

Computed tomography scan in the evaluation of patients with stroke

P. K. Chhetri¹, S. Raut²

¹Assistant Professor, ²Resident, Department of Radiodiagnosis, College of Medical sciences-Teaching Hospital, Bharatpur, Chitwan

ABSTRACT

Cerebrovascular accident is a frequent cause of death and disability in most parts of the globe. The incidence of cerebrovascular diseases increases with age and the number of strokes is projected to increase as the elderly population grows. This study was undertaken to determine the type of stroke and also to relate the risks factors associated with stroke. Hundred consecutive patients presenting with stroke in the emergency department / neurology clinic were subjected to computed tomography scan of the brain. Sixty – four patients had ischemic infarct and 36 had intracranial hemorrhage which included 2 patients with subarachnoid hemorrhage and 1 with underlying cavernous angioma. Ischemic stroke was thus commoner than hemorrhagic stroke. Previous infarct or lacunes were noted in 25 % of the patients presenting with stroke. Risk factors were present in most (77 %) of the patients presenting with stroke. Among the various risk factors, the single most common was smoking seen in 38 cases, followed by hypertension in 28 cases, obesity in 8 cases and diabetes in 3 cases. Twenty- one patients had multiple combinations of the above risk factors. Among the multiple risk factors, combination of alcohol and smoking was the commonest seen in 11 cases, followed by hypertension and obesity in 8 cases and alcohol with obesity in 2 cases. Change in lifestyle and eating habits may thus help reduce the incidence of stroke.

Key words: CT scan, stroke, risk factors.

Introduction

Stroke and cerebrovascular disease are terms that relate to a broad category of conditions characterized by sudden onset of a neurological deficit. Stroke is defined by the WHO as “The rapidly developing clinical symptoms and signs of focal (at times global) disturbance of cerebral function with symptoms lasting

more than 24 hours or leading to death with no apparent cause other than that of vascular origin.”¹ Stroke and cerebrovascular disease are terms, which are used interchangeably.

Cerebrovascular disease (stroke) is a frequent cause of death and disability and is a major health problem in most parts of the world. They cause ~200,000 deaths

Correspondence: P K Chhetri

E-mail: pramodchhetri@rediffmail.com

each year in the United States and are a major cause of disability. The incidence of cerebrovascular diseases increases with age and the number of strokes is projected to increase as the elderly population grows, with a doubling in stroke deaths in the United States by 2030.²

The four major types of stroke are cerebral infarction, intracerebral hemorrhage, primary subarachnoid hemorrhage and venous occlusion.

Normal cerebral blood flow is in the range of 50 to 55 mL/100g brain tissue/min. A severe perfusion deficit with cerebral blood flow values below 10 mL/100g/min may lead to infarction within a matter of minutes, whereas more moderate levels of ischemia (10 to 20 mL/100g/min) may be reversible for a period of hours after the start of the ischemic insult.³

Many of the imaging changes observed with computed tomography (CT) and magnetic resonance imaging depend on abnormal accumulation of water that occurs after brain injury. Cytotoxic edema begins to occur within minutes after the onset of infarction with water accumulation in the intracellular environment. The overall tissue water content increases to about 3 % to 5 % with the development of cytotoxic edema. With the breakdown in the blood brain barrier, vasogenic edema occurs, and proteins and water flood into the extracellular space from the intracellular environment and is generally believed to begin 4 to 6 hours after the ischemic insult. This results in much greater levels of tissue edema and brain swelling.⁴

The sensitivity of standard CT for brain ischemia increases after 24 hours. However, in a systematic

review involving 15 studies where CT scans were performed within six hours of stroke onset, the prevalence of early CT signs of brain infarction was 61 percent (standard deviation +/- 21 percent).⁵

The appearance of intracranial hemorrhage on CT is comparatively straightforward. There is a linear relationship between CT attenuation and hematocrit, hemoglobin concentration and protein content. Because the hematocrit of an acute retracted clot is around 90% and the globin (protein) component of hemoglobin has a high mass density, fresh intracerebral blood clots typically appear hyperdense on CT when compared to normal brain.⁶ The attenuation of intracerebral hematomas decreases with time, diminishing at an average of 1.5 HU per day.⁷

CT scan is widely available, affordable, non-invasive and relatively accurate investigation used in cases of stroke and is the modality of choice as an initial investigation in patient with stroke. The purpose of CT is to differentiate ischemic stroke from intracranial hemorrhage and to rule out other pathological processes such as tumour and vascular malformations which may present as stroke.

