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A Study of Clinical Profile and Etiological Factors of Stroke in Age Group 18-60 Years: A Study from Tertiary Care Centre in India

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Abstract

Background and Objective: Stroke in adults aged 18-60 years presents unique clinical and etiological profiles. This study aims to examine the clinical presentations and identify various etiological factors contributing to stroke in this age group**.**

Methodology: A prospective observational study was conducted in the Department of General Medicine, Balrampur Hospital, Lucknow, from September 2019 to December 2020. A total of 144 patients, aged 18-60 years with a first-ever stroke (ischemic, haemorrhagic, or cerebral venous thrombosis), were included. Stroke subtypes in ischemic cases were classified according to TOAST criteria. Detailed clinical evaluations, including history, examination, and relevant investigations (basic and specific, such as vasculitis and prothrombotic workups), were performed. Stroke outcomes were assessed using the National Institute of Health Stroke Scale (NIHSS) and the modified Rankin Scale (mRS). Data analysis was conducted with SPSS 15.0, with significance set at $p < 0.05$.

Results: Among the 144 cases, 68.7% were ischemic, 13.2% haemorrhagic, and 18.1% cerebral venous thrombosis (CVT). Large-artery atherosclerosis was the predominant ischemic subtype (47.5%), followed by small-vessel occlusion (27.3%). Ischemic strokes were most frequently associated with hypertension, dyslipidemia, and alcohol use, while haemorrhagic strokes were linked with hypertension and low hemoglobin levels. CVT was more prevalent in younger patients, especially females, with hyperhomocysteinemia and protein S deficiency being common findings. Migraine was a notable risk factor in a subset of ischemic strokes. Haemorrhagic strokes predominantly presented as lobar bleeds, with females affected disproportionately.

Patients with CVT had the best functional outcomes, with a high rate of recovery across stroke types**.**

Conclusion: This study highlights ischemic strokes as the most common type among young adults, followed by haemorrhagic strokes and CVT. Key risk factors include hypertension, dyslipidemia, alcohol consumption, and prothrombotic states. The findings underscore the need for targeted interventions addressing lifestyle-related risks and coagulation abnormalities in young adults to improve stroke prevention and outcomes**.**

Keywords: Young stroke, Clinical profile, Stroke etiology, Ischemic stroke, Haemorrhagic stroke, Cerebral venous thrombosis (CVT).

Introduction

Stroke is a leading cause of morbidity and mortality worldwide, defined by the World Health Organization (WHO) as an event caused by the interruption of blood supply to the brain, often due to a blood vessel rupture or blockage. This disruption deprives brain tissue of oxygen and nutrients, leading to neuronal damage[1]. The most common symptom of stroke, observed in approximately 90% of patients, is sudden weakness or numbness in the face, arm, or leg, typically on one side of the body[2]. Other symptoms include confusion, difficulty in speaking or understanding speech, vision problems, loss of balance or coordination, severe headache, and, in extreme cases, fainting or unconsciousness. The clinical presentation of a stroke depends on the affected area of the brain and the severity of the damage. In severe cases, stroke can lead to sudden death.

Globally, stroke is a significant cause of death and disability, with ischemic strokes accounting for up to 80% of cases[3]. In 1999, WHO reported approximately 5.54 million stroke-related deaths worldwide, with the majority occurring in less developed countries. In India, stroke prevalence ranged from 136 to 220 per 100,000 population in the last decade[4-7]. Historically, epidemiological studies on stroke have largely focused on developed countries, but the rising burden of hypertension, changing lifestyles, and population aging suggest an increasing incidence of stroke in developing nations[8].

Data on stroke in patients first appeared in the 1940s-1950s through small case series and has expanded notably in the past two decades, largely due to advances in diagnostics and patient evaluation[9].

Stroke etiology in patients is highly diverse, with the exact cause unknown in up to one-third of cases. Common risk factors are significant but often complicated by cryptogenic, cardioembolic, and venous strokes. This diversity makes diagnosis in this age group more challenging[10].

The pathophysiology of stroke in younger adults can be categorized into five basic principles[3]: (a) the causes of stroke are distinct from those in older populations, with less frequent large-artery atherosclerosis and atrial fibrillation; (b) risk factors for stroke differ significantly between adults and the elderly; (c) stroke in younger patients often leads to significant socio-economic consequences due to the loss of productivity; (d) genetic causes may play a larger role in this population, suggesting a need for genetic counselling; and (e) effective treatment and prevention in younger adults can yield more quality-adjusted life years than in the elderly.

Despite its importance, stroke in the patients has long been understudied and current data originate mostly from small patient series.[9,11,12]

Studies show that 21-48% of strokes in patients are due to atherosclerotic large artery occlusion, 10-33% to non-atherosclerotic large artery occlusion (including 10-20% dissections), 13-35% to cardioembolism, 3-18% to small artery disease, 8-15% to prothrombotic states, and 4-15%

to other causes. Cryptogenic strokes account for 7-40% of cases[10].

A study by Lee et al. found that vascular risk factors, such as hypertension, diabetes, and hyperlipidemia, were more common in patients with large artery atherosclerotic and small vessel occlusive diseases[13].

Simply identifying cerebral infarction as the cause of neurological deficit in stroke patients is no longer adequate. Advances in technology and knowledge increase the chances of uncovering underlying causes. The approach to investigating stroke in these patients is similar to that in older adults, though additional studies focusing on cardiac, hematologic (such as hypercoagulable states), infectious, inflammatory, and metabolic disorders are often required[14].

