Section: Anatomy

Research

A STUDY OF HAEMATOLOGICAL VALUES IN CEREBROVASCULAR ACCIDENTS

Dandangi Taraka Lakshmi[1], Mudduluru. Revathi[2], Ragam Ravi Sunder[3], Barika Sireesha[4]

[1]Assistant Professor, Department of Physiology, Rangaraya Medical College, Kakinada, India.
[2]Associate Professor, Department of Physiology, Andhra Medical College, Visakhapatnam, India.
[3]Professor & HOD, Department of Physiology, GITAM Institute of Medical Sciences & Research, Visakhapatnam, India.
[4]Assistant Professor, Department of Anatomy, Government Medical College, Vizianagaram, India.

Received : 16/12/2022
Received in revised form : 19/01/2023
Accepted : 30/01/2023

Keywords:
Cerebrovascular accidents, haemodynamic ischaemic insult, hematological disorders.

Corresponding Author:
Dr. Barika Sireesha
Email: sireeshabarika@gmail.com
ORCID: 0000-0003-2065-8072

DOI: 10.47009/jamp.2022.5.1.160

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2023; 5 (1); 766-771

Abstract

Background: Stroke is defined as a focal Neurological deficit of sudden onset due to non-traumatic vascular lesion. Stroke rank first in frequency and important among all the neurological disorders of Adult life. It is the most frequent cause of death of neurological origin. It is the third leading cause of death after heart disease and cancer in developed countries. **Objectives**: To study the physiological variants of clinical importance by examining the blood for complete blood picture, platelet count, ESR and blood indices. **Materials and Methods:** Hospital based observational study. Study area: Department of Physiology and General Medicine, Andhra Medical college, Visakhapatnam. Study Period: April 2021 – March 2022. Study population: Patients admitted in acute Medical care unit who met the diagnostic criteria of acute stroke and proved after CT scan were taken up for study. Sample size: study consisted a total of 50 cases. Sampling method: Simple random method. Study tools and Data collection procedure: Patients admitted with an acute stroke for first time were assessed by a detailed clinical history to detect the risk factors (blood pressure, diabetes, smoking and alcohol intake), and physical examinations as outlined in the proforma. The blood of the patient for complete haemogram, Leucocyte count, Differential leucocyte count, packed cell volume, erythrocyte sedimentation rate, platelet count, bleeding and clotting time, blood sugar, and serum cholesterol were taken and sent for the above investigations. **Result:** The age wise distribution of hematocrit among the patients was 38% (young) 39.1% (middle) 37.66% (elderly) in those who had infarction and 40% (young), 39.66% (middle age) 39.99% elderly in those who had haemorrhage. The average MCV in infarct group was 94.90 fl and in haemorrhage group was 87.68 fl with and aggregate average of 92.20 fl. Average MCH was low in female (29.99 Pg) compared to males (30.54 pg). **Conclusion:** In particular, with a history of cerebral ischemia event, it was determined that haematological characteristics may be able to affect cerebral microcirculation. Individuals with a previous cerebral ischemic stroke may have continuously changed haematological markers. Events suggest that this haematological parameter might be interpreted as a straightforward inflammatory response that takes place as ischemia damage develops.

INTRODUCTION

Stroke is defined as a focal Neurological deficit of sudden onset due to non-traumatic vascular lesion. Stroke rank first in frequency and important among all the neurological disorders of Adult life. It is the most frequent cause of death of neurological origin. It is the third leading cause of death after heart disease and cancer in developed countries. It presents as mild weakness to coma. Etiology ranges from primary abnormality in the vessels to hematological disorders. Multiple risk factors are involved in the pathogenesis. There is unpredictable prognosis especially in strokes due to hemorrhage and in advanced age group. Almost all elderly people have blockage of some small arteries in the brain, and has many as 10% eventually have enough blockage to cause serious disturbance of brain function a condition called stroke. Normal functions of the brain are dependent upon a constant

