Original Article

ROLE OF SMOKING AND ALCOHOLISM IN CEREBROVASCULAR ACCIDENTS

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ABSTRACT

Though previously stroke was considered an old man disease, it is increasing among the young and middle aged people are not likely to die of stroke as they are of heart attack, but they are likely to be disabled for rest of their lives.

As the cases of stroke admitted to medical wards, formed nearly 1/10th to 1/8th of total bed strength at any time, and their prolonged stay at hospital attracted our attention. To study the etiological factors for the present study we have thoroughly evaluated 50 pts, with acute cerebrovascular disease and focused more attention in the revaluation of physiological variants of clinical importance by examining the blood for complete blood picture, which may help a long way in near future to predict the risk group in developing an acute cerebrovascular stroke and to avoid risk factors and to take measures to rectify the variations so as to minimize the mortality due to stroke.

KEYWORDS: Cerebrovascular Accidents, Stroke, Mortality, Heart Attack.

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INTRODUCTION

Many studies revealed a close relation between smoking and alcoholism with stroke. Carbon monoxide inhalation produces an increase in the alveolar to arterial oxygen gradient on combining with hemoglobin Carbon monoxide causes a functional anemia by decreasing the amount of Hb available for conveying oxygen.[1]

Strokes rank foremost of all the disorder of CNS. If the blood flow to the brain is reduced below is 15 ml./100 gm/min the resulting ischemia with hypoxia, when sufficiently prolonged may cause death of neurons.[2]

Lipid content mainly cholesterol esters and phospholipids increasing with age in arterial wall. LDL conc. is seen to be increasing with age in arterial wall, increasing rigidity of vessels. The amount of external support also determines the ability of vessels weakened by loss of elasticity to withstand hydrostatic pressure. The unsupported cerebral arteries may be particularly vulnerable in this regard.[3]

Hemo concentration has been attributed to cigarette smoking and was considered a possible explanation of elevated leucocytes counts. Smokers also had higher average hemoglobin conc. Mechanism by which smoking could increase the leucocytes count include nicotine induced release of catecholamines which can raise the leukocyte count and an irritant effect of smoke on the respiratory tree with resultant inflammation.[5] S.GILL reported that among men the relative risk of stroke was lower in light drinkers them in nondrinkers, but was four times higher in heavy drinker than in nondrinkers. Heavy alcohol consumption is an important and under recognized independent risk factor for stroke in men.[6]

R.W.Howell, reported that ESR is about 10% higher in cigarette smokers. Smoke of one cigarette provide enough toxic material to inhabit completely the function of exposed oral leucocytes.[7] Regular use of three or more drinks of alcohol per day is a risk factor for hypertension.[9] Number of leukocytes is increased in smokers notably in those who inhale. The increase is above 30% for heavy smokers who inhale, compared with a non-smoker.[10]

In alcoholics there is abnormal platelet function, prolonged bleeding time, impaired primary and secondary aggregation, reduced Pf_3 availability and subnormal nucleotide release occurred during ethanol ingestion. Ethanol inhibited the aggregation of normal platelets induced by norepinephnine. Ethanol may alter the reactivity of platelet membranes by acting like a local anesthetic since it has an oil water partition co-efficient compatible with such a role and it has been shown to alter membrane current. The life span of platelets is decreased to 50% on more in alcoholics.[12]

MCV and MCH is distinctly higher in smokers, due to impaired deoxy nucleoprotein synthesis. There are more than 1150 different chemicals identified in tobacco smoke to choose from a possible antagonist to this vulnerable process. The cyanide or tobacco smoke is detoxified by path ways involving Vit.B12 depleting the vitamin with subsequent alternate detoxification by production of thiocyanate.

The elevated Hb and hematocrit in smokers could be interpreted at a compensatory response to chronic carbon monoxide induced hypoxia which produces a demand for more erythrocytes.[13] Thus excessive and sustained exposure to carbon monoxide from smoking produces hypoxemia which can intern cause polycythemia.[15] Increased viscosity of blood in smokers cause the velocity gradient to decrease. Also increased attraction of cells to adhere to one another to form rolls of cells, or rouleux, groups of which eventually stick to one another.[19]

(MCV) Macrocytosis is the most typical morphological abnormality induced by excessive ethanol consumption. Acetaldehyde can bind to protein and cellular constituents forming stable addicts, which has adverse consequences of cellular function.[21] The changes in maturation are likely to due to toxic effect of alcohol on nuclear metabolism of bone marrow cells.