Materials and methods

This study was carried out on a prospective basis in the Department of Radiodiagnosis, College of Medical Sciences – Teaching Hospital, Bharatpur during the period January, 2010- January, 2012. All patients presenting to the emergency or neurology unit with clinical features of stroke was referred to the radiology department for CT scan of the brain. Non contrast CT scan of the head was performed using Siemens Somatom Spirit. Initial 5 mm axial sequential scan for

posterior fossa and 8 mm axial sequential scans was taken for the supratentorial region. Contrast was given only when indicated.

A detailed cardiovascular and neurological examination was done by the attending physician. Important clinical history and risk factors was taken from each patient (ie hypertension, diabetes mellitus, coronary artery disease, smoking, alcohol intake, previous history of TIA/ stroke). USG of the carotid / vertebral artery was also done where advised.

Any patients with suspected stroke were included in this study regardless of cause like tumoral bleed or secondary to vascular malformation. Infarct with secondary hemorrhagic was included under primary infarct. Repeat CT scan after 24 hour was done in any patient with clinical stroke and normal early CT scan findings.

The following two group of patients were excluded from this study (1) patient presenting with history of onset more than 7 days because after 7-10 days intracerebral hematoma may become iso-hypodense and then is difficult to differentiate from an infarct and (2) patient presenting with intracranial stroke secondary to trauma, bleeding diathesis, coagulation disorder.

Observation and results

100 consecutive patients presenting to the emergency or the neurology clinic with a clinical sign of stroke and subjected to CT scan in the radiology department were included in this study.

Among the 100 patients with stroke, 72 (72 %) were males and 28 (28 %) were females. The age ranged from 42 to 85 years. Sixty – four of the cases were

due to infarctions and 36 were due to hemorrhagic stroke. Of the patient with hemorrhagic stroke, 2 had subarachnoid haemorrhage and 1 had underlying vascular malformation. Infarction was thus the most common cause of stroke and both infarction and hemorrhage were more commonly seen in males than females presenting with stroke as shown in table 1.

Table 1: Distribution of cases according to the type of stroke and sex of the patients

Sex	Infarction	Hemorrhage	Total
Male	48	24	72
Female	16	12	28
Total	64	36	100

Of the 64 patients with infarction, the middle cerebral artery was involved in approx 75 %, the posterior cerebral artery in approx 15 %, the anterior circulation in approx 10 %. Thus most of the stroke was supratentorial (approx 90 %) and involved the middle cerebral arterial territory (approx 75 %). The posterior fossa was involved in 9 cases (approx 10 %) out of which 6 had cerebellar infarct and 3 had pontine infarct.

Among the patients with infarct, most (75 %) had a single isolated vascular territorial infarct while 25 % had a prior episode of infarct or small lacunar infarct/s. The commonest region involved was internal capsule and the basal ganglia, followed by the temporo-parietal cortex, occipital lobe, frontal lobe and finally the posterior fossa.

Of the patients presenting with infarcts, as mentioned above 25 % (16 patients) had lacunar infarctions (size < 1.5 cm). All the lacunar infarcts were round to ovoid and showed no mass effect.

Table 2: Location of lacunar infarctions

Location	No.of cases with lacunar infarctions n=16	
Internal capsule	6	(37.5 %)
Basal ganglia	5	(31.25 %)
Centrum semiovale	3	(18.75 %)
Thalamus	2	(12.5 %)

Intracerebral haemorrhage was seen in 36 patients. Thirty-three patients (91.66 %) had primary intracranial bleed. The most common involved region was the basal ganglia and the internal capsule (66.66 %), followed by the thalamus (21.21 %), temporo-parietal lobe (9.09 %) and the posterior fossa (3.03 %).