ISSN (O): 2687-5365; ISSN (P): 2753-6556

supply of oxygen and glucose, as well as nutrients derived from the blood perfusing it (55 to 70 ml blood per 100 g of brain per min). The principal source of energy is almost exclusively oxidation of glucose. If for any reason the blood flow is critically reduced below 15 ml per 100 g per min, the resulting ischemia with hypoxia, when sufficiently prolonged, may cause death of neurons and glial infarction). The mean arterial blood pressure, cerebrovascular resistance, local metabolic products (PH, PaO2 PaCO2 etc) together with several known and unknown factors help to maintain the critical threshold of blood flow for brain metabolism. Furthermore, the blood flow varies in different areas of the brain and a self-regulatory mechanism (autoregulation) determines the regional flow to meet local metabolic needs. For example, with an increase in partial pressure of CO2, the arterioles dilate to increase blood flow. The precise role of vasoconstrictor (sympathetic) and vasodilator nerve impulses in the regulation of vascular tone and local blood flow is much debated, but circulating neurochemical transmitters (serotonin, catecholamines etc.) do modify the local needs. To protect the brain from such haemodynamic ischaemic insult, nature has provided several collateral pathways. The four major extracranial arteries (one pair each of carotid and vertebral arteries) carrying oxygenated blood from the aortic arch, form at the base of the brain a good caliber, low resistant cranial anastomosis (the circle of Willis). In addition, extracranial anastomosis between the cervical branches of the ipsilateral external carotid, subclavian and vertebral arteries have also been identified. These arterial anastomoses help to maintain cerebral blood supply in individuals even with occlusion of a major artery in the neck. Likewise, large precapillary anastomosis exists between the anterior, middle and posterior cerebral arteries and various cerbellar arteries. Such post-willisian anastomoses further protect cerebral tissue from the effects of occlusion of single cortical branches. Thus, in an individual with symmetrical circle of willis, despite major extracranial arterial occlusions, sufficient blood may still reach the territory through the collateral pathways to prevent on coming cerebral ischaemic insult. On the other hand, in the presence of generalized arterial disease (atherosclerosis), congenital variations and multiple skipped stenotic lesions, these collateral pathways may prove inadequate in maintaining normal blood flow and predispose to cerebral ischaemia or infarction. If for any reason, such as cardiac arrest of prolonged hypotension (systolic BP below 70 mmHg). the brain tissue is significantly deprived of its nutrition for more than three minutes, death of brain parenchyma (cerebral infarction) ensues. Such cerebral infarcts are either pale (ischaemic infarction in thrombosis) or may show petechial haemorrhages in the cortical mantle (haemorrhagic infarction in embolism). Hence the present study was undertaken to study the hematological parameters in the cerebrovascular accident patients.

Objectives

To study the physiological variants of clinical importance by examining the blood for complete blood picture, platelet count, ESR and blood indices.

MATERIALS AND METHODS

Study Design: Hospital based observational study.
Study area: Department of Physiology and General Medicine, Andhra Medical college, Visakhapatnam.
Study Period: April 2021 – March 2022.
Study population: Patients admitted in acute Medical care unit who met the diagnostic criteria of acute stroke and proved after CT scan were taken up for study.
Sample size: study consisted a total of 50 cases.
Sampling method: Simple random method.
Inclusion criteria: All patients with a clinical diagnosis of acute cerebrovascular stroke with obvious vascular cause were taken up for study.
Exclusion criteria:
Patients who are on antiplatelet agents excluded from the study.
Patients who had second stroke were stroke were also not included.
Ethical consideration: Institutional Ethical committee permission was taken prior to the commencement of the study.
Study tools and Data collection procedure:
Patients admitted with an acute stroke for first time were assessed by a detailed clinical history to detect the risk factors (blood pressure, diabetes, smoking and alcohol intake), and physical examinations as outlined in the proforma. The blood of the patient for complete haemogram, Leucocyte count, Differential leucocyte count, packed cell volume, erythrocyte sedimentation rate, platelet count, bleeding and clotting time, blood sugar, and serum cholesterol were taken and sent for the above investigations.

Statistical Analysis

Once all the observations had been recorded, the data collected was transferred to a master chart and analyzed. Data analyzed by using SPSS version 20. The association between different parameters analyzed with Chi-square test. Percentage and frequencies of patients was compared using Chi-square. A P-value <0.05 is considered statistically significant.

