Original Article

Clinicoradiological Features and Long-term Cognitive and Functional Outcome in Patients with Deep Cerebral Venous Thrombosis

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Abstract

Background: Deep cerebral venous thrombosis (DCVT) can have long-term functional and cognitive sequelae. Although literature exists on cognitive impairment after arterial stroke, cognitive sequelae after cerebral venous thrombosis (CVT) are much less studied. **Methods:** Clinical records of 29 patients diagnosed with DCVT were reviewed. The Modified Telephonic Interview for Cognitive Status (TICS-M) was adapted and validated in the regional language (Kannada) and applied to 18 patients with DCVT, at a mean follow-up duration of 5.32 years. Screening for depression was done via telephonic Patient Health Questionnaire-9 (PHQ-9)-Kannada version, and functional status was screened by applying the modified Rankin Scale (mRS). **Results:** DCVT had a mortality rate of 10.34% due to acute complications. mRS scores of 0–1 were achieved at follow-up in all patients who survived. Receiver operating characteristic (ROC) analysis revealed a cutoff of ≤44.5 (maximum score of 49) for the diagnosis of cognitive impairment via TICS-M (Kannada version) in DCVT patients. Evidence of cognitive dysfunction was seen in eight patients (42.10%), and three patients (16.66%) had evidence of depression. **Conclusions:** Survivors of acute DCVT can potentially have long-term cognitive sequelae. Screening for cognitive dysfunction, depression, and functional status can be effectively done using telephonically applied scales that are adapted to the local language. Neuropsychological evaluation and early cognitive rehabilitation can be initiated for patients in whom deficits are identified on cognitive screening.

Keywords: Cerebral venous thrombosis, cognitive screening, neuropsychology, telemedicine, TICS-M, vascular dementia

Introduction

Cerebral venous thrombosis (CVT) accounts for 0.5%–1% of all strokes per year, with an incidence of five per million adults per year.[1] In comparison with arterial stroke, the outcome of CVT is better with approximately 80% of patients, with CVT regaining functional independence, when assessed on the modified Rankin Scale (mRS).[2] Thrombosis can occur in superficial cerebral veins, deep venous systems, or cortical veins of the brain. Deep cerebral venous thrombosis (DCVT) refers to thrombosis of the internal cerebral vein, vein of Galen, and basal vein of Rosenthal.[3] Approximately 10% of cases of CVT are because of thrombosis of deep cerebral veins.[4]

Vascular dementia is overall the second most common cause of cognitive impairment.[5] The role of age-related dysregulation of the cerebral venous circulation in contribution to vascular cognitive impairment is being increasingly recognized.[6] Anatomical structures involved in DCVT include the thalamus, caudate nucleus, and deep white matter of the brain, which are implicated in cognitive processes such as attention, motivation, and speed of cognitive processing.[7] DCVT offers an attractive model to study the long-term impact of factors affecting the venous drainage of the brain on cognitive function. Although literature exists on cognitive impairment after arterial stroke, cognitive impairment after CVT is much less studied. We aimed to study the impact of acute dysfunction of the cerebral venous system, as occurs in DCVT, on long-term cognitive functions of the brain.

Whereas many screening tools for detecting cognitive decline require in-person assessment, telephonic cognitive assessment has gained importance in the pandemic era and has been done through validated scales such as Short Portable Mental Status Questionnaire (SPMSQ), Structured Telephone Interview for Dementia Assessment (STIDA), Telephonic Montreal Cognitive Assessment (MOCA), Minnesota Cognitive Acuity Screen, modified Telephonic Interview for Cognitive Status (TICS-M) and TICS.[8] TICS-M assessment have been well studied and validated in subjects with dementia and post-stroke cognitive impairment.[8-10] Telephonic-based

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cognitive assessment can improve the breadth of coverage and reduce no-show rates.[11] TICS-M is particularly useful when the goal is to identify patients with potential cognitive impairment who could later be assessed in detail via an in-person neuropsychological examination.[12] We aimed to study the cognitive and functional status post deep CVT by using TICS-M adapted and validated in the regional language (Kannada).

