



RESEARCH ARTICLE

CLINICAL AND COMPLICATION PROFILE OF GERIATRIC PATIENTS WITH ACUTE ISCHEMIC STROKE

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ABSTRACT

Aims and Objectives: To study the clinical and complication profile of geriatric patients with CT or MRI proven ischaemic stroke and to describe the factors (clinical and laboratory parameters) associated with ischaemic stroke.

Methodology: A total of 120 subjects were included in the study, out of which 80 were patients of ischaemic stroke. 40 healthy subjects served as controls in this study. The data was analysed and following clinical, demographic and laboratory observations were made.

Results: The results of our study shows that 105 (8) people suffered from monoparesis, 62.5% (50) cases suffered from right hemiparesis and 27.5% (22) cases suffered from hemiparesis of the left side. In this study, 19 cases (23.75%) and 6 controls (15%) were suffering from diabetes mellitus as per the American diabetes association criteria of diagnosing diabetes. 61 cases and 34 controls showed euglycemia.

Conclusion: There is a significant relationship of hypertension, diabetes mellitus, age, BMI and lipid derangements with lipoprotein (a) levels and occurrence of acute ischaemic stroke as such.

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INTRODUCTION

Over 2,400 years ago, Hippocrates (460-370 B.C) was first to describe the phenomenon of sudden paralysis, which we know is called by stroke. Apoplexy from the Greek word meaning "struck down with violence," first appeared in Hippocratic writings to describe stroke symptoms (Thompson JE, 1996, Kopito, Jeff, 2001). In 1658, in his Apoplexia, Johann Jacob Wepfer (1620-1695) identified the cause of hemorrhagic stroke as well as the ischaemic stroke when he suggested that apoplexy might be caused due to bleeding or blockage in arteries supplying the brain (NINDS, 1999). Stroke by definition means a sudden, non convulsive loss of neurologic function due to an ischaemic or hemorrhagic intracranial vascular event. In general cerebrovascular accidents are classified by anatomic location in the brain, vascular distribution, etiology, age of the affected individual, and hemorrhagic versus non-hemorrhagic nature. (Adams *et al*, 2005). Stroke can also be defined as rapidly developing clinical symptoms and / or signs of focal and at times global loss of cerebral function with symptoms lasting more than 24 hours or leading to death with no apparent reason other than that of vascular origin.

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After coronary heart disease and cancer, stroke is the 3rd most common cause of death in western countries and is the most common cause of severe disability in people. Stroke occurs most frequently in the hour or two after waking in the morning as well as stroke mortality is higher in winter than summer. Stroke and its incidence in the U.K is about 2/1000/year (Bamford, Sanderock *et al*). About 10000 patients have a first stroke every year, one every five minutes or so. For India, community surveys have shown a crude prevalence of hemiplegia in the range of 200/100000 persons, nearly 1.5% of all urban hospital admissions, 4.5% of all medical admissions and about 20% of all neurological admissions (Dalal, 1982). Stroke causes 10% of the world wide deaths (the world health report, 2004)

Classification of stroke (Smith S Wade *et al*, 2001):

Stroke is broadly divided in to two groups:

1. Ischaemic stroke
2. Hemorrhagic Stroke Causes of Ischaemic stroke:
 - i. Arterial Thrombosis
 - ii. Embolism
 - iii. Vasoconstriction
 - iv. Venous

Commonest cause of cerebral Ischaemia and infarction are atherosclerosis with thromboembolism and cardiogenic thromboembolism. Clinical manifestations of Ischemic stroke:- Typical Ischaemic stroke presents with the abrupt onset of a focal neurological deficit and is characterized by subsequent clinical course. Accordingly Ischaemic Stroke can be, Reversible Ischaemic neurological deficit

- Completed stroke
- Progressive stroke or stroke in evolution.

Pathophysiology

Within 10 seconds after cerebral flow ceases, metabolic failure of brain tissue occurs. The EEG shows slowing of electrical activity and brain dysfunction becomes clinically manifest. If circulation is immediately restored, there is abrupt and complete recovery of brain function. If perfusion abnormality persists for a few minutes, neuronal injury results. With restoration of flow, recovery of function takes several minutes or hours and may be incomplete. More prolonged periods of ischaemia result in frank tissue necrosis.

Ischaemic stroke is defined into two broad categories

- Thrombotic.
- Embolic.

