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Original Article

Increased intracranial pressure in migraine? Neuroimaging study on a cohort of migraineurs

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Abstract

Aim: To investigate the frequency of papilledema and radiological signs of increased intracranial pressure (ICP) in our cohort of migraineurs, and investigate the possible clinical predictors.

Material and Methods: This was a cross-sectional study in which we have included all the patients with migraine who applied to our neurology clinic during January 2020 and accepted to involve in the study. The demographic and clinical characteristics including migraine subtype (episodic/chronic), headache frequency per month, headache characteristics were evaluated in all the study group. Besides, the presence of chronic fatigue syndrome (CFS) was noted. The fundus examination was performed in all the patients. Previously defined eight findings of increased ICP were evaluated by our radiologist. We have classified the migraineurs into two groups as migraineurs with clinical suspicion of increased ICP and those without. We have identified the patients with papilledema and/or positivity of at least one of the neuroimaging signs of increased ICP as migraineurs with suspicion of increased ICP. The remaining patients were defined as migraineurs without suspicion of increased ICP.

Results: Ultimately, 63 migraineurs were included in this study. The median age was 36.8 ± 11.3 and the F/M ratio was 51/12. Papilledema was determined in 11 (17.4%) of the patients and 19 (30.1%) of the migraineurs had at least one of the neuroimaging signs of increased ICP. Twenty-six (41.3%) of the overall group were evaluated as migraineurs with suspicion of increased ICP and 37 of the patients were those without suspicion of increased ICP. The comparisons between these two patient groups revealed that obesity was more prevalent in the migraineurs with suspicion of increased ICP. Logistic regression analyses revealed obesity (OR: 0.090, P = 0.014) and radicular pain (OR: 7,647, P = 0.030) as clinical predictor factors for the patients with suspicion of increased ICP.

Conclusion: We determined a high rate of patients with either a positive neuroimaging sign of increased ICP and/or papilledema in our cohort of migraineurs. Our results may suggest obesity and radicular as potential risk factors in the prediction of increased ICP in migraineurs. The results of this study need to be confirmed in future prospective-studies with the evaluations of the lumbar puncture opening pressures in the patient subgroup with required indication.

Keywords: Increased intracranial pressure; migraine; MRI; papilledema; pathophysiology

INTRODUCTION

Migraine is a common primary, headache disorder characterized by recurrent episodes of headache often associated with nausea, vomiting, photophobia, and phonophobia. It is the third most common disease in the world with a prevalence between 2.6% and 21.7%, which leads to a substantial clinical and economic burden (1). Chronic migraine (CM) is a subtype of migraine which results from the progressive worsening of attacks frequency, up to a daily or near-daily pain, that may develop, more or less progressively, in a part of episodic migraine (EM) sufferers (2). Recent researches have remarked to the coexistence of increased intracranial pressure (ICP) as a crucial mechanism that might contribute to the pathophysiology of progression of migraine a transformation from EM to CM (3-6). However, there are also other authors supporting that these co-occurrences are actually rare and exaggerated (7). Taken together, the association between increased ICP and migraine still needs to be clarified in many aspects (8). However, the investigation of migraineurs routinely by spinal tap test is technically difficult and may constitute ethical problems for researches as there is no medical indication for this intervention in any type of migraines. In this context, we aimed to indirectly investigate the prevalence of increased ICP systematically in migraineurs by performing fundus examinations and evaluating the signs of increased ICP on magnetic resonance imaging. We also aimed to

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investigate the significance and predictive value of some clinical features in differentiating the migraineurs with possible coexistence with increased ICP.