AIM:

About 50 patients with acute cerebrovascular disease are thoroughly evaluated for the evidence of risk factors. Focus, was more on revaluation of physiological variants of clinical importance by examining the blood for complete blood picture, platelet count, ESR and blood indices which may help to predict the risk group in developing an acute cerebrovascular stroke and to avoid the risk factors.

MATERIALS AND METHODS

Patients admitted with an acute stroke for the first time were assessed by a detailed clinical history to detect the risk factors and by physical examination.

The blood of the patient, for complete hemogram, leukocyte count, differential leukocyte count, packed call volume, erythrocyte sedimentation rate, plate let count, bleeding and clothing time, blood sugar, and serum cholesterol is evaluated.

RESULTS

Of the 50 patients 32 males and 18 females

The age of the patients ranged from 30 – 80 yrs, with a mean age of 52 – 55 yrs.,

Of the 50 patients, 30 (60%) had cerebral infarction and 20 (40%) had cerebral hemorrhage.

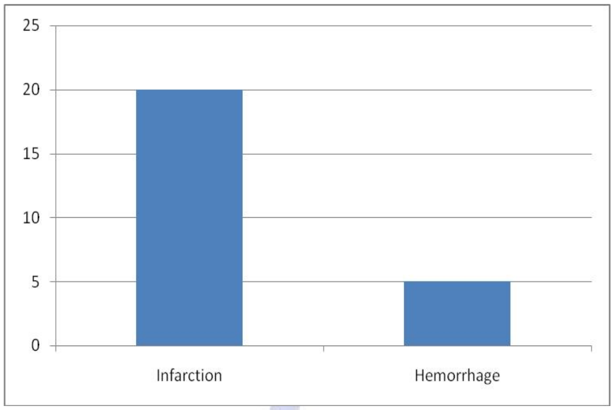
30 of the patients gave history of hypertension in the past, and 10 more patients were recorded with high blood pressure (140/90).

18 patients gave history of diabetes mellitus and 12 more had blood sugar more than 140 mg% at the time of admission.

Hyper cholesterolemia (serum cholesterol >250 mg) was detected in 28 (56% patients.

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 (80%) 20 patients (80%) had Infarction and 5 patients (5%) had hemorrhage.

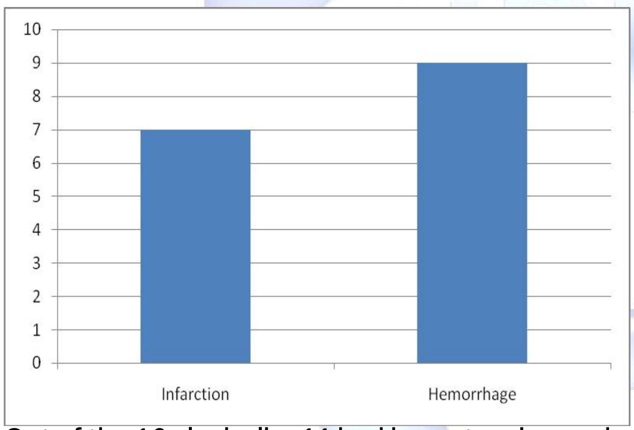
Fig. 1: Causes for Cerebrovascular accidents in smokers.



Out of the 25 patients with smoking habit 16 patients were hypertensive and 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 and 9 (56.25%) were in hemorrhage group.

Fig. 2: Causes for Cerebrovascular accidents in Alcoholics.



Out of the 16 alcoholics 11 had hypertension and 5 had hyper cholesterolemia. Out of the 11 hypertensives 5 developed infarct and 6 developed haemorrhage and out of 6 hyper cholestrolemia 3 had haemorrhage and 2 had infarct.

Table 1: Showing the no. alcohol consumed patients effected with CVA.

	Alcohol	
	Infarct	**Hemorrhage**
Hypertension	5	6
Hyper cholesterolemia	2	3

Out of the 32 diabetics 21 had associated hypertension and 11 had hypercholesterolemia.

