The Egyptian Journal of Hospital Medicine (January 2023) Vol. 90 (2), Page 3159-3161

Studying Effect of Therapeutic CSF Tapping on Outcome of Patients with Idiopathic Intracranial Hypertension through Blink Reflex

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ABSTRACT

Background: About 39-59% of individuals with idiopathic intracranial hypertension (IIH) have cranial nerve impairments of some kind, including single or multiple cranial nerve (CN) palsies. **Objective:** The aim of the current study was to assess clinical outcome after cerebrospinal fluid (CSF) tapping through blink reflex (BR).

Patients and methods: A comparative study was carried out on 40 female patients with IIH, aged between 18 and 60 years, and 40 age and sex matched normal volunteers as a control group, between 2019 and 2021. Participants were subjected to blink reflex before and after therapeutic CSF tapping at Neuro-Diagnostic & Research Center (NDRC), Beni-Suef University Hospital. **Results:** Patients with IIH after CSF tapping showed significant decrease of latencies of BR {the ipsilateral R2 (R2i) from 31.6 (2.8) to 30.6 (2.1) ms (P=0.019) and contralateral R2 (R2c) from 35.2 (3.3) to 33.8 (2.5) ms (P=0.023). After CSF tapping, when compared patients to controls, the latencies of R2i 30.6 (2.1) to 29 (2.6) ms (P=0.005) and R2c 33.8 (2.5) to 32.2 (2.8) ms (P=0.007).

Conclusion: The therapeutic CSF tapping improves latencies of R2i and R2c of BR and clinical outcome IIH.

Keywords: Idiopathic Intracranial Hypertension, CSF tapping, Blink reflex, Comparative study, Beni-Suef University.

INTRODUCTION

In the absence of tumors or venous drain obstructions, patients with idiopathic intracranial hypertension (IIH) have high intracranial pressure (ICP), or cerebrospinal fluid (CSF) abnormalities. For the clinical management and follow-up of patients with IIH, the ability for non-invasively monitoring changes in ICP over time could potentially reduce the need for invasive diagnostic lumbar drainages (LD) [(1)].

There have also been reports of different motility problems in addition to sixth nerve palsies. Some of them are the result of incorrect assumptions that were made in reaction to the little vertical ocular motor imbalance that is known to be present in sixth nerve palsies. Bell's palsies of the cranial nerve (CN) VII are infrequent and frequently temporary. CNs II, VI, and VII that almost create a 90° bend appear to be vulnerable to damage at the point of the bend, which is a recurring theme in these studies. Patients experiencing ocular movement difficulties other than sixth nerve palsies should be cautious when considering the diagnosis of IIH [(2)].

When assessing the trigeminal (sensory afferent), face (motor efferent), and linked interneurons at the level of the pons and the lateral medulla, blink reflex (BR), an electrodiagnostic neural reaction similar to the corneal reflex, is used [(3)]. The aim of the current study was to assess clinical outcome after CSF tapping through BR.

PATIENTS AND METHODS

A comparative study was conducted Beni-Suef University Hospital's Neurology Outpatient Clinics between 2019 and 2021. A total of 40 female patients with IIH, aged between 18 and 60 years, together with 40 age and sex matched normal volunteers as a control group, were recruited for the present study.

According to the modified Dandy criteria from 1985, with the exception of abducent (sixth) nerve palsy, IIH is characterized by symptoms of increased ICP (headache, nausea, vomiting, temporary visual obscurations, or papilledema), lumbar puncture (LP) opening pressure of more than 200 mmH2O in non-obese patients, and likely greater than 250 mmH2O in obese patients, and normal biochemical and cytological composition of CSF. Patients with recent or ancient CN nerve palsies, lesions in the brainstem, cerebrovascular disease, and systemic illnesses causing cranial neuropathy, such as diabetes mellitus, hepatic or renal failure, or toxic exposures were excluded from the study.

All IIH patients underwent a general and neurological examination, a CNs examination, and a history taking procedure regarding the severity of the disease, the use of medications, and the presence of symptoms of increased ICP, such as headaches, vomiting, tinnitus, and temporary visual obscurations. routine laboratory testing, such as tests for the liver function, kidney, INR, and full blood count; Fundus For reducing ICP, papilledema severity was examined and assessed, and therapeutic CSF draining was performed with an examination of its biochemical components and opening pressure.

Using Nihon Kohden EMG/EP ® apparatus, neurophysiological tests were conducted at the Neuro-Diagnostic &Research Center (NDRC), Beni-Suef University Hospital.

Received: 22/09/2022
Accepted: 25/11/2022

Blink reflex study was carried out through supraorbital nerve stimulation on each side separately with percutaneous electrical stimulation placed in the superior orbital fissure with interstimulus interval of 10 seconds to avoid habituation and recording by surface electrodes from the inferior orbicularis–oculi muscles bilaterally. The R1 response was marked ipsilaterally to the side being stimulated and the R2 response was marked bilaterally; R2 ipsilateral (R2i), and R2 contralateral (R2c). The absolute latencies of these responses were measured. We took the average of two responses over each side.