MATERIAL and METHODS

We have initially included all the patients with migraine who applied to the neurology clinic of Yozgat City Hospital during January 2020 and accepted to involve in this study. The diagnosis of migraine in our study group was based on the four diagnostic criteria of the International Classification of Headache Disorders 3rd edition (9). The demographic and clinical characteristics including migraine subtype (episodic/chronic), headache frequency per month, headache characteristics were evaluated in all the patients. Besides, based on the previous hypotheses linking the pathophysiological mechanisms of migraine and chronic fatigue syndrome (CFS), the presence of CFS was also noted. Possible causes of secondary intracranial hypertension including hypertension and obesity were evaluated, body mass indexes (BMI) were calculated for all the migraineurs. Patients with refractory hypertension, chronic sinusitis, and obstructive sleep apnea were excluded from the study. All patients with migraine were asked to perform the self-reported questionnaire of headache impact test for the assessment of headache severity. In addition, the presence of symptoms including cognitive impairment, radicular pain those have been associated with increased intracranial pressure in previous reports were noted (10). Fundus examination was performed in all the patients and those with papilledema were noted. In some of the patients with papilledema and/or encouraging evidence of the neuroimaging signs of increased ICP, LP was suggested. However, our study was not in a prospective design, and data regarding the CSF opening pressure was not available in those patients undergoing LP. On the other hand, we have classified the migraineurs into two groups as migraineurs with clinical suspicion of increased ICP and those without. We have identified the patients with papilledema and/or positivity of at least one of the neuroimaging signs of increased ICP as 'migraineurs with suspicion of increased ICP'. The remaining patients without papilledema and a neuroimaging sign of increased ICP were defined as 'migraineurs without suspicion of increased ICP'.

Headache Impact Test

The Headache Impact Test (HIT-6) (11) is a validated tool to assess the impact of headache (range: 36–78) on the lives of respondents. HIT-6 scores ≤48 reflect little impact; scores between 56 and 59 reflect substantial impact, and scores ≥60 reflect severe impact.

Neuroimaging

Cranial MRI was performed in a single session in all the patient groups using a 1.5 Tesla Magnetom Amira MRI scanner using (5-mm slice thickness, Siemens). The images were assessed by a radiology specialist (GG) who is particularly interested in neuroimaging. Conventional cranial MRI studies were specifically investigated for the presence of optic nerve head protrusion, posterior scleral flattening, increased peri-optic cerebrospinal fluid (CSF), optic nerve tortuosity, partial-empty sella, tonsillar herniation, enlargement of Meckel cave, meningoceles (*). A total radiological score (TRS) was calculated for all the patients by adding up the scores of all the above-mentioned parameters. The presence of the findings of increased ICP was evaluated according to the criteria, defined in the previous reports (12-15). This study was conducted in accordance with the Declaration of Helsinki.

Statistical Analyses

All statistical analyses were performed using the SPSS statistics 20 programs. Continuous variables are expressed as mean ± standard deviation. The compliance of the variables with normal distribution was assessed by the Kolmogorov-Smirnov test. Inter-group analyses were performed with Student's t-test for normally distributed variables and the Mann-Whitney U test for non-parametric variables. The chi-square test was used for the comparison of qualitative data. Binay logistic regression analyses were conducted to evaluate the utility of some clinical variables in the prediction of migraineurs with suspicion of increased ICP. A p-value of <0.05 was considered to be statistically significant.

Table 1. The demographic and clinical characteristics of the overall group

	Overall migraineurs (n=63)
Age [Mean ± SD)]	36.8 ± 11.3
Gender (F/M)	51/12
BMI (mean ± SD)	28.6 ± 5.5
Obesity (n, %)	24 (38.0%)
Papilledema (n, %)	11 (17.4%)
Positivity of at least one of the neuroimaging signs of increased ICP	19 (30.1%)
Episodic/Chronic	48 (76.2%) /15 (23.8%)
Hypertension (n, %)	4 (6.3%)
HIT-6 [Median (IQR)]	22(19-26)
Days with headache per month [Median (IQR)]	10 (4-12)

RESULTS

Ultimately, 63 migraineurs were included in this study. The median age was 36.8 ± 11.3 and the F/M ratio was 51/12. Papilledema was determined in 11 (17.4%) of the patients and 19 (30.1%) of the migraineurs had at least one of the neuroimaging signs of increased ICP (Table 1). Detailed investigation of the neuroimaging signs of increased ICP reveaed that the most common finding was optic nerve tortuosity (n=11, 17%). Nine of the patients had at least two of the neuroimaging signs of ICP. One patient received 4 points on TRS (Figure 1). Among these patients, episodic migraineurs were more common and papilledema was present in only two of them (Table 2). However, arrestingly, eight of the patients were evaluated as obese (Table 3). Some of the neuroimaging parameters of increased ICP are demonstrated in Figure 2.