Thrombotic strokes occur without warning symptoms in 80-90% of patients. 10-20% is heralded by one or more transient Ischaemic attacks. Thrombotic strokes often present with stuttering, fluctuating symptoms that worsen over several minutes or hours. Embolic strokes usually present with a neurologic deficit that is maximum at onset. Lacunar infarcts are small infarcts in deep white matter, usually due to hypertension induced Lipohyalinosis or arteriosclerosis of small perforating arteries.

Risk factors of Ischaemic stroke

Various risk factors for Ischaemic stroke are

Age: Age is the strongest risk factor for cerebral infarction. Risk of Ischaemic stroke in people aged 75-85 is 25 times the risk in people aged 45-54. Patients above 80 years with stroke were more likely to present in coma, and more likely to have severe strokes (Di Carlo *et al.*, 1999).

Sex: There is a small increase in stroke in males as compared to females. Men are 1.25 times more likely to suffer cerebrovascular accidents than women (NINDS, 1999).

Blood Pressure: Stroke risk doubles with each 7.5 mm Hg increase in the usual diastolic blood pressure. (Cutler *et al* 1990)

Smoking: There is an increased incidence of stroke in smokers as compared to non smokers (Hankey, 1999).

Blood Lipids: There is an increased incidence of stroke in patients with increased serum lipid levels (Di Mascio *et al.*, 2000)

Diabetes Mellitus: Diabetes Mellitus almost doubles the risk of stroke. Stroke in diabetes is more likely to be fatal. (Burchfiel *et al.*, 1994).

Coronary Heart Disease: It is clearly associated with stroke. The evidence comes from postmortem, case control and cohort studies (Lamlam 1991).

Hyperhomocysteinemia: Hyper homocysteinemia increases the risk of stroke due to its possible atherogenic propensity due to endothelial dysfunction and injury followed by platelet activation and thrombus formation. (Eikelboom *et al.*, 2000, Ribo *et al.*, 2004).

Lipoprotein (a): Lipoprotein (a) [Lp (a)] consists of low-density Lipoprotein (LDL) particle to which is attached a unique glycoprotein called apolipoprotein (a) [apo (a)]. (Utermann, 1989) Density of Lp (a) ranges from 1.050- 1.120 gm/ml size of Lp

(a) is 25nm. Lp (a) is attached to apo (a) and apo B-100 which are synthesized in liver and attached to each other by disulfide linkage. Plasma levels of Lp (a) vary over a 1000 fold range (0.1- > 100 mg/dl) between individuals and differ significantly between ethnic groups [Albers *et al*, 1990; Bovet *et al*; 1994].

MATERIALS AND METHODS

The present study was carried out in the Department of Medicine, SBHGMC, Dhule, Maharashtra, India for a period of 12 months. SBHGMC is a tertiary care hospital.

Subject/patient population

Patients were recruited from indoor wards of general medicine, outpatient clinics, and emergency department. All the eligible patients were explained the purpose of study.

Methodology

All patients were subjected to detailed history and clinical examination. While taking history of our patients their data was entered into risk groups with respect to Age, Male sex, hypertension, DM, Hypercholesterolemia, CHD, Smoking, use of oral contraceptives, drug intake. The following points shall be considered while taking the history of our patients.

- Clinical presentation which includes mode of onset (Sudden, insidious)
- Progression of symptoms
- H/o TIA.
- Stroke risk factors
- Personal history including Dietary habits, smoking, alcohol intake, Occupation, shall be taken.
- Family medical history shall be taken. The details of history and clinical examination shall be recorded in a predesigned proforma.

Inclusion criteria

All patients aged 65 years and above who have suffered a cerebrovascular accident and confirmed as cerebral infarction

which shall be confirmed by CT Head or MRI brain (where ever required).

Exclusion criteria

1. All patients where CT scan head shows:
2. ICH
3. Tumor
4. Age below 65 years

The investigation that shall be carried out other than plasma Lipoprotein (a) levels include

- Complete Haemogram
 - Hb%
 - TLC
 - DLC
 - PBF
 - Platelet Count
- ESR. (Fasting)
- Blood sugar
 - Fasting
 - Post Pyrandial
- Complete Urine Examination.
- Renal function tests
 - Serum urea
 - Serum Creatinine.
- Serum Electrolytes
 - Sodium
 - Potassium
- Serum Uric acid
- Lipid profile
 - Total cholesterol
 - HDL
 - LDL
 - VLDL
 - Triglycerides.
- ECG
- ECHOCARDIOGRAPHY
- X RAY (CHEST). PA VIEW.
- CT SCAN (BRAIN)
- MRI (BRAIN) (WHERE EVER REQUIRED).

