Original Article

A Study on Clinical Efficacy of Propranolol, Topiramate and their Combination in Migraine Prophylaxis

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Abstract:

Background: Migraine ranks as one of the most incapacitating disorders. A variety of drugs are recommended for the preventive management of migraines. **Objective:** The objective of this study was to assess the comparative effectiveness of Propranolol, Topiramate and their combination in the prophylaxis of migraines within the Bangladeshi demographic. **Methods:** This randomized control trial with a 2×2 factorial design on 120 diagnosed cases of migraine, divided into four equal intervention groups to receive placebo, Propranolol, Topiramate and combination of Propranolol and Topiramate. The reduction of headache frequency, proportion of ≥50% headache reduction and headache severity were compared among the groups. **Results:** Groups received Propranolol, Topiramate and combination of these two showed significantly greater reduction in all three variables studied than placebo group (p-values <0.001 in all comparisons, 0.02 between placebo and Topiramate-treated groups in proportion of ≥50% reduction in headache frequency). Propranolol-treated and Topiramate-treated groups did not show any significant difference. A greater reduction in frequency was observed in the group treated with both Propranolol and Topiramate (p-value 0.03 and 0.02) and proportion of ≥50% reduction in frequency (p-value 0.03 and 0.01) than Propranolol and Topiramate-treated groups. **Conclusion:** Propranolol and Topiramate, whether administered separately or in conjunction, demonstrate efficacy in the prevention of migraines. The combination of Propranolol and Topiramate is superior to the individual agents in mitigating the reducing headache frequency.

Key words: Migraine prophylaxis, Propranolol, Topiramate

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Introduction:

Migraine is among the most prevalent headache disorders, impacting more than a billion individuals around the world, and ranked as the second leading cause of disability on a global scale.[1,2] The exact pathogenesis of migraine is still not completely understood. The most recent hypothesis proposes that migraine begins with the activation of the hypothalamus by a trigger factor, which then activates the cerebral cortex, leading to a cortical spreading depression. This process subsequently stimulates the trigeminal sensory nucleus, thalamus, and chemoreceptor trigger zone. The activation of the trigeminal nucleus results in the release of neurotransmitters such as serotonin, calcitonin gene-related peptide (CGRP), and pituitary adenylate cyclase-activating peptide (PACAP) from trigeminal nerve endings at the dura mater, leading to neurogenic inflammation and headache. The activation of the thalamus is responsible for photophobia and phonophobia, while the activation of the chemoreceptor trigger zone results in nausea and vomiting.[3,4]

According to the third edition of the International Classification of Headache Disorders (ICHD-3), migraine is defined as a headache lasting between 4 and 72 hours, which must include any two of the following four characteristics: unilateral location, pulsating quality, moderate or severe pain intensity, and aggravation by routine physical activity, along with at least one of the following symptoms: nausea/vomiting, photophobia, or phonophobia.[5]

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Migraine is categorized into two types: migraine without aura and migraine with aura, based on whether an aura is present or absent. Aura is described as brief focal neurological symptoms, frequently visual or sensory, that generally precede, although they can also coincide with, the headache phase of a migraine episode. Chronic migraine is characterized by experiencing 15 or more headache days each month for a duration exceeding three months, along with meeting the ICHD-3 criteria for migraine on at least eight days per month. A headache frequency of fewer than 15 days per month in a migraine patient is referred to as episodic migraine.[5]

Preventive therapy aims to lower the frequency, duration, or severity of migraine attacks. Furthermore, beyond these main objectives, it may also improve the efficacy of acute treatments, boost patients' functional abilities, and lessen overall disability. For the prophylaxis of migraines, the following pharmacological agents are recommended: Propranolol, Metoprolol, Topiramate, Candesartan, Valproate, Topiramate, Amitriptyline, and CGRP monoclonal antibodies.[6]

Propranolol functions by inhibiting the release of noradrenaline and reducing the firing rate in both the locus coeruleus and periaqueductal gray matter, mediated by GABAergic action[7]. The prophylactic effect of Topiramate on migraines is achieved through the enhancement of GABA, the inhibition of glutamate, the blockage of sodium channels, and the inhibition of carbonic anhydrase. These actions collectively reduce excitatory neurotransmitters and the secretion of vasoactive peptides like CGRP [7]. Recommended doses of Propranolol and Topiramate in migraine prophylaxis are 40-160 mg and 50-100 mg daily, respectively[8].

