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STUDY ON ETIOPATHOGENESIS CLINICAL PRESENTATION AND OUTCOME IN 100 YOUNG STROKE PATIENTS

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ABSTRACT **Introduction:** stroke in the young is one of leading cause of death and disability worldwide that requires recognition and aggressive treatment to prevent long term morbidity and mortality. **Methods:**This was an observational studyof 100 cases of completed stroke patients in the age group less than 45 years with regard to etiopathogenic risk factors, clinical presentation and their outcome in further follow upThis study was conducted among 100 Youngstroke patients who were admitted in government Rajaji hospital, Madurai from March 2013 to February 2014. **Results:** Young stroke is common in the age group 35-45 years. Males are affected more than females in young stroke. Ischemic stroke is more common than hemorrhagic stroke. Thrombotic stroke was more common than embolic stroke. Among ischemic strokesanterior circulation strokes more common than posterior circulation strokes. Motor weakness is the common presentation of young stroke followed by unsteadiness, unconsciousness and speechdisturbances. Right sided weakness more common than left sided weakness. Hemiparesis was more common than hemiplegia. Motor aphasia most common type of aphasia in our study. Smoking and alcohol are the most common risk factor for ischemic stroke followed by dyslipidemia, diabetes mellitus and hypertension. Atrial fibrillation is the most common risk factor for embolic stroke. Rheumatic heartdisease is the most common cardiac disease causing embolic stroke. Hypertension is the single most important factor for hemorrhagic stroke. Vasculitis,hypercoagulablestates, homocystinemia, APLA are rare risk factors for young stroke. Death were seen in 12 % of patients. **Conclusion:** young stroke carries modifiable and non modifiable risk factors,recognizing pattern of clinical manifestations and outcome with long term follow up is necessary to reduce morbidity and mortality.

KEYWORDS : young stroke, ischemic, MCA, ICH

INTRODUCTION

Stroke is the leading cause of death and disability world wide.WHO defines stroke as rapidly developing clinical signs of focaldisturbances of cerebral function lasting >24 hrs or leading to deathwith no apparent cause other than vascular origin.Stroke is a morbid life threatening condition that requires rapid andaggressive treatment to prevent progression from cerebral ischemia tocerebral infarction.In developing countries like India, the incidence of stroke under45years is increasing. Stroke in young age is relatively uncommonwhen compared to old age but has serious impact on the affected familyas well as society. The causes are more diverse and differ as comparedto the elderly. Overall prognosis is better than elderly but there is stillsignificant morbidity and mortality.Knowledge of the risk factors that leads to stroke under 45 yearsand evaluation of them is necessary for better primary prevention and health. Non modifiable risk factors include age, gender, genetics, race and non modifiable risk factors include hypertension, transient ischemic attack,diabetes mellitus, priorstroke, obesity, dyslipidemia .homocysteinemia,increased fibrinogen, cardiac disease,carotid stenosis.

The incidence of stroke gradually increases with age increasing.Data from the Framinghom study shows that incidence of stroke risesfrom 2-5/1000 in 45-55yrs, 10/1000 in 56-65 yrs, 20/1000 in 65-85 yrs.The incidence of stroke under 45 years is 2 in 10000. In the Framingham study men aged 45-55 years have a strokeincidence rate of 20/10000, women aged 45-55 years have a stroke incidence rate 11/10000.Male developed strokes at a higher rates than females up to the age of 75 yrs. The rate of incidence of stroke in blacks higher than white s blacks had been thought to have higher rates of intracranialatherosclerotic occlusive disease. Auckland, Newzealand, Pacificisland people have higher mortality with in 28 days of stroke whencompared to Europeans.Chinese, Koreans, Japanese have higher incidence ofintracerebral hemorrhage when compared to whites.In developing countries like India, the average age of patients with stroke is 15 years younger than that in developed countries. In India, nearly one fifth of patient with first ever stroke admitted to hospitals are under 45 years Trivandrum stroke registry showing stroke of about 9.5% under 45 years.Indian studies have shown that about10to15% strokes occur in people below the age of 40 years.

Heredity seems to play a minor role in the pathogenesis ofcerebral infarction. However an increased risk is seen with familyhistory of stroke among first degree relatives. Number of inheriteddisease like Ehlerdanlos syndrome, Marfan syndrome, Osler RenduWeber disease, Sturge weber syndrome, Hereditary dyslipoprotenemia, deficiency of Protein C, Protein S, Antithrombin III, MELAS, Fabry'sdisease, Homocystinuria can cause stroke.