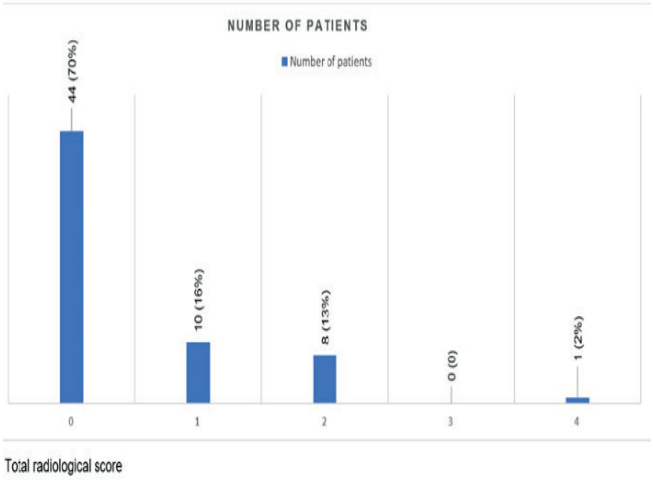

Figure 1. The figure showing the distribution of the total radiological scores of the patients

Table 2. The table showing the results of the comparative analyses of of the presence of a neuroimaging sign of increased ICP and the total radiological score between migraineurs with papilledema and those without papilledema

Total number of patients, n=63		Papilledema, n=11 (17.4%)		P-value
		Present	Absent	
Positivity of at least one of the neuroimaging signs of increased ICP, n=19 (30.1%)	Present (n, %)	4 (21)	15 (79)	0.721
	Absent (n, %)	7 (16)	37 (84)	
Total radiological score [median (range)]		0 (2)	0 (4)	0.630

Table 3. The clinical features of the subgroup of migraineurs with a total radiological score of 2 or more (n=9)

Case number	Age	Gender	BMI	E/C	HIT-6	Papilledema	TRS	Positive MRI findings
1	42	K	33.4 (O)	E	27	-	2	Increased perioptic CSF, optic nerve tortuosity
2	36	K	23.3	E	22	-	4	Increased perioptic CSF, optic nerve tortuosity, Partial emp. Sella, Enlargement of Meckel c
3	31	K	36.7 (O)	E	25	+	2	Partial emp. Sella, Enlargement of Meckel c
4	59	K	33.8 (O)		14	-	2	Increased perioptic CSF, Partial empty s.
5	32	K	33.3 (O)	E	19	+	2	Optic nerve tortuosity, Posterior scleral flat.
6	53	K	34.9 (O)	E	21	-	2	Optic nerve tort, Partial empty s.
7	46	K	33.2 (O)	E	28	-	2	Increased perioptic CSF, Partial empty sella
8	30	K	35.3 (O)	C	28	-	2	Increased perioptic CSF, Optic nerve tortuosity
9	47	K	31.2 (O)	C	23	-	2	Partial empty sella, Enlargement of Meckel c

E/C:Episodic/Chronic, BMI:Body Mass Index, HIT-6:Headache Impact Test, TRS: Total Radiological Score

Table 4. The comparisons of the demographic data, clinical features and radiological parameters between the migraineurs with suspicion of increased ICP and those without

	Migraineurs with suspicion increased ICP (n=26, 41.3%)	Migraineurs without suspicion of ICP (n=37, 58.9%)	P-value
Age [Median (Range)]	36.8 ± 10.0	36.7 ± 12.2	0.973
Gender (F/M)	23/3	28/9	0.329
Episodic/Chronic	20/28	6/9	1.00
BMI (median ± SD)	30.7 ± 5.7	27.1 ± 4.9	0.009*
Obesity (n, %)	15 (57.6)	9 (24.3)	0.015*
Hypertension (n, %)	1 (3.8)	3 (8.1)	0.637
HIT-6 [Median (Range)]	22.7 ± 4.7	21.4 ± 3.7	0.200
Days with headache per month [Median (Range)]	9.1 ± 6.0	9.8 ± 5.9	0.555
Wake-up headache (n, %)	15 (57.6)	14 (37.8)	0.194
Exacerbation of headache during lying (n, %)	7 (26.9)	13 (35.1)	0.679
Blurred vision (n, %)	16 (61.5)	22 (59.4)	1.00
Tinnitus (n, %)	12 (46.1)	23 (62.1)	0.317
Chronic fatigue syndrome (n, %)	23 (88.4)	35 (94.5)	0.641
Chronic fatigue syndrome score (n, %)	29.9 ± 10.0	31.4 ± 9.1	0.551
Cognitive symptoms (n, %)	18 (69.2)	27 (72.9)	0.968
Radicular pain (n, %)	10 (38.4)	19 (51.3)	0.451