A few comparative studies comparing the efficacy of Propranolol and Topiramate were published. Ashtari et al. found Topiramate better than Propranolol in migraine prophylaxis[9]. A recent study in Andhra Pradesh, India also concluded in favour of Topiramate over Propranolol as migraine prophylactic agent[10]. However, Kumar et al., in a recent study in Bihar, India did not find any significant difference in the efficacy of these two drugs in migraine prophylaxis[11]. Chowdhury et al. revealed similar efficacies of Propranolol and Topiramate in migraine prophylaxis[12]. Tayeb et al. in a study on Bangladeshi migraine patient observed comparable efficacies of Propranolol and Topiramate[13]. Ghahramani et al. found significant improvement of quality of life in migraine patients treated with Propranolol-Topiramate combination in an Iranian study[14]. Pascual et al. demonstrated the efficacy of Propranolol-Topiramate combination in migraine patients not adequately controlled with the single agents[15]. There is a scarcity of publications on the head-to-head comparison of the efficacy of Propranolol-Topiramate combination to the individual agents. The objective of this study was to assess the comparative effectiveness of Propranolol, Topiramate and their combination in the prophylaxis of migraines within the Bangladeshi demographic.

Methods:

This was a randomized control trial (RCT) conducted on 120 migraine patients diagnosed as per ICHD-3 criteria[5] attending the Medicine outpatient of Shaheed Monsur Ali Medical College Hospital from April 2024 to March 2025. Recommendations by the International Headache Society (IHS) on clinical trials on migraine treatment were followed in this study[16]. The participants aged 18-45 years, of both genders, with a history of migraine for at least 1 year and a headache frequency of 2-8 and <15 headache days per month. Migraineurs with co-existing other headache disorders, bronchial asthma, COPD, heart failure, bradycardia, peripheral vascular disease and pregnancy were excluded. Exclusion from the study also applied to those who had utilized migraine prophylactic medication, antidepressants, and antipsychotics within the previous three months, as well as females of reproductive age who were not practicing contraception.

A 2×2 factorial design was employed in this RCT[17]. Participants were randomly allocated into four groups. Group A was administered a placebo. Group B was prescribed a slow-release preparation of Propranolol 40 mg every morning. Group C received Topiramate at a dosage of 25 mg, taken twice a day. Group D was treated with both Propranolol and Topiramate, using the same dosages as in groups B and C. All participants received Rizatriptan 5 mg as required to terminate acute migraine episodes[18].

The frequency and severity of migraine were recorded at baseline and after 4 weeks. Frequency was expressed as the number of migraine attacks occurring in a 4-week interval. Distinct attacks were defined as consecutive acute migraine episodes that were separated by at least 24 hours[16]. Severity was measured subjectively using a verbal rating scale (VRS) that coded 0=no pain, 1=mild pain, 2=moderate pain and 3=severe pain[16,19]. The primary end-points were reduction in frequency and VRS score. proportion of reduction of headache frequency by ≥50% was set as a secondary end-point. The duration of headache was not documented since the IHS guideline does not recognize it as a valid endpoint in migraine prophylaxis studies[16].

The age, reduction in frequency and VRS score had symmetrical distributions, were expressed in terms of mean and standards deviation (SD) and assessed across all groups using ANOVA and compared between pairs of groups with the application of Student's t-test. The baseline and follow-

up headache frequency and VRS exhibited significant skewness in the data were expressed as median and inter-quartile range (IQR) and analyzed across the groups using the Kruskal-Wallis test. Gender and the proportion of reduction of headache frequency by ≥50% were expressed as frequency (n) and percentage (%) and compared employing Chi-square test. A p-value <0.05 was considered statistically significant.

Results:

The participants' ages ranged from 18 to 45 years, with a mean±SD of 32.3±6.2 years, where 77.5% of them were in the 25-40 years age category (Figure-1). Number of patient in each group is 30 without any missing during follow up.