Of the remaining 3 patients with haemorrhage, two (5.55 %) had primary subarachnoid bleed secondary to an aneurysm involving the proximal MCA and one (2.77 %) had an underlying giant cavernoma causing the bleed (which was later confirmed by neurosurgery).

Table 3. Location of primary intracranial haemorrhage

Location	No. of cases with hemorrhagen=33	
Basal ganglia + internal capsule	22	(66.66 %)
Thalamus	7	(21.21%)
Temporo-parietal lobe	3	(9.09 %)
Posterior fossa (cerebellar)	1	(3.03%)

Risk factors were present in most (77 %) of the patients presenting with stroke. Among the various risk factors, the single most common was smoking seen in 38 cases, followed by hypertension in 28 cases, obesity in 8 cases and diabetes in 3 cases. Twenty- one patients had multiple combinations of the above risk factors. Among the multiple risk factors, combination of alcohol and

smoking was the commonest seen in 11 cases, followed by hypertension and obesity in 8 cases and alcohol with obesity in 2 cases.

In cases with cerebral infarction, smoking was seen in 39 % whereas in cases with intracranial hemorrhage, smoking was seen in 43 %. Thus infarction was more common in smokers (24.96 %) compared to hemorrhage (15.48 %).

In untreated cases of hypertension, hemorrhage was more common (57.88 %) than infarction (42.12 %). In treated cases infarction was more common (54.5 %) than hemorrhage (45.5 %).Among obese patients infarction was more common 56.8 % than hemorrhage 43.2%. Hemorrhage was commoner in moderate drinkers 61.5 % compared to infarction 38.5 %. In heavy drinkers, infarction and hemorrhage were equally common (10 cases each).In treated cases of diabetes, infarction was commoner 66.66 % than hemorrhage 33.34 %. However in untreated cases, hemorrhage was commoner 75 % than infarction 25 %.Only 2 of the cases with stroke had cardiac disease causing cerebral infarct. One had dilated cardiomyopathy and another had rheumatic mitral stenosis.

Discussion

Cerebrovascular accident is a frequent cause of death and disability in most parts of the globe. Among the stroke subtypes, cerebral ischemia and infarction constitute about 85 to 90 % of the total stroke subtypes in western countries, with only 10 to 15 % of patients with cerebral haemorrhage.⁸ Sotaniemi KA et al⁹ in their study of 154 cases of stroke found 102 (66.2 %) were due to infarcts, 30 (19.5 %) were due to hemorrhage and 16 (10.4 %) were due to subarachnoid

hemorrhage. In contrast to the western population, hemorrhagic stroke constituted a larger percentage of stroke subtypes on this side of the globe as seen in countries like Japan and China. It has been postulated that the higher percentage of hemorrhagic stroke in Asian countries could be because of poorly controlled hypertension.¹⁰ In this present study also haemorrhage as a cause of stroke constituted 36 % of the total cause. A number of classification systems for ischemic stroke have been proposed. The most commonly used clinical systems divide ischemic stroke into three major stroke subtypes: large artery or atherosclerotic infarctions, cardioembolic infarctions and small vessel or lacunar infarctions. Clinical studies may refer to this as the TOAST classification because it was adopted for the clinical evaluation of patients participation in a trial of a low molecular weight heparinoid, the Trial of Org 10172 in Acute Stroke Treatment.¹¹ Accurate determination of the relative frequencies of these stroke subtypes are difficult to determine because incidence varies from study to study. In addition, it can be difficult in some clinical settings to precisely determine the stroke subtypes, and many clinical strokes are reported to be of undetermined etiology.¹² In a large series the incidence of large artery or atherosclerotic infarction can vary from approximately 15 % to 40 %, cardioembolic strokes from 15 % to 30 %, and lacunar infarctions also from approximately 15 % to 30 % of the infarction reported.¹³