Conditions: A sweep speed of 5 or 10 msec per division was used. The high cut filter was set at 10 Hz, the low cut filter at 5 KHz, and the notch filter was set at 50 Hz. The initial sensitivity was 500 microvolts per division. The R2 threshold was established as the lowest stimulus intensity that elicited R2 responses with at least 50 V of amplitude. The stimulus intensity used in the investigation was set at two to three times the R2 threshold.

Ethical Approval:

This study was ethically approved by the Institutional Review Board of the Faculty of Medicine, Beni-Suef University. Written informed consent was obtained from all participants. This study was executed according to the code of ethics of the World Medical Association (Declaration of Helsinki) for studies on humans.

Statistical Analysis

The collected data were introduced and statistically analyzed by utilizing the Statistical Package for Social Sciences (SPSS) version 25 for windows. Qualitative data were defined as numbers and percentages. Chi-Square test and Fisher's exact test were used for comparison between categorical variables as appropriate. Quantitative data were tested for normality by Kolmogorov-Smirnov test. Normal distribution of variables was described as mean and standard deviation (SD), and independent sample t-test was used for comparison between groups. Pearson's and Spearman's correlation coefficients were employed to check for statistical significance in the observed discrepancies. P value ≤0.05 was considered to be statistically significant.

RESULTS

The demographic data of patients and controls are illustrated in **Tables 1**.

Table 1**: Age and body mass index of the studied groups.**

Items	Patients (mean ± SD)	Controls (mean ± SD)	P-value
Age	35.4 ± 10.5	33.8 ± 8.9	0.508
BMI	26.7 ± 1.5488	26 ± 2.2	0.237

BR data of patients were compared before the LP and 2 after that. There were significant decreases of the R2i and R2c latencies (**Table 2)**. Also, when comparing data of patients after LP and controls, there was still increase of latencies of R and R2c **(Table 3)**.

Table 2: Effect of lumbar puncture on the Blink reflex parameters.

Blink reflex Parameters		Mean ± SD	P-value
R1 Latency (milliseconds)	**Before**	10.4 ± 1	0.397
	After	10.3 ± 0.7	
R2i Latency (milliseconds)	**Before**	31.6 ± 2.8	0.019
	After	30.6 ± 2.1	
R2c Latency (milliseconds)	**Before**	35.2 ± 3.3	0.023
	After	33.8 ± 2.5	

Table 3: Comparison between patients (after the intervention) and normal healthy controls regarding Blink reflex parameters.

Blink reflex Parameters	Patients	Controls	P-value
R1 Latency (milliseconds)	10.3 ± 0.7	10.5 ± 0.6	0.249
R2i Latency (milliseconds)	30.6 ± 2.1	29 ± 2.6	0.005
R2c Latency (milliseconds)	33.8 ± 2.5	32.2 ± 2.8	0.007

Table 4 shows significant positive correlation between patients' age and latencies of R1 (P=0.047) and R2i (P=0.095); positive correlation between R2c latencies and BMI (P=0.092); negative correlation between R1 latency and duration of illness (P=0.039).

Table 4: Correlation between blink reflex parameters and age, BMI and duration of illness of patients with IIH.

Variable		Age	BMI	Duration of illness
Blink reflex parameters				
R1 Latency (milliseconds)	**R**	0.316	-0.091	-0.328
	P-value	0.047	0.577	0.039
R2i Latency (milliseconds)	**R**	0.268	0.262	0.160
	P-value	0.095	0.102	0.323
R2c Latency (milliseconds)	**R**	0.138	0.270	0.184
	P-value	0.396	0.092	0.256

Table 5 shows no significant linear correlation between the opening pressure and the studied parameters before tapping (P>0.05)**.**

Table 5: Correlation between the opening pressure and the percent of change of Blink reflex parameters before tapping.

Percentage of change		Opening pressure
Blink reflex parameters		
R1 Latency (milliseconds)	**R**	-0.205
	P-value	0.211
R2i Latency (milliseconds)	**R**	-0.058
	P-value	0.727
R2c Latency (milliseconds)	**R**	-0.003
	P-value	0.987

Table 6 shows significant negative correlation between the opening pressure of CSF and R1 latency of blink reflex (P=0.001) after tapping.

Table 6: Correlation between the opening pressure and the parameters under study after tapping.

Percentage of change		Opening pressure
Blink reflex parameters		
R1 Latency (milliseconds)	**R**	-0.519
	P-value	0.001
R2i Latency (milliseconds)	**R**	-0.151
	P-value	0.353
R2c Latency (milliseconds)	**R**	-0.119
	P-value	0.466

DISCUSSION

Patients with IIH show raised intracranial strain without even a trace of growths, venous channel blockages, or CSF irregularities. It could be feasible to lessen the requirement for obtrusive symptomatic lumbar wastes for the clinical consideration and follow-up of patients with IIH by having the option to painlessly screen changes in ICP over the long run [(1)].