Ischemic stroke in adults has numerous and varied causes, often requiring extensive investigation to identify underlying issues. A thorough search is essential, as many underlying disorders are treatable. Key treatable causes include extracranial arterial dissection, cardio embolism, premature atherosclerosis, hematologic and immunologic disorders, and migraine. Drug abuse is a growing risk factor, while stroke risk during pregnancy remains unclear. Isolated CNS angiitis, heritable connective tissue disorders, and other genetic disorders (like mitochondrial cytopathies) account for a small number of cases. If a full investigation finds no clear cause, the future stroke risk is generally low[14].

This study recruited stroke patients aged 18-60 years from Balrampur Hospital. They were examined and investigated according to standard protocols, and ischemic strokes were sub-classified using the TOAST criteria[15].

In this study, we also investigated stroke patients for thrombophilic states, which predispose to thrombosis. While thrombophilia is a known cause of venous thrombosis, it rarely causes arterial occlusions. Even in those with a positive thrombophilia screen and arterial thrombosis, thrombophilia may not be the primary cause[16].

Detecting thrombophilic disorders aids in management, prognosis, family screening, and possible prevention. These disorders are classified as inherited or acquired, including deficiencies in natural anticoagulants (e.g., protein C, protein S, antithrombin III) and genetic mutations like factor V Leiden and prothrombin gene 20210G/A variant[16].

Material and Methods

Study Design: This prospective observational study was conducted in the OPD ward and ICU of the General Medicine Department at Balrampur Hospital, Lucknow. It aimed to examine the clinical presentations and etiological factors in stroke patients aged 18-60 years.

Study Population: The study included all patients aged 18-60 with a first-ever stroke (ischemic, haemorrhagic, or cerebral venous thrombosis). Ischemic strokes were sub-classified according to TOAST criteria.

Study Duration: September 2019 to December 2020.

Inclusion Criteria

- First-ever stroke patients (ischemic, haemorrhagic, cerebral venous thrombosis) aged 18-60.
- Ischemic stroke subtypes included: large-artery atherosclerosis, cardio-embolism, small-vessel occlusion, other determined etiology, and undetermined etiology.

Exclusion Criteria

- Stroke patients under 18 or over 60 years.
- Recurrent strokes.
- Transient Ischemic Attack (TIA).

Sample Size: Based on an estimated 16% incidence of young stroke in India, with a 5% alpha error and 8% margin, a sample size of 100 was computed. Ultimately,

144 eligible patients presented during the study period and were included.

Study Protocol: All cases underwent comprehensive history-taking, clinical examination, and both basic and specific investigations, including radiological workups, vasculitis and prothrombotic workups. Outcomes were assessed on follow-up using the NIH Stroke Scale (NIHSS) and modified Rankin Scale (mRS) scores.

Data Analysis: All data were entered into a Microsoft Excel spreadsheet and analysed using SPSS 15.0 for Windows. Continuous numerical data were summarized as means, standard deviations, medians, minimum, maximum, and standard error of the mean. Means were compared across three groups with ANOVA, and distributions with Pearson's Chi-Square Test. A p-value of < 0.05 was considered statistically significant.

Parameters Studied

- (i) Clinical features of stroke in patients aged 18-60 years
- (ii) Etiological factors of stroke in patients aged 18-60 years

Study Format

- Patient's name, address, and personal details were recorded.
- Detailed history and examination were conducted at initial presentation.
- Significant alcohol use was defined as >70 gm per week, and a smoker had a history of >1 pack-year of smoking.
- High altitude area (HAA) was defined as over 9,000 feet above sea level.
- Normal blood pressure was set at 140/90 mmHg.
- Baseline tests included Hb, TLC, lipid profile, RBS, platelets, PT, INR, chest X-ray, and ECG.

Specific investigations were conducted based on initial presentation and baseline findings, including:

- **CT scan**: Performed with the Philips Brilliance CT 16-slice system, providing high-quality images and fast 0.4-sec scan times.
- **MRI, MRA, and MRV scans**: Performed with the Siemens Harmony 1-Tesla machine for brain and neck imaging.
- **Color Doppler**: Conducted with the Wipro GE Logic PS ultrasonic system.
- **2D Echocardiography**: Performed using the Philips iE33 intelligent diagnostic ultrasonic system.

Vasculitis Workup

- **Anti-Nuclear Antibody/Factor (ANA/ANF)**:Useful in diagnosing autoimmune diseases like SLE, rheumatoid arthritis, and CREST syndrome. Low-titer ANA positivity can increase with age.
- *Procedure*: 2 ml of serum collected after overnight fasting and processed by enzyme immunoassay.
- **C-Reactive Protein (CRP)**: A sensitive acute-phase reactant for inflammation, with levels rising sharply after trauma, infection, or inflammation.
- *Procedure*: 2 ml of serum collected after overnight fasting and processed by immune-turbidometry.
- **Rheumatoid Factor**: An IgM antibody found in conditions like rheumatoid arthritis, SLE, and chronic illnesses.
- *Procedure*: 2 ml of serum collected after overnight fasting and processed by enzyme immunoassay.
- **Erythrocyte Sedimentation Rate (ESR)**: Indicates inflammatory activity but is not specific to any disease. Useful in monitoring inflammatory disease progression.
- *Procedure*: 3 ml of whole blood collected after overnight fasting and processed by capillary photometry.