Lacunar infarctions are deep subcortical infarctions that generally involve perforating arteries supplying regions such as deep gray matter and brainstem. Lacunar infarctions are generally less than 1 cm and are not larger than 1.5 cm in diameter.¹⁴ In the anterior circulation, the perforating arteries are usually the

lenticulostriate arteries. In the posterior circulation they are the thalamoperforating arteries and small perforating arteries from the main stem of the basilar artery. Lacunar infarctions are predominantly in the basal ganglia, the white matter of the internal capsule, the brainstem, and the deep white matter of the hemispheres. Perforating arteries responsible for lacunar infarctions have been demonstrated to have microatheromata, lipohyalinosis, and fibrinoid necrosis.¹⁴ Many of these pathologic changes have been attributed to hypertension, and a high percentage of patients with lacunar infarctions have hypertension. Patients with diabetes mellitus also are predisposed to the development of these small, deep infarctions. Many of these multiple lesions are in fact clinically silent and may be seen in elderly patients, particularly those at risk for the development of cerebral vascular disease.⁴

Most nontraumatic spontaneous intracranial hemorrhages in adults are associated with systemic hypertension. In some cases, ruptured microaneurysm (Charcot-Bouchard aneurysm) on deep perforating vessels, particularly the lateral lenticulostriate arteries, have been implicated.¹⁵

Hypertensive intracranial hemorrhage has a predilection for areas supplied by penetrating branches of the middle cerebral and basilar arteries. Hypertensive hemorrhages therefore preferentially involves the external capsule and putamen, thalamus and pons. The approximate location of hypertensive hemorrhages are putamen / external capsule (60-65%), thalamus (15-25 %), pons (5 -10%), cerebellum (2 – 5 %) and subcortical white matter (1-2 %).¹⁶ In this present study, basal ganglia / internal capsule was involved in approx 66 %, thalamus in 21 %, subcortical white matter in 9 % and posterior fossa in 3 %.

Lobar white matter hemorrhage is seen in 15 to 20 % of intracranial hemorrhage cases, some of these spontaneous lobar hemorrhages are due to amyloid angiopathy, but most are still hypertensive.¹⁷ As mentioned approx 9 % of the patients in this study had lobar hemorrhage involving the temporo-parietal lobe. Jose Luis Ruiz-Sandoval et al¹⁸ in 1999 studied intracerebral hemorrhage in 200 young patients whose age ranged from 15 to 40 years. They found that the most frequent risk factors were hypercholesteremia (35 %), tobacco use (20 %), hypertension (13 %) and alcohol abuse (10 %). The locations of hemorrhage were lobar (55 %), basal ganglia / internal capsule (22 %) and others (24 %). They observed that the majority of patients with hemorrhage that resulted from hypertension were aged >31 years and those with hemorrhage that resulted from vascular malformation were aged <20 years.

Konrad J et al¹⁹ found that consumption of meat more than 4 times weekly was a factor associated with increased risk for all strokes in general. In this study all the patients were non-vegetarians.

It is traditionally believed that intracerebral hemorrhage is more closely associated with hypertension than infarction but the Farmingham study showed that the incidence of atherothrombotic lesions also closely correlated with the blood pressure levels. It has been observed that sudden, prolonged and profound hypertension in a hypertensive can lead to small vessel infarct.²⁰

Potential risk factors for stroke include cardiac disease, hypertension, smoking, diabetes mellitus and alcohol abuse.²¹ While hypertension, smoking and cardiac disease are established risk factors, newer risk factors

are being reported and their association and relative risk still being ascertained. Obesity as a cause for stroke has insufficient supporting data. However, obese persons have higher levels of blood pressure, glucose and atherogenic serum lipids and on that account could be expected to have increased stroke incidence.²²

Horowitz et al²³ in their study of 50 patients with cerebral infarctions found hypertension in 58 %, smoking in 48 %, diabetes mellitus in 18 % and alcohol abuse in 16 %. They found that while hypertension, smoking and cardiac disease are established risk factors, patients having them did not suffer more severe infarctions and that a history of diabetes mellitus significantly affected early clinical status and radiographic findings. They observed that patients with diabetes mellitus had an increased incidence of hemorrhagic infarctions, had larger infarcts, significant mass effect and developed greater neurological deficits. They concluded that combining the history of diabetes mellitus with CT findings is useful in evaluating early cerebral infarctions within the first five hours of stroke and that hypertension, smoking and cardiac disease do not affect the clinical severity in this period.

