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Relation of post-stroke headache to cerebrovascular pathology and hemodynamics

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Abstract

Despite high prevalence of cerebrovascular stroke, headache attributed to ischemic strokes is often undertreated and overlooked. The aim is to detect the relation of a post-stroke headache to cerebrovascular pathology and changes in hemodynamics through a high-resolution duplex ultrasound examination. The present study was a case-control study conducted among 239 patients, who presented with an acute ischemic stroke. Patients were sub-divided into two groups: group I included patients with headache attributed to ischemic stroke (cases) and group II included headache-free stroke patients (controls). History consisted of headache characteristics and risk factors. Clinical and radiological examination were preformed to detect the type of stroke. Ultrasound duplex examination of extra-cranial and intra-cranial cerebrovascular system was carried for both groups. Group I included 112 patients (mean age, 57.66 ±6.59 years), and group II included 127 patients (mean age 57.73 ±7.89 years). Post-stroke headache was more frequent in patients with posterior circulation infarction (58%). Post-stroke headache was reported within 7 days post-stroke in 61.6% of patients. Pre-stroke headache was an independent predictor for post-stroke headache occurrence (OR = 28.187, 95% CI: 6.612-120.158%, $p < 0.001$). Collateral opening and various degrees of intra-cranial vascular stenosis were strong predictors of headache occurrence (OR = 25.071, 95% CI: 6.498-96.722%, $p < 0.001$). In conclusion, post-stroke headache is a common phenomenon, especially in patients with pre-stroke headache, history of old stroke, posterior circulation infarction, and large artery disease. This headache was of moderate-intensity with clinical characteristics of tension-type. Intra-cranial cerebrovascular pathological changes including opening of collateral channels and variable degrees of stenosis of cerebrovascular systems were implicated in the production of that headache.

Key words: *post-stroke headache, cerebrovascular, hemodynamics, duplex ultrasound.*

Introduction

Cerebrovascular stroke is a medical hazard that is responsible for many morbidities and mortalities. Despite its' high prevalence, headache attributed to ischemic stroke is often undertreated and overlooked [14]. It is an important symptom that may be severe and persistent after ischemic stroke [8]. The prevalence of headache attributed to ischemic stroke is ranging between 7.4% and 34% [23].

The clinical criteria of headache attributed to ischemic stroke were introduced by the International Classification of Headache Disorders (ICHD), which was finally updated for the third time in 2018 [4]. These new criteria more accurately reflected the

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close temporal relationship between the onset of headache and the development of focal signs and symptoms of an ischemic stroke, an improvement of headaches, and better classified the entity of persistent post-stroke headaches [17].

The exact pathophysiology of such headache is still unclear [24]. Till now, many theories have been introduced to justify such headache. Activation of the trigemino-vascular pathway is the most accepted theory for headache production [11,24]. However, there were many conflicting data in the literature regarding the triggers that activate this pathway [3,11,24].

Atherosclerosis and collateral opening can be considered as another triggering factors in the pathogenesis of post-stroke headache. Despite that the trigemino-vascular system could be activated by biochemical triggers, opening collaterals could be involved. Due to difficulty in the assessment of opening of collaterals, the literature does not contain enough data to explain its' role in the production of post-stroke headache till now [14].

Duplex ultrasound is considered an easy, feasible, safe, more accurate, and non-invasive tool for the assessment of cerebrovascular changes, including the collateral system and hemodynamics [6,12].

This study aimed to understand the relation of a post-stroke headache to cerebrovascular pathology and hemodynamic changes by using a high-resolution duplex ultrasound examination.

Material and methods

The current study was an observational retrospective case-control study that was conducted in Kasralainy hospital, Cairo University, and Al-Azhar University hospitals from January 2021 to August 2021. We included 239 patients, with a recent ischemic stroke within three months from the onset. Patients were further sub-divided into two groups: Group I (cases) included patients fulfilling criteria of the ICHD-3 for headache attributed to ischemic stroke, and group II (controls) consisted of headache-free stroke patients. We excluded patients with intra-cranial hemorrhages, transient ischemic attacks, artery dissections, superficial temporal arteritis, unconscious, confused, and/or aphasic patients.

