# Magnetic Resonance Imaging and Neuropsychological Correlates of Subcortical Vascular Dementia

#### **Abstract**

Common understanding of dementia is mostly based on the Alzheimer's disease model. However, in subcortical vascular dementia (SVaD), several other cognitive and behavioral dysfunctions predominate over what is commonly noticed in Alzheimer's disease. There are inconsistencies in literature regarding the profile of cognitive impairments in vascular dementia. Objective: In the current study, different cognitive functions pertaining to different lobes of the brain along with neuropsychiatric symptoms we explored in a holistic manner. Materials and Methods: A sample of 12 patients diagnosed with SVaD without any comorbidity were recruited for the study. All the patients underwent magnetic resonance imaging (MRI) scanning and different standardized neuropsychological tests were administered. Results: Patients reported various neuropsychiatric symptoms with varied severity, mostly falling in moderate range, reflecting organic personality change. Most of the cognitive functions pertaining to different lobes of the brain were impaired with parietal lobe being intact mostly. Comparison of MRI findings and neuropsychological findings revealed that there is involvement of cortical functions with the impairment in subcortical structure. Conclusion: These findings emphasis need to look beyond clinical diagnosis and MRI findings for better rehabilitation of the patients by including cognitive dysfunction as well as emotional disturbances prominent in SVaD which might me quite distressing for caretakers.

**Keywords:** Magnetic resonance imaging, neuropsychological functioning, sub-cortical vascular dementia

# Introduction

Subcortical vascular dementia (SVaD) is one of the subtypes of dementia that is believed to arise from arteriosclerotic changes in small vessels of the brain. SVaD is characterized by extensive white matter hyperintensities and multiple lacunar infarcts that can be observed in magnetic resonance imaging (MRI) scans.[1,2] Our current diagnostic understanding of dementia is greatly influenced by research on Alzheimer disease, which places major emphasis on memory dysfunction.[3-5] However, in SVaD. frontal-subcortical circuits which consist of frontal lobe, basal ganglia, and thalamus are commonly affected due to cerebrovascular hemorrhage which in turn results in frontal executive dysfunction and other extrapyramidal symptoms. [2,5,6]

The pace of understanding of dementia has increased markedly from the past few decades. Among all the dementias, vascular dementia is probably one the most common types in elderly people. However,

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: reprints@medknow.com

studies have largely focused on diagnosis, treatment, and underlying pathological substrates. Cognitive functioning domains focusing on deficits in learning and memory rather than other prominent cognitive dysfunctions were emphasized.[3-5] With time, studies reported distinctive profile of cognitive functions in SVaD. In addition to memory, other mental faculties affected in dementias include language, visuospatial ability, calculation, judgment, problem-solving.[3-5] Neuropsychiatric and social deficits resulting in depression, withdrawal, hallucinations, delusions, agitation, insomnia, and disinhibition usually interferes with daily living of the patients in SVaD. They are usually more depressed and physically dependent than patients with Alzheimer's disease.[7]

Our study differs from earlier studies which have usually focused on a particular domain of cognitive functioning especially different aspects of memory. Hence, it becomes difficult to get a full picture of the cognitive profile in SVaD. Hence, we used a comprehensive battery of tests pertaining

**How to cite this article:** Paul MA, War FA, Sharma V, Kushwaha S. Magnetic resonance imaging and neuropsychological correlates of subcortical vascular dementia. Asian J Neurosurg 2018;13:631-5.

# Mohd Altaf Paul, Firdous Ahmad War<sup>1</sup>, Vibha Sharma<sup>2</sup>, Suman Kushwaha<sup>3</sup>

Department of Psychiatry,
Institute of Mental Health and
Neurosciences, Kashmir,
Jammu and Kashmir,
¹Department of Humanities
and Social Sciences, Indian
Institute of Technology, Kanpur,
Uttar Pradesh, Departments
of ²Clinical Psychology and
³Neurology, Institute of Human
Behaviour and Allied Sciences,
New Delhi, India

Address for correspondence: Mr. Firdous Ahmad War, Room No. A235, Hall XI, IIT Kanpur, Kanpur - 208 016, Uttar Pradesh, India. E-mail: mailfaw@gmail.com



to all the four lobes of the brain. Due to heterogeneity in the underlying pathologies in SVaD, we focused on patients with diffuse subcortical ischemic leukoencephalopathy. Thus, the main aim of the present study was to identify the profile of cognitive impairment in SVaD. It will play a vital role in identifying, diagnosing, quantifying, and establish better precision of the type and extent of cognitive and behavioral deficits which is usually missed in MRI findings. It will help in detecting favorable cognitive effects of drugs and will help in improving the well-being of patients through educating the caretakers about possible cognitive and behavioral outcome of the disorder.