Patients and Methods

This study was conducted in the Department of Neurology of the institute after obtaining approval from the institute's ethics committee and after obtaining informed telephonic consent from the patients or their caregivers during the COVID-19 pandemic. Patient records from the stroke ward registry for DCVT patients admitted and evaluated between 2013 and 2021 were screened. Patients over the age of 18 years and diagnosed to have DCVT based on clinical and neuroimaging findings, either via contrast-enhanced computerized tomography (CECT) or magnetic resonance imaging (MRI) of the brain, were included. The following categories of patients were excluded: patients with isolated superficial CVT, significant hearing dysfunction that could potentially affect telephonic cognitive assessment, patients with any other major neurological disorder or significant psychiatric illness, history of significant head injury, and patients consuming drugs that may interfere with motor and cognitive outcome assessment leading to falsely high scores on the PHQ-9.

First, demographic details, clinical parameters, and radiological features of patients with DCVT were documented. Where available, treatment history and comorbid medical, psychiatric, and neurologic illnesses were documented.

Subsequently, the Kannada version of TICS-M was validated using the following steps. The Telugu language version of TICS-M, which is validated and widely studied in the Indian context (unpublished thesis data), was translated, back-translated, and adapted to Kannada. This version was applied in 10 normal subjects and in 10 patients with a diagnosis of vascular dementia who already had their cognitive scores documented by Addenbrooke cognitive evaluation (ACE) or Mini-mental state examination (MMSE). A cross-validation was performed, and the correlation between the TICS-M Kannada version and MMSE/ACE scores was studied. The TICS-M Kannada version has 10 sections with questions pertaining to the patient's name, orientation to time and place, counting backward, wordlist immediate recall, naming, repetition, wordlist delayed recall, praxis, and word opposites. ROC analysis revealed a cutoff of ≤41.5 for the diagnosis of cognitive impairment on TICS-M for vascular dementia patients. However, because the age of vascular dementia patients was significantly higher than that of DCVT patients in the study group, a cutoff of 44.5 was derived for the diagnosis of cognitive impairment in the DCVT cohort after correcting for age.

Finally, cognitive assessment was done using the TICS-M Kannada version after obtaining informed consent over the telephone. This was done by an experienced rater (AP), a neurologist with formal training and experience in conducting the cognitive assessment of patients. Because depression can masquerade as cognitive dysfunction, we adopted the telephonic Patient Health Questionnaire-9 (PHQ-9)[13] to screen for depression as well. Functional assessment using the mRS was performed over the telephone.

The data collected were tabulated, and the correlation between clinical–demographic characteristics, radiological features, TICS-M Kannada, and PHQ-9 scores was analyzed. The mean and standard deviation for continuous variables and categorical variables were calculated and expressed as frequencies and percentages, respectively. The analysis of categorical variables was done using the Pearson Chi-Square test. The strength of the association was tested between the two continuous variables by using Pearson's correlation coefficient or Spearman rank correlation depending upon the normality of the data. A P value of ≤0.05 was considered significant.

The study methodology is shown in Figure 1.

Results

Clinical and demographic characteristics

The initial record search yielded 29 patients (21 females and eight males) with a diagnosis of DCVT fulfilling the inclusion criteria with a mean age of 29.67 ± 8.65 years. Three patients had expired during the hospital stay due to acute complications.

The most common clinical symptoms were headache in 27 (93%) patients, followed by altered sensorium in 26 (89.65%), and vomiting in 24 (79.31%). Less common manifestations included hemiparesis in 10 (31.03%), seizures in four (13.79%), and aphasia in four patients (13.79%). Rare features were extrapyramidal symptoms (parkinsonism) in two patients and visual impairment in one patient.

The risk factors for CVT were as follows: anemia, that is, Hb <12 g/dL in 22 patients (75.8%), followed by hyperhomocysteinemia (serum homocysteine levels >15 mg/dL) in 13 (42.3%), vitamin B12 deficiency (serum vitamin B12 levels <200 pg/mL) in nine (31%), and thrombocytosis (platelet count >450/10^9 L) in four patients (13.79%). Four patients were in postpartum, and three were on treatment with oral contraceptive pills. Moreover, three patients had history of alcohol dependence, and one patient had human immunodeficiency virus positivity. The majority of the patients (73%) had more than one risk factor.