Demographic data collected included age, gender, history of pre-stroke headache, and associated comorbidities (hypertension, diabetes mellitus, ischemic heart disease, atrial fibrillation, dyslipidemia, smoking, presence of old stroke, and using anti-hypertensive medications). Clinical symptoms and signs, initial computed tomography, and/or magnetic resonance imaging of the patient's brain and cerebrovascular system were obtained. Strokes were classified according to TOAST (Trial of Org 10172 in Acute Stroke Treatment) classification [1]. As regards to vascular distribution of stroke, Oxfordshire Community Stroke Project (OCSP) was applied to classify the patients into partial anterior circulation infarcts (PACI), lacunar circulation infarcts (LACI), and posterior circulation infarcts (POCI) [25]. The presence of headache attributed to ischemic stroke was considered if the patient reported newly developed headache or changing in pre-stroke headache character. Features of post-stroke headache, including character (tightening, pulsatile, or stabbing), intensity (mild to moderate, if not interfere with daily activities; or moderate to severe, if interfere with daily activities), side (ipsilateral to the infarction, contralateral, unilateral alternating, or bilateral), and location [diffuse, anterior (frontal, temporal, or parietal); posterior (occipital)] were collected. In addition, we identified the time of post-stroke headache with the onset of stroke (72 hours before stroke onset, within 7 days post-stroke, or after 7 days post-stroke) and headache-accompanying symptoms (nausea, vomiting, photophobia, and phonophobia).

Ultrasound duplex assessment was carried out for all patients using Phillips iU 22 ultrasound equipment, with a low frequency of 3-9 MHz linear probe for extra-cranial vascular systems (carotid and vertebrobasilar systems), and low frequency of 1-5 MHz phased array for trans-cranial cerebrovascular system. In case of carotid stenosis, data are interpreted according to the Society of Radiologists in Ultrasound Consensus Criteria for Carotid Stenosis [12]. Parameters obtained transcranially were interpreted according to Baumgartner criteria [6].

This study was conducted in agreement with the declarations of Helsinki, and the institutional review board approval was obtained from the ethics unit of Faculty of Medicine, Al-Azhar University. Each patient was requested to sign a written informed consent before enrollment into the study.

The sample size calculation was conducted using MedCalc software for windows after applying the prevalence of carotid atherosclerosis among patients with stroke-associated headache that was reported by Paciaroni *et al.* [24] to be 59.1%, with alpha error

as 5% and beta error 20%. These resulted in recruitment of 238 patients. Statistical analysis was conducted using SPSS platform for Windows, version 26. Data were presented according to their type as mean ± standard deviation (SD), in normally distributed continuous data. Categorical data were presented as numbers and percentages (%). Comparison between the case and control groups was conducted using Student's *t*-test and chi-square (χ^2) test, according to type of data. Multivariate logistic regression analysis was applied to identify the significant predictors of post-stroke headache. *P*-values of less than 0.05 were considered statistically significant.

Results

Group I included 112 patients and group II consisted of 127 matched patients. Pre-morbid risk factors did not show statistically significant differences between the two groups. Pre-stroke headache and type of stroke showed a statistically significant difference between the two groups ($p < 0.001$) (Table I).

The tightening character of headache was the most prevalent type (69.6%); however, it was of moderate intensity in most of the cases (73.2%). Around two-thirds (61.6%) of patients started headache within seven days post-stroke, while in 31.3% occurred after seven days. Headache was ipsilateral to infarction in 58% of patients (Table II).

Despite most of the extra-cranial atherosclerotic diseases were mild in most of the examined arteries, there was a considerable fraction (19.6%) reported with severe internal carotid artery (ICA) stenosis in the case group. Similarly, severe stenosis up to total occlusion of the vertebral artery (VA) was reported in 28.6% of patients with post-stroke headache, with significant difference when compared with patients without headache. A statistically significant increase

Table I. Baseline characteristics of the included 239 patients with acute ischemic stroke