RESULTS

A total of 120 subjects were included in the study, out of which 80 were patients of ischaemic stroke. 40 healthy subjects served as controls in this study. The data was analysed and following clinical, demographic and laboratory observations were made;

In the above table, 26.25% (21) patients were 60-65 years of age. 31.25% (25) were in the 65-70 years age group and 42.5% patients (34) patients were above 70 years of age. Out of 80 cases, 60% (48) were males and 40% (32) patients were females. The BMI was below 25 kg/m² in 33.75 (27) patients in the case group and BMI was >25 kg/m² in 66.25% (53) patients in the case group where as it was <25 kg/m² in 42.5% controls and >25 kg/m² in 57.5% controls. 50% cases had smoking history and Diabetes was seen in 23.7% (19) patients of the case group.

Table A. Clinical and demographic profile of subjects included in the study

		Cases			Controls		
		Total	Males	Females	Total	Males	Females
Age	60	21	11	10	10	5	5
	65	25	17	8	13	9	4
	70	34	20	14	17	10	7
	All	80	48	32	40	24	16
BMI	<25	27	15	12	17	8	9
	>25	53	33	20	23	13	10
Smokers		40	32	8	14	11	3
Diabetic		19	13	6	6	4	2
HTN		57	35	22	16	10	6
RHD		4	1	3	0	0	0
Cad		8	7	1	3	2	1
Paralysis/ Parasis	unilat	8	5	3	0	0	0
	RT	50	29	21	0	0	0
	Left	22	13	9	0	0	0

In the control group Diabetes was seen in 155 of the subjects only. In the case group 71.255 (57) patients were hypertensive where as only 40% control subjects suffered from hypertension. It was noted that 105 (8) people suffered from monoparesis, 62.5% (50) cases suffered from right hemiparesis and 27.5% (22) cases suffered from hemiparesis of the left side.

Age Distribution

Table 1.

Age group	Cases	Controls
60-65 Years	21	10
65-70 years	25	13
> 70 Years	34	17
Total	80	40

As shown in Table 1, present study included a total of 80 patients of ischaemic stroke and 40 healthy sex and age matched controls. 21 cases of ischaemic stroke and 10 controls were 60-65 years of age, 25 patients and 13 controls belonged to the age group between 65-70 years, whereas 34 patients and 17 controls were above 70 years of age.

Sex Distribution

Table 2.

Sex group.	Cases	Controls
Males	48	24
Females	32	16

Table No.2 shows the sex distribution in the present study. Among the controls 24 were males and 16 were females. Out of 80 cases 60 % (48) patients were males and 40% (32) patients were females.

Blood pressure status Distribution

Hypertension was diagnosed when systolic blood pressure was 140 mmHg or more and diastolic blood pressure was 90 mmHg or more as per the guidelines of seventh US Joint National Committee on prevention, detection, evaluation and treatment of high blood pressure (JNC-7).

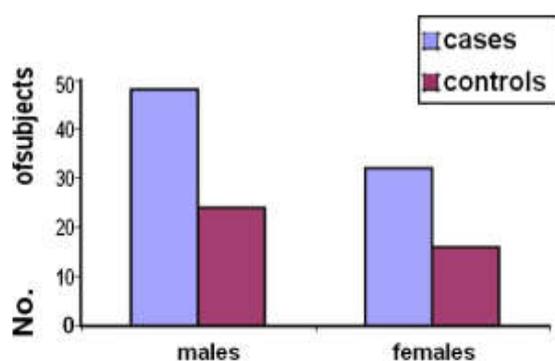


Fig. A.

Table 3.

Blood pressure status	Cases	Controls
Hypertensives	57	16
Non hypertensives	23	24

Table No. 3 shows the blood pressure status distribution in the present study. Among cases 57 were hypertensive and 23 were not suffering from hypertension. Among controls 16 were hypertensive and 24 were non-hypertensive.