ISSN (O): 2687-5365; ISSN (P): 2753-6556

RESULTS

Table 1: Showing age wise Distribution.

Age group in years	No of Subjects
21 – 30	4
31 – 40	6
41 – 50	13
51 – 60	16
61 – 70	6
71 – 80	5
Total	50

The age of the patients ranged from 30 – 80 years.

Table 2: Showing Sex Wise Distribution.

Subjects Studied	50
Males	32
Females	18

Of the 50 pts there were 32 males & 18 females with a male female ratio of 1.8: 1.

Table 3: Showing age wise incidence of hemorrhage & infarction.

Age group in years	Infarction	Haemorrhage
21 -30	1	3
31 – 40	4	2
41 – 50	9	4
51 – 60	11	5
61 – 70	3	3
71 – 80	2	2
Total	30	20

The peak incidence of stroke was seen in fifth decade in both categories of haemorrhage & infarction5 patients (10% of total cases) were 40 years or less in age falling into the category of stroke in young. 29 patients out of 50 were between the age of 41 – 60 making about 58% of all strokes & were strokes in middle age. Out of 50 patients 11 were between the age of 61 & above making 22% of total strokes & fell into the group of strokes in elderly. Of the 50 patients 30 (60%) had cerebral infarction & 20 (40%) had cerebral haemorrhage.

There were 22 (67%) males & 8 (25%) Females in infarction group & 11 (55%) & 9 (45%) in haemorrhage groups. 30 (60%) persons gave history of hypertension in the past & were on treatment, apart from the 10 more patients were recorded a high blood pressure (>140/90) at the time of admission making a cumulative tally of 40 (80%) patients with hypertension. Out of 40 cases of hypertension 30 (75%) were in the infarction group & 10 (25%) were in the Haemorrhage. 18 patients gave history or diabetes Mellitus, & 12 more had blood sugar more than 140 mg% at the time of admission making a total of 30 (60%) of all cases. Out of which 20 (66.6%) had infarction & 10 (33.3%) had Haemorrhage.

Table 4: Showing cases who revealed a laboratory evidence of Hypercholesterolemia.

Infarction	15 (53.5%)
Haemorrhage	13 (46.4%)
Total	28

Hyper cholesterolemia (Serum Cholesterol > 250 mg) was detected in 28 (56%) patients out of which 15 (53.5%) had infarction & 13 (46.4%) had Haemorrhage. Out of 50 patients 25 (50%) patients were in the habit of regular smoking at a frequency of 5 – 25 Cigarettes. Out of 25 patients 20 patients (80%) & 5 patients (5%) had Haemorrhage. Out of the 25 patients with smoking habit 16 patients were hypertensive & 9 patients revealed a raised serum cholesterol level. 16 Patients (32%) out of the total cases were alcoholics. Out of them 7 (43.7%) were in infarction group & 9 (56.25%) were in Haemorrhage group. Out of the 16 Alcoholics 11 had Hypertension & 5 had Hypercholesterolemia. Out of the 11 Hypertensives 5 developed infarct & 6 developed Haemorrhage & out of 7 Hypercholesterolemia 3 had Haemorrhage & 2 had infarct. Out of the 32 daibetics, 21 had associated Hypertension & 11 had hypercholesterolemia. Of the 21 hypertensives 12 had infarct & 9 had Haemorrhage. Of the 11 having Hypercholesterolemia 8 had infarcts 3 had Haemorrhage.

Table 5: Showing Hb concentration in relation with infarction & Haemorrhage

Patient Groups	Hb gm/dl
Infarct	11.15 gm/dl
Haemorrhage	9.80 gm/dl

$t = 14.96$ $p < 0.05$

ISSN (O): 2687-5365; ISSN (P): 2753-6556

The average Hb concentration in patients with infarct was 11.15 gm / dl & in Haemorrhage cases was 9.8 gm / dl. Age wise distribution of Hb among the patients was 12.31 gm/dl in young age (20-40 years), 10.82 gm/dl in middle age (41-60 years) and 9.74 gm/dl in elderly (>60 years) with gradual decrease in Hb % as a age advanced.