Prothrombotic Testing Summary

- **VDRL and HIV Tests**: Conducted in selected cases.

- **Prothrombotic Factors**: Tested in all cerebral venous thrombosis (CVT) cases and ischemic strokes without hypertension or diabetes at Lal's Pathology National Reference Lab, New Delhi (CAP, ISO, and NABL accredited).
- **Homocysteine**: A sulfur-containing amino acid linked to vascular disorders. Normal range: 5.46-16.2 µmol/l.
- *Procedure*: 2 ml serum collected after fasting, processed by chemiluminescent immunoassay.
- **Protein S, Functional**: Evaluates congenital/acquired protein S deficiency, indicating risk of thrombosis. Normal range: 70-140%.
- *Procedure*: 4 ml platelet-poor plasma in two sodium citrate tubes, prepared within 1 hour after fasting, processed by electromechanical clot detection.

Prothrombotic Testing Overview:

- **Protein C, Functional**: Screens for congenital protein C deficiency, particularly in patients with a family history of thrombosis.
- *Normal Range*: 73-143%.
- *Procedure*: 4 ml platelet-poor plasma (PPP) in sodium citrate tubes, processed by chromogenic assay.
- **Antithrombin III**: Diagnoses acquired or congenital antithrombin deficiency.
- *Normal Range*: 80-120%.
- *Procedure*: 4 ml PPP, processed by chromogenic assay.
- **Factor V Leiden Mutation Analysis**: Detects Factor V Leiden mutation causing resistance to degradation by activated protein C.
- *Procedure*: 5 ml whole blood in EDTA, processed by real-time PCR.
- **Antiphospholipid Antibodies (IgG, IgM)**: Tests for unexplained arterial/venous thrombosis or other conditions like recurrent fetal loss, livedo reticularis, or SLE.
- *Normal Range*: 0.50-10 MPL U/ml for IgM; 0.50-10 GPL U/ml for IgG.
- *Procedure*: 2 ml serum after fasting, processed by enzyme immunoassay.

Result and observation

In the study, 144 stroke patients were analyzed. Among them, 99 (68.7%) had ischemic strokes, 19 (13.2%) had hemorrhagic strokes, and 26 (18.1%) were diagnosed with cerebral venous thrombosis (CVT).[figure 1]

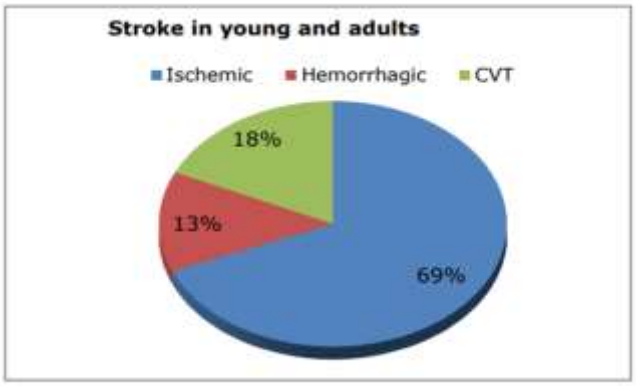
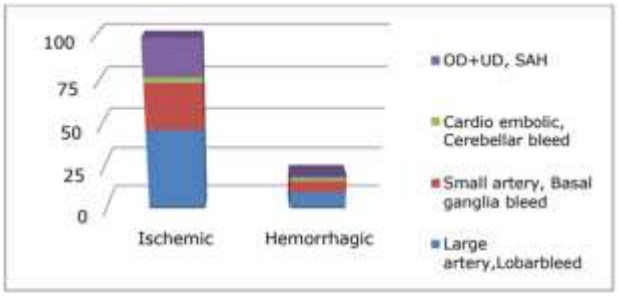


Figure 1:

Out of 99 ischemic group, 65 (65.7%) strokes were in anterior circulation, 18 (18.2%) strokes were in posterior circulation (Figure 2), 16 (16.1%) involved both anterior and posterior circulations.

According to TOAST criteria out of 99 ischemic stroke subtypes 47 (47.5%) were large artery strokes, 27 (27.3%) were small artery strokes, 3 (3.0%) were cardio-embolic strokes, 18 (18.2%) were strokes of other determined causes (16 with prothrombotic states, 4 with normal MRI, 6 with migraines, and 1 each with SLE and malignancy), 4 cases were of undetermined etiology.[figure 2]



OD- stroke of other determined etiologies
UD- strokes of undetermined origin, where no cause was found despite an extensive evaluation or a most likely cause could not be determined because more than one plausible cause was found.
SAH – subarachnoid hemorrhage

Figure 2:

Among the 19 haemorrhagic stroke cases (13.2% of the total) 8 (47.1%) were lobar bleeds, 7 (41.2%) were basal ganglia bleeds, 2 (11.8%) were cerebellar bleeds. No cases of thalamic bleeds were observed.[figure 2]

The mean age for different stroke types showed significant differences, in Cerebral Venous Thrombosis (CVT) mean age was 32.54 ± 7.11 years, indicating a younger demographic, Ischemic Strokes mean age was 40.08 ± 6.38 years (p = 0.000), haemorrhagic Strokes mean age was 41.00 ± 6.73 years (p = 0.000). This highlights that CVT cases predominantly occurred in a younger population compared to ischemic and haemorrhagic strokes.