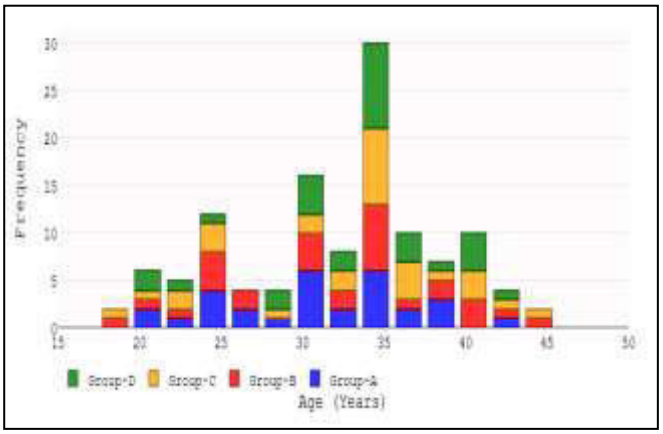

Figure-1: Age distribution of the participants

Majority of the participants were females (97, 80.8%). The age and gender distributions were comparable across the groups (p-value 0.56 and 0.8, respectively) (Table-I). Aura was identified in 26(21.7%) participants. Aura was found in 26 (21.7%) participants and did not differ in among the four groups (p-value 0.62).

Table-I: Age and gender of the participants.

Variables	Group-A (n_1=30)	Group-B (n_2=30)	Group-C (n_3=30)	Group-D (n_4=30)	p-value
Age (Years)[1]	31.0±5.6	32.0±6.8	32.7±6.7	33.2±5.7	0.56[a]
Female gender[2]	24 (80)	26 (86.7)	24 (80.0)	23 (76.7)	0.80[b]
Aura[2]	4 (13.3)	7 (23.3)	7 (23.3)	8 (26.7)	0.62[b]
1-mean±SD, 2-n(%), a-ANOVA, b-Chi-square test					

Table-II: Headache frequency and VRS score at baseline and follow-up among the participants.

Variable	Record time	Group-A (n_1=30)	Group-B (n_2=30)	Group-C (n_3=30)	Group-D (n_4=30)	p-value
Headache frequency /4 weeks	Baseline	8(6-8)	7(4-8)	8(6-8)	8(6-8)	0.76[a]
	Follow-up	6(6-8)	4(2-6)	4(2-6)	2(0-6)	<0.001[a]
VRS score	Baseline	3(2-3)	3(2-3)	3(2-3)	3(2-3)	0.99[a]
	Follow-up	3(2-3)	1(1-2)	1(0-3)	1(0-2)	<0.001[a]
1- median(IQR), a- Kruskal-Wallis test						

Table-II illustrates the headache frequency and VRS score at baseline and follow-up. Headache frequency and VRS score were comparable among the four groups at baseline (p-value 0.76 and 0.99, respectively). The frequency of headaches and the VRS score exhibited significant differences across the four groups at the follow-up (p-value <0.001).

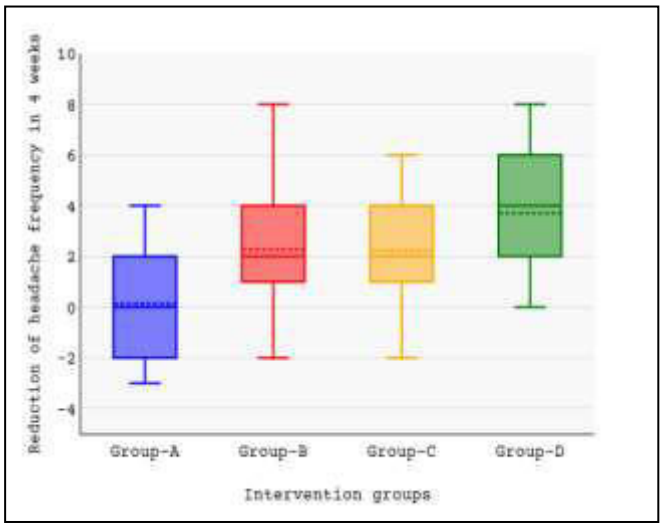

Figure-2: Reduction of headache frequency in the four groups

Figure-2 and Table-III show the reduction of headache frequency among the participants. Mean±SD of reduction of the headache frequency per 4 weeks was 2.1±2.6. The mean±SD reduction of headache frequency was 0.1±1.9, 2.3±2.3, 2.3±2.0 and 3.7±2.5 per 4 weeks in group-A, group-B, group-C and group-D, respectively that significantly differed among the four groups (p-value<0.001).