Table 6: Showing PCV in hemorrhage & infarct groups.

Infarction	**40.56%**
Haemorrhage	38.26
Average	35.95

X2= 3.92 $p < 0.05$

Average packed cell volume of all patients was 35.95% with 56% infarct group & 38.26% in haemorrhage group.

Table 7: Showing age wise distribution of haematocrit in both groups.

Age in years	**Infarction**	**Hemorrhage**
20-40 years	38%	40%
41-60years	39%	39.66%
>61 years	37.66%	39.99

X2= 4.62 $p > 0.1$

The age wise distribution of hematocrit among the patients was 38% (young) 39.1% (middle) 37.66% (elderly) in those who had infarction and 40% (young), 39.66% (middle age) 39.99% elderly in those who had haemorrhage. The average MCV in infarct group was 94.90 fl and in haemorrhage group was 87.68 fl with and aggregate average of 92.20 fl. Average MCH was low in female (29.99 Pg) compared to males (30.54 pg).

There was a gradual decrement of MCH as age advances (30pg) young, (29.91pg) middle and (28.1pg) in elderly in those having infarction and 30.41 (young), 29.0 (middle) and 29 (elderly) in haemorrhage group.

There was slight variation in MCHC in males (32.85gm/dl) when compared to females (32.39gm/dl). Smokers has a slight excess MCV (90.44fl) when compared to non-smokers (85.72fl) & alcoholics revealed still more MCV (95.66fl) & alcoholics who concomitantly smoked had MCV of (99.34fl).

Table 8: Showing age wise distribution of total leucocytes counts in stroke Patients

Age group	**Infarct**	**Hemorrhage**
20 – 40 yrs	1700/Cumm	13600/Cumm
41 – 60	11650/Cumm	14100/Cumm
>60	10400/Cumm	14600/Cumm

Females with stroke had a slight excess of leucocytes (12400/Cumm) when compared with males (11980/Cumm). A significant leucocytosis was noted in persons who had hemorrhage in all age groups. (13,600/Cumm in young, 14100 in middle age, 14600 in elderly) as compared to infarction (1700 in young, 11650 in middle age, 10400 in elderly). Females had a slight neutrophilia (80%) compared to males (77%) & Neutrophilia was a dominant feature in cases of Hemorrhage (85%) as compared to infarct (75%).

Table 9: Showing sex wise & lesion wise comparison of platelet count.

Males	**3.20 Lakhs/Cumm**
Females	3.55 Lakhs/Cumm
Infarct	3.45 Lakhs/Cumm
Hemorrhage	2.95 Lakhs/Cumm

Platelet count was more in females (3.55 lakhs/Cumm) than in males (3.20 lakhs/Cumm), there was a significant thrombocytosis (3.45 lakhs/Cumm) in infarct group when compared to hemorrhage (2.95lakhs / Cumm).

Females had more ESR (25.1 mm/1st hour) compared to males (23.73 mm/1st hour) though the difference was not much. Bleeding time was more (5.1min) in hemorrhage group than in infarct group (4.3 min). Clotting time showed no much change although it was more in hemorrhage cases (5.45 min) than in infarct cases (4.96 min**).**

DISCUSSION

Cerebrovascular diseases have been studied by a number of People extensively in various aspects & still there are debatable questions like in cerebral auto regulations. We had attempted to identify factors predisposing to stroke in patients getting admitted with evidence of stroke in acute medical care unit with special attention towards blood indices. 29 strokes out of 50 developed in middle aged and elderly constituting about 58% of total strokes. Hence age itself is a major risk factor leading to development of acute cerebrovascular event. Shaper AG et al.[1] in their study concluded that about half of the first stroke occurs in people aged 76 yrs & above. The changes that do occur with ageing include a slow, apparently continuous, symmetric increase in the thickness of intima due to