ICP: Intracranial Pressure, HIT-6: Head Ache Impact Test-6

Table 5. The results of the binary Logistic Regression Analyses: Predicting the patients with suspicion of increased ICP

Independent Variables	B	S.E.	Wald	Sig.	Exp(B)	95% CI.for EXP(B)	
						Lower	Upper
Age	-.015	.036	.189	.664	.985	.918	1.056
Gender	1.195	.908	1.730	.188	3.303	.557	19.596
Hypertension	.995	1.651	.363	.547	2.705	.106	68.723
Obesity	-2.404	.934	6.619	.010*	.090	.014	.564
Episodic/chronic	-.527	.826	.408	.523	.590	.117	2.978
Radicular pain	2.034	.937	4.718	.030*	7.647	1.220	47.938
Wake-up headache	-.822	.646	1.618	.203	.440	.124	1.560
Exacerbation of headache during lying	.428	.713	.359	.549	1.534	.379	6.209
Blurred vision	-1.476	.860	2.946	.086	.229	.042	1.233
Tinnitus	1.265	.740	2.920	.088	3.544	.830	15.125
Chronic failure syndrome	.894	1.243	.517	.472	2.445	.214	27.940
Constant	-.870	3.076	.080	.777	.419		

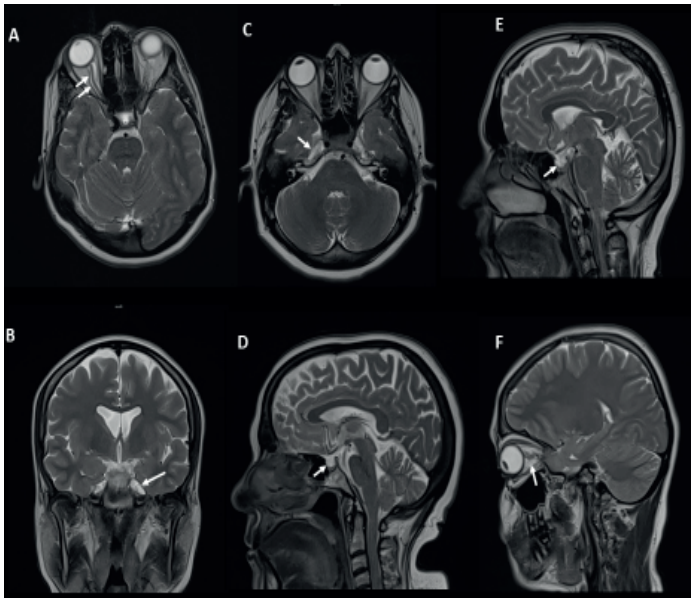

Figure 2. A, Increased peri-optic CSF (Case 1). B, The enlargement of Meckel's cave (Case 2). C, The enlargement of Meckel's cave (case 3). D, Partial empty sella (Case 4). E, Partial empty sella (Case 3). F, Optic nerve tortuosity (Case 5) [arrows]

It was determined that 26 (41.3%) of the overall group were migraineurs with suspicion of increased ICP and 37 of the patients (58.9%) were those without suspicion of increased ICP. The comparisons between these two patient groups revealed that obesity was more prevalent in the migraineurs with suspicion of increased ICP. There was no difference in the clinical parameters including the demographic data, migraine severity, migraine subtype (E/C), and some other clinical features between groups (Table 4). Logistic regression analyses revealed obesity (OR: 0.090, P = 0.014) and radicular pain (OR: 7,647, P = 0.030) as possible clinical predictors for the patients with suspicion of increased ICP (Table 5).