Table-III: Reduction of headache frequency among the participants

Comparison groups	Reduction in frequency[1]	p-value
All groups[1]	0.1±1.9, 2.3±2.3 2.3±2.0, 3.7±2.5	<0.001[a]
A – B[1]	0.1±1.9, 2.3±2.3	<0.001[b]
A – C[1]	0.1±1.9, 2.3±2.0	<0.001[b]
A – D[1]	0.1±1.9, 3.7±2.5	<0.001[b]
B – C[1]	2.3±2.3, 2.3±2.0	0.95[b]
B – D[1]	2.3±2.3, 3.7±2.5	0.03[b]
C – D[1]	2.3±2.0, 3.7±2.5	0.02[b]
1-Mean±SD, a-ANOVA, b-Student's t-test		

Compared to the other groups, Group-A showed a considerably lesser reduction in the frequency of headaches (p-value <0.001 in all cases). No substantial difference was observed between group-B and group-C (p-value 0.95). Group-D demonstrated a greater decline in the frequency of headaches relative to both group-B and group-C (p-value 0.03 and 0.02, respectively).

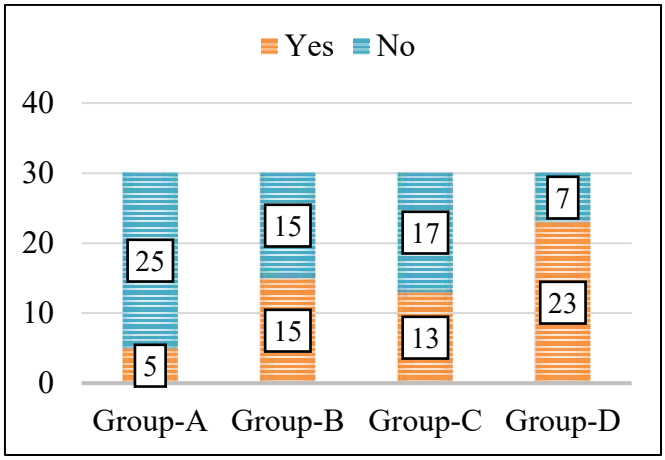

Figure-3: Percentage of ≥50% reduction of headache frequency in the four groups

In Figure-3 and Table-IV, the proportion of participants who experienced a reduction in headache frequency of at least 50% is presented. In group-A, group-B, group-C, and group-D, the proportion of participants achieving a reduction of headache frequency by 50% or more was 16.7%, 50.0%, 43.3%, and 76.7%, respectively. There was a considerable disparity among the four groups (p-value <0.001).

Table-IV: Proportion of participants achieving ≥50% reduction of headache frequency

Comparison groups	≥50% reduction in frequency[1]	p-value
All groups[1]	5(16.7), 15(50.0) 13(43.3), 23(76.7)	<0.001[a]
A – B[1]	5(16.7), 15(50.0)	<0.001[a]
A – C[1]	5(16.7), 13(43.3)	0.02[a]
A – D[1]	5(16.7), 23(76.7)	<0.001[a]
B – C[1]	15(50.0), 13(43.3)	0.61[a]
B – D[1]	15(50.0), 23(76.7)	0.03[a]
C – D[1]	13(43.3), 23(76.7)	0.01[a]
1-n(%), a-Chi-square test		

Group-A exhibited a notably smaller proportion compared to the other groups (p-values <0.001, 0.02 and <0.001 against group-B, group-C and group-D, respectively). Proportions were comparable between group-B and group-C (p-value 0.61). Group-D demonstrated a higher proportion compared to group-B and group-C (p-values 0.03 and 0.01, respectively).