The average Hb concentration in patients with infarct was11 – 15 gm./dl and in hemorrhage cases was 9 – 8 gm/dl.

Average packed cell volume of all patients was 35.95% with 40.56% in infarct gp and 38.26%in hemorrhage group.

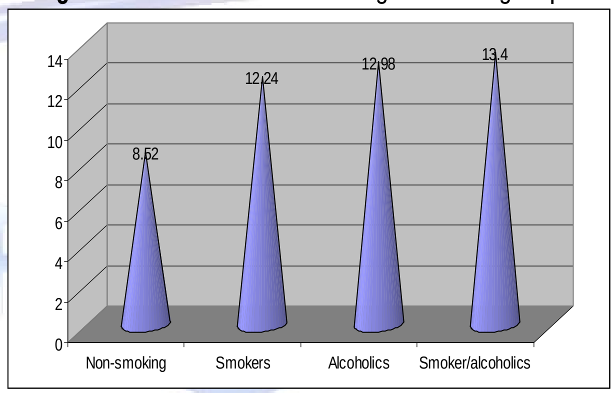
The average MCV in infarct gp was 94.90 fl. and in hemorrhage group was 87.68 Fl. With an aggregate average of 92.90 fl.

Smokers with stroke revealed a Hb concentration (12.24 g%) when compared to 8.53% of non smokers.

Smokers who had the habit of alcohol showed a slight more Hb concentration (13.40 gm%) when compared to smoking alone.

Persons who only had the habit of alcohol intake revealed a comparatively less Hb conc. (12.98 gm%).

Fig. 3: Hb Concentration among different groups.



Smokers has a slight excess MCV (90.44 fl.) when compared to nonsmokers (85.72 Fl) and alcoholics revealed still more MCV (95.66 Fl) and alcoholics who concomitantly smoked had MCV of 99.34 Fl.

Table 2: Showing the MCV among the various groups.

	MCV
Non-smokers	85.72 Fl
Smokers	9.44 Fl
Alcoholics	95.66 Fl
Smokers & alcoholics	99.34 Fl

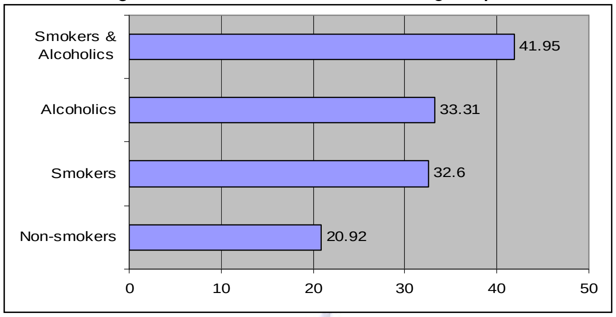
Smokers has a slight excess MCH (35.40) when compared to nonsmokers (20.72) smokers who also had the habit of alcohol intake had still more MCH (40.72) and only alcoholics had MCH of (38.42).

Table 3: Showing the MCH among the various groups.

	MCH
Non-smokers	20.72
Smokers	35.4
Alcoholics	38.42
Smokers & alcoholics	40.72

MCHC was also excess in smokers (32.80) as compared to non-smokers (20.92 and MCHC still increased in alcoholics (33.31) and still more in alcoholics and smokers who had stroke (41.95).

Fig. 4: MCHC levels in Various groups.



Females with stroke had a slight excess of leucocytes (12400/cumm) when compared with males (11980/cumm).

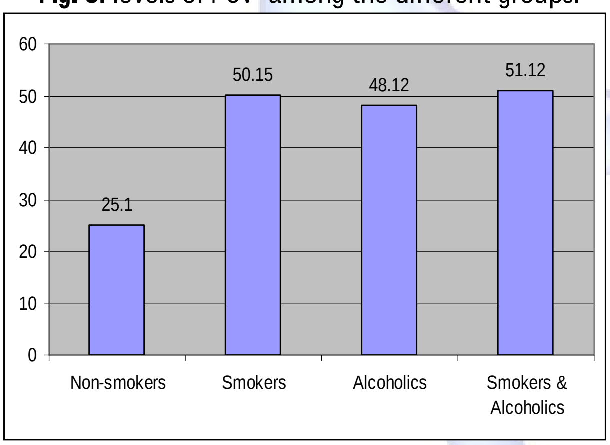
A significant leucocytes was noted in persons who had hemorrhage in all age gps as compared to infarction.