DISCUSSION

In this study, we found that 17% of the migraineurs had papilledema and 30% of the patients had at least of the neuroimaging signs of increased intracranial pressure (ICP). Remarkably, a considerably high rate of the patients (41.3%) had papilledema and/or a neuroimaging sign of increased ICP. Idiopathic intracranial hypertension, which is classified under the secondary headaches, can be defined as an entity of elevated intracranial pressure with no distinguishable etiology. IIH, particularly the subtypes without papilledema, and chronic migraine (CM) are often clinically indistinguishable (8). The increasing incidence of IIH by paralleling the global rise of obesity and the increased cooccurrences of IIH and chronic migraine has been emphasized in many reports (8-10). The researchers also remarked that the comorbid IIH is frequently underdiagnosed in the routine clinical practice which may result in the delayed diagnosis and potential for permanent neurological deficit including blindness (6,16,17). In a crucial study, Mathew et al. evaluated 85 patients with a refractory chronic headache with migrainous features by spinal tap and they found elevated CSF pressure in 12 patients (3). Remarkably, none of these patients had papilledema; and adding medications such as acetazolamide and furosemide resulted in better control of symptoms (3). More recently, De Simone et al. evaluated the LP opening pressure in all 44 consecutive patients diagnosed with unresponsive chronic/transformed migraine and evidence of sinus stenosis at magnetic resonance venography (17). They found that 86.4% of the patients had an LP opening pressure of 200 mmH2O or higher. Furthermore, the lumbar puncture-induced normalization of intracranial pressure resulted in prompt remission of pain in 77.3 % of the patients which supported the increased ICP as a crucial etiological agent of pain. Based on these observations, it has been suggested that proven unresponsiveness to medical treatment may predict the presence of a raised ICP in the clinical series of chronic migraine patients (17). However, this issue is controversial in the literature, as there are also authors proposing that this coexistence is actually rare, not clinically significant, and exaggerated. They emphasized acting according to these observations which have not been validated in any large-scale studies, may lead to unnecessary invasive interventions (7,8). Ergo, there still needs to be further efforts to clarify the possible role of increased ICP in the pathophysiology of migraine and propose possible risk factors for these coexistences. In this study, we preferred to evaluate the possible coexistence of increased ICP in migraineurs by performing fundus examination and evaluating the neuroimaging signs of increased ICP on MRI. Papilledema in the absence of a cause of secondary intracranial hypertension on MRI constitutes a crucial diagnostic clue for IIH (18). On the other hand, recent studies have proposed some imaging findings such as vertical tortuosity of the optic nerves, posterior flattening of the globes, optic nerve protrusion, distention of the optic nerve sheath, and inferior tonsillar displacement as supportive findings in the diagnosis of IIH (19-21). Based on the study results remarking the high reliability of these findings in the determination of increased ICP, some authors suggested that these findings may provide sufficient clues for the diagnosis of IIH without the need to measure lumbar puncture opening pressure (19-21). We found that 30% of the patients had at least one of the neuroimaging signs of increased ICP. However, a crucial point may be that a high rate of patients with neuroimaging signs of increased ICP had no papilledema (79%). Such that, 7 of the 9 patients with a TRS of two or more had no papilledema. These results should be interpreted cautiously. In the literature, the diagnostic sensitivity of these neuroimaging signs for IIH has been reported to vary from 46% to 87% differing according to the parameter investigated (19,20,22). However, high specificity values have been reported for all the neuroimaging parameters in several previous studies, which strengthen the value of our study results in this regard (20,21,23). We think that these patients without papilledema, but a positive neuroimaging sign of increased ICP may reflect a subgroup of migraineurs with a possible

coexistence of IIH without papilledema. The disease of IIH without papilledema is a well-known entity in literature and its possible coexistence with migraine has been firstly investigated by Mathew et al. in detail (3). The diagnosis of this entity is strictly challenging and the definitive diagnosis is impossible without spinal tap investigation. On the other hand, a crucial limitation was that we conducted our research in a cross-sectional design and a spinal tap test was not performed in any of the patients to confirm the increased ICP. However, we believe that the results of our study, albeit this limitation, are certainly important and may provide crucial perspectives.