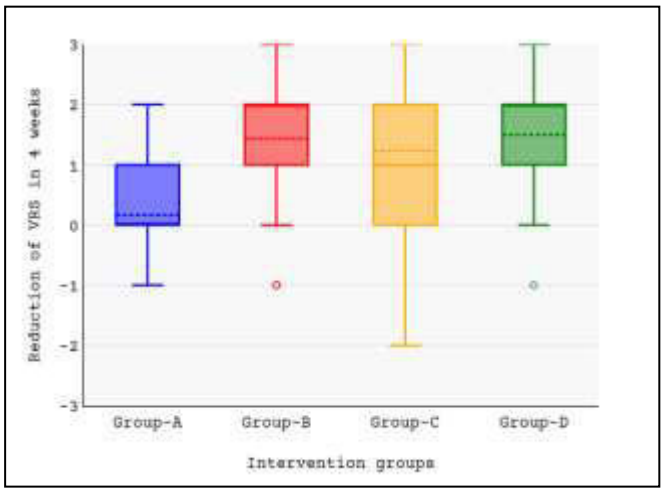

Figure-4: Reduction of VRS score in the four groups

Figure-4 and Table-V illustrate the reduction of VRS score among the participants. The Mean±SD of the reduction of VRS score was 1.1±1.2. Mean±SD VRS score reduction was 0.2±0.6, 1.4±1.1, 1.2±1.3 and 1.5±1.1 in group-A, group-B, group-C and group-D, respectively with significant difference across the four groups (p-value <0.001). Group-A revealed a significantly smaller reduction in VRS score relative to the other three groups (p-value <0.001 in all cases). No significant difference was found between group-B and group-C, group-C and group-D, and group-B and group-D (p-values 0.53, 0.82 and 0.4, respectively).

Table-V: Reduction of VRS score among the participants

Comparison groups	Reduction in VRS score[1]	p-value
All groups[1]	0.2±0.6, 1.4±1.1 1.2±1.3, 1.5±1.1	<0.001[a]
A – B[1]	0.2±0.6, 1.4±1.1	<0.001[b]
A – C[1]	0.2±0.6, 1.2±1.3	<0.001[b]
A – D[1]	0.2±0.6, 1.5±1.1	<0.001[b]
B – C[1]	1.4±1.1, 1.2±1.3	0.53[b]
B – D[1]	1.4±1.1, 1.5±1.1	0.82[b]
C – D[1]	1.2±1.3, 1.5±1.1	0.40[b]
1-Mean±SD, a-ANOVA, b-Student's t-test		

Discussion:

The participants had a mean age of 32.3±6.2 years, with a strong female predominance (77.5%). This age distribution aligns with findings from Majumder et al., Ahmed et al., and Haque et al. in their studies of Bangladeshi individuals with migraines[20-22]. A female predominance was also observed, with a ratio of 3:1. The higher female to male ratio observed in this study is possibly due to the fact that the participants were recruited from the outpatients that operates between 8 am and 2 pm, when majority of the males remain engaged in their workplaces. Aura was found in 21.7% participants, similar to found in the recent studies[20,21].

The median baseline headache frequency among the participants was 8 per 4 weeks. In their studies, Rafi et al. and Tayeb et al. reported mean headache frequencies of 9.5 and 7.0 per 4-week period, respectively[23,24]. The participants in this study had a median VRS score of 3. In their studies, Ahmed et al. and Tayeb et al. reported mean Visual Analogue Scale (VAS) scores of 8 and 7 (in a 0-10 scale), respectively which correspond to a VRS score of 3[19,21,24].

Participants on Propranolol and Topiramate, either singly or combined, had a greater reduction of frequency of migraine and VAS score and a greater proportion of achieving ≥50% reduction of frequency than the placebo group. It indicates both Propranolol and Topiramate are more effective than placebo. Linde et al. in their meta-analysis on 58 studies concluded that Propranolol was more effective than placebo in reducing the frequency of migraine[25]. Another meta-analysis by Versijpt et al. drew a similar conclusion about the efficacy of Propranolol in migraine prophylaxis[26]. Raffaelli et al. in their meta-analysis on 8 RCTs found Topiramate as an effective drug in reducing migraine frequency[13]. In a meta-analysis that included 8 RCTs, Guo et al. found that Topiramate is effective in the prophylaxis of migraines[27].

Propranolol and Topiramate did not differ in reducing either frequency or severity. A recent study in Bangladesh conducted by Tayeb et al. found Propranolol and Topiramate as equally effective in migraine prophylaxis[13]. Two recent Indian studies conducted in Bihar and New Delhi also concluded the same[11,12]. The findings of this study contrasts with those of two studies in Isfahan, Iran and Andhra Pradesh, India who found Topiramate superior to Propranolol in migraine prophylaxis[9,10].