ISSN (O): 2687-5365; ISSN (P): 2753-6556

accumulation of smooth cells probably from the media, leading to loss of elasticity. And these vessels fail to withstand hydrostatic pressure. The unsupported cerebral arteries may be particularly vulnerable in this regard leading to occurrence of stroke in elderly people. In this study 40 (80%) of the total 50 cases were detected to be hypertensives making the first & the leading cause of stroke in all age groups. The mean arterial systolic & diastolic blood pressure was comparatively high in hemorrhage cases as compared with patients with infarction. The role of high blood pressure is not only a risk factor, but also a treatable aetiological factor in preventing development of stroke.[2,3] Elevated B.P. as a precursor of a stroke has been an almost universal finding in all the prospective studies.[4,5,6] The raised systolic B.P can readily be detected & reduction of which reduced the incidence of strokes.[7] A large study has emphasized the superiority of systolic B.P. as indicator of risk over diastolic B.P.[8,9] In our study there were 30 patients (60%) who were diabetic out of which 20 (66.6%) had infarction & 10 (33.3%) had hemorrhage. Consolidating the view point of previous studies, in a variety of population have shown an association of hyperglycemia with clinically evident atherosclerotic disease, suggesting a role of hyperglycemia in atherosclerosis. There is increased tendency towards cerebral thrombosis & infarction but not towards cerebral hemorrhage in diabetics. In our study smokers accounted 25 (50%) & 20 (60%) among smokers had ischaemic stroke & only 5 (20%) had hemorrhage, indicating that smokers are at the risk of developing ischeamic stroke than hemorrhagic stroke. Robert. D. Abbolt et al.[10] in their study came with a conclusion that was compared with nonsmokers. Cigarettes smokers had 2 – 3 times risk of thromboembolic or hemorrhagic stroke, & stopping smoking showed significant benefits. Cigarrette smoking increases the risk of stroke by about 3 folds.[11] It may be because of an increase in the Hemoglobin Concentration & raised hematocrit as it is evident by the study of Ehrly et al.[12] In my study I could come across 28 (56%) patients who revealed a serum cholesterol > 250 mg% at admission, among which 15 (53.5%) had infarction which is supported by studies in which men screened for the multiple risk factor intervention trial, the risk of death from thrombosis stroke increased significantly with increased cholesterol concentration. For cerebral hemorrhage the death rate was highest in those with lower blood cholesterol level. In my study the packed cell volume was low in females & it stills very low in females who had hemorrhage. Smokers had a comparatively more PCV (50.15) when compared to nonsmokers (25.10). Elevated hematocrit or HB level were considered to be a well-documented risk factor in the review by Dyken et al.[13,14] certain other studies have found no association between the hematocrit & risk of stroke among men.[15] & infarct a negative association has been found among women, those with low hematocrit had the highest risk of stroke. The hematocrit hemoglobin concentration was found to be high in person who indulge in habit of smoking.[16] PCV is among the factors that may cause a rise in blood viscosity, which may affect microcirculation more seriously.[17] leading to development of infarction. The risk factor of elevated hematocrit can be eliminated within a week of stopping smoking.[18] In the present study MCV gradually increased with age of person & smokers & alcoholics had an increased erythrocyte size than nonsmokers. In the present study women showed a slight raised leucocyte count & the leucocytes were found to be highest in hemorrhagic stroke, with neutrophilia. Due to any variety of vascular injury to nervous system there occurs a leucocytic response and by 24-48 hrs there is a variable neutrophil infiltration marked by peripheral neutrophilic leukocytosis.[19] The magnitude of leucocytic response depends upon the severity of vascular insult & leucocytosis is more pronounced in cerebral haemorrhage than infarction. It is also reported that the leucocyte counts gradually increase with age & intensity of smoking. In the present study the platelets count was more in females compared to males and a significant finding suggesting an overall low platelet count although in normal limits was present in patient who had hemorrhagic stroke. Stevan RF et al.[21] through a study of platelet count came to a conclusion that a higher mean count exists in woman compared to men. Altered platelet number, Metabolism and function leads to an impairment in coagulation process, that could increase the trauma of cerebral hemorrhage. In the present study BT & CT were found to be comparatively prolonged in hemorrhagic stroke when compared with non-hemorrhagic stroke which probably may be due to activation of fibrinolytic system.[22]

CONCLUSION

In particular, with a history of cerebral ischemia event, it was determined that haematological characteristics may be able to affect cerebral microcirculation. Individuals with a previous cerebral ischemic stroke may have continuously changed haematological markers. Events suggest that this haematological parameter might be interpreted as a straightforward inflammatory response that takes place as ischemia damage develops.