# **Materials and Methods**

# **Participants**

Twelve patients with SVaD, within the age range of 50–70 years, were recruited from Neurology Outpatient Department at Institute of Human Behaviour and Allied Sciences (IHBAS), India. Out of 12 patients, 7 were males and rest were females. All the patients were screened and diagnosed by a consultant Neurologist as per the International Classification of Diseases-10 criteria for SVaD. The duration of illness was from 1 year to 5 years. Patient with any comorbid psychiatric diagnosis was excluded from the study. All the patients had undergone MRI scanning.

# **Measures**

Standardized neuropsychological tests were administered. Tests measuring different functions of the four lobes of the brain were selected so as to get a holistic understanding of the brain functioning. These tests included: frontal assessment battery[8] to assess different functions pertaining to frontal lobe. Rey's auditory verbal learning test<sup>[9]</sup> to assess functions mainly pertaining to temporal lobe. Rey's complex figure test[9] to assess functions mainly pertaining to occipital lobe. Focal signs<sup>[9]</sup> to assess functions mainly pertaining to parietal lobe. These focal signs include finger agnosia, visual agnosia, color agnosia, tactile agnosia, ideational apraxia, ideomotor apraxia, dressing apraxia, kinetic apraxia, spatial apraxia, and construction apraxia. Addition to this, neuropsychiatric questionnaire[10] was used to assess the severity of behavioral symptoms commonly observed in patients with dementia.

#### **Procedure**

This study was approved by the IHBAS ethics committee. Written informed consent was obtained from participants before applying tests. All the participants were assessed individually. Adequate rest and pauses were given in between the testing. Testing was mostly conducted during the morning and early afternoon hours.

# **Results**

Table 1 shows various neuropsychiatric symptoms reported by the patients along with their severity. Symptoms varied in their severity, mostly falling in moderate range. In addition, these symptoms reflect organic personality change in patients.

Table 2 shows most of the patients on different subscales fall in the range of moderate to severe impairment on frontal lobe functions mostly pertaining to prefrontal cortex. However, on the full-scale score, most patients fall within moderate level of impairment.

Table 3 shows impairment on the different dimensions of verbal learning and memory with higher number of patients falling in the severe impairment range.

Table 4 clearly indicates most of the patients fall in severe range on a different dimension of visual learning and memory.

Table 5 clearly shows that constructional apraxia was found more than other types of apraxias. Among agnosia only visual agnosia was found.

Table 6 shows common MRI findings were cerebral atrophy, multiple infarcts, ischemic demyelination, and hyperintensities of frontoparietal lobe while

Table 1: Number and percentage of patients having different degrees of impairment on various neuropsychiatric symptoms

	Mild	Moderate	Severe	Total
	n (%)	n (%)	n (%)	n (%)
Delusions	0 (0%)	1 (0.08%)	1 (0.08%)	2 (17%)
Hallucinations	0 (0%)	0 (0%)	3 (25%)	3 (25%)
Aggression	2 (17%)	3 (25%)	5 (42%)	10 (83%)
Depression	3 (25%)	5 (42%)	1 (0.08%)	9 (75%)
Anxiety	1 (0.08%)	5 (42%)	1 (0.08%)	7 (58%)
Elation	1 (0.08%)	0 (0%)	0 (0%)	1 (0.08%)
Apathy	0 (0%)	6 (50%)	2 (17%)	8 (67%)
Disinhibition	4 (33%)	2 (17%)	0 (0%)	6 (50%)
Irritability	1 (0.08%)	6 (50%)	1 (0.08%)	8 (67%)
Motor disturbance	0 (0%)	1 (0.08%)	1 (0.08%)	2 (17%)
Sleep	2 (17%)	5 (42%)	1 (0.08%)	8 (67%)
Appetite	1 (0.08%)	5 (42%)	1 (0.08%)	7 (58%)