All patients received treatment in the form of anti-edema measures and initial anticoagulation with unfractionated heparin, which was switched over to oral anticoagulation over 1–2 weeks. Oral anticoagulants were continued for 1–2 years. Patients who had seizures received anti-epileptics (levetiracetam/phenytoin). Drug doses were

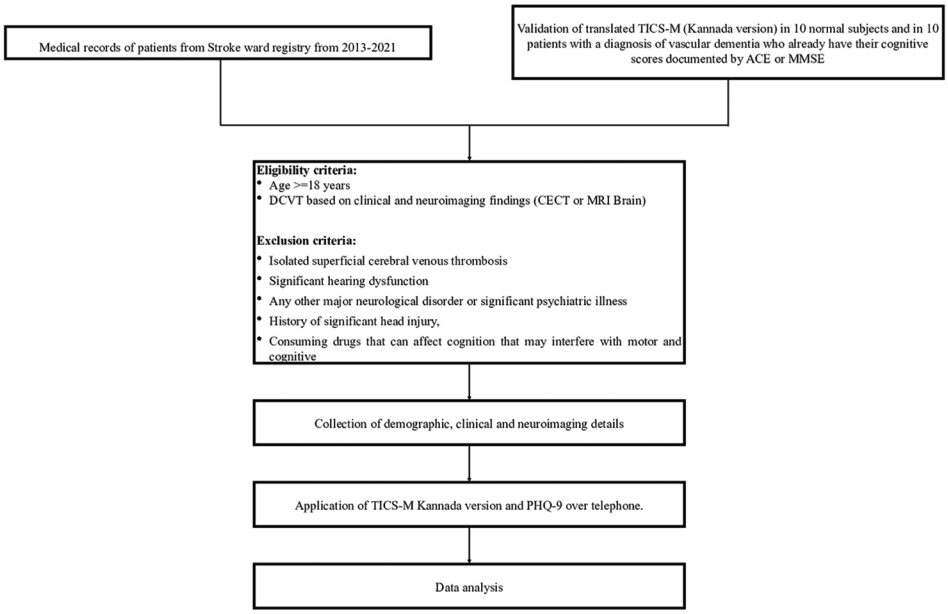

Figure 1: Flowchart of study methodology. (TICS-M: Modified Telephonic Interview for Cognitive Status; ACE: Addenbrooke cognitive evaluation; MMSE: Mini-mental state examination; DCVT: Deep cerebral venous sinus thrombosis; CT: Computed tomography; MRI: Magnetic resonance imaging; PHQ-9: Patient Health Questionnaire-9)

individualized. In addition, patients received treatment for risk factors such as anemia and vitamin B12 deficiency.

In the patients who survived the acute stage of illness (n = 26), long-term telephonic follow-up and cognitive screening were done. The clinical and demographic details and radiological characteristics of these 26 patients are shown in Table 1.

Radiological characteristics

Most patients (96.15%) had a brain parenchymal lesion (hemorrhagic venous infarction). This was mainly subcortical, with the thalami the most common structure involved in 24 patients (96%), followed by basal ganglia in 18 (72%), midbrain in four (12%), and cerebellum in one (4%). Cortical involvement was seen in two patients (8%). Bilateral lesions were seen in 21 patients (84%). The straight sinus was involved in all 26 (100%) patients, followed by the vein of Galen in 24 (92.3%) and internal cerebral veins in 18 (69.2%). Additional involvement of transverse and sigmoid (superficial venous sinuses) occurred in 11 (42.3%) and cortical vein thrombosis in one patient. Hydrocephalus was seen in 20 (76.9%) patients. Midline shift was seen in three patients (11.53%).

A follow-up CT/MRI of the brain was available for eight patients with a median follow-up time of 24 months (SD: ±11.31). Complete recanalization of the involved sinuses was seen in four (50%) patients, whereas two patients had partial recanalization, and one had recurrence of CVT later due to non-compliance to treatment. Two patients did not have contrast imaging to ascertain recanalization