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Clinical Profile of Posterior Circulation Stroke

Vaibhav Bhat, Poonam Ashok Kamath*, BA Shastry***

Abstract

***Background:** The vascular territory associated with posterior circulation supplies a compact region of the brain which controls vital body functions, hence a stroke in this area can lead to significant morbidity and mortality. Due to overlapping symptomatology, it is often misdiagnosed as a peripheral cause of vertigo. Our study was aimed at studying the risk factors, symptomatology, anatomical localisation, artery involved, and outcomes related to posterior circulation stroke (PCS). We believe this study can lead to earlier diagnosis and prompt management which is very crucial in deciding outcomes of posterior circulation strokes.*

***Methods:** A tertiary care hospital-based cross-sectional time bound study was conducted among patients attending OPD or admitted in ward in from September 2021 to October 2022 meeting the inclusion-exclusion criteria. 43 patients admitted with PCS under departments of General Medicine and Neurology in Kasturba Hospital, Manipal were analysed for the study.*

***Results:** 22 patients were older than 65 years. 14 were aged 45 to 65 years. 28 were males. Among risk factors, diabetes and hypertension were found to be present in 23 and 21 patients, respectively. 21 subjects had significant smoking history. 26 subjects presented with ataxia or vertigo. 26 subjects had cerebellar signs on examination. Vertebral artery and posterior cerebral artery were the most common implicated vessel (12 and 6 subjects respectively). As per TOAST classification, small artery type was found in 14 subjects and large artery stroke was present in 11 subjects.*

***Conclusions:** Diabetes mellitus was the most common risk factor for PCS in our study followed by hypertension. Ataxia and vertigo were the most common presenting features of PCS in the present study. Cerebellum was the most common anatomical area involved. In our study, vertebral artery was most commonly affected. Small vessel disease was the main aetiological factor followed by large vessel disease.*

Introduction

Cerebrovascular accident (stroke) is a major cause of morbidity and mortality affecting the neurological system. Strokes can be classified based on the involved circulation into anterior and posterior circulation strokes. Posterior circulation of the brain supplies the brainstem, cerebellum, occipital lobe, thalamus, parts of parietal and temporal lobe. As posterior circulation supplies a compact of area of structure controlling important functions and containing neurological pathways, strokes involving this territory often result in significant morbidity and mortality with catastrophic outcomes. They account for 9.2% of all the strokes[1]. PCS presentation can be misdiagnosed as a peripheral cause; it mimics vestibular disease, thereby causing significant delay in diagnosis and treatment[2]. These factors also add on to the morbidity associated with the disease. PCS can also mimic anterior circulation stroke[3]. The clinical presentation and risk factor stratification for PCS have not been extensively studied in the Indian population for PCS. The aim of our study was to study the risk factors, clinical presentation and anatomical areas involved in PCS.

Material and Methods

A tertiary care hospital-based, cross-sectional, time bound study was conducted among patients attending OPD or admitted in ward in from September 2021 to October 2022 meeting the inclusion-exclusion criteria. Ethical clearance was obtained from the Institutional Ethics Committee (IEC) of KMC (Kasturba Medical College) and Hospital, Manipal (IEC 255/2021). The study has been registered with Clinical Trials Registry of India (CTRI/2022/09/045676). Written informed consent was taken.

Patients aged above 18 years of age presenting with clinical features and radiological evidence of PCS were included. Patients having radiological evidence of haemorrhage or infarct in anterior circulation or pre-existing infarcts were excluded from the study.

Detailed questionnaires were filled regarding clinical features, co-morbidities, risk factors, and radiological imaging. Data were entered in MS Excel and analysis was done using SPSS 21.0 version. Data was presented as mean and standard deviation for continuous variables and as percentages for categorical variables. Histograms were used

**Senior Resident, **Professor, Department of General Medicine, Kasturba Medical College, Manipal Academy of Higher Education, Manipal - 576 104, Karnataka.*

Corresponding Author: Dr Poonam Ashok Kamath, Senior Resident, Department of General Medicine, Kasturba Medical College, Manipal Academy of Higher Education, Manipal - 576 104, Karnataka. Tel: 8317402192, E-mail: kamath.poonam@manipal.edu

to plot the continuous data, whereas bar diagrams and pie charts for qualitative data.