Data from Framingham study shows that men and womenwith definite hypertension have a three fold greater risk of stroke thannormotensive individuals.Female with hypertension who use oral contraceptive pills has 10fold increased risk of stroke compared with female nonusers of oralcontraception pills without hypertension. Diabetes increases the risk of stroke an estimated 3 to 4 fold as compared with risk in people without diabetes. In addition diabetes increases mortality and morbidity after stroke. The mechanism of stroke secondary to diabetes caused by accelerating atherosclerosis,cardiac embolism, rheological abnormalities, low HDL and increasingplatelet adhesiveness. High total cholesterol and high low density lipoprotein are correlated with atherosclerosis. Recent meta analysis however have suggested that ischemic stroke increases with increasing cholesteroland reduction in stroke risk associated with statin therapies is related toreduction in LDL.Smoking is a biologically plausible independent determinant ofstroke. Smoking has been associated with 70% increased risk of stroke.Stroke risk was greatest in heavy smokers and reduced within 5 yearsamong those quit. It is also a independent determinant of carotid arteryplaque thickness, intra cerebral hemorrhage, overall the stroke riskattributed to cigarette smoking is greatest for subarachnoid hemorrhage,

intermediate for cerebral infarction and lowest for cerebral hemorrhage.Alcohol consumption has been shown to be a risk factor for bothintracranial and subarachnoid hemorrhage. The relative risk of strokeincreased with heavy alcohol consumption (five or more drinks / day) and decreased with light drinking when composed to non drinkers.Heavy alcohol consumption induces cardiac arrhythmias,cardiomyopathy and hypertension increases clotting factors, increasesplatelet aggregation, also causes activation of sympathetic nervoussystem.

Presence of Cardiac disease is the most common etiology forembolic stroke. In the Framinghom study only 13.6% of patients werefree of any heart disease, 80% were hypertensive, 32.7% had priorcoronary artery disease 14.5% had cardiac failure, 13.5% had Atrial fibrillation.Coronary artery disease is an indicator of diffuse atherosclerotic vascular disease and act as a potential source of emboli from muralthrombi due to congestive heart failure, dilated cardiomyopathy,

myocardial infarction. Atherosclerotic lesions of the carotid artery bifurcation are common cause of stroke. Asymptomatic carotid artery disease carries agreater risk for stroke. It carries the risk of stroke 1.5% at 1 yr and 7.5at 5 yrs. Asymptomatic carotid artery stenosis of < 75% caries a strokerisk of 1.3% annually, stenosis of > 75% carries a risk of 10.5% peryear. Plaque structure rather than degree of carotid artery stenosis maybe a critical risk factor for stroke. Ulcerated, echolucent,

andheterogenous plaque with a soft core represent unstable plaque at high risk for producing arterio arterial embolism. Patients who suffer TIA are greater risk than normal controls forstroke. The risk for stroke is approximately 3 times higher.Approximately 10 to 15% of those experiencing a stroke have TIAbefore their stroke. The interval from the last TIA is an importantpredictor of stroke risk. Of all patients who subsequently experience stroke, 21% do so in 1 month, 51% do so in 1 year of the last TIA.

Elevated plasma homocysteine level is an independent risk factorfor atherosclerotic disease. Patient with high homocysteine levelincreased risk of thrombotic stroke and PVD in family in youngpatients. Antiphospholipid antibodies are marker for an increased risk ofthrombosis including TIA and stroke particularly in younger patients. The component required for ACL binding is B2glycoprotein 1 (B2 GPI). It is the B2GP1 dependent ACL of the IGGisotype that has been significantly associated with stroke .It alsoassociated with ocular ischemia, cerebral venous thrombosis, migraine,dementia, chorea, and transverse myelopathy.Paradoxical embolism caused by Right toLeft shunt through PFO or ASA can be responsible for stroke.

Antiplatelets, oral anticoagulants, trans catheter or surgical closure of PFO have been recommended.Coexistence of PFO and ASAincreases the risk of embolic stroke. The evidence from case control studies suggest that migraineparticularly migraine with aura is associated with increased risk ofischemic stroke in young women under 45 years of age.

Cerebral autosomal dominant arteriopathy with subcortical infarct and leucoencephalopathy is a familial non arteriosclerotic, nonamyloidangiopathy characterized by migraine with aura, recurrentischemic strokes leading to pseudo bulbar palsy, cognitive decline,subcortical dementia, and white matter Hyperintensities on MRI.CADASIL is caused by simple missense mutation in Notch 3 gene onchromosome 19q12. Pathologically there is characteristic granular osmophilic material in arterial walls including dermal arteries. Familial hemiplegic migraine characterized by transientweakness or frank paralysis during the aura, has also been mappedclosed to the CADASIL locus. It accounts for upto one fifth of ischemic stroke in young and middle aged patients . In majority of cases the specific etiology remains unknown. Trauma, Infection Migraine, Ehlerdanlos syndrome,Fibro muscular dysplasia, familial are the main causes ofdissection. like episodes (MELAS), Reversible vasoconstriction syndrome, MoyaMoya disease, Sneddon syndrome, Fabry's disease and malignancy includes causes of non atherscleroticarteriopathy. The mostcommon of those in young stroke patient is cervical artery dissectionwhich has been implicated in upto 20-25% of cases of young strokefollowed by vasculitis, infection and Moyamoya in Asian population.