Variable	Headache patients (*n* = 112)	Headache-free patients (*n* = 127)	*P*-value*
Age (years), mean ±SD	57.66 ±6.59	57.73 ±7.89	0.939
Gender (males), *n* (%)	69 (61.6)	69 (54.3)	0.256
Risk factors, *n* (%)			
Diabetes mellitus	65 (58.0)	66 (52.0)	0.347
Hypertension	72 (64.3)	74 (58.3)	0.341
Ischemic heart disease	39 (34.8)	35 (27.6)	0.226
Atrial fibrillation	21 (18.8)	24 (18.9)	0.977
Dyslipidemia	77 (68.8)	78 (61.4)	0.236
Smoking	49 (43.8)	61 (48.0)	0.508
History of an old stroke	49 (43.8)	38 (29.9)	0.027
Use of anti-hypertensive medication	43 (38.4)	45 (35.4)	0.636
History of pre-stroke headache, *n* (%)			
Total number	73 (62.2)	19 (15.0)	< 0.001
Migraine	19 (26.0)	6 (31.6)	0.889
Tension-type headache	51 (69.9)	13 (68.4)	
Cluster	3 (4.1)	0 (0.0)	
Clinical state of patients, *n* (%)			
Partial anterior circulation infarct	28 (25.0)	62 (48.8)	< 0.001
Posterior circulation infarct	65 (58.0)	25 (19.7)	< 0.001
Lacunar infarct	19 (17.0)	40 (31.5)	0.009
Etiology of stroke, *n* (%)			
Large artery disease	65 (58.0)	28 (22.0)	< 0.001
Small vessel disease	32 (28.6)	61 (48.0)	0.002
Cardio-embolism	12 (10.7)	25 (19.7)	0.056
Undetermined or other causes	3 (2.7)	13 (10.2)	0.02

** p < 0.05 was considered statistically significant*

Table II. Headache characteristics in patients with post-stroke headache

Characteristics	Headache patients (n = 112)
Character, *n* (%)	
Pulsatile	30 (26.8)
Stabbing	4 (3.6)
Tightening	78 (69.6)
Intensity, *n* (%)	
Mild to moderate	82 (73.2)
Moderate to severe	30 (26.8)
Temporal relation of post-stroke headache with focal signs, *n* (%)	
72 hours before stroke onset	8 (7.1)
Within 7 days post-stroke	69 (61.6)
After 7 days post-stroke	35 (31.3)
Location, *n* (%)	
Anterior	57 (50.9)
Posterior	35 (31.3)
Diffuse	20 (17.9)
Side, *n* (%)	
Ipsilateral to infarction	65 (58.0)
Contralateral to infarction	15 (13.4)
Unilateral alternating	7 (6.3)
Bilateral	25 (22.3)
Association, *n* (%)	
Nausea and vomiting	46 (41.1)
Photophobia	30 (26.8)
Phonophobia	12 (10.7)

in the proportion of large artery stenosis was detected in group I (Table III).

The frequency of collateral opening was higher in the case group. Headache was more frequent in patients with the leptomeningeal collateral opening ($p < 0.001$). Group I showed an increased frequency of intra-cranial stenosis (irrespective of the grade of stenosis) in both anterior and posterior circulation (Table IV).

The multi-variable regression analysis revealed that the presence of pre-stroke headache was an independent predictor for post-stroke headache occurrence (OR = 28.187, 95% CI: 6.612-120.158%, $p < 0.001$). The presence of any intra-cranial cerebrovascular pathological changes, regardless its' nature, can also predict headache development (OR = 25.071, 95% CI: 6.498-96.722%, $p < 0.001$). Mild stenosis (< 50%) of either PCA or VA segment 4 and opening collaterals were also strong predictors for post-stroke headache ($p < 0.001$, for all) (Table V).

Discussion

The intra-cranial cerebrovascular pathological changes, including opening of the collateral channels and variable degrees of stenosis of cerebrovascular systems, were implicated in the production of the post-stroke headache. Headache is a commonly reported symptom in patients with acute ischemic stroke. Despite the literature being crowded with data suggesting that the activation of the trigemino-vascular system due to cortical spreading depression (CSD) triggered by cerebral ischemia, the pathophysiology of headache attributed to ischemic stroke has not been well-established until now [9,23]. The literature suggested headache attributed to ischemic stroke was not related to intra-cranial vasodilation or increased flow through collateral circulation [15]. This could be assigned to the lack of association between post-stroke headache and angiographically demonstrated collateral circulation [11].

Assessment of cerebrovascular structures and hemodynamics in this study using duplex ultrasound has added valuable data in understanding its' pathophysiology. It was shown that post-stroke headache was more common in patients with pre-morbid headache attacks, yet with a significant change of character following the cerebrovascular event. Large artery atherosclerotic stenosis was strongly related to the headache precisely in the territory of the posterior circulation. Patients with collaterals opening, particularly leptomeningeal one, showed a higher frequency of post-stroke headache. Regarding the history of pre-stroke headache, our results were concomitant with previous literature, which revealed the presence of pre-stroke headache in 22-57% of patients with stroke-attributed headache [26]. This could be attributed to stroke-associated pathophysiological changes, which could affect the pattern of patient's usual type of headache [22]. In our patient's sample, tension headache was the most prevalent type in the pre-morbid state coming in the first place before the vascular headache. This could be explained through a release of common headache-associated chemical mediators in both vascular and tension-type headache, vasoactive substances, amino acid neurotransmitters, and platelet activation factor [10,11,16]. Subsequently, ischemic stroke could affect the production of these substances making changes in the character of the pre-stroke headache [10]. There was a significant association between previous ischemic strokes and development of post-stroke headache. The association of old ischemic stroke and development of