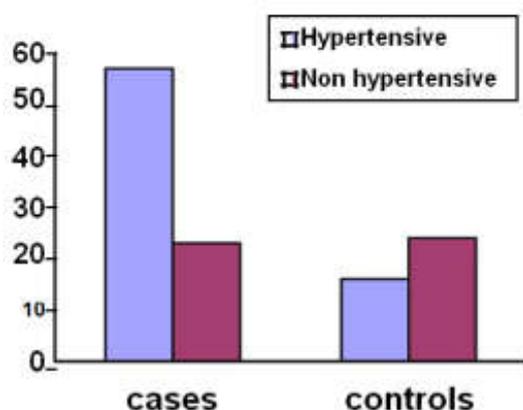


Fig. B. Bar chart showing Blood Pressure status

Smoking Habit distribution

Table 4.

Group	Cases	Controls
Smokers	40	14
Non smokers	40	26

As per Table No 4. Out of 80 cases 40 had a history of smoking and out of 40 controls 14 were smokers.

Lipoprotein a levels distribution in serum

Table 5.

Study Group	Cases	Controls
Mean Plasma Lp(a)	26.7 mg/l	17.04 mg/dl
Observations	80	40

Table No.5 shows the mean plasma Lipoprotein (a) values in cases and controls. The plasma Lp (a) values in the case group ranged from 3.4-98.7 mg/dl with a mean value of 26.7 mg/dl.

In the control group plasma Lp (a) values ranged from 3.5-48 mg/dl with a mean of 17.04 mg/dl. Statistical analysis was done to assess the significance of difference of mean of plasma Lipoprotein(a) values in 80 cases of ischaemic stroke and 40 healthy age and sex matched controls, p value was <0.000 which suggests a statistically significant difference in mean values of plasma Lipoprotein (a) in cases versus controls.

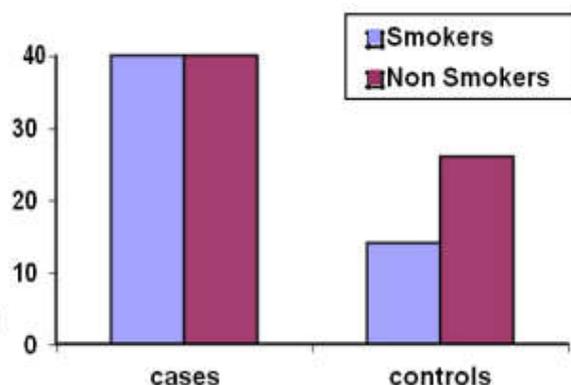


Fig. C.

Lipid Profile Distribution

Lipid profile was evaluated by an enzymatic calorimetric assay. The value of low density lipoprotein cholesterol was derived by using FRIEDEWALD'S

FORMULA: (low density lipoprotein = total cholesterol – high density lipoprotein – [triglycerides/5])

Table 6.

Lipid Profile	Cases (Mean) mg/dl	Controls (Mean) mg/dl
Serum Cholesterol	180.9	154.2
Sr. Triglycerides	108.65	97.9
Sr. HDL	35.80	39.92
Sr. LDL	102.15	91.29

Table No. 6 gives mean blood cholesterol value distribution in the present study. In 80 cases mean serum cholesterol levels were 180.9 mg/dl whereas, in 40 controls mean serum cholesterol levels were 154.2 mg/dl. In cases serum Triglycerides concentration was having a mean value of 108.65 mg/dl whereas in controls, mean value of 97.9 mg/dl was found. Mean serum HDL levels in 80 cases were 35.80 mg/dl and in 40 controls were 39.92 mg/dl. Mean serum LDL levels in 80 cases were 102.15 mg/dl and in 40 controls were 91.29 mg/dl.

BMI Status Distribution

BMI [weight in kg/height in meter²] was calculated and BMI of 25 or more was taken as reference level.

Table 7.

Group	Cases	Controls
<25 kg/sqm	27	17
>25 kg/sqm	53	23

In the present study, 27 cases and 17 controls had BMI values of <25 kg/sqm and 53 cases and 23 controls showed BMI

values of >25 kg/sqm as shown in Table.3. The BMI was below 25 kg/m² in 33.75 % (27) patients in case group as compared to 42.5% (17) in the control group. BMI value of >25 Kg/m² was found in 66.25% (53) of the case group where as it was <25 kg/m² in 57.50% (23) of control group.

Diabetes Mellitus status

Group	Cases	Controls
Diabetics	19	6
Non diabetics	61	34

In this study, 19 cases (23.75%) and 6 controls (15%) were suffering from diabetes mellitus as per the American diabetes association criteria of diagnosing diabetes. 61 cases and 34 controls showed euglycemia.