Haemorrhagic strokes occurred in 26.3% of females, ischemic strokes in 7.1% of females, and CVT in 3.8% of females. [figure 3]

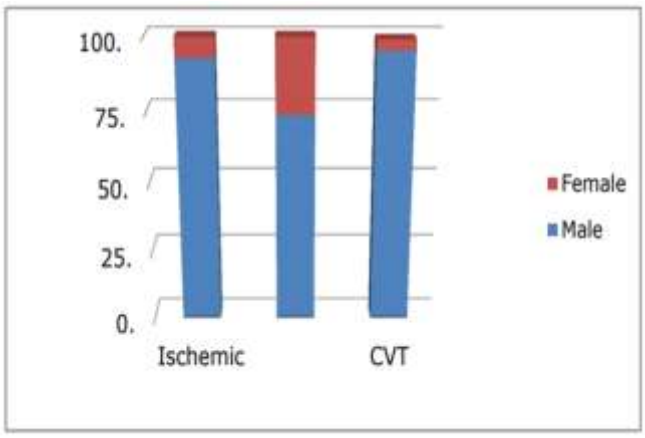


Figure 3:

Hemiplegia was the most common symptom in 83 (83.8%) ischemic stroke cases. Facial weakness occurred in 68 (68.7%), speech difficulty in 66 (66.7%), vomiting in 21 (21.2%), and seizures at onset in 6 (6.1%) cases.

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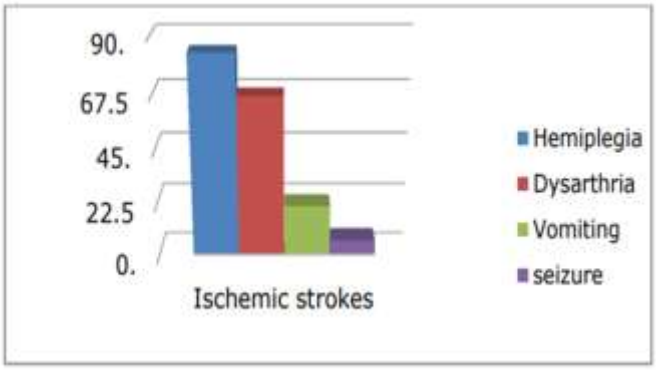


Figure 4:

Haemorrhagic strokes presented with hemiplegia in 14 (73.7%) cases, facial weakness in 13 (68.4%), vomiting in 11 (57.9%), headache in 10 (52.6%), and altered sensorium in 9 (47.4%). Seizures occurred in 3 (15.7%) cases at onset. [figure 5]

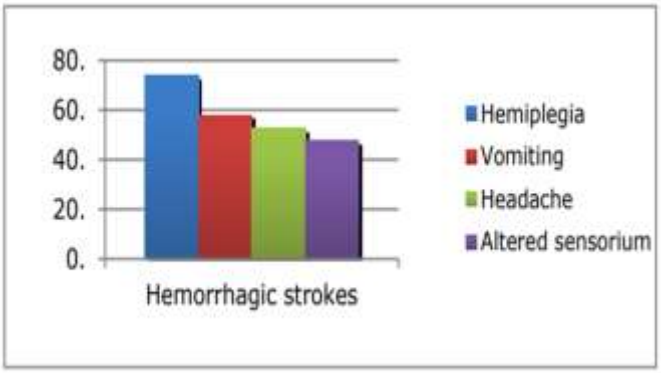


Figure 5:

CVT presented as headache in 24 (92.3%) cases, the most common symptom, followed by vomiting in 17 (65.4%) cases. Seizures occurred in 11 (42.3%) cases,

and 6 (23.1%) patients had hemiplegia at presentation. [figure 6]

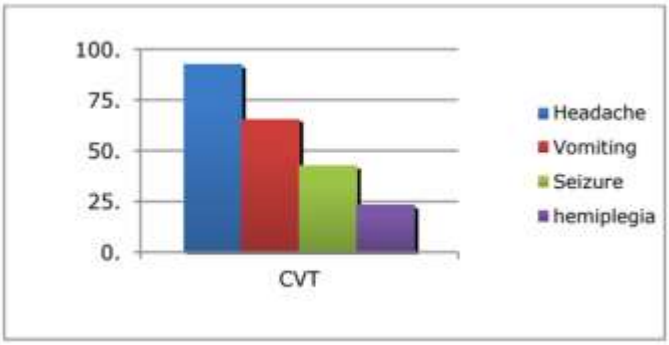


Figure 6:

Summary of above common symptoms-

	Ischemic strokes	Hemorrhagic strokes	CVT
Hemiplegia	83.8%	73.7%	23.1%
Dysarthria	66.7%	52.1%	19.2%
Vomiting	21.2%	57.9%	65.4%
Seizure	6.1%	15.7%	42.3%
Headache	49.5%	52.6%	92.3%