Konrad Jamrozik et al¹⁹ in 1994 studied the role of lifestyle factors in the etiology of stroke. They found that smoking, consumption of meat more than 4 times a week and a history of hypertension were each associated with increased risk in multivariate models for all strokes and for all first ever strokes. Consumption of 1 to 20 grams /day of alcohol in the preceding week was associated with a significant increase in the risk of all strokes, all ischemic strokes and of primary intracerebral hemorrhage, while eating fish more than 2 times a month appeared to have a protective effect. Diabetes mellitus was associated with a significantly

increased risk of ischemic stroke but a decreased risk of hemorrhagic stroke. They concluded that risk factors for ischemic and hemorrhagic stroke are not exactly the same and that change in lifestyle relating to tobacco and diet might make important contributing in reducing the incidence of stroke.

Hemorrhage was more common in untreated hypertensive (57%), moderate alcohol consumption (61.5%), tobacco chewers (60.46%) and untreated diabetics (75%). Diabetics are known to have increased susceptibility to coronary, femoral and cerebral artery atherosclerosis. Diabetes increases the concentration of prothrombotic factors in blood and increases platelet aggregability. Increased frequency and severity of atherosclerosis in the circle of Willis as well as in the middle cerebral, basilar and carotid arteries is observed in diabetics.²⁴

Matti Hillborm et al²⁵ in 1999 investigated the effect of alcohol as a risk factor for acute ischemic stroke. They found that recent (past week) heavy drinking (151-300g) but not former heavy drinking was an independent risk factor for stroke. Light drinking (<40mg) did not increase the risk for stroke.

There are a number of mechanisms by which heavy alcohol consumption may predispose to and moderate alcohol consumption may protect against stroke. Cigarette smoking is more frequent in heavy alcoholics and there is additive hemoconcentration. Alcohol and cigarette smoking have been known to increase both the blood hematocrit and viscosity and thus thrombocytosis.²⁶

Conclusion

Although infarction is a more common cause of stroke than hemorrhage, the incidence of hemorrhage was

found to be higher than that in western countries. Smoking was the commonest risk factor associated with stroke and is also seen in combination with alcohol consumption in many cases. There are multiple associated risk factors associated with stroke. Change in lifestyle and eating habits may help reduce the incidence of stroke.

References

1. S. Hatano. Experience from a multicentre stroke register: A preliminary report. *Bulletin WHO* 1976;**54**:541-53.
2. A.S. Fauci, E. Braunwald, D.L. Kasper, et al, editors. *Harrison's principles of internal medicine*. 17th Ed. New York: Mc Graw Hill, Health professional division. 2008. chapter 370.
3. W.D. Heiss, G. Rosner. Functional recovery of cortical neurons as related to degree and duration of ischemia. *Ann Neurol*. 1983;**14**:294-301.
4. P. Michael. Marks. Cerebral Ischemia and Infarction. In *Scott W Atlas MRI of the brain and spine*. Third edition Lippincott Williams and Wilkins, 2002;**18**:919-79.
5. J.M. Wardlaw, O. Mielke, Early signs of brain infarction at CT: observer reliability and outcome after thrombolytic treatment-systematic review. *Radiology*. 2005;**235**:444.
6. R.A. Brooks, G. DeChiro, N. Patronas: MR imaging of cerebral hematoma at different field strengths: theory and applications. *J Comp Asst Tomogr*. 1989;**13**:194-206.
7. W.A. Cohen, L.A. Wayman. Computed tomography of intracranial hemorrhage. *Neuroimaging Clinics N Amer*. 1992;**2**:75-87.
8. J. Bamford, P. Sandercock, M. Dennis et al. A prospective study of acute cerebrovascular disease in the community: The Oxfordshire community stroke project, 1981-86, 2. Incidence, case fatality rates and overall outcome at one year of cerebral infarction, primary intracerebral and subarachnoid hemorrhage. *Journal of neurology, Neurosurgery, Psychiatry*. 1990;**53**:16-22.