Table 2: Number with percentages of patients having different degrees of impairment in frontal lobe functions

	Mild n (%)	Moderate n (%)	Severe n (%)	<b>Total</b> <i>n</i> (%)
Conceptualization	5 (42%)	2 (17%)	5 (42%)	12 (100%)
Mental flexibility	8 (67%)	2 (17%)	2 (17%)	12 (100%)
Programming	1 (0.08%)	1 (0.08%)	5 (42%)	7 (58%)
Interference	2 (17%)	3 (25%)	4 (33%)	9 (75%)
Inhibitory control	2 (17%)	4 (33%)	3 (25%)	9 (75%)
Full scale	2 (17%)	5 (42%)	0 (0%)	7 (58%)

Table 3: Number with percentages of patients having different degrees of impairment on verbal learning and memory

	Immediate	Delayed	Long term	Recognition
	recall	recall	percent retention	
Mild	1 (0.08%)	2 (17%)	1 (0.08%)	3 (25%)
Moderate	3 (25%)	1 (0.08%)	2 (17%)	0 (0%)
Severe	3 (25%)	5 (42%)	5 (42%)	4 (33%)
Total	7 (58%)	8 (67%)	8 (67%)	7 (58%)

Table 4: Number with percentages of patients having different degrees of impairment on visual learning and memory

		•	
	Сору	Immediate recall	Delayed recall
Mild	1 (0.08%)	1 (0.08%)	1 (0.08%)
Moderate	1 (0.08%)	1 (0.08%)	1 (0.08%)
Severe	8 (67%)	9 (75%)	9 (75%)
Total	10 (83%)	11 (92%)	11 (92%)

Table 5: Number with percentages of patients having different degrees of impairment on focal signs

		Ideational apraxia		Constructional apraxia	Visual agnosia
Difficulty	2 (17%)	0 (0%)	0 (0%)	8 (83%)	0 (0%)
Impairment	0 (0%)	2 (17%)	2 (17%)	3 (25%)	2 (17%)

neuropsychological tests showed dysfunction in different lobe with parietal lobe mostly being intact. In addition to it, comparison of MRI and neuropsychological findings revealed that there is involvement of cortical functions with the impairment in subcortical structures of the brain.

# **Discussion**

Prior studies have usually looked into some particular aspect of neuropsychological functioning of SVaD patients. These studies have suggested that SVaD patients exhibit greater impairment of higher cognitive functions than the basic functions. However, discrepancies in findings were observed among these studies. In the present study, a comprehensive battery of neuropsychological tests assessing major functions of different lobes of the brain along with neuropsychiatric symptomatology checklist was used. Results revealed that all the lobes are affected in SVaD with parietal lobe being largely intact. The emotional outburst of the patients was the main concern of the caretakers which was observed during the clinical interview and in the symptom checklist as well.

On the neuropsychiatric symptom checklist, most of the symptoms were found in the mild to severe range of severity [Table 1]. These symptoms include aggression, depression, anxiety, apathy, irritability, sleep, and appetite. While delusions, hallucination, elation, motor disturbances, and disinhibition were mostly in mild range of severity.<sup>[11,12]</sup> Studies show that our frontal lobes have extensive connections with portions of the limbic system, the basal ganglia, and the thalamus which are impaired in SvaD.<sup>[5]</sup> Thus, the neurological mechanism for such dysfunction is most likely due to the disruption of these frontal-subcortical connections.<sup>[13]</sup>

Such symptomatology reflects more of emotional dysregulation and is suggestive of organic personality change, which was also evident during the interview with the patients. [14] Such symptoms were the main concern of the caretakers than other cognitive symptoms. In Indian culture, aged people are highly respected and regarded in the decision-making process regarding family matters; however, such symptoms limit their capacity to fulfill demands of their position in family resulting in an increase in their level of frustration. On the one hand, it impacts their wellbeing and worsen the illness, and on the other hand, it enhances expressed emotion of caretakers. Psychological techniques may be helpful in managing these emotional outbursts. [15]

On neuropsychological test of frontal lobe, most of the patients were found to have dysfunction in frontal functioning which includes conceptualization, mental flexibility, programming, interference, and inhibitory control [Table 2]. Overall, 58% of patients report dysfunction on full scale with conceptualization, mental flexibility, interference, and inhibitory control being main affected, which is usually found in later stages of Alzheimer's disease. These findings suggest involvement of prefrontal areas, which is a part of frontal lobe. Similar findings have been reported in other studies.<sup>[5,16]</sup> Executive functions in SVaD are not entirely understood. However, intra- and inter-hemispheric circuit communication is usually said to play an important role in normal functioning. This communication is often mediated by myelinated axons that travel in the subcortical white matter, and damage to the white matter can induce dysfunction. In MRI findings, demyelination of the axons was commonly reported.