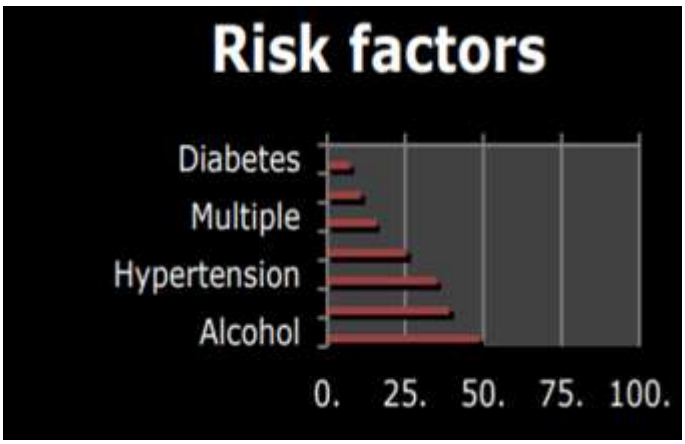
The mean systolic and diastolic blood pressures were higher in haemorrhagic strokes (156.42 ± 21.93 mmHg and 95.47 ± 14.27 mmHg) compared to ischemic strokes (134.44 ± 21.67 mmHg and 86.83 ± 13.04 mmHg) and CVT (126.81 ± 10.83 mmHg and 80.38 ± 10.46 mmHg). This difference was statistically significant ($p < 0.001$ for systolic, $p = 0.010$ for diastolic between ischemic and haemorrhagic strokes; $p < 0.001$ for both systolic and diastolic between CVT and haemorrhagic strokes). [table 1]

Table 1:

	Ischemic strokes	Hemorrhagic strokes	CVT	Test Used independent Sample t test		
	Mean ± Std. Deviation	Mean ± Std. Deviation	Mean ± Std. Deviation	P Value between ischemic & hemorrhagic strokes	P Value between CVT & ischemic strokes	P Value between CVT & hemorrhagic strokes
Systolic blood pressure	134.44 ± 21.67	156.42 ± 21.93	126.81 ± 10.83	<0.001	0.085	<0.001
Diastolic blood pressure	86.83 ± 13.04	95.47 ± 14.27	80.38 ± 10.46	0.010	0.022	<0.001

Alcohol consumption was found in 70 (48.6%) cases, smoking in 36 (25%) cases, hypertension in 50 (34.7%), diabetes in 10 (6.9%), obesity in 15 (10.4%), and dyslipidemia in 56 (38.8%). Multiple risk factors were present in 22 (15.3%) cases.

Table 2: risk factors in percentage



Diabetes was found in 10 (10.1%) ischemic stroke cases, but none had diabetes in CVT or haemorrhagic stroke cases. This was not statistically significant ($p = 0.091$ and $p = 0.148$). [table 3] Hypertension was present in 12 (63.2%) haemorrhagic stroke cases, 38 (38.4%) ischemic stroke cases, and absent in CVT cases. Hypertension was statistically significant for haemorrhagic and ischemic strokes compared to CVT ($p < 0.001$). [table 4]. Alcohol consumption was found in 11 (42.3%) CVT, 10 (52.6%) haemorrhagic, and 49 (49.5%) ischemic stroke cases. Smoking was present in 2 (7.7%) CVT, 3 (15.8%) haemorrhagic, and 31 (31.3%) ischemic stroke cases.

However, alcohol and smoking were not significant risk factors for any stroke type (p > 0.555). [table 5]

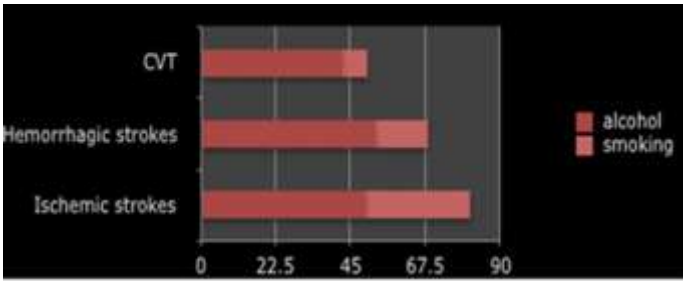
Table 3**:**

		Strokes			Total	P Value between Ischemic & Haemorrhagic strokes	P Value between CVT & Ischemic strokes	P Value between CVT & Haemorrhagic strokes
		Ischemic	Hemorrhagic	CVT				
DM	No	89	19	26	134	0.148	0.091	NA
		89.9%	100.0%	100.0%	93.1%			
	Yes	10	0	0	10			
		10.1%	.0%	.0%	6.9%			
Total		99	19	26	144			
		100.0%	100.0%	100.0%	100.0 %			

Table 4:

		Strokes			Total	P Value between Ischemic & Haemorrhagic strokes	P Value between CVT & Ischemic strokes	P Value between CVT & Haemorrhagic strokes
		Ischemic	Hemorrhagic	CVT				
HTN	No	61	7	26	94	0.045	<0.001	<0.001
		61.6%	36.8%	100.0%	65.3%			
	Yes	38	12	0	50			
		38.4%	63.2%	.0%	34.7%			
Total		99	19	26	144			
		100.0%	100.0%	100.0%	100.0%			

Table 5:



Hemoglobin levels were lowest in haemorrhagic strokes (12.8±1.92 gm/dl), followed by ischemic strokes (13.7±1.77 gm/dl), and CVT (14.3±1.62 gm/dl). Lower hemoglobin levels were significantly associated with haemorrhagic strokes (p = 0.010), while higher levels were linked to CVT compared to ischemic strokes (p = 0.035).[table 6]