Trauma is the leading cause of cerebrovascular mortality in thedeveloping and developed countries. Blunt or penetrating trauma mayresult in cervicocephalic arterial dissection, arterial thrombosis, arterialrupture, pseudo aneurysm formation or development of AV fistula. Internal carotid artery thrombosis may also follow maxillary andmandibular angle fractures. Carotid artery hematoma may causehematoma formation over the lateral neck, retinal or hemispheric ischemia and a Horner syndrome. Primary hypercoagulable states are Antithrombin deficiency,protein C deficiency, protein S deficiency,Factor V leiden mutation, Activated protein C resistance, Prothrombin G 20210 mutation. Hypo or dysfibrinogenemia, hypoplasminogen, Lupusanticoagulant and anticardiolipin antibodies. Secondary hypercoagulable states are malignancy, pregnancy, puerperium, oral contraceptive use, ovarian hyper stimulation syndrome, nephrotic syndrome, polycythemia rubravera, paroxysmalnocturnal hemoglobinuria, heparin induced thrombocytopenia, homocystinuria, sickle cell disease, thrombotic thrombocytopenic purpura.

METHODS:

This was a cross sectional observational study conducted at Government Rajaji Hospital, Madurai Medical College, Madurai during the period between March 2013 and February 2014.The inpatients admitted in Neurology and Medical ward during oneyear period between March 2013 to February 2014 were taken up forthe study. The clinical details on demographic profile, stroke history,and treatment history were collected by interviewing the patient and from the case history with the help of standard questionnaire. Associated symptoms,risk factors for stroke were noted in proforma. The diary will bescrutinized monthly during the period of the survey.Patients with age <45 yrs, arterial and venous strokes were included.Paediatric age group patients and patients above 45years,patients who developed post ictal weakness following seizures, weakness due to infections (TB, Neurocysticercosis, Toxoplasmosis), Tumour like meningioma, weakness due to trauma,with focal neurological deficits due to HIE, attempted hanging, poisoning were excluded.A total of 100 patients of young stroke admitted in neurology and medical wards were included and analysedbetween March 2013 to February 2014. Data analysis was done with the help of computer by using SPSS software and Sigma Stat 3.5 version (2012). Using thissoftware, range, frequencies, percentage, mean, standard deviation and 'p'value were calculated through One way ANOVA, Chi square, Pearson andSpearman Correlation test and P value of < 0.05 was taken as significant.

RESULTS:

In the present study which was conducted over 1 year observation period, totally 100 patients completed the study and they wereanalysed. Their mean age was 38.9 yrs and 61 of them were male and39 were females.Young stroke (under 45 years) is more common in the age groupbetween 35 to 45 years. Total no. of cases is in 35 to 45 yrs is 63. The percentage is 63%. total no of cases in the age group 25 to 35 yrs was 27(27%.), no of cases in the age group 15 to 25 yrs was10(10%). Our study correlated with Mayotudy. Atherosclerosis is the chief cause of cerebral ischemia. Asatherosclerosis is an age related process incidence of stroke is probablyless common in age group 25 to 35 yrs when compared to 35 to 45 yrs. Males constitute 63% of young stroke. Females constitute 37% of young stroke in our study. Our study correlate with the Chan MT Morris et al study. Males were prone for stroke than females Ischemic stroke more common than hemorrhagic stroke. Ischemic stroke contribute 84%,hemorrhagic stroke contributes 16% cases in our study Among ischemic strokes thrombotic stroke(53%) was common than cardio embolic stroke(33%) ,cerebral venous thrombosis contributes 14% cases.