The assessment of verbal and visual aspects of learning and memory revealed deficits in most of the patients, however, the visual aspect was more affected than verbal [Tables 3 and 4]. [16,17] Indicating that right temporal lobe of the brain is more affected than the left temporal (medial) lobe. Lesion studies have revealed that acquisition of new verbal information is mediated by a wide network of structures including the anterior temporal cortex, amygdale, hippocampus, prefrontal cortex, and retrosplenial cortex. [18]

Parietal lobe functioning in SVaD has received little attention than other cognitive functions. In the present study, only few patients showed dysfunction on different parietal lobe focal signs such as constructional apraxia, ideational apraxia, spatial apraxia, visual agnosia, and

	Table 6: Comparison of neuropsychological test findings and MRI findings					
Patient	Neuropsychological test findings			MRI - Findings		
	<b>Executive functions</b>	Verbal learning and memory	Visual learning and memory	Focal signs		
1	Intact	Impaired	Impaired	Intact	Diffuse cerebral atrophy with periventricular ischaemic demyelination.	
2	Impaired	Impaired	Impaired	Impaired	Few supratentorial areas of ischaemic demyelination of fronto-parietal deep white matter and few chronic lacunar infarcts with diffuse cerebral atrophy.	
3	Impaired	Impaired	Impaired	Impaired	white matter (ischemic demyelination/leukoaraiosis) with chronic lucaner ischemic foci and chronic microhemorrhages in bilateral cerebral hemispheres and ganglio-thalamic regions. Possibility of chronic hypertensive encephalopathy appears likely.	
4	Intact	Impaired	Impaired	Intact	Mild cerebral and cerebellar atrophy	
5	Intact	Intact	Intact	Intact	Mild generalized age related cerebral atrophy with changes of ischemic leucoaraiosis and old lacunar infarct in left parieto-occipital periventricular region.	
6	Impaired	Impaired	Impaired	Intact	Diffused cerebral atrophy with dep matter hyperintensities.	
7	Intact	Intact	Impaired	Intact	Diffuse cerebellar and mild cerebral atrophy with nonspecific deep white ischemic changes.	
8	Impaired	Intact	Impaired	Intact	Cerebral and cerebellar atrophy with hyper intensities in bilateral cerebral white matter (ischemic demyelination, leukoaraiosis) with chronic lucanar ischemic foci and micro hemorrhages in bilateral cerebral hemispheres and ganglio-thalamic regions.	
9	Impaired	Impaired	Impaired	Impaired	Lacunar infarcts and atrophic changes consistent with age. FLAIR intensity seen in bilateral deep white matter brainstem and right external capsule.	
10	Intact	Intact	Impaired	Intact	Diffuse cerebral atrophy with nonspecific T2/fast FLAIR hyper intensities in bilateral fronto-parietal deep white matter.	
11	Impaired	Impaired	Impaired	Intact	Mild lucanar infarcts with micro haemorrhage in bilateral cereberal hemispheres.	
12	Impaired	Impaired	Impaired	Intact	Multiple supratentorial and pontine areas of ischemic demyelination and chronic lacunar infarcts with diffuse cerebral atrophy.	

kinetic apraxia. Among all these, constructional apraxia was most affected. These findings are suggestive of the right parietal lobe involvement and occipital lobe being intact mostly.<sup>[19]</sup> In Alzheimer's disease agnosia and apraxia are usually reported.