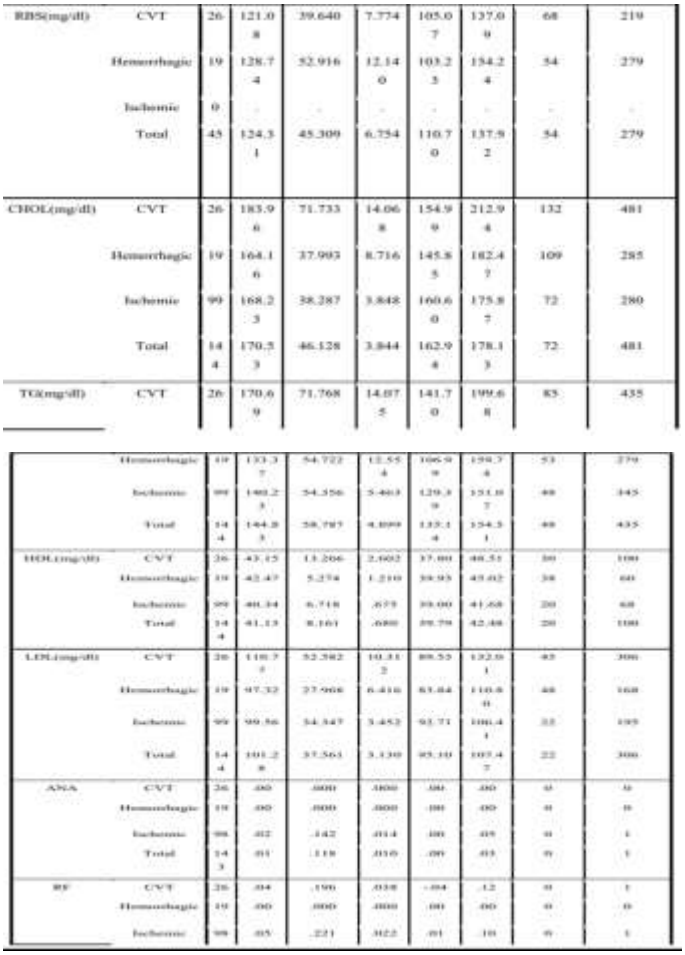
Platelet counts were similar across stroke types, with ischemic strokes having a mean of 2.29±0.74 lacs/mm³, CVT 2.07±0.74 lacs/mm³, and haemorrhagic strokes 2.10±0.65 lacs/mm³. No significant differences were found (p > 0.05). [table 6]

Blood sugar levels did not show significant differences at presentation (p > 0.05). [table 6]

Dyslipidemia analysis showed higher triglyceride levels in CVT (170.69±71.73 mg/dl) compared to ischemic strokes (140.23±54.36 mg/dl, p = 0.034) and haemorrhagic strokes (133.37±54.72 mg/dl, p = 0.018). Higher triglycerides were significantly associated with CVT (p = 0.040). Cholesterol levels were higher in CVT (183.96±71.3 mg/dl), but differences were not statistically significant (p = 0.246). HDL and LDL levels did not show significant differences across stroke types (p = 0.221 and p = 0.356, respectively). [table 6]

Table 6:

		N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
						Lower Bound	Upper Bound		
AGE	CVT	26	32.54	7.112	1.395	29.67	35.41	22	45
	Hemorrhagic	19	41.00	6.723	1.543	37.76	44.24	28	48
	Ischemic	99	40.08	6.382	.641	38.81	41.35	22	49
	Total	144	38.84	7.166	.597	37.66	40.02	22	49
Hb(gm/dl)	CVT	26	14.23 4	1.6226	.3182	13.58 8	14.88 9	8.4	17.0
	Hemorrhagic	19	12.86 3	1.9394	.4449	11.92 8	13.79 8	8.2	16.0
	Ischemic	99	13.74 3	1.7697	.1779	13.39 0	14.09 6	8.0	18.2
	Total	144	13.71 9	1.7972	.1498	13.42 3	14.01 5	8.0	18.2
Platelets (Lacs/mm³)	CVT	26	2.073 9	.74854	.14680	1.771 1	2.375 8	1.00	3.88
	Hemorrhagic	19	2.100 0	.65939	.1512 8	1.782 2	2.417 8	1.30	4.00
	Ischemic	99	2.292 9	.74722	.07511 0	2.143 9	2.442 0	.89	4.42



RBS(mg/dl)	CVT	26	121.08	39.640	7.774	105.07	137.09	68	219
	Hemorrhagic	19	128.74	52.916	12.140	103.23	154.24	54	279
	Ischemic	0	-	-	-	-	-	-	-
	Total	45	124.31	45.309	6.754	110.70	137.92	54	279
CHOL(mg/dl)	CVT	26	183.96	71.733	14.068	154.99	212.94	132	481
	Hemorrhagic	19	164.16	37.993	8.716	145.85	182.47	109	285
	Ischemic	99	168.23	38.287	3.848	160.60	175.87	72	280
	Total	144	170.53	46.128	3.844	162.98	178.13	72	481
TG(mg/dl)	CVT	26	170.69	71.768	14.075	141.70	199.68	85	435
	Hemorrhagic	19	133.37	54.722	12.554	106.99	159.74	53	279
	Ischemic	99	140.23	54.356	5.463	129.39	151.07	49	345
	Total	144	144.83	58.787	4.899	135.14	154.51	49	435
HDL(mg/dl)	CVT	26	43.15	13.206	2.592	37.80	48.51	30	100
	Hemorrhagic	19	42.47	5.274	1.210	39.93	45.02	34	60
	Ischemic	99	40.34	6.718	.675	39.00	41.68	20	68
	Total	144	41.13	8.161	.680	39.79	42.48	20	100
LDL(mg/dl)	CVT	26	110.73	52.582	10.312	89.53	132.01	43	306
	Hemorrhagic	19	97.32	27.906	6.416	83.84	110.80	48	168
	Ischemic	99	99.86	34.347	3.452	93.71	106.41	33	195
	Total	144	101.28	37.563	3.130	95.10	107.47	22	306
ANA	CVT	26	.00	.000	.000	.00	.00	0	0
	Hemorrhagic	19	.00	.000	.000	.00	.00	0	0
	Ischemic	98	.02	.142	.014	.00	.05	0	1
	Total	143	.01	.118	.010	.00	.03	0	1
RF	CVT	26	.04	.196	.038	-.04	.12	0	1
	Hemorrhagic	19	.00	.000	.000	.00	.00	0	0
	Ischemic	99	.05	.221	.022	.01	.10	0	1