Table III. Characteristics of extra-cranial vasculature

Variable	Headache patients (*n* = 112)	Headache-free patients (*n* = 127)	*P*-value*
Basilar artery, *n* (%)			
Mild stenosis < 50%	23 (20.5)	4 (3.1)	< 0.001
Moderate stenosis > 50%	12 (10.7)	5 (3.9)	0.042
Vertebral arteries, *n* (%)			
Mild stenosis < 50%	29 (25.9)	3 (2.4)	< 0.001
Moderate stenosis > 50%	25 (22.3)	5 (3.9)	< 0.001
Severe stenosis > 70%	18 (16.1)	4 (3.1)	0.001
Occlusion	14 (12.5)	2 (1.6)	0.001
Incomplete subclavian steal, *n* (%)	15 (13.4)	4 (3.1)	0.003
Complete subclavian steal, *n* (%)	14 (12.5)	3 (2.4)	0.002
ICA stenosis, *n* (%)			
< 50%	24 (21.4)	9 (7.1)	0.001
50-69%	14 (12.5)	7 (5.5)	0.06
> 70%	22 (19.6)	6 (4.7)	< 0.001
ICA total occlusion, *n* (%)	12 (10.7)	5 (3.9)	0.042
Common carotid artery, *n* (%)			
Mild atherosclerosis causing no hemodynamic changes bilaterally	76 (67.9)	23 (18.1)	< 0.001

** p < 0.05 was considered statistically significant; ICA – internal carotid artery*

post-stroke headache could be explained through an opening of more pain-sensitive collaterals' channels as a consequence of each ischemic event [15]. The time of this collateral opening was reported to be in close temporal relation with the onset of ischemic stroke [14]. That is why our results showed that post-stroke headache was more likely to occur within the first seven days post-stroke. These results were in agreement with a previous study, which revealed that post-stroke headache occurred mostly within the first 3-4 days post-stroke [19]. This could be also explained by the endothelial release of bradykinins, prothrombotic factors, glutamate, interleukin-6, nitric oxide metabolites, calcitonin gene-related peptides, substance P, and excitotoxic substances during the acute phase of ischemic stroke [13,18]. Despite that supratentorial lesions are usually accompanied by headache in ophthalmic distribution of the trigeminal nerve [3], our data revealed that the anteriorly-located headache was more associated with posterior circulation infarcts. This may indicate a relationship between the richly innervated posterior circulation vasculature through the trigeminal system [20].

Activation of the trigemino-vascular pathway in posterior circulation affection could be attributed to ischemia of the trigeminal nucleus, dysfunction of the serotoninergic nuclei of the brainstem, or ischemia of the dura of the posterior fossa [2,7,27]. Furthermore, the enhanced trigemino-vascular pathway, in addition to collaterals opening, occur mainly ipsilaterally to the infarction, explaining the high frequency of ipsilateral post-stroke headache [13]. However, due to large receptive area of the trigeminal system, some intra-cranial vessels have contralateral trigeminal innervation [21]. This gives an acceptable explanation for reporting contralateral headache in some patients. The association of headache with ischemic stroke related to carotid artery disease revealed in our study was reported in previous research. Its' prevalence ranged between 26% and 35% [5]. Being involved in the development of thrombotic stroke [8], carotid artery disease could explain the significant association between thrombotic, large, and small vessel diseases, stroke, and post-stroke headache, rather than cardioembolic stroke. However, the prevalence of headache associated with large vessel disease was higher than that linked to small vessel disease, indicating an implication of arterial caliber in headache production. Occlusion of a large artery produces large infarction, which in turn produces large inflammation and irritation to pain-sensitive structures, causing headache [5]. Our study could be limited by selection bias, as the patients with disturbed level of consciousness or those