The comparison of MRI and neuropsychological test findings did not contribute much in our understanding of the relation of cognitive functions and affected brain structures. In most of the MRI scans, diffuse cerebral atrophy, multiple infarcts, and ischemic demyelination was reported without specifying much about the region. These findings can only help in arriving to the diagnosis of SVaD. However, neuropsychological tests findings help in the diagnosis as cognitive profile of these patients varies from that of other dementia especially Alzheimer's disease

#### **Conclusion**

In the present study, the profile of cognitive impairment in SVaD was studied. Findings revealed almost all the lobes were affected in different degrees with parietal lobe mostly intact. Executive functions were also compromised. Patients exhibited behavioral and emotional problems mostly aggression and depression. Hence, the findings of the present study would help in formulating a plan to retrain the patients according to their cognitive deficits which would help in preventing further deterioration. These findings can help in educating the caretakers regarding the cognitive deficits and outcome.

# Financial support and sponsorship

Nil

# **Conflicts of interest**

There are no conflicts of interest.

# References

- Lee JH, Kim SH, Kim GH, Seo SW, Park HK, Oh SJ, et al. Identification of pure subcortical vascular dementia using 11C-Pittsburgh compound B. Neurology 2011;77:18-25.
- Román GC, Erkinjuntti T, Wallin A, Pantoni L, Chui HC. Subcortical ischaemic vascular dementia. Lancet Neurol 2002;1:426-36.
- Desmond DW. The neuropsychology of vascular cognitive impairment: Is there a specific cognitive deficit? J Neurol Sci 2004;226:3-7.

- Graham NL, Emery T, Hodges JR. Distinctive cognitive profiles in Alzheimer's disease and subcortical vascular dementia. J Neurol Neurosurg Psychiatry 2004;75:61-71.
- Sellal F, Wolff V, Marescaux C. The cognitive pattern of vascular dementia and its assessment. Semin Cerebrovasc Dis Stroke 2004;4:79-86.
- Cummings JL. Frontal-subcortical circuits and human behavior. Arch Neurol 1993;50:873-80.
- Gure TR, Kabeto MU, Plassman BL, Piette JD, Langa KM. Differences in functional impairment across subtypes of dementia. J Gerontol A Biol Sci Med Sci 2010;65:434-41.
- 8. Dubois B, Slachevsky A, Litvan I, Pillon B. The FAB: A frontal assessment battery at bedside. Neurology 2000;55:1621-6.
- Rao SL, Subbaakrishna DK, Gopukumar K. NIMHANS Neuropsychological Battery Manual. Bangalore: National Institute of Mental Health and Neurosciences; 2004.
- Kaufer, DI, Cummings JL, Ketchel P, Smith V, MacMillan C, Shelley T, et al. Validation of the NPI-Q, a brief clinical form of the Neuropsychiatric Inventory. J Neuropsychiatry Clin Neurosci 2000:12:233-9.
- Moretti R, Torre P, Antonello RM, Cattaruzza T, Cazzato G, Bava A. Frontal lobe dementia and subcortical vascular dementia: A neuropsychological comparison. Psychol Rep 2005;96:141-51.

- Levy JA, Chelune GJ. Cognitive-behavioral profiles of neurodegenerative dementias: Beyond Alzheimer's disease. J Geriatr Psychiatry Neurol 2007;20:227-38.
- Price CC, Jefferson AL, Merino JG, Heilman KM, Libon DJ. Subcortical vascular dementia: Integrating neuropsychological and neuroradiologic data. Neurology 2005;65:376-82.
- Cummings JL, Petry S, Dian L, Shapira J, Hill MA. Organic personality disorder in dementia syndromes: An inventory approach. J Neuropsychiatry Clin Neurosci 1990;2:261-7.
- Hersch EC, Falzgraf S. Management of the behavioral and psychological symptoms of dementia. Clin Interv Aging 2007;2:611-21.
- Jokinen H, Kalska H, Mäntylä R, Pohjasvaara T, Ylikoski R, Hietanen M, et al. Cognitive profile of subcortical ischaemic vascular disease. J Neurol Neurosurg Psychiatry 2006;77:28-33.
- Suades-González E, Jódar-Vicente M, Pérdrix-Solàs D. Memory deficit in patients with subcortical vascular cognitive impairment versus Alzheimer-type dementia: The sensitivity of the 'word list' subtest on the Wechsler Memory Scale-III. Rev Neurol 2009;49:623-9.
- Vann SD, Aggleton JP, Maguire EA. What does the retrosplenial cortex do? Nat Rev Neurosci 2009;10:792-802.
- Slotnick SD. Visual memory and visual perception recruit common neural substrates. Behav Cogn Neurosci Rev 2004;3:207-21.