Vasculitis Workup

- **Antinuclear Antibodies**: Positive in 2 (2.02%) ischemic stroke cases, none in CVT or haemorrhagic stroke (p = 0.633).
- **Rheumatoid Factor**: Positive in 5 (5.05%) ischemic stroke cases, 3.85% in CVT, none in haemorrhagic stroke (p = 0.600).
- **C-Reactive Protein**: Positive in 15.2% of ischemic strokes, 15.4% of CVT, and 5.3% of haemorrhagic strokes (p = 0.506).
- **VDRL**: Non-reactive in all cases.

Prothrombotic State

- In ischemic strokes, 64 patients tested: 47 (73.4%) had hyper-homocysteinemia, 44 (68.3%) had protein S deficiency, and 36 (56.3%) had multiple prothrombotic states.
- In CVT, 24 patients tested: 21 (87.5%) had hyper-homocysteinemia, 16 (66.7%) had protein S deficiency, and 17 (70.8%) had multiple prothrombotic states.

Statistical Comparisons

- **Homocysteine**: Higher in CVT (29.43±20.02 µmol/l) compared to ischemic strokes (23.18±10.97 µmol/l), but not significant (p = 0.069).
- **Factor V Leiden Mutation**: More common in CVT (12.5%) than ischemic strokes (6.3%), but not significant (p = 0.462).
- **Protein S**: Lower in CVT (54.6±25.3%) than ischemic strokes (60.47±26.71%), with no significant difference (p = 0.373).
- **Protein C**: Similar levels in both CVT (98.29±36.27%) and ischemic strokes (98.13±25.89%), with no significant difference (p = 0.984).
- **Antithrombin III**: Lower in ischemic strokes (96.36±17.90%) compared to CVT (104.42±22.34%), but not significant (p = 0.092).

Antiphospholipid antibodies were negative in both ischemic strokes and CVT.

Discussion

This prospective observational study was conducted in the OPD, ward, and ICU at the Department of General Medicine, Balrampur Hospital, Lucknow. Its primary aim was to examine the clinical presentations and etiological factors of strokes in patients aged 18-60 years. Patients were followed up throughout their hospital stay to assess outcomes, mortality, morbidity, and complications. Data collected during the study were analysed to understand the patterns of stroke in this age group.

This study had a higher male-to-female ratio of 10:1, differing from previous research on young stroke patients. For example, Putaala et al[3]. reported a ratio of

1.7:1, while Razzaq et al[31]. found a ratio of 1.8:1. Interestingly, this study revealed a greater proportion of females in the haemorrhagic stroke group compared to ischemic stroke and cerebral venous thrombosis (CVT). Furthermore, CVT was more prevalent in the younger age group compared to ischemic and haemorrhagic strokes, a trend consistent with other studies highlighting the relative youthfulness of CVT patients.

In this study, highest incidence of ischemic strokes (69%) was found as compared to haemorrhagic strokes (13.2%) and CVT (18.1%), which is corroborated by previous studies from Asia and other parts of the world[3,33,35,36,38]

According to the TOAST criteria for ischemic stroke subtypes, most cases in this study were classified as large artery strokes (47.5%), which is consistent with findings from other.[10,17] South Asian study[43] where only 12.6% of ischemic strokes were large artery strokes, and a decade-old Baltimore-Washington[46], which reported just 3.8% of large artery strokes. The possibility that some of artery strokes in this study could have been due to arterial dissection remains unconfirmed, as conventional angiography was not used.

This study found 27.3% of cases to be small artery strokes, higher than the 13.8% reported by Putaala et al. 3. Another study of Southeast Asians in the UK found only 7% had lacunar strokes[43].

In this study, 18.2% of cases had another determined etiology, lower than the 25.7% found by Putaala et al. 3 and higher than the 11.2% in a study of Southeast Asians in the UK. 6.1% of patients had migrainous strokes, compared to 1.4% in the Baltimore Washington study and 1-2% according to the WHO. However, a case series from Rome reported 26% of young ischemic stroke patients had migraine.[47]

This study found 3% cardio-embolic strokes, which is lower than the incidence found in previous studies [3, 10, 13, 44, 46]. This differs from a recent study in Pakistan, where 20% of young stroke patients had cardio-embolic strokes.[44.]

In this study, 4.04% of cases had an undetermined etiology, lower than the 22.4% in the Helsinki Young Stroke Registry and 7-40% in other studies[10]. A Southeast Asian study found 23.4%, while two stroke centre-based studies reported 33%. The lower percentage in this study may be due to more extensive evaluation of prothrombotic states, reducing the cases classified as undetermined etiology.[13,52]

In this study, most haemorrhagic strokes were lobar bleeds, followed by putaminal bleeds, similar to findings by Ruiz-Sandoval et al[53]., who reported 55% lobar bleeds in intracerebral haemorrhage (ICH) patients. This study also showed a higher incidence of haemorrhagic strokes in females compared to ischemic strokes and CVT.