Females had a slight neutrophilia (80%) compared to males (77%) and neutrophilia was a dominant feature in cases of hemorrhage (85%) as compared to infarct (75%).

Smokers revealed a high PCV (50.15) when compared to non-smokers (25.10) and smokers who had the habit of alcohol intake had still more PCV (51.12).

Alcoholics had relatively less PCV (48.12) as compared to smokers alone.

Fig. 5: levels of PCV among the different groups.



Platelet count was more in females (3.55 l lakhs/cumm) than in males (3.20 lakhs/cumm), there was a significant thrombo cytosis (3.45 lakhs/cumm.) in infarct gp when compared to hemorrhage (2.95 lakh/cum.)

Table 4: Showing the platelet count among the various groups.

	Platelet count
Males	3.20 lakh/cumn.
Females	3.55 lakh/cumn.
Infarct	3.45 lakh/cumn.
Hemorrhage	2.95 lakh/cumn.

Females had more ESR (25.1 mm/1st hr.) compared to males (23.73 mn/1st hr.) though the difference was not much.

Bleeding time was more (5.1 min.) 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

Stroke is a focal neurological deficit of sudden onset due to non traumatic vascular lesion.

Normal functions of brain are dependent upon a constant supply of oxygen and glucose, as well as nutrients derived from the blood per fusing it (55to 70 ml./100 gm. of brain/min.

The blood varies in different areas of brain and a self-regulatory mechanism (auto regulation) determines the regional flow to meet local metabolic needs.

Conversely, in regions of cerebral ischaemia, there is "paralysis of auto regulation" and the microvasculature agents fail to respond to chemical and to other forms of stimuli. The cerebral vasculature in this ischemic zone becomes permeable to protein and fluid leaks in the vicinity (extra cellular cerebral edema). Such events also lead to local haemo concentration and vascular stasis. Thus, cerebral infarction is not the mere result of ischaemia from occluded blood vessels, but an end result of a series of highly complex ischaemia modifying agents.

If for any reason, such cardiac arrest of prolonged hypotension (systolic BP below 70 mmg Mg) 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 (ischemic infarction in thrombosis) or may show petechial hemorrhages in the cortical mantle (hemorrhagic infarction in embolism).

An attempt is made to identify factors predisposing to stroke in patients getting admitted with evidence of stroke in acute medical care unit with special attention towards risk factors like blood pressure diabetes mellitus, smoking and alcoholism.

AGE FACTOR

58% of total strokes developed in middle aged and elderly. The changes that do occur with ageing include a slow, apparently continuous symmetric increase in the thickness of intima due to 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.[16]

B.P. as an etiological factor:

The mean arterial systolic and diastolic BP 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 a etiological factor in preventing development of stroke.[11,17]

Diabetes mellitus its role:

Studies 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 and infarction but not towards cerebral hemorrhage in diabetes.[3]

Smoking and cerebrovascular studies:

Smokers are at the risk of developing ischemic stroke than hemorrhagic stroke. Cigarette smokers had 2 – 3 times risk of thrombo embolic or hemorrhagic stroke and stopping smoking showed significant benefits. Cigarette smoking increases the risk of stroke by about 3 folds. It may be because of an increase in the hemoglobin concentration and raised hematocrit as it is evident by our studies.[4]

ALCOHOL AND STROKE:

In the present study 16 cases were alcoholics and among them 9 developed an intra cerebral hemorrhage confirming the fact that alcohol is the risk factor for developing hemorrhagic stroke. The risk factor for hypertension, independent of age, sex and race. The risk of intracranial hemorrhage increasesmonotonically with increase in alcohol intake such that heavy drinkers are at three times the risk of stroke compared with non drinkers.[9]

CHOLESTEROL AND STROKE:

Patients with serum cholesterol >250 mg% reported more incidents of infarction. The risk of death from thrombosis increased significantly with increased cholesterol concentration. For cerebral hemorrhage the death rate was highest in those with lower blood cholesterol level.

RAISED HEMOGLOBIN CONC & PCV AS A RISK FACTOR FOR DEVELOPING STROKE:

In my study smokers had a comparatively more PCV when compared to non-smokers.