Sample size was estimated at 9%prevalence of PCS to be 68. In our centre in that year there were 804 stroke patients, out of which 68 had PCS. However, 19 had simultaneous anterior circulation involvement as well hence were excluded. 6 patients got discharged against medical advice hence investigations and detailed assessment could not be done, and they were not included in analysis. Since it was a time bound study, 43 subjects were studied.

Results

43 patients admitted with PCS under departments of General Medicine and Neurology in Kasturba Hospital, Manipal were analysed for the study. Hence n = 43.

Table I: Baseline characteristics.

Characteristic		Total
Age (years) Mean: 61.3 ± 14	< 45	7
	45 - 65	14
	Above 65	22
Gender	Male	28
	Female	15
Co-morbidities	Diabetes	23
	Hypertension	21
	Dyslipidaemia	11
	BMI > 30 kg/m^2	11
	Ischemic heart disease	5
	Hypothyroidism	4
	Rheumatic heart disease	1
Modifiable risk factors	Smoking	21
	Alcoholism	7

Among the cranial nerves involved, 10 subjects had IX and X nerve palsy. 4 subjects each had III nerve and VII nerve (UMN and LMN) palsy. 2 patients had optic (II) pathway involvement (visual field loss). No involvement of abducent

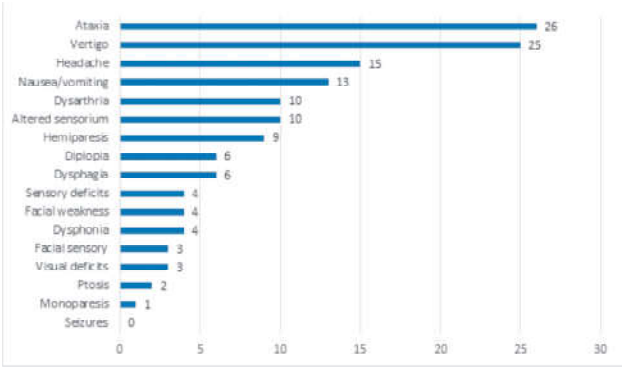

Fig. 1: *Bar graph showing presenting symptoms in posterior circulation stroke.*

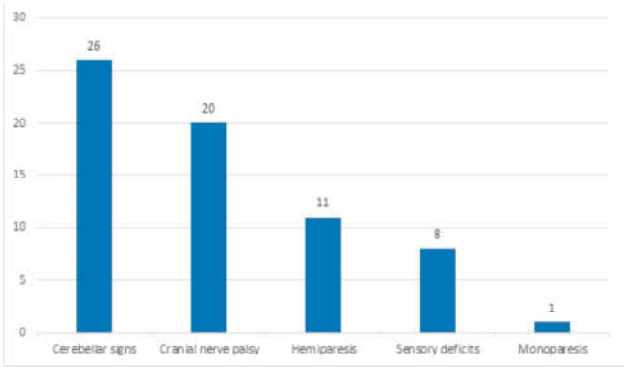

Fig. 2: *Bar graph showing clinical signs on examination.*

(IV) or hypoglossal (XII) nerves were noted in the study.

MRI brain was conducted for all patients in the study. It showed ischemic stroke in 40 subjects and haemorrhage in 3 patients. Out of the three, 2 had pontine bleed and 1 subject had cerebellar bleed.

Among the anatomical areas involved, cerebellum was most commonly implicated, in 19 subjects. Occipital lobe was involved in 14 subjects and 12 had medulla involvement. Pons was involved in 9 patients. 6 patients each had midbrain and temporal lobe involvement. 23

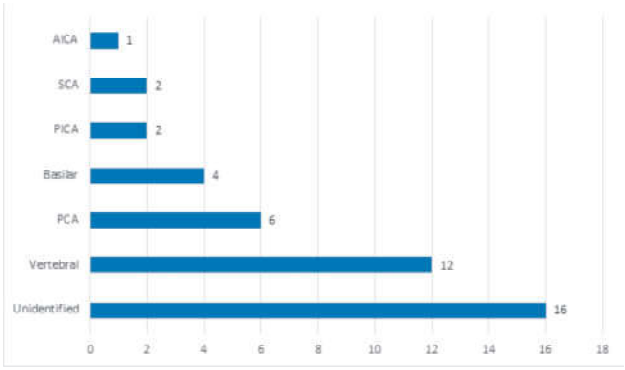

Fig. 3: *Bar graph showing arteries involved in PCS. AICA – anterior inferior cerebellar artery, SCA – superior cerebral artery, PICA – posterior inferior cerebellar artery, PCA – posterior cerebral artery.*