In this study, the clinical presentations of stroke in young patients were similar to those seen in older individuals, reflecting the anatomical distribution of brain damage. Hemiplegia was the most common presentation in ischemic and haemorrhagic strokes, while headache was the predominant symptom in CVT, as also observed in a study by Razzaq et al[31].

In this study, significantly higher systolic and diastolic blood pressures were found in ischemic and haemorrhagic strokes compared to CVT. Haemoglobin levels were significantly lower in haemorrhagic strokes than in ischemic strokes and CVT, consistent with previous research linking anemia to stroke risk in young individuals[17]. Additionally, higher haemoglobin levels were associated with CVT, which aligns with data suggesting polycythemia as a known risk factor for CVT[55].

This study found that common stroke risk factors in adults were dyslipidemia (38.8%), hypertension (34.7%),

multiple risk factors (15.3%), and diabetes (10.4%). Previous studies, such as those by Nayak et al[39]. (42%) and Sridharan et al.[42] (highlighting hypertension, dyslipidemia, and diabetes as significant risk factors), support these findings. Lee et al. reported hyperlipidemia in 53.1% and hypertension in 45.8% of stroke cases. A study in Switzerland[45] found hypercholesterolemia in 39% and hypertension in 19% of young stroke patients. The Helsinki Young Stroke Registry also noted multiple causes in 21 out of 1008 cases. Similarly, a study from Pakistan[44] identified hypertension in 14% and miscellaneous causes in 4% of young stroke cases. This study also supports diabetes as a risk factor primarily for ischemic stroke, as noted in prior research[3,13,42].

In this study we found alcohol consumption in 48.6% and smoking in 25% of strokes in young. Previous studies also showed that alcohol and smoking were important risk factors. shorten sentences.

In this study we found alcohol consumption in 48.6% and smoking in 25% of strokes in young. Previous studies also showed that alcohol and smoking were important risk factors. shorten sentences[13,39,40,45].

A significant number of patients without classical cardiovascular risk factors exhibited prothrombotic states in both CVT and ischemic strokes, indicating these factors are important stroke risks in young people in India. This finding aligns with past studies on stroke in young populations, highlighting the relevance of prothrombotic conditions as key contributors[3,10,13,17,43]

In our study, high homocysteine levels were found in 87.5% of CVT cases and 73.4% of ischemic strokes. The ICMR workshop on stroke in 2006, supported by the WHO, reported that 54.6% of young stroke patients in the Guwahati Stroke Registry had elevated homocysteine levels. However, a study from Pakistan identified hyper-homocysteinemia in only 4% of cases, suggesting regional variations in prevalence[44]

In this study, protein S deficiency was found in 68.3% of ischemic stroke cases and 66.7% of CVT cases. This is much higher than previous studies, which reported isolated protein S deficiency in about 10% of young ischemic stroke cases. For example, Lee et al. found protein S deficiency in 6.8% of young ischemic stroke patients[13].

In this study, protein C deficiency was observed in 12.5% of ischemic strokes and 29.2% of CVT cases. This is higher than findings from Nedeltchev et al[45]., who reported a 0.5% incidence of protein C deficiency in young ischemic strokes, and Lee et al.[13], who found 2.5% of young ischemic stroke cases with protein C deficiency.

In this study, antithrombin III deficiency was found in 12.5% of ischemic strokes and 8.3% of CVT cases. This is higher than the 1.9% incidence reported by Lee et al. in young ischemic strokes[13].

In this study, factor V Leiden mutation was found in 6.3% of ischemic strokes and 12.5% of CVT cases, which is higher than the 0.9% incidence noted by Nedeltchev et al. and in two stroke centre-based studies[52]

In our study, haemorrhagic strokes presented with greater disability (mean NIHSS 8.74, mean mRS 3.18) but showed significant recovery in most cases. CVT patients had the best outcomes (mean NIHSS 0.29, mean mRS 0.08), which aligns with findings from global studies on stroke outcomes[45,66,67].

Summary and Conclusion

This prospective observational study at Balrampur Hospital, Lucknow, aimed to analyse clinical presentations and etiological factors of stroke in patients aged 18-60 years. All cases were followed throughout their hospital stay to assess outcomes, mortality, morbidity, and complications. Key findings include:

1. The study was male-dominated, likely due to referral bias at the tertiary care centre,
2. Ischemic strokes had the highest incidence, with CVT being more common in younger patients.
3. Alcohol, smoking, hypertension, dyslipidemia, and obesity were significant lifestyle risk factors for stroke in adults.
4. Many patients had multiple risk factors.
5. According to TOAST criteria, the majority of ischemic strokes were large artery strokes, followed by small artery strokes and other/undetermined etiologies.
6. A significant proportion of CVT and ischemic stroke patients had prothrombotic states, highlighting its importance as a risk factor in young stroke patients.
7. Many ischemic stroke patients without classic cardiovascular risk factors showed hyper-homocysteinemia and protein S or C deficiencies, a pattern also seen in CVT. Further studies are needed to confirm a causal relationship.
8. About one-third of ischemic strokes with other determined etiologies were linked to a history of migraine.
9. Haemorrhagic strokes were predominantly lobar bleeds, followed by putaminal bleeds.
10. Haemorrhagic strokes were more common in females.
11. Hypertension and low haemoglobin levels were associated with haemorrhagic strokes, while high haemoglobin levels were linked to CVT.
12. Most patients showed good functional recovery, with CVT having the best outcomes.

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