P. K. Chhetri, *Computed tomography scan in the evaluation of patients with stroke*

9. K.A. Sotaniemi, J. Phytinen, V.V. Myllyla. Correlation of clinical and computed tomographic findings in stroke patients. *Stroke*. 1990; **21**:1562-66.
10. C.Y. Huang, F.L. Chan, Y.L. Yu et al. Cerebrovascular disease in Hong Kong Chinese. *Stroke*. 1990; **21**:230.
11. H.P. Adams, B.H. Bendixen, L.J. Kappelle et al. Classification of subtype of acute ischemic stroke-definitions for use in a multicenter clinical trial. TOAST. Trial of Org 10172 in Acute Stroke Treatment. *Stroke*. 1993; **24**:35-41.
12. R.L. Sacco, J.A. Ellenberg, J.P. Mohr et al. Infarction of undetermined cause: the NINCDS Stroke Data Bank. *Ann Neurol*. 1989; **25**:382-90.
13. R. Sacco. Classification of stroke. In: Fisher M, ed. Clinical atlas of cerebrovascular disorders. London: Wolfe, 1994; **2**:1-2.25.
14. C.M. Fisher. Capsular infarcts. *Acta Neuropathol* 1979; **36**:65-73.
15. S. Wakai, N. Kumakura, M. Nagai: Lobar intracerebral hemorrhage. *J Neurosurg*. 1992; **76**:231-38.
16. G. Anne Osborn. Intracranial Hemorrhage. Diagnostic Neuroradiology. Mosby. 1994 Chapter; 7: 154 -98.
17. J. Broderick, T. Brott, T. Tomsik et al: Lobar hemorrhage in the elderly: the undiminishing importance of hypertension. *Stroke*. 1993; **24**:49-51.
18. Jose Luiz Ruiz-Sandoval, Carlos Centu, SCM Fernando Barinagarrementeria. Intracerebral hemorrhage in young people *Stroke*. 1999; **30**:531-41
19. Konrad Jamrozik, Robyn J Broadhurst, Craig S Anderson et al. The role of lifestyle factors in the etiology of stroke. *Stroke*. 1994; **25**:51-9.
20. W.B. Kannel, P.A. Wolf, J. Vesta et al. Epidemiological assessment of the role of BP in stroke. The Farmingham study *JAMA*. 1970; **219**:301-10.
21. J.W. Norris. Steroid therapy in acute cerebral infarction. *Archives Neurology*. 1976; **33**:69-71.
22. WHO task force on stroke and other cerebrovascular disorders: Recommendations on stroke prevention, diagnosis and therapy. *Stroke*. 1989; **20**:1407-31.
23. S.H. Horowitz, J.L. Zito, Domarumma R et al. Clinico-radiologic correlation within the first five hours of cerebral infarction. *Acta Neurology Scand*, 1992; **86**:207-14.
24. K.J. Asplund, E. Hagg, C. Helmers. The natural history of stroke in diabetic patients. *Acta Medicine Scand*. 1980; **207**:417-24.
25. Matti Hillbom, Heikki Numminen, Seppu Juvela. Recent heavy drinking of alcohol and embolic stroke. *Stroke*. 1999; **30**:2307-12.
26. A.S. Leon, H. Blackburn: physical inactivity page 86 in Kaplan NM, Stamler J (eds): Prevention of coronary heart disease, practical management of the risk factors. WB Sanders, Philadelphia 1983.