The rate of papilledema was also strictly high (17%) in our cohort. Basically, papilledema occurs when increased pressure from the brain and cerebrospinal fluid is placed on the optic nerve. Idiopathic intracranial hypertension is a crucial cause of papilledema; however, there are also other several conditions such as brain tumor, inflammatory disease of the central nervous system, refractory hypertension, etc. which may lead to papilledema. The strength of our study was that, in all our study group, a conventional cranial MRI was routinely performed, which did not reveal a space-occupying lesion or inflammatory lesion in any of the subjects that might lead to papilledema. Besides, patients with refractory hypertension were not enrolled in this study. Moreover, we have also excluded other conditions including chronic sinusitis and obstructive sleep apnea that may cause papilledema (24,25) which also strengthens the value of our study results.

The results of the comparative analyses between migraineurs with and without suspicion of increased ICP revealed that obesity was more common in migraineurs with suspicion of increased ICP. Binary logistic regression analyses also revealed obesity as a clinical predictor factor for suspicion of increased ICP in our cohort with migraineurs. The incidence of IIH is known to be higher in obese patients, such that, over 90% of patients with IIH are obese or overweight (26-28). Furthermore, weight loss has been established as an effective treatment in IIH also suggesting a strong linkage of IIH with obesity (28). On the other hand, obesity has been acknowledged as a risk factor in chronification of migraine (29), and it has also been shown to a risk factor for comorbid IIH in a few, recent studies on migraine patients (29,30). Our study, which was methodologically distinct from those, revealed coherent results with the previous studies in this context.

Another crucial result was that we found radicular pain as a significant clinical predictor for the suspicion of increased ICP. In a limited number of reports, radiculopathy has been proposed as one of the clinical manifestations of IIH which is propably an under-recognized one. However, those reports consisted of only a few case presentations (10,31-33), and no systematic study addressing the existence of this manifestation in IIH has been conducted up to date. Clinical manifestations reported in these case reports varied from radicular pain to severe motor deficit due to severe radiculopathy (10,31-33). In the crucial report by Obeid et al. (33), two remarkable patients presenting with severe quadriparesis due to intracranial hypertension were reported who had been misdiagnosed as Guillain-Barre syndrome and received intravenous immunoglobulin therapy which had not provided an amelioration. However, after re-evaluation, the diagnosis of intracranial hypertension was made and they responded well to lumboperitoneal shunting. The most likely mechanism is the mechanical compression of nerve roots, due to elevated CSF pressure distending the subarachnoid space which has also been proposed as the mechanism of neuropathies of other cranial nerves in IIH. However, radiculopathy secondary to increased ICP has been reported almost exclusively in patients with IIH (31-34). In light of these reports, we think that the results of our research may provide crucial perspectives to be kept in mind in the clinical practice. We hypothesize that the interrogation of radicular pain in migraineurs with atypical features may provide substantial clues in the clinical practice. We think that it would probably reasonable to investigate the presence of radicular involvement in IIH, and in migraineurs with comorbid IIH in future-prospective studies including electrophysiological examinations.

There may be some limitations of this study. First, the study group was small avoiding to propose certain conclusions. Second, although papilledema is a sensitive and specific sign of increased ICP, and neuroimaging signs those evaluated in this study are shown to be highly specific for increased ICP (20,21,23); the definitive diagnoses of IIH were lack, as a spinal tap was not conducted in this study. We have just formed a classification as patients with suspicion of increased ICP and those without, which has no validation. Therefore, the confirmation of these results in future-prospective studies including patients with a definitive diagnosis of IIH is required in this regard.

CONCLUSION

We determined a high rate of patients with papilledema and/or positive neuroimaging signs of increased ICP in our cohort of migraineurs. In accordance with the few previous studies, obesity was found as a clinical predictor of patients with suspicion of increased ICP, and remarkably, radicular pain was also found as another clinical predictor. We think that the frequent coexistence of these entities, associated clinical clues and possible pathophysiological links between increased ICP and migraine should be investigated in larger patient cohorts in future studies. The results of these studies may provide substantial perspectives regarding the unknown aspects of the pathophysiology of migraine.

Competing interests: The authors declare that they have no competing interest.

Financial Disclosure: There are no financial supports.

Ethical approval: This study was approved by the Institutional Ethics Committee and conducted in compliance with the ethical principles according to the Declaration of Helsinki.

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