Table IV. Characteristics of intra-cranial vasculature

Variables	Headache patients (n = 112)	Headache-free patients (n = 127)	P-value*
Collateral opening, n (%)	80 (71.4)	14 (11)	< 0.001
Leptomeningeal	34 (30.3)	7 (5.5)	< 0.001
ACA	25 (22.3)	4 (3.1)	< 0.001
Posterior communicating artery	21 (18.8)	3 (2.4)	< 0.001
Diffuse intra-cranial stenosis, n (%)	21 (18.8)	3 (2.4)	< 0.001
Mild stenosis < 50%, n (%) [a]			
MCA	59 (52.7)	11 (8.7)	< 0.001
ACA	14 (12.5)	6 (4.7)	0.03
ICA	21 (18.8)	3 (2.4)	< 0.001
PCA	47 (42.0)	4 (3.1)	< 0.001
BA	23 (20.5)	4 (3.1)	< 0.001
VA4	36 (32.1)	1 (0.8)	< 0.001
Moderate stenosis > 50%, n (%)			
MCA	45 (40.2)	5 (3.9)	< 0.001
ACA	15 (13.4)	4 (3.1)	0.003
ICA	24 (21.4)	3 (2.4)	< 0.001
PCA	20 (17.9)	0 (0.0)	< 0.001
BA	12 (10.7)	5 (3.9)	0.042
VA4	33 (29.5)	1 (0.8)	< 0.001
Severe stenosis > 70%, n (%)			
MCA	39 (34.8)	5 (3.9)	< 0.001
ACA	14 (12.5)	2 (1.6)	0.001
ICA	9 (8.0)	1 (0.8)	0.007
PCA	22 (19.6)	0 (0.0)	< 0.001
VA4	26 (23.2)	0 (0.0)	< 0.001

*MCA – middle cerebral artery, ACA – anterior cerebral artery, ICA – internal carotid artery, PCA – posterior cerebral artery, BA – basilar artery, VA4 – vertebral artery segment 4, [a] arteries were presented bilaterally, but considered separately when different degrees of stenosis were present in the same patient; *p < 0.05 was considered statistically significant*

Table V. Multivariate logistic regression to detect independent predictors of post-stroke headache

Predictor variables	OR	95% CI		P-value
		Lower	Upper	
Pre-stroke headache	28.187	6.612	120.158	< 0.001
PCA stenosis < 50%	84.657	10.418	687.947	< 0.001
VA4 stenosis < 50%	842.472	50.262	14121.06	< 0.001
Intra-cranial cerebrovascular system pathological changes	25.071	6.498	96.722	< 0.001
Collateral opening	60.826	13.003	284.541	< 0.001

PCA – posterior cerebral artery, VA4 – vertebral artery segment 4; p < 0.05 was considered statistically significant

with aphasia could not provide information regarding the presence of headache. Therefore, patients with poor prognosis were not included.

Conclusions

Headache attributed to ischemic stroke is a common phenomenon, especially in patients with pre-stroke headache, history of old stroke, posterior circulation infarction, and large artery disease. This headache was of moderate-intensity with clinical characteristics of tension-type, and it was in close temporal relation with clinical symptoms and signs in most of the cases. The intra-cranial cerebrovascular pathological changes, including opening of the collateral channels and variable degrees of stenosis of cerebrovascular systems, were implicated in the produc-

tion of that headache. Our results could help further studies for better understanding and hence, provide better management for post-stroke headache. Further prospective studies assessing the effect of headache attributed to ischemic stroke on the prognosis and outcome of ischemic stroke and post-stroke disability and quality of life are also needed.

Disclosure

The authors report no conflict of interest.