DISCUSSION

Our study included more number of male patients (60%) than female patients (40%) as supported by the observation of Kurtzke JF (20) who reported that the incidence of atherothrombotic cerebrovascular disease is about 30% higher in males as compared to females. In our study, age range was 60– 90 years, median length of hospital stay (irrespective of outcome) was 15 days (3-30 days), mean Lipoprotein(a) levels in plasma were found to be higher in cases of ischaemic stroke (26.7 mg/dl) as compared to controls (17.04 mg/dl). There was no correlation of serum Lipoprotein (a) levels with the outcome in patients of ischaemic stroke. The results of our study are also in agreement with a study done by Mehndiratta MM *et al* who studied stroke in adults and concluded that in ischaemic stroke patients, 29.4% had cardioembolic strokes followed by atherosclerotic vascular disease in 15.6% subjects. 13 patients had a metabolic etiology, 17.36% of the young patients had no risk factors whereas 27.55 patients had multiple risk factors as hypertension, diabetes, tobacco use and higher cholesterol levels. It has recently been established that Lp (a) has a pivotal role in atherogenesis by facilitating LDL-C oxidation, thus impairing endothelial function and inducing plaque formation. Our results favouring increased lipoprotein (a) level association with metabolic derangements as increased BMI and dyslipidemias has been established in many recent studies. Lindgren *et al* (1992) determined lipid variables in 131 patients six months after their stroke. They found that these patients had higher TG and Lp (a) values and lower TC, LDL and HDL concentrations compared with controls. Haberland ME *et al* (1992) explained that Lp(a) provides a carrier system for LDL-C and promotes cholesterol accumulation in the cells and foam cell formation. Sorensen K.E. *et al* (1994) concluded that Lp(a) has a role in atherogenesis by facilitating LDL-C oxidation, thus impairing endothelial function.

Conclusion

Stroke ranks first in frequency and importance among all adult neurological disorders. Keeping in view the magnitude of morbidity and mortality associated with ischaemic stroke, it seems prudent to search for risk factors that predispose to stroke. After obtaining the findings and evaluating these for statistical significance, we conclude that there is a significant relationship of hypertension, diabetes mellitus, age, BMI and lipid derangements with lipoprotein (a) levels and occurrence

of acute ischaemic stroke as such. Though it is proven and only based on evidence, further long term prospective studies are required in Indian population before we can concretely prove our point.

REFERENCES

- Adams *et al*, Principles of neurology 2005; 777-810.
- Albers, J.J., Marcovina, S.M., Lodge, M.S. 1990. The unique Lipoprotein (a); properties and immunochemical measurement. *Clin Chem.*, 36; 2019-26.
- Baba, Y., Ishikawa, Shizukiyo *et al.*, 2011. high pulse pressure is associated with increased risk of stroke in Japanese: The JMS Cohort Study., 20(1); 10-14.
- Bovet, P., Rickenbach, M., Wietlisbach, V., *et al.* 1994. Comparison of serum Lipoprotein distribution and its correlates among black and white populations. *Int. J Epidemiol.*, 23; 20-27.
- Burchfiel, C.M., Curb, J.D., Rodriguez, B.L *et al.* 1994. Glucose intolerance and 22 year stroke incidence. The Honolulu Heart program. *Stroke.*, 25: 951-57.
- Das, S.K., Biswas, A., Roy, J. *et al.* 2008. Prevalence of major neurological disorders among geriatric population in the metropolitan city of Kolkata. *J Assoc Physicians India*, 56:175-81.
- Dhamija, R.K., Arora, S., Gaba, P., *et al.* 2007. Role of Lipoprotein (a) - a genetic risk factor in patients of acute Ischaemic stroke: a case- control study in North Indian Urban population. *Case Rep Clin Pract Rev.*, 8:112-117.
- Di Carlo, A., Lamassa, M., Pracucci, G., *et al.* 1999. Stroke in the very old: Clinical presentation and determinants of 3 month functional outcome : a European perspective. *Stroke*, 30: 2313-19.
- Di Mascio, R., Marchioli, R., Tognoni, G. 2000. Cholesterol reduction and stroke occurrence: an overview of randomized clinical trials. *Cerebrovasc Dis.*, 10: 85- 92.
- Eikelboom, J.W., Hankey, G.J., Anand, S.S. *et al.* 2000. Association between high homocysteine and ischaemic stroke due to large and small artery disease but not other etiological sub types of ischemic stroke. *Stroke*, 31: 1069-75.
- Hankey, G.J. 1999. Smoking and risk of stroke. *J cardiovasc Risk.*, 6 : 207-211.
- Jacob, S.E., S. Claiborne J. 2003. Thirty-Year Projections for Deaths from Ischemic Stroke in the United States. *Stroke*, 34:2109.
- Kario, K., Matsuo, T., Kobayashi, H., *et al.* 1996. "Silent" cerebral infarction is associated with hypercoagulability, endothelial cell damage, and high Lp (a) levels in elderly Japanese. *Arterioscler Thromb Vasc Biol.*, 16: 734-41.
- Kopito, Jeff, 2001. "A stroke in Time".Merginet.com (number 9) KurtzkeJF. Epidemiologyof cerebrovascular disease. In cerebrovascular survey report for joint council Sub committee on cerebrovascular disease. NINDS and Stroke NHLI (Revised): Whiting Press: 1980; 135-176.
- Lindgren, A., Nilsson-Ehle, P., Norrving, B., *et al.* 1992. Plasma lipids and lipoproteins in subtypes of stroke. *Acta Neurol scand.*, 86: 572-78.
- Mehndiratta, M.M., Puneet, A., Kaushik, S., *et al.* 2004. Stroke in young adults. A Study from University hospital in North India. *Med Sci Monit.*, 10(9): CR535-41.
- National Institute of Neurological disorders and Stroke (NINDS, 1999) Stroke: Hope through research.