In our study anterior circulation strokes commonly involved than posteriorcirculation. In the anterior circulation middle cerebral artery more commonly involved than anterior cerebral artery. MCA stroke contributes 70%, ACA stroke contributes 14% and Posterior circulation stroke contributes 16%. Aphasiaseen in 38 cases accounts for 38% of total young stroke cases. Of the sepure aphasia seen in 10 cases(26%), aphasia with weakness seen in 28 cases(74%).Of all the aphasia motor aphasia more commonly involved than other a phasia. Motor aphasia contributes 63%. Sensory aphasia contributes16%, global aphasia contributes 21 % of total cases. Motor weakness more common than others. Weakness contributes 54%, Unsteadiness contributes 13%cases, unconsciousness contributes 15% cases, Pure Language disturbances seen in 9% cases, Headache alone contributes 4% of cases, Seizures alone contributes1%cases, lower cranial palsies in 2% of cases, sensory symptom in 1% of case. Right sided weakness (65%) most commonly present than left(35%). Hemiparesis (power>3) (62%)most commonly presented than hemiplegia (power<3)(38%). Males are most commonly affected than females. Males accounts for 63 % of cases, Smoking, and alcohol is the most common risk factors in male. Smoking alone seenin 48% cases, alcoholism alone in 38%, both smoking and alcoholism seen in 68% of cases. Hypertension, dyslipidemia, Coronary artery Disease, valvularlesions are the most common risk factors for stroke. Of these Hypertension, Dyslipidemia and Coronary artery disesae are common in males whereas Valvular lesions are common in females in our study. Positive ANA and positive Rheumatoid factors, and positive APLA are the risk factors present only in females in our study. AN A positivity seen in 1 cases, Rheumatoid factor positivity in 3 cases, APLA positivity seen in one case. Homocystinemia seen in 1 case responsible for thrombotic stroke in male patient.

Hypercoagulable status was positive in 4 cases, and causes CVTin 4 patients. Of these 4 patients one patient was male others 3 were female. Protein C deficiency was seen in one male patient. Two female patients had protein C deficiency and another female patient had protein S deficiency. The other remaining 8 patients were female all are in postpartum state Hypertension is the most common cause for hemorrhagic stroke. Of the 16 hemorrhagic stroke cases 15 cases were hypertension induced and one cases of hemorrhagic stroke was due to Hemophilia induced hemorrhagic stroke seen in 11 males cases, and in 5 female cases. cardiac cause as a risk for cardiembolic stroke was seen in28 cases. Of these Valvular lesions seen in 26 cases. 2 cases

presentedwith cardiomyopathy. Of the 26 valvular cases RHD accounts for 20 cases. In all these RHD cases atrial fibrillation seen. ASD seen in 6cases. Atrial fibrillation present in 5 cases. Hence our study concludethat presence of atrial fibrillation in heart disease increases the risk ofcardio embolic stroke.. In our study > 50% of our young stroke patients were comes under metabolic syndrome and responsible for both stroke and CAD.

Clinical outcome of weakness at 90 days were assessed, onadmission more than 50% of cases were clustered in MRS scoring of3-4.after 90 days follow up more than 50%of cases were clustered inMRS grading of 1-2 showing significant improvement in motorweakness.

On admission most ofthe were clustered in modified BARTHAL index SCORING of moderate to severe stage (25-74 score). After 90 days follow up most ofthe cases were clustered in mild MBI staging (score 75-96). Speech disturbances at 180 days were assessed. Motor aphasia was improved in 21 cases(87.5%) and persisted in 3 cases(12.5%). Sensory aphasia improved tonnominal aphasia in 5 cases (83.3%) persistence of sensory aphasia seenin 1 cases (16.7%). Global aphasia improved to motor aphasia in 6cases (75%) and it was persisted in 2 cases (25%).Unsteadiness wasimproved only in 3 cases(23.1%). Persistence of unsteadiness was seenin 10 cases(76%) during 1 year follow up.Total number of death was 12 cases. Immediate death seenin 8 cases(66.6%) and late death seen in 4 (33.3%)cases.

DISCUSSION

The stroke is the leading cause of death and morbidity worldwide. A higher proportion of younger individuals suffer from strokeamong developing countries as compared with developed countries.By knowing the prevalence of risk factors in both stroke andstroke subtypes we can improve the primary and secondary preventivestrategies.Stroke in the young requires a different approach toinvestigations and management than stroke in the elderly givendifferences in the relative frequencies of possible underlying cause.In the following discussion I explore the different etiologicalrisk factors in young stroke, their clinical presentation and their outcome in further follow up.

According to Carolei et al, Kwon et al, Varona et al, of all subtypes of stroke in young adults found that thrombotic strokewas the most common subtype followed by embolic and hemorrhagic.Our study correlates with above study. In our study thromboticstrokewere seen in 44 cases (44%) cardio embolic stroke in 28 cases (28%)hemorrhagic strokes in 16 cases (16%), CVT in 12 cases (12%)Overall there is males are most commonly affected in stroke.

Studies(Nayak et al, Lipska et al) performed on ischemic stroke amongthe 15-45 age group from India also reported a malepreponderance.The proportion of cases in our study is higher in the age groupbetween 35-45 yrs which is similar to findings reported by Nayak et al .Presenting symptoms are similar to Chopra, Prabakar and Nayaket al study, In our study embolic stroke presented with profound loss of power in limbs.Mostly they occur in the afternoon Thrombotic stroke occurs mostly in the early morning while people wake up.