Participants on Propranolol-Topiramate combination demonstrated a more considerable decrease in headache frequency and a larger percentage of achieving a ≥50% reduction than those who were treated with either drug alone. Nonetheless, the decrease in VRS score was comparable between the single agent and dual agent groups. Research by Pascual et al. found Propranolol-Topiramate combination more efficacious than either of the single agents in reducing headache frequency in migraine[15].

There were no differences in any of the three variables studied between the groups treated solely with Propranolol or Topiramate. Propranolol and Topiramate showed equal efficacy in reducing both the frequency and the severity of headache in migraine. A combination of Propranolol and Topiramate is superior to either of the single agents in reducing the frequency.

Conclusion:

Propranolol and Topiramate are equally effective in reducing frequency and severity of migraine. Their combination outperforms each single agent in terms of reducing frequency.

Limitations and recommendations:

This research was carried out with a small group collected from one center. The follow-up time was also shorter than the recommended 12 weeks. Further multi-centre studies with large samples on the efficacy of Propranolol, Topiramate and their combination on migraine prophylaxis are recommended.

Conflict of interest:

The authors have no conflict of interest to reveal.

References:

1. Amiri P, Kazeminasab S, Nejadghaderi SA, Mohammadinasab R, Pourfathi H, Araj-Khodaei M, et al. Migraine: a review on its history, global epidemiology, risk factors, and comorbidities. Front Neurol. 2022;12:800605. doi: 10.3389/fneur.2021. 800605.
2. Steiner TJ, Stovner LJ, Jensen R, Uluduz D, Katsarava Z. Lifting the burden: the Global Campaign against Headache. Migraine remains second among the world's causes of disability, and first among young women: findings from GBD2019. J Headache Pain. 2020;21(1):137. doi: 10.1186/s10194-020-01208-0.
3. Ashina M. Migraine. N Engl J Med. 2020;383(19): 1866-76. doi: 10.1056/NEJMra1915327.
4. Khan J, Asoom LIA, Sunni AA, Rafique N, Latif R, Saif SA, et al. Genetics, pathophysiology, diagnosis, treatment, management, and prevention of migraine. Biomed Pharmacother. 2021;139:111557. doi: 10.1016/ j.biopha.2021.111557.
5. Headache Classification Committee of the International Headache Society (IHS) The International Classification of Headache Disorders, 3rd edition. Cephalalgia. 2018;38(1):1-211. doi: 10.1177/0333102417738202.
6. Diener HC, Holle-Lee D, Naegel S, Dresler T, Gaul C, Gobel H, et al. Treatment of migraine attacks and prevention of migraine: guidelines by the German Migraine and Headache Society and the German Society of Neurology. Clin Translat Neurosci. 2019;3(1):1-40. doi: 10.1177/2514183X18823377
7. Sprenger T, Viana M, Tassorelli C. Current prophylactic medications for migraine and their potential mechanisms of action. Neurotherapeutics. 2018;15(2):313-23. doi: 10.1007/s13311-018-0621-8.
8. Ray JC, Macindoe C, Ginevra M, Hutton EJ. The state of migraine: an update on current and emerging treatments. Aust J Gen Pract. 2021;50(12):915-21. doi: 10.31128/AJGP-01-21-5807.