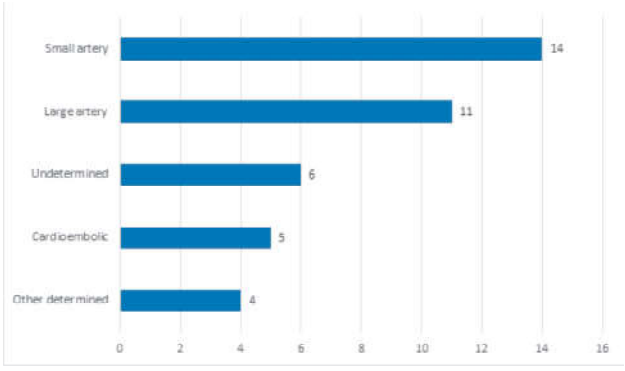

Fig. 4: *Bar graph showing aetiology of stroke as per TOAST classification.*

individuals had multiple territories involved in posterior circulation in the form of lacunar stroke.

Discussion

Prevalence

In our hospital, 8.6% of all strokes in the study period were PCS. It was similar to other Indian studies (Manmohan *et al*)[4] that showed 11.3% of PCS among all strokes. But these were comparatively lesser when compared to Western population studies in Europe and USA, where PCS were ranging from 26 - 39.8%[5,6]. Europe and USA have a higher average life expectancy when compared to India. In older adults, diabetes mellitus, hypertension, dyslipidaemia, metabolic syndrome and other risk factors have a higher chance of causing atherosclerotic vessel wall damage. These metabolic diseases-induced atherosclerotic disease causes hardening and narrowing of arteries which is prominent in posterior circulation system[7].

Age and gender

In the current study, 51% (22) patients were over the age of 65 years with an average age of 61 years and SD of 14 years. The predominance of PCS in older age groups is due to calcium deposition and metabolic disease like diabetes mellitus, dyslipidaemia, hypertension causing atherosclerotic damage and plaque formation. The results are similar to another study by Joshi *et al*[8] with an average age of 59.8 years. In the study by Manmohan *et al*[4], the average age was 51 years as this study had included patients from paediatric age groups. Male predominance was seen in the current study with 65.1% (25) being males. Stroke pathology is influenced by gender in numerous ways and understanding the same still remains incomplete. In females, Oestrogen reduces atherosclerosis by its action on intravascular adhesion process, tone of the smooth muscle and monocyte differentiation[9]. Also, in males, testosterone levels act variably in association with stroke risk. There is a correlation of increased risk in older men with decreased testosterone and younger men with elevated levels of testosterone[10]. Male predominance is also seen in closely associated risk factors like diabetes mellitus and hypertension which predispose male gender to a higher risk of stroke. Among other studies, Manmohan *et al*[4] and Joshi *et al*[8] showed a male predominance. It was also comparable to the New England PCS registry[11] which also showed a male predominance of 65% (256).

Risk factors

In the present study, diabetes mellitus was the predominant risk factor for PCS seen in 23 patients. Diabetes mellitus being a metabolic disease, plays a role in different mechanisms for cardiovascular disease. Long-standing DM can cause early onset arteriosclerotic changes with endothelial dysfunction leading to adhesion defect and thickening of capillary basement membrane. These factors lead to non-compliance of the vessel and atherosclerotic plaque formation due to turbulent flow and systemic inflammation. This predisposes patients with diabetes mellitus to stroke[12]. In the current study, diabetes mellitus was predominant when compared to Manmohan *et al*[4] (30%) and Joshi *et al*[8] (28%), as the present study was done in South Indian population where incidence and prevalence of diabetes mellitus is higher when compared to North Indian population[13].