According to Mehindiratta MM et al Smoking, Alcoholism, hypertension have been found to be significantly associated with ischemic stroke. Our study also correlates with this study. Smoking seen in 43%, Alcoholism seen in 38%, Hypertension seen in 28% of ischemic stroke cases. Diabetes was not found to be a risk factor in Sweden and Taiwan but in out study (25%) Diabetes is a significantly risk factor. It corresponds to Lipska study. Dyslipidemia (hyper cholesterolemia, hyper tryglyceredemia) known to be associated with ischemic stroke in young adults. Dyslipidemia significantly seen in 33 out of 52 ischemic stroke contributing to atherosclerosis in our study. Our study correlates with the Arnold M et al study. Elevated homocysteine level seen in one case responsible for thrombotic stroke in male patient. Antiphospholipid antibody syndrome seen in one caseresponsible for thrombotic stroke in female. Vasculitis is also one of the cause of young stroke. Vasculitiscausing stroke seen in 4 patients in our study. Of these Rheumatoidarthritis contributes 3 cases .SLE seen in 1 case (1.68%). All 4 caseswere seen in females. Hypercoagulable status is a rare cause of young stroke. In ourstudy hypercoagulable status seen in 4 cases. All contributes CVT inour study. Among 4 patients, 1 patient was male and remaining 3were females.

Stroke may presented with various symptoms and signs. Like weakness, numbness, confusion, speech disturbances, slurring ofspeech, hemianopia, visual loss, headache, convulsion, unsteadinessgiddness and unconsciousness.Of the 100 cases of stroke Weakness (Loss of power) mostcommonly seen. Weakness seen in 82 cases. Of the weakness Rightsided weakness is more common seen in 52 cases (63%). Lt sidedweakness seen in 30 cases (37%).Speech disturbances seen in 38 cases. Pure speech disturbancesseen in 10 cases (26.3%)) speech disturbances with weakness in28cases(73.7%). Of the various speech disturbances motor aphasiamore common than other aphasias seen in 24 cases (63.2%) Sensoryaphasia seen in 6 cases (15.7%) global aphasia seen in 8 cases (21.1%).Headache is a common symptom in hemorrhagic stroke. In ourstudy headache seen in 4 cases. All cases are hemorrhagic stroke. Unsteadiness is a common symptoms in posterior circulationstroke. In our study, Unsteadinessseen in 13 cases and were due to posterior circulation stroke. Among these 7 patients also had lowercranial nerve palsy (9, 10 cranial nerves)Unconsciousness seen in 15 cases in our study, of these 15 cases,11 cases were due to hemorrhagic stroke other 4 cases were due tomassive infarct.Convulsions are uncommon in stroke. It can occur in embolicstroke, hemorrhagic stroke and in cerebral venous thrombosis. In ourstudy convulsions alone are the least symptom seen in one case due toembolic stroke. Convulsions with weakness usually seen in CVT. Our 4patients had similar pattern all are due to CVT.Obesity is one of the features of the metabolic syndromeresponsible for stroke and CAD in both younger and older individuals.In our study obesity was common due to sedentary life style. Obesity inour study seen in > 50% (55 cases). Over weight individualscontributes 27% of young stroke cases.RHD is the most common etiology for cardio embolic stroke inour study. RHD seen in 20 cases of all cardioembolic strokes (28cases). ASD seen in 6 cases. Cardiomyopathy seen in 2 cases. Amongthe 28 cardio embolic cases Atrial fibrillation seen in 24 cases. HenceAtrial fibrillation is the potential risk factor for embolic stroke.Hypertension is the major risk factor for hemorrhagic stroke.Among the 16 cases of hemorrhagic stroke hypertension was present in15 cases. One case was due to bleeding diathesis (Hemophilia).Secondary causes of hypertension was assessed. Renal artery stenosisseen in 2 cases. Adrenal tumor seen in 2 cases. Coarctation of aortaseen in 1 case. Idiopathic hypertension seen in 2 cases. Chronickidney diseasewas seen in 7 cases.

During followup study over a period of 6 months to 1 yearweakness, speech disturbances and unsteadiness were assessed.Weakness and speech disturbances were improved significantly more than unsteadiness.Motor weakness were improved significantlyaccording to Modified RANKIN scale and modified BARTHEL index.Motor aphasia recovered to normal speech in 21 cases. Sensoryaphasia recovered to nominal aphasia seen in 5 cases. Global aphasiarecovered to motor aphasia seen in 6 cases after intense speech therapyafter a period of 6 month to 1 year..

Unsteadiness was not much improved when compared to motorweakness. In our study unsteadiness seen in 13 cases. Only 3 caseswere improved partially and able to walk independently. Remaining 10cases were not improved required assistance to walk.