9. Ashtari F, Shaygannejad V, Akbari M. A double-blind, randomized trial of low-dose topiramate vs propranolol in migraine prophylaxis. Acta Neurol Scand. 2008;118 (5):301-5. doi: 10.1111/j.1600-0404.2008.01087.x.
10. Rajkumar K, Kumar PA. A prospective randomized single blind study to compare the safety and efficacy of propranolol and topiramate in migraine prophylaxis. Int J Basic Clin Pharmacol. 2021;10(4):425–8. doi: 10.18203/2319-2003.ijbcp20211027.
11. Kumar R, Haque SMI, Singh A, Kumar M. Comparison of propranolol and topiramate for the prevention of chronic migraine. Int J Pharmacet Clin Res. 2024;16(2):1035-8. doi: 10.18203/2319-2003.ijbcp 2021 1027
12. Chowdhury D, Bansal L, Duggal A, Datta D, Mundra A, Krishnan A, et al. TOP-PRO study: A randomized double-blind controlled trial of topiramate versus propranolol for prevention of chronic migraine. Cephalalgia. 2022;42(4-5):396-408. doi: 10.1177/0333 1024211047454.
13. Tayeb M, Hasanuzzaman M, Karim AMMR, Sanaullah M, Islam MA. Topiramate versus Propranolol in the prophylaxis of migraine in Bangladeshi population. Chattagram Maa-O-Shishu Hosp Med Coll J. 2020;18(2),12–7. doi: 10.3329/cmoshmcj.v18i2.47764
14. Ghahrami S, Hadi N, Shariat A, Memer Z, Montazeri A. A quality of life of migraine patients treated with combined propranolol and topiramate. Shiraz-E-Med J. 2014;15(4):e23277. doi: 10.17795/semj23277.
15. Pascual J, Rivas MT, Leira R. Testing the combination beta-blocker plus topiramate in refractory migraine. Acta Neurol Scand. 2007;115(2):81-3. doi: 10.1111/j.1600-0404.2006.00772.x.
16. Tfelt-Hansen P, Pascual J, Ramadan N, Dahlof C, D'Amico D, Diener HC, et al. Guidelines for controlled trials of drugs in migraine: third edition. A guide for investigators. Cephalalgia. 2012;32(1):6-38. doi: 10.1177/0333102411417901.
17. Aggarwal R, Ranganathan P. Study designs: part 4 - interventional studies. Perspect Clin Res. 2019;10(3): 137-9. doi: 10.4103/picr.PICR_91_19.
18. Cameron C, Kelly S, Hsieh SC, Murphy M, Chen L, Kotb A, et al. Triptans in the acute treatment of migraine: a systematic review and network meta-analysis. Headache. 2015;55 (Suppl 4):221-35. doi: 10.1111/head.12601.
19. Aicher B, Peil H, Peil B, Diener HC. Pain measurement: visual analogue scale (VAS) and verbal rating scale (VRS) in clinical trials with OTC analgesics in headache. Cephalalgia. 2012;32(3):185-97. doi: 10.1177/ 03331024111430856.
20. Majumder B, Paul D, Barman KK, Ferdoushi SS, Islam MR, Roy NR. Demographic and clinical patterns of headache in migraine patients. Bangladesh J Neurosci. 2019;35(2):63-8. doi: 10.3329/bjn.v35i1.57628.
21. Ahmed KGU, Mahmud R, Islam MR, Chowdhury AH, Alam I, Russel M, et al. Migraine headache: A Bangladesh perspective. Bangladesh J Neurosci. 2019; 35(1):1-9. doi: 10.3329/bjn.v35i1.57618.
22. Haque R. Migraine pain location in adult: the Bangladeshi experience. J Headache Pain. 2014;15 (Suppl 1):D27. doi: 10.1186/1129-2377-15-S1-D27.
23. Rafi A, Islam S, Hasan MT, Hossain G. Prevalence and impact of migraine among university students in Bangladesh: findings from a cross-sectional survey. BMC Neurol. 2022;22(1):68. doi: 10.1186/s12883-022-02594-5.
24. Linde K, Rossnagel K. Propranolol for migraine prophylaxis. Cochrane Database Syst Rev. 2017;2 (2):CD003225.doi:10.1002/14651858.CD003225. pub3.
25. Versijpt J, Deligianni C, Hussain M, Amin F, Reuter U, Sanchez-Del-Rio M, et al. European Headache Federation (EHF) critical re-appraisal and meta-analysis of oral drugs in migraine prevention -part 4: propranolol. J Headache Pain. 2024;25(1):119. doi: 10.1186/s10194-024-01826-y.
26. Raffaelli B, García-Azorín D, Boucherie DM, Amin FM, Deligianni CI, Gil-Gouveia R, et al. European Headache Federation (EHF) critical reappraisal and meta-analysis of oral drugs in migraine prevention - part 3: topiramate. J Headache Pain. 2023;24(1):134. doi: 10.1186/s10194-023-01671-5.
27. Guo Y, Han X, Yu T, Yao G. Meta-analysis of efficacy of topiramate in migraine prophylaxis. Neural Regen Res. 2012;7(23):1806-11. doi: 10.3969/j.issn.1673-5374. 2012.23.007.