REFERENCES

1. SHAPER AG, PHILLIPS an, POCOCK Sj, WALKER M. MACFARLANE PW Risk factors for stroke in middle aged men. BMJ 1991; 302; 111-1115.
2. Hypertension detection and follow up programme co-operative group. Five year findings of the hypertension detection follow up programme III. Reduction in stroke

ISSN (O): 2687-5365; ISSN (P): 2753-6556

incidence among persons with high blood pressure. JAMA 1982; 247: 633-38.
3. THE AUSTRALIAN THE RAPEUTIC TRIAL in Mild hypertension report by management committee LANCET 1980; 1: 1261-67.
4. GRIGOLEIT HG, LEONARD H, JACOB1 G.Mikrorheologische untersuchungen an Gesunden Dtsch Med Wochenschr 1978; 103: 329-34.
5. ISBEN H, CHRISTENSEN NJ, RASMUSSEN S, HOLNOGAC H, NIELSEN MD> Influence of alcohol intake on Blood pressure, plasma nor-adrenalin concentration, and plasma rennin concentration. Cli Sci 1981; 61: 377.
6. STEVENS RF, ALEXANDER MK. A sex difference in platelet count. Indian J. Haematol 1977; 37: 295-300.
7. KULLER LH. Epidemiology of stroke. Adv Neurol 1978;296 1194- 2000.
8. MENOTTE A et al: The predictive role of systolic, diastolic and mean blood pressures on all causes of death. J.Hypertens 1989: 7 : 595-9.
9. SIMON RABKIN W, FRANCIS AL, MATHEWSON et al: Predicting risk of ischemic heart disease and cerebrovascular disease from systolic and diastolic blood pressures. Ann int. Med 1978; 88: 342- 45.
10. ROBERT D. ABBOTT, YIN YIN, DWAYNE et al: Risk of stroke in male cigarette Smokers. NEJM 1986; 315: 717 – 20.
11. BLACKWELDER WC, YANO K, RHOADS GG, KAGAN A, GORDAN T, PALESH Y. Alcohol and mortality: The Honolulu Heart study. Am J Med 1980; 68. 165 – 9.
12. EHRLY AM, SCHIMPF WJ et al: 1978 : 10: 245 – 6.
13. DYKEN ML, WOLF PA, BARNETT HMJ et al. Risk factors in stroke a statement for physicians by subcommittee on risk factors and stroke o the stroke council. Stroke 1984; 15: 1105 – 11.
14. KANNEL WB, WOLF PA, VERTER J. Mainfestations of coronary disease predisposing to stroke. The Framingham Study. JAMA 1983; 250: 2942-46.
15. KIYOHARA Y, UADA K, HASUO Y et al: Hematocrit as a risk factor for cerebral infarction. Stroke 1986; 75: 687- 92.
16. GALEA G, DAVIDSON RJL. Haematological and Hemorrheological changes associated with cigarette smoking. J.Clin Pathol 1985; 38: 978-84.
17. WELLS R, Syndromes of hyperviscosity. NEJM 1970; 283; 183-86.
18. EISEN ME, HAMMOND EC, Effect of smoking on packed cell volume, red cell counts, haemoglobin and platelet counts. Can Med Assoc J 1956; 75: 520-23.
19. JAMES H MORRIS. Vascular disease. The nervous system – Basic pathology – Robbins – Kotran – Kumar 1992; 5th ed: 713-18.
20. FRIEDMAN GD, SIEGELAUB AB, SELTZER CC et al: Smoking habits and leucocyte count. Arch Environ Health. 1973; 26: 137 – 43.
21. GILL JS, ZEZULKO AV, SHIPLEY MJ, GILL SK, BEEVERS DG. Stroke and alcohol consumption NEJM 1986; 315: 1041-46.
22. WILLUM E GANONG Review of Medical physiology 18th ed. 1997. 506.

ISSN (O): 2687-5365; ISSN (P): 2753-6556