No. of cases of death were 12 in our study. Immediate death seenin 8 cases. Of these 4 cases due to massive cerebral edema, 2 caseswere due to associated systemic disease. 2 cases were due to electrolyte imbalances. 4 cases were died during follow up study. Ofthese 2 cases were due to aspiration pneumonia and respiratory failure.2 cases were due to bedsores and septicemia.

CONCLUSION

Stroke under 45 yrs is common in the age group between 35-45 years. Males are affected more than females in young stroke. Ischemic stroke more common than hemorrhagic stroke. Thrombotic stroke more common than embolic stroke in ischemic strokes. Anterior circulation strokes more common than posterior circulation strokes.MCA more commonly involved than ACA and PCA Motor weakness is the common presentation of young stroke followed by unsteadiness, unconsciousness and speech disturbances. Right sided weakness more common than left sided weakness. Hemiparesis more common than hemiplegia. Motor aphasia most common type of aphasia in our study. Smoking and Alcohol are the most common risk factor for is chemic stroke followed by Dyslipidemia, Diabetes mellitus and Hypertension. Atrial fibrillation is the most common risk factor for embolic stroke. Rheumatic heart disease is the most common cardiac disease causing

embolic stroke. Hypertension is the single most important factor for hemorrhagic stroke.Vasculitis,Hypercoagulable states,Homocystinemia,APLA are rare risk factors for young stroke.Weakness and speech disturbances were improved significantly.Unsteadiness not improved significantly..Death were seen in 12 cases.

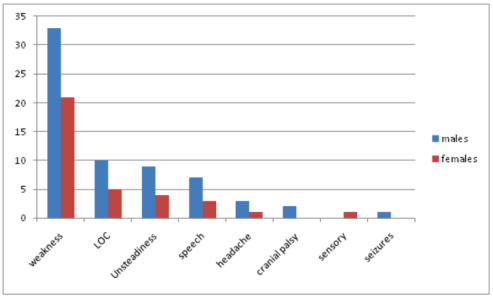

Figure1:clinical presentation of stroke in young

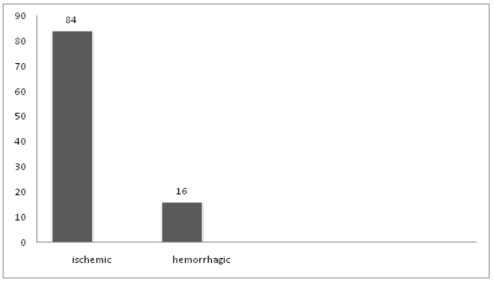

Figure2:types of stroke in young

Table 1:Ischemic stroke subtypes(TOAST classification)

Types of stroke	No of patients	percentage
Large vessel	36	42.9
cardioembolic	28	33.3
Small vessel	8	9.5
CVT/others	12	14.3

Table 2:Hemorrhagic stroke etiology

idiopathic	hypertension					Bleeding diathesis
3	12					1
	Renal parenchymal disease	RAS	Adrenal tumour	Coarctation of aorta	others	
	7	2	2	1	-	

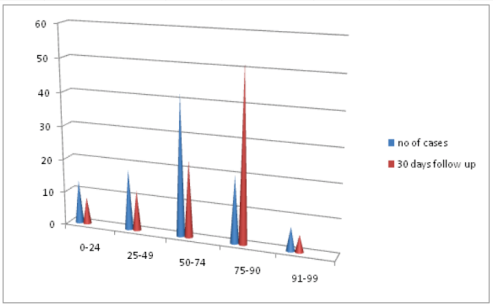

Figure 3:Barthel index score:outcome of patients with stroke

REFERENCES:

1. KielyDK,WolfPA,Cupples LA et al, Familial aggregation of stroke.TheFraminghom Study. Stroke 1993.
2. V.L.Feign,"Stroke in developing countries; can the epidemic bestopped and outcomes improved. Lancet Neurology, vol.6 V.L.Feigin, Stroke in developing countries can the epidemic, No.2 pp 94-97, 2007.
3. Poungrarin, "Stroke in the developing world, Lancet vol 35,supplement 3, pp III 19 – S111 22, 1998.
4. J.D. Pandian, A Jaison, SS Deepak et al, Public awareness of warningsymptoms risk factors and treatment of stroke in North west India,Stroke vol 36, No.3, pp 644-648, 2005.
5. K. Lipska, P.N. Sylaja, P.S. Sarma et al, Risk factors for acuteischemic stroke in young adults in south India, Journal of Neurology,Neurosurgery, and psychiatry vol 78, No.9, pp 959-963, 2007.
6. S.K. Das, T.K. Banerjee A, Biswas et al, A prospective communitybased study of stroke in Kolkatta, India, Stroke vol 38, no.3, pp 906-910, 2007.
7. S.Razdan,R.LKoul ,A.Motta,andS.kaul ,cerebro vascular disease inrural Kashmir, India, Stroke vol 20, no.12, pp 1691-1693, 1989.
8. Chobanian AV, Bakrish GL, Black HR,Cushman WC et al, Theseventh report of joint national committee on prevention,detection,evaluation and treatment of high blood pressure;TheJNCReport JAMA;2003;2560-2572
9. Gray, C.S., Hildreth, A.J., Alberti, GK., and O'connel, J.E., Gistcolloborators, 2004, Post stroke hyperglycemia, natural history andimmediate management, stroke, vol 35, pp 122- 126.
10. Bruno, A., Saha, C., Williams, LS., Shankar, R 2004, IV insulinduring acute cerebral infarction in diabetic patients. Neurology, vol.62, pp 1441-1442.
11. Van den Berghe, G., Wilmer, A., Hermans, G., et al 2006, Intensivetherapy in the medical ICU, N Engl. J Med, vol. 354, pp 449-446.
12. AmarencoP,LabraucheJ,Lavalide P,&Touboul RJ 2004.Statins instroke prevention and carotid atherosclerosis,systemic review andmeta analysis,Stroke vol .39,pp.2902-2909.
13. Grundy S.M, Cleeman J.I, MerzZ.N,et al 2004.Implications ofclinical trials for the national cholesterol education programme Adulttreatment panel 3 guidelines circulation,vol 110,pp.227-239
14. M. Sharma and N.K. Ganguly, Premature coronary artery disease inIndians and its associated risk factors, Vascular health and riskmanagement, vol1, no.3 pp 217-225, 2005.
15. P. Muennig, H. Jia, and K. Khan, Hospitalisation for heart disease,stroke and diabetes mellins among Indian, born persons : a small areaanalysis, BMC cardiovascular disorders, vol 4, no.1, p 19, 2004.
16. Johnston, SC, Gress, D.R., Browner, W.S., and Sidney. S. 2000.Short term prognosis after emergency department diagnosis of TIA,AMA, vol 284, pp 2901-2906.
17. Coutts, SB., Simon, J.E., Eliasziw, M., et al, 2005. Triaging Transientischemic attack and minor stroke patients using MRI. Ann Neurol,vol 57, pp 848-854
18. Norrving B, Magnusson, M. Cu Holtas, S. 1995. SES 1995. IsolatedAcute vertigo in the elderly; vestibular or vascular disease? ActaNeurolScand, vol 91, pp. 43-48.
19. Giles, WH., Kittner, S.J. Anda, R.F., et al, 1995, Serum folate and riskfor ischemic stroke, first national health and nutrition examinationsurvey epidemiological follow up study, stroke, vol 26, pp. 1166-1170.
20. R. Cervera, J.C. Piette, J Font et al, Antiphospholi syndrome, clinicand immunologic manifestations and patterns of disease expression ina cohort of 1000 patients, Arthritis and rheumatism, vol 46, no.4, pp1019-1027, 2002.
21. P. Nagaraja, R. Christopher, and T. Manjari, "AnticardioLipinantibodies in ischemic stroke in the young : Indian experience, Journalof the neurological sciences, vol 150, No.2, pp 137-142, 1997.
22. Bray, R.L., abbott, R.D., Curb, J.D., et al 2001. Beta (2) Glycoprotein1 – dependent anticardiolipin antibodies and risk of ischemic strokeand myocardial infarction; the Honolulu heart programme, stroke, vol32, pp 1701-1706.
23. D.E. Thaler and J.L. Saver, Cryptogenic stroke and patent foramenovale, Current opinion in cardiology, vol 23, No.6, pp 537-544, 2008.
24. L. Cebanes, J.L. Mas A, Cohen et al, Atrial septal aneurysm andpatent foramen ovale as risk factors for cryptogenic stroke in patientsless than 55 yrs of age ; a study using trasnsoesophageal echocardiography" stroke, vol 24, No. 12, pp 1865-1873, 1993.
25. Homma, S., Saoro, RL., Di Tullio, M.r., et al for PFO in cryptogenicstroke studyinvestigations 2002. Effect for medical treatment instroke with patent foramen ovale: Patent foramen ovale in cryptogenicstroke study, circulation, vol 105, pp 2625-2631.