Elevated hematocrit or Hb level was considered to be a well documented risk factor for stroke. The hematocrit hemoglobin conc. were found to be high in person who indulge in habit of smoking.[8, 6,1]

PCV is among the factors that many cause a rise in blood viscosity, which may affect microcirculation more seriously leading to development of infarction. The risk factor of elevated hematocrit can be eliminated with in a week of stopping smoking.

MCV, MCH & MCHC IN STROKE:

In my present study MCV gradually increased with age of person and smokers and alcoholics had a increased erythrocyte size than non-smokers. The erythrocytes enlarge as people's age with an addition of hemoglobin content leading to a raised MCV & MCH. Persons with stroke and habit of smoking have large erythrocytes than non-smokers in both sexes and all ages smokers have correspondingly high Hb, hematocrit and MCH values than non-smokers.[14,18]

The macrocytosis is usually a result of deoxy nucleoprotein synthesis. There are more than 1150 different chemicals identified in tobacco smoke and one among them is hydrogen cyanide in cigarette smoke and cyanide of tobacco is detoxified by pathway involving Vit.B12 leading to depletion of Vit.B12 and its non availability for maturation of RBC and a resultant formation of large erythrocyte.

Elevated MCV, macrocytosis is the most typical morphological abnormality induced by excessive ethanol consumption. Acetaldehyde, the first metabolite of ethanol, may play a role in hematological derangements in peripheral blood cells and in bone marrow of alcoholic patients.

Studies have shown that acetaldehyde can bind to proteins and cellular constituents forming stable adducts. Elevated adducts levels have been found from the erythrocytes of alcohol abusers, which may also be associated with ethanol induced effects in haematopoieres and advance consequences in cellular function.

The most common hematological abnormalities in alcoholism are raised mean corpuscular volume of erythrocyte and thrombocytopenia.

Macrocytosis may be considered as an early marker for alcoholism. The changes in maturation are likely to be caused by a toxic effect of alcohol on nuclear metabolism of bone marrow cells.[22]

LEUCOCYTES IN STROKE:

Due to any variety of vascular injury to nervous system there occurs a leucocytic response and by 24 -28 hrs. there is a variable neutrophil infiltration marked by peripheral neutrophilic leucocytosis. The magnitude of leucocytic response depends upon the severity of vascular insult and leucocytosis is more pronounced is cerebral hemorrhage than infarction. It is also reported that the leucocyte count gradually increase with age and intensity of smoking.

PLATELETS IN STROKE:

Altered platelet number, metabolism and function leads to an impairment in coagulation process that could increase the trauma of cerebral hemorrhage. Alcohol drinkers demonstrate thrombocytopenia which is caused by ineffective thrombopoiesisis and by shortened life span of platelets as direct effect of ethanol functional impairments of thrombocytes.[6, 23]

BLEEDING AND CLOTTING TIME IN STROKE:

Bleeding and clothing time 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.[20]

SUMMARY AND CONCLUSIONS:

Aetiopathology of acute cerebrovascular disease is not usually because of a simple risk factor as their presence in isolation for long duration could not result in disease.

Males are none susceptible compared to females, may be due to multivariate etiological factors.

Among the multiple risk factors hypertension took the lead role followed by diabetes mellitus, with predilection for hemorrhage and infarction respectively.

Hypercholesterolemia is a risk factor along with smoking lined at third spot and showed a predilection for non-hemorrhagic stroke.

Alcohol intake with risk of hemorrhagic stroke stood next.

There was considerable increases in hemoglobin conc., PCV, MCV and MCH in stroke patients.

Leucocytosis with predominantly polymorphonuclear leucocytosis was noted in the study with a gross increase in patients who had hemorrhagic stroke.

A lower platelet count, a prolonged bleeding and clotting time was noted in hemorrhagic strokes requiring further study.

Cigarette smoking is of considerable importance in development of stroke. The last advice which can be given to patients with mild hypertension is that they should not smoke.[19]

As age advances and the habits of cigarette smoking and alcohol continue there occur a gradual progressive change in the blood indices and hematocrit of an individual. A careful and continuous monitoring of these parameters may aid in advising discontinuation of such practices where ever necessary.

Conflicts of interest: None

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