- Peng, D.Q, Zhao, S.P., Wang, J.L. 1999. Lipoprotein (a) and apolipoprotein E e4 as independent risk factor in ischaemic stroke. *J Cardiovasc Risk.*, 6: 1-6.
- Phyo K. Myinta, Shubhada S. 2008. Risk factors for first-ever stroke in the EPIC-Norfolk prospective population-based study, *Eur J Cardiovasc Prev Rehabil.*, 15:663– 669
- Prospective studies collaboration. Cholesterol, diastolic blood pressure, and stroke: 13,000 strokes in 45,000 people in 45 prospective cohorts. *Lancet* 1995; 346: 1647-53.
- Ribo, M., Montaner, J., Monasterio, J., Mozinac, 2004. Role of homocysteine in acute phase of stroke. *Neurologia*, 19 (1): 10-14.
- Sivenius, J., Cunha, L., Diener, H.C., *et al.* 1999. Second European Stroke Prevention Study: antiplatelet therapy is effective regardless of age. *Acta Neurologica Scandinavica*, 99; 1; 54–60.
- Smith S. Wade, Hauser, L., Stephen *et al.* 2001. Cerebrovascular diseases Harrison's principles of internal medicine, 15th ed ; Vol. 2, 2369-90.
- Sorensen, K.E., Celermajer, D.S., Georgakopoulos, *et al.* 1994. Impairment of endothelium dependent dilation is an early event in children with familial hypercholesterolemia and is related to the Lipoprotein (a) level. *J Clin Invest.*, 93:50-55.
- Sujata, D., Avijit, H., Biman, K.R. *et al.* 2010. Burden among Stroke Caregivers. Results of a Community-Based Study From Kolkata, India. *Stroke*, 41:2965.
- Teasdale, T.W., Engberg, A.W. 2001. Suicide after a stroke: a population study. *J Epidemiol Community Health*, 55:863-866.
- Thompson, J.E. 1996. the evolution of surgery for the treatment and prevention of stroke. The Willis Lecture. *Stroke* 27(8): 1427-34.
- Van Kooten, F., Van Krimpen, J., Dippel, D.W., *et al.* 1996. Lipoprotein (a) in patients with acute cerebral ischaemia. *Stroke*, 27: 1231-35.
- Vavernova, H., Novotny, D., Ficker, L., *et al.* 1993. Lipoprotein (a): a genetic risk factor for early ischaemic cerebrovascular stroke. *Vnitř Lek.*, 39: 979-87.
- Vogel, J. 1994. 5 year's follow-up study of stroke patients over 65 years of age. *Rehabilitation*, Aug; 33(3):155-7.
- Warlow, C.P., Dennis, M.S., Van Gijn, J., *et al.* 2000. A practical guide to management. Oxford: Blackwell scientific publications *Stroke.*, 34; 45-47.
- Yannick, B., Olivier, R., Agnès, J., *et al.* 2010. Stroke in the Very Old: Incidence, Risk Factors, Clinical Features, Outcomes and Access to Resources – A 22-Year Population-Based Study. *Cerebrovasc D.*, 29:111-121.