Our outcomes showed tremendous impact off lumbar cut exhaustive critical diminishing in latencies of R2i from 31.6 ms (±2.8) to 30.6 ms (±2.1) with significant difference (P-value=0.019) and R2c from 35.2 ms (±3.3) to 33.8 ms (±2.5) with significant difference (P-value=0.023), yet at the same time above mean upsides of sound controls {R2i 29 ms (±2.6) with (P-value=0.005) and R2c 32.2 ms (±2.8) with (P-value=0.007)}.

Other creators' discoveries were as opposed to our own, and they found that the ipsilateral and contralateral R2 latencies were a lot of lower in cases than in controls. This disparity could be made sense of by the more modest number of controls and cases utilized in their investigation [(4)].

In our study, the ipsilateral and contralateral R2 was higher in cases than controls might be because of presence of unpretentious facial nerve, its core and additionally cerebrum stem warmth. These outcomes were upheld by case reports, albeit few yet affirmed, which found one-sided or respective facial nerve paralysis related with IIH patients [(5)]. Speculations connecting IIH with facial nerve paralysis incorporate the likelihood that the facial nerve might be packed inside the fallopian channel because of the raised intracranial tension [(6)]. Various investigations have characterized it as a deceptive restricting sign that may be brought about by raised ICP [(7)]. It is believed that the extra strain pulls on the extra-hub facial nerve [(8)].

Different investigations made sense of this relationship with provocative and hereditary variables which are related straightforwardly or by implication with expanded ICP. These components could modify the brainstem pathways that BR can investigate in people with IIH [(9)]. However, different examinations showed no association between blink reflex attributes and CSF opening tension [(10, 11)].

CONCLUSION

The therapeutic CSF tapping improves latencies of R2i and R2c of BR and clinical outcome IIH.

DECLARATIONS

- **Consent for publication:** I attest that all authors have agreed to submit the work.
- **Availability of data and material:** Available
- **Competing interests:** None
- **Funding:** No fund
- **Conflicts of interest:** no conflicts of interest.

REFERENCES

1. **Jerin C, Wakili R, Kalla R *et al*. (2015):** The Effect of Increasing Intracranial Pressure on Ocular Vestibular-Evoked Myogenic Potential Frequency Tuning. Ear and Hearing, 36(6):336-41. doi: 10.1097/AUD.0000000000000190
2. **Rowe F (2011):** Assessment of visual function in idiopathic intracranial hypertension. Br J Neurosurg., 25(1):45-54. doi: 10.3109/02688697.2010.544783.
3. **Kimura J (2006):** Electrodiagnosis of the cranial nerves. Acta Neurologica Taiwanica, 15(1):2-12.
4. **Samanci B, Altıokka-Uzun G, Kocasoy E *et al*. (2018):** Trigeminal Hyperexcitability in Idiopathic Intracranial Hypertension. A Blink Reflex Study. Journal of Clinical Neurophysiology, 35(5):408-14. doi: 10.1097/WNP.0000000000000497
5. **Samara A, Ghazaleh D, Berry B *et al*. (2019):** Idiopathic intracranial hypertension presenting with isolated unilateral facial nerve palsy. A case report. Journal of Medical Case Reports, 13(1):94. doi: 10.1186/s13256-019-2060-5
6. **Brackmann D, Doherty J (2007):** Facial palsy and fallopian canal expansion associated with idiopathic intracranial hypertension. Otology & Neurotology, 28(5):715-8. doi:10.1097/01.mao.0000281801.51821.27
7. **Kearsey C, FernandoP, Benamer H *et al*. (2010):** Seventh nerve palsy as a false localizing sign in benign intracranial hypertension. Journal of the Royal Society of Medicine, 103(10):412-4. doi: 10.1258/jrsm.2010.100049
8. **Tzoufi M, Makis A, Grammeniatis V *et al*. (2010):** Idiopathic intracranial hypertension and facial palsy: case report and review of the literature. Journal of Child Neurology, 25(12):1529-34. doi: 10.1177/0883073810375849
9. **Samancı B, Samancı Y, Tüzün E *et al*. (2017):** Evidence for potential involvement of pro-inflammatory adipokines in the pathogenesis of idiopathic intracranial hypertension. Cephalalgia: An International Journal of Headache, 37(6):525-31. doi: 10.1177/0333102416650705
10. **Baheti N, Nair M, Thomas S (2011):** Long-term visual outcome in idiopathic intracranial hypertension. Annals of Indian Academy of Neurology, 14(1):19-22. doi: 10.4103/0972-2327.78044
11. **Kesler A, Hadayer A, Goldhammer Y *et al*. (2004):** Idiopathic intracranial hypertension. Risk of recurrences. Neurology, 63(9):1737-9. doi: 10.1212/01.wnl.0000143067.40281.16