26. Messe, SR. Silverman I.E., Kizer, IR, et al 2004, Practice Parameter :Recurrent stroke with patent foramen ovale and atrial septalaneurysm, Report of the quality standards subcommittee of theAmerican Academy of Neurology,Neurology, vol 62, pp 1042-1050.
27. M.G. Bousser, Estrogen, migraine and stroke. Stroke, vol 35, no.11,pp 2662-2656, 2004.
28. C.L. Chang, M. Donaghy, and N. Poulter, "Migraine and stroke inyoung women, case control study. British medical Journal, vol 318,no. 7175, pp 13-18, 1999.
29. S.Schwag, D.G, Nabari, A. Fresa, IW Husseted, and S. Evers, "Theassociation between migraine and Juvenile stroke, headache, vol 43,No.2, pp 90-95, 2003. .
30. Carolei, A., Marini C.,& De Matteis, G 1966. History of migraineand risk of cerebral ischemia in young adults. The Italian nationalresearch council study group on stroke in young, Lancet, vol. 347, pp.1503-1506.
31. Kalimo H., Ruchoux MM., Vitanen, M., et al 2002, CADASIL : acommon from of hereditaryarteriopathy causing brain infarcts and dementia, Brain Pathol, vol 12, pp. 371-384.
32. Hutchinson, M., O'Riovdan, S., Javed M., et al, 1995, FamilialHemiplegic migraine and autosomal dominant arteriopathy withencephalopathy (CADASIL). ANN Neurovol 38, pp. 2418-2424.
33. Verin, M., Rolland,Y, Landgraf, F, et al 1995. New phenotype ofcerebral autosomal dominant arteriopathy mapped to chromosome 19migraine as the predominant clinical feature. J NeurolNeuroSurgpsychiatry, vol 59, pp 579-585.
34. S.M. Rubinstein, S.M., Peerdeman, M.W., Van Tuldep I, Riphagen,and S. Haldeman, A systematic, review of the risk factors for cervicalartery dissection, Stroke vol 36, No.7, pp 1575-1580, 2005
35. S. Debette and H.S. Markus, The genetics of cervical arterydissection, a systematic review, Stroke, vol 40 No.6, pp 459-466,2009.
36. W.S. Chan, J.Ray, E.K., Wai et al, Risk of stroke in women exposedto low dose oral contraceptite a critical evaluation of the evidence,Archieves of Internal medicine, vol. 164, No.7, pp 741-747, 2004.
37. Nunenz, D.B. Jr., Torres – Leon, M., et al 2004. Vascular injuries of the neck and thoracic inlet. Helical CT angiographic correlation,Radiographics, vol 24, pp 1087-1098, discussion pp. 1099-1100.
38. Fields, M.C. & Levine, SR, 2005, Thrombophilia and stroke :Diagnosis treatment and prognosis, J thromb thrombolysis, vol. 20.Pp. 113-126.
39. Cushman, M, Rosendaal, FR, Psaty, B.M., et al, 1998, Factor 5 leidenis not a risk factor for arterial vascular disease in the elderly. Resultsfrom the cerebro vascular health study, thrombhaemostasis, vol 79,pp, 912-915.
40. Loh, E., Sutton, M.S., Won, CC, et al 1997, Ventricular dysfunctionand risk of stroke after myocardial infarction, N Engl J Med, vol 336,pp 252-257.
41. Mattioli, AV., Castellani, ET, Fusco, A., et al 1997, Stroke in paed.patients with sick sinus syndrome; relevance of atrial mechanicalfunction, pacing mode and clinical characteristics, cardiology, vol 88,pp 264-270.
42. Seidl, K., Hauer,B., Schwick, N.G., et al, 1998, Risk of thromboembolic events in patients with atrial flutter, Am J cardiol, vol 82, pp.580-5
43. Oliveria – Filho, J., Massaro, AR., Yamamoto, F et al 2000, stroke asthe first manifestation of calcific aortic stenosis, cerebrovasc. Dis, vol10, pp 413-416.
44. J.L. Rulz. Sandoval, C. Centu and E. Braringarrementeria, Intracerebral hemorrhage in young people : analysis of risk factors,Location, causes and Prognosis, Stroke, vol. 30, No.3, pp 537-541,1999.
45. O' Donnell, H.C., Rosand, I., Knudsen,KA., et al 2000.Apoliproprotein E genotype and the risk of recurrent. Lobar intracerebral hemorrhage. N Engl J med. Vol 342, pp

240-245.
46. Woo, D. Sauerbeck, LR, Kissen, BM et al 2002, Genetic andenvironmental risk factors for intra cerebral hemorrhage. Primaryresults of a population based study, stroke, vol 33, pp 1190-1196.
47. Casseta, I, Granleri, E, Portaluppi, E, and Majfredini, R 2002, Circadianvariability in hemorrhagic stroke, JAMA, vol 287, pp 1260-1267.
48. Rosand,J., Hylek, EM., O' Donell, HC and Greenberg, SM, 2000,Warfarin associated hemorrhage and cerebral amyloid angiopathy: agenetic and pathologic study, Neurology, vol 55, pp 947-951.
49. Furlan, A Higaswida R., Wechsler, L. et al 1999, Intra arterial Prourokinase for acute ischemic stroke, a randomized controlled trial,SAMA, vol. 282, pp 2003-2011.