References

1. Adams Jr HP, Bendixen BH, Kappelle LJ, Biller J, Love BB, Gordon DL, Marsh 3rd EE. Classification of subtype of acute ischemic stroke. Definitions for use in a multicenter clinical trial. TOAST. Trial of Org 10172 in Acute Stroke Treatment. Stroke 1993; 24: 35-41.
2. Afridi S, Goadsby P. New onset migraine with a brain stem cavernous angioma. J Neurol Neurosurg Psychiatry 2003; 74: 680-682.
3. Arboix A, Massons J, Oliveres M, Arribas MP, Titus F. Headache in acute cerebrovascular disease: a prospective clinical study in 240 patients. Cephalalgia 1994; 14: 37-40.
4. Arnold M. Headache classification committee of the international headache society (IHS) the international classification of headache disorders. Cephalalgia 2018; 38: 1-211.
5. Atkinson RA, Appenzeller O. Headache in small vessel disease of the brain: a study of patients with systemic lupus erythematosus. Headache 1975; 15: 198-201.
6. Baumgartner RW, Mattle HP, Schroth G. Assessment of ≥ 50% and < 50% intracranial stenoses by transcranial color-coded duplex sonography. Stroke 1999; 30: 87-92.
7. Bonvento G, MacKenzie E T, Edvinsson L. Serotonergic innervation of the cerebral vasculature: relevance to migraine and ischaemia. Brain Res Rev 1991; 16: 257-263.
8. Chen PK, Chiu PY, Tsai IJ, Tseng HP, Chen JR, Yeh SJ, Yeh SJ, Sheu JJ, Chung CP, Wu MH, Hu CJ. Onset headache predicts good outcome in patients with first-ever ischemic stroke. Stroke 2013; 44: 1852-1858.
9. Dohmen C, Sakowitz OW, Fabricius M, Bosche B, Reithmeier T, Ernestus RI, Brinker G, Dreier JP, Woitzik J, Strong AJ, Graf R. Spreading depolarizations occur in human ischemic stroke with high incidence. Ann Neurol 2008; 63: 720-728.
10. Edmeads J. Complicated migraine and headache on cerebrovascular disease. Neurol Clin 1983; 1: 385-397.
11. Edmeads J. The headaches of ischemic cerebrovascular disease. Headache 1979; 19: 345-349.
12. Grant EG, Benson CB, Moneta GL, Alexandrov AV, Baker JD, Bluth EI, Carroll BA, Eliasziw M, Gocke J, Hertzberg BS, Katarick S. Carotid artery stenosis: grayscale and Doppler ultrasound diagnosis – Society of Radiologists in Ultrasound consensus conference. Ultrasound Q 2003; 19: 190-198.
13. Hamdy SM, Barrada HO, Fahmy MS, Samir H. Headache associated with transient or permanent cerebrovascular disease. J Headache Pain 2002; 3: 101-104.
14. Harriott AM, Karakaya F, Ayata C. Headache after ischemic stroke: a systematic review and meta-analysis. Neurology 2020; 94: e75-e86.
15. Iwasawa E, Ichijo M, Ishibashi S, Yokota T. Acute development of collateral circulation and therapeutic prospects in ischemic stroke. Neural Regen Res 2016; 11: 368-371.
16. Jensen R. The tension-type headache alternative. Peripheral pathophysiological mechanisms. Cephalalgia 1999; 19 (25 suppl): 9-10.
17. Lai J, Harrison RA, Plecash A, Field TS. A narrative review of persistent post-stroke headache – a new entry in the International Classification of Headache Disorders. Headache 2018; 58: 1442-1453.
18. Leira R, Davalos A, Aneiros A, Serena J, Pumar JM, Castillo J. Headache as a surrogate marker of the molecular mechanisms implicated in progressing stroke. Cephalalgia 2002; 22: 303-308.
19. Maino A, Algra A, Koudstaal PJ, Van Zwet EW, Ferrari MD, Wermer MJ. Concomitant headache influences long-term prognosis after acute cerebral ischemia of noncardioembolic origin. Stroke 2013; 44: 2446-2450.
20. Marinkovi SV, Gibo H. The blood supply of the trigeminal nerve root, with special reference to the trigeminocerebellar artery. Neurosurgery 1995; 37: 309-317.
21. O'Connor TP, van der Kooy D. Pattern of intracranial and extracranial projections of trigeminal ganglion cells. J Neuroscience 1986; 6: 2200-2207.
22. Olesen J, Friberg L, Olsen TS, Andersen AR, Lassen NA, Hansen PE, Karle A. Ischaemia-induced (symptomatic) migraine attacks may be more frequent than migraine-induced ischaemic insults. Brain 1993; 116: 187-202.
23. Oliveira FA, Sampaio Rocha-Filho PA. Headaches attributed to ischemic stroke and transient ischemic attack. Headache 2019; 59: 469-476.
24. Paciaroni M, Parnetti L, Sarchielli P, Gallai V. Headache associated with acute ischemic stroke. J Headache Pain 2001; 2: 25-29.
25. Pittock SJ, Meldrum D, Hardiman O, Thornton J, Brennan P, Moroney JT. The Oxfordshire Community Stroke Project classification: correlation with imaging, associated complications, and prediction of outcome in acute ischemic stroke. J Stroke Cerebrovasc 2003; 12: 1-7.
26. Vestergaard K, Andersen G, Nielsen MI, Jensen TS. Headache in stroke. Stroke 1993; 24: 1621-1624.
27. Weinstein M, Stein R, Pollock J, Stucker TB, Newton TH. Meningeal branch of the posterior cerebral artery. Neuroradiology 1974; 7: 129-131.