, the infratemporal segment of the facial nerve, the bony part, is the most vulnerable part of this nerve to pressure ischemia. Electrophysiological studies have shown that Bell's palsy mostly involves this part of the nerve, but the proximal segments, while abnormality in multiple segments, may be detected due to GBS [30]. Due to the limited knowledge about blink reflex in GBS, further studies are strongly required.

CONCLUSION

Based on the findings of the current study, at least one of the blink reflex indices was abnormal in up to 30.90% of the patients, it seems that the blinking reflex cannot be considered a reliable early diagnostic test for GBS, but this fact cannot completely rule out this method and additional studies should be done with more patients in other populations.

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