Hypertension was the second most common risk factor in this study with 21 patients being previously diagnosed with the same. Long-standing hypertension causes accelerated atherosclerosis. These structural modifications in the arterial vessel wall protect the microcirculation from pressure change related injury. However, these changes lower the vessel's response to vasodilation related changes and are seen more in tortuous vessels with higher turbulent flow like vertebral artery. This response associated with intraluminal plaque plays a major role in stroke[13]. It was the predominant risk factor in Manmohan *et al*[4] and Joshi *et al*[8] studies. These studies were done in metropolitan cities. Environmental factors and lifestyle changes may have a role in hypertension which is not fully understood at present[14].

In the current study, 11 subjects had dyslipidaemia at presentation, which is one of the established factors for ischaemic stroke. Dyslipidaemia causes increased fatty acid oxidation. This causes elevated generalised inflammation which also affects arteries. Inflammation with elevated LDL and altered ApoB levels leads to intravascular plaque formation[15]. Role of dyslipidaemia in haemorrhagic strokes remains inconclusive and no significant association is established. Serum triglyceride levels are associated with higher neurological decline post-CVA and poorer recovery. The exact mechanism and role of serum triglyceride in this process is still under evaluation, but significant association has been established[16]. In the Manmohan *et al* study[4], 21.5% of the patients had dyslipidaemia, which is similar to the current study.

Obesity is an independent risk factor for stroke[17]. In this study, 11 patients had obesity while the Manmohan *et al* study[4] had 10% patients with obesity. Inclusion of paediatric strokes may have resulted in this difference as obesity is seen more commonly in adults in Indian population when compared to the Western world where paediatric obesity is significantly present.

In our study, 5 patients had ischaemic heart disease, which

is known to contribute to stroke by various pathophysiological means. It causes endo-myocardial ischaemia which alters surface adhesion process and causes decreased contractility, due to myocardial ischaemia[18]. These factors provide an environment for formation of microthrombi in the intramural surface. Ischaemic heart disease causing atrial fibrillation, leads to an intra-atrial clot which embolises and causes stroke[19]. But coronary artery disease and stroke are more of an association rather than risk factors for each other. In the Manmohan *et al* study, a higher prevalence of ischaemic heart disease was seen at 22.5% when compared to the current study. This can be due to epidemiological variation occurring with different study populations, as stroke is associated with numerous risk factors and confounders.

In the study we conducted, 21 patients were smokers. Stroke – first presentation and recurrent stroke, both are associated with smoking. Vessel elasticity and compliance is altered in smokers[20]. Smoking raises haematocrit, which increases blood viscosity. It causes damage to the endothelial lining, which stimulates platelet aggregation and hence encourages thrombosis. The persistent endothelial inflammation induced by smoking and secondary polycythaemia predispose chronic smokers to recurrent CVA. 31% patients were chronic smokers in the Manmohan *et al* study[4] and 34% patients in the Joshi *et al* study[8]; there is a difference which was attributed to socio-demographic variations. 2 patients in the current study had secondary polycythaemia (in known chronic smokers).

Alcoholism is the next factor in substance abuse that is an established risk factor for CVA. In this study, 7 patients were diagnosed with alcohol abuse as per alcohol use disorder criteria[18]. Alcohol in moderation (1 - 2 drinks/ week) was associated with decreased hazard ratio for stroke. At higher levels of consumption, alcohol cases were positively correlated with stroke incidence. Direct correlation between alcohol consumption and stroke is not known. 6.25% patients in the Manmohan *et al* study were diagnosed as alcohol abusers as per CAGE criteria. The data are similar between the current study and the Manmohan *et al* study.

In our study, 3 patients were diagnosed as chronic kidney disease. A higher risk of ischaemic and haemorrhagic stroke is linked to chronic kidney disease (CKD) as it can lead to platelet dysfunction, coagulation abnormality, endothelial dysfunction, and inflammation in addition to common risk factors like hypertension caused by reno-parenchymal disease[21]. In the Manmohan *et al* study[4], 3.75% of the patients had CKD. This difference is due to the increased prevalence of diabetes in the current study.

1 patient in the current study had anti-phospholipid antibody (APLA) syndrome – a 20-year-old female with recurrent abortions presenting with features of cerebellar CVA. APLA although rare, is a significant risk factor for CVA. APLA leads to formation of anti-phospholipid antibodies like lupus anticoagulant and anticardiolipin antibody that causes decreased formation of prostacyclin. This process causes increased aggregation of platelets leading to plaque or thrombus, hence causing endothelial injury.

Symptomatology

Most common presentation was with features of acute vestibular syndrome, presenting as sudden onset ataxia in 26 and vertigo in 25 patients. In PCS, stroke involving cerebellar peduncles, cerebellum and vestibular nuclei causes ataxia with vertigo[22]. It was also the predominant complaint in the Manmohan *et al*[4] and Joshi *et al*[8] studies. It was noted that in this study, 9 patients were primarily evaluated under ENT for peripheral vertigo and later referred to medicine for further evaluation of acute vestibular syndrome, which was further diagnosed as PCS. This delay in diagnosis and gap in knowledge in PCS presentation leads to delayed initiation of anti-thrombotic agents and poorer neurological outcomes. Headache with nausea/vomiting was the second most predominant complaint in PCS in our study. Headache was predominantly posterior, characterised by a dull aching sensation. This occurs due to irritation of trigeminovascular neurons located in brainstem arteries[23]. If headache is present in acute vestibular syndrome presentation, PCS must be ruled out. The other differential diagnosis for such a presentation is vestibular migraine, a rare entity. In PCS, nausea/vomiting are attributed to 2 mechanisms. First is involvement of vestibular nuclei or fasciculus, this is usually accompanied with vertigo. Second mechanism is involvement of CTZ vascular supply, where no significant vertigo or ataxia is seen[24]. Dysarthria was the next common symptom of presentation in the current study, observed in 10 subjects. It was noted in facial nerve nuclear/ fascicular lesions and cerebellar lesions. 10 patients also had altered sensorium at presentation, which can be attributed to involvement of RAS (reticular activating system) centre in brainstem strokes. Hemiparesis was present in 9 patients. It was mainly seen in brainstem strokes involving basal pons and pyramids of medulla. 1 case of hemiparesis was noted in midbrain stroke as a part of Weber's syndrome. In the Manmohan *et al* study, the predominant presenting symptomatology was vertigo and ataxia in 56% and 48% of the patients respectively. It was followed by vomiting and headache in 33% and 25% of the patients. In the Joshi *et al* study, 70% patients had vertigo and 62% patients had ataxia, followed by 58% patients with headache and 54% patients with vomiting. These findings are similar to the current study.

Anatomical localisation

All the studies had localised infarcts with MRI Brain-DWI sequence and ICH by NCCT brain imaging. 40 patients had ischaemic lesions in the current study. Joshi *et al* study showed increased haemorrhagic strokes in 37% (n = 15) patients. In this study, 4 patients with haemorrhagic stroke were discharged against medical advice, therefore they could not be included in the analysis. This might have led to decreased representation of haemorrhagic strokes in our study. Our study showed predominance of cerebellar strokes(19 cases). In PCS studies, cerebellar strokes were predominant, but the exact mechanism of this predominance remains unknown. Possible hypotheses include branches of multiple arteries from the posterior circulation supply various parts of cerebellum and involvement of these gives a higher chance of involvement of cerebellum. In the Joshi *et al* study, cerebellum was predominantly involved at 37.5% of all strokes. Occipital lobe involvement was the second most common stroke in the present study seen in 14 patients. They most commonly present with headache. In the Joshi *et al* study, 24% of the patients had occipital strokes, which is comparable and similar to our study. 12 patients had medullary involvement in the present study. Among them 8 patients had "lateral medullary syndromes" with 4 of them having hemiparesis or extension into pyramids causing "Babinski-Naegotte Syndrome". 1 patient had "hemi-medullary syndrome" involving complete left half of the medulla. 9 patients presented with pontine lesions in the present study. 2 of them had pontine haemorrhage with others having ischaemic lesions. Most common presentation was with 7th LMN cranial nerve involvement and hemiparesis as a part of multi-infarct status. 1 patient with pontine lacunar infarct presented with "Dysarthria-Clumsy Hand syndrome". 1 patient with pontine haemorrhage was diagnosed as "Locked-in Syndrome" presented with quadriparesis with preserved vertical eye movements and loss of other conjugate vision. 6 patients had midbrain strokes. 2 patients presented with involvement of 3rd cranial nerve nuclei causing ptosis with one patient being diagnosed as Weber's Syndrome. A total of 53.5% (23) of the patients had brainstem involvement in the present study and 23 patients also had multi-infarct presentation due to artery-to-artery embolisation (in large artery disease) or by cardio-embolic phenomena.

Artery involved

CT angiography was used for diagnosis of arterial involvement, measure of degree of occlusion and status of the plaque. Occlusions of >50% were considered as clinically significant. In this study, vertebral artery was predominantly involved, in 12 patients. The tortuous extracranial course of the vertebral artery along with commonly occurring hypoplasia predispose them for endothelial damage and large artery disease in older adults. Our study had a higher representation of older adults, hence showed a higher predominance for vertebral artery involvement. This is followed by PCA in 6 patients. However, in the Manmohan *et al* study, 53% patients had involvement of PCA with only 2.5% of the patients showing vertebral artery involvement. In the Manmohan *et al* study, extracranial vertebral artery lesions were excluded. As extracranial course is predominantly involved, the Manmohan *et al* study showed significantly low incidence of vertebral artery involvement.

TOAST Classification

Our study had predominantly small artery disease/lacunar strokes in 14 patients, followed by large artery disease in 11. In the Manmohan *et al* study, majority of the patients had large artery disease in 61% followed by cardio-embolic phenomena in 10%. Higher age is associated with small artery disease due arterial decreased compliance that progresses with age. This can be noted, as predominant small vessel disease was observed. New England PCS registry[11] showed a predominance of large artery disease.

Outcome

Mortality due to PCS were similar in the present study compared to national and international studies. Predominant causes of death were secondary hospital acquired infections like ventilator associated pneumonia in 4 patients. 1 patient suffered concomitant myocardial infarction followed by cardiogenic shock. 1 patient with cerebellar bleed who had delayed presentation to hospital, had features of brainstem herniation on CT imaging, and eventually led to cardiopulmonary compromise. Posterior circulation infarcts have been generally considered to have a poor outcome with high mortality and morbidity[25], but several studies have conflicting results. The comparative functional outcome of patients with posterior *versus* anterior circulation stroke has been little studied, especially that of the subset of patients with minor initial deficits. The current evidence suggests that minor PCS exhibited more frequent disability at 3 months than minor ACS. Especially, the presence of vertebrobasilar large vessel disease in minor PCS had a substantially higher risk of disability when compared to anterior circulation strokes[24].

Conclusion

Diabetes Mellitus was the most common risk factor for PCS in our study followed by hypertension. Ataxia and vertigo, with clinical features of "Acute Vestibular Syndrome" were

the most common presenting features of PCS in the present study. Cerebellum was the most common anatomical area involved. Vertebral artery was most commonly affected and small vessel disease was the main aetiological factor.

Limitations

Our study had a relatively smaller sample size compared to other studies. Large number of PCS had prior or concomitant anterior circulation involvement, hence could not be included as per our criteria. Hence, few variables like cardioembolic phenomenon, large artery disease could not be assessed adequately and might have affected the results. In the COVID pandemic, subtle or self-limiting acute vestibular syndromes may not have been diagnosed adequately or may not have sought healthcare.

References

1. Vasculature of the Central Nervous System and the Cerebrospinal Fluid. In: Martin JH. eds. Neuroanatomy: Text and Atlas, 5e. McGraw Hill; 2021. Accessed October 16, 2023.
2. Kim JT, Park MS, Choi KH *et al*. Clinical outcomes of posterior versus anterior circulation infarction with low national institutes of health stroke scale scores. *Stroke* 2017; 48 (1): 55-62.
3. Tao WD, Liu M, Fisher M *et al*. Posterior versus anterior circulation infarction: how different are the neurological deficits? *Stroke* 2012; 43 (8): 2060-5.
4. Mehndiratta M, Pandey S, Nayak R. Posterior circulation ischaemic stroke-clinical characteristics, risk factors, and subtypes in a north Indian population: a prospective study. *Neurohospitalist* 2012; 2 (2): 46-50.
5. Kim H, Kim JT, Lee JS *et al*. Stroke of Other Determined Aetiology: Results From the Nationwide Multicenter Stroke Registry. *Stroke* 2022; 53 (8): 2597-2606.
6. Ng AC. Posterior Circulation Ischaemic Stroke. *Am J Med Sci*. 2022; 363 (5): 388-98.
7. Shuaib A, Hachinski VC. Mechanisms and management of stroke in the elderly. *CMAJ* 1991; 145 (5): 433-43.
8. Rawat KJ, Korde BS, Joshi KS. Clinical profile and prognosis of patients with posterior circulation stroke. *Inter J Res Med Sci* 2016; 4 (12): 5159-64.
9. Haast RA, Gustafson DR, Kiliaan AJ. Sex differences in stroke. *J Cereb Blood Flow Metab* 2012; 32 (12): 2100-7.
10. Bhadra J, Seth S, Kulshrestha M *et al*. Testosterone and estradiol in men with acute ischaemic stroke: A North Indian case control. *Curr J Neurol* 2021; 20 (4): 202-7.
11. Caplan L, Chung CS, Wityk R *et al*. New England medical center posterior circulation stroke registry: I. Methods, data base, distribution of brain lesions, stroke mechanisms, and outcomes. *J Clin Neurol* 2005; 1 (1): 14-30.
12. Tun NN, Arunagirinathan G, Munshi SK. Diabetes mellitus and stroke: A clinical update. *World J Diabetes* 2017; 8 (6): 235-48.
13. Kuriakose D, Xiao Z. Pathophysiology and Treatment of Stroke: Present Status and Future Perspectives. *Int J Mol Sci* 2020; 21 (20): 7609.
14. Valham F, Mooe T, Rabben T *et al*. Increased risk of stroke in patients with coronary artery disease and sleep apnea: a 10-year follow-up. *Circulation* 2008; 118 (9): 955-60.
15. Dong H, Chen W, Wang X *et al*. Apolipoprotein A1, B levels, and their ratio and the risk of a first stroke: a meta-analysis and case-control study. *Metab Brain Dis* 2015; 30 (6): 1319-30.
16. Deng Q, Li S, Zhang H *et al*. Association of serum lipids with clinical outcome in acute ischaemic stroke: A systematic review and meta-analysis. *J Clin Neurosci* 2019; 59: 236-44.
17. Vicente VS, Cabral NL, Nagel V *et al*. Prevalence of obesity among stroke patients in five Brazilian cities: a cross-sectional study. *Arq Neuropsiquiatr* 2018; 76 (6): 367-72.
18. Kranzler HR, Soyka M. Diagnosis and Pharmacotherapy of Alcohol Use Disorder: A Review. *JAMA* 2018; 320 (8): 815-24.
19. Smith WS, Johnston S, Hemphill III J. Ischaemic Stroke. In: Loscalzo J, Fauci A, Kasper D *et al*. eds. *Harrison's Principles of Internal Medicine*, 21e. McGraw Hill; 2022.
20. Kool MJ, Hoeks AP, Struijker Boudier HA *et al*. Short- and long-term effects of smoking on arterial wall properties in habitual smokers. *J Am Coll Cardiol* 1993; 22 (7): 1881-6.
21. El Husseini N, Kaskar O, Goldstein LB. Chronic kidney disease and stroke. *Adv Chronic Kidney Dis* 2014; 21 (6): 500-8.
22. Choi JH, Kim HW, Choi KD *et al*. Isolated vestibular syndrome in posterior circulation stroke: Frequency and involved structures. *Neurol Clin Pract* 2014; 4 (5): 410-8.
23. Vestergaard K, Andersen G, Nielsen MI. Headache in stroke. *Stroke* 1993; 24 (11): 1621-4.
24. Stroke and Cerebrovascular Diseases. In: Ropper AH, Samuels MA, Klein JP, Prasad S. eds. *Adams and Victor's Principles of Neurology*, 11e. McGraw Hill; 2019.
25. Kim JT, Park MS, Choi KH *et al*. Clinical outcomes of posterior versus anterior circulation infarction with low national institutes of health stroke scale scores. *Stroke* 2017; 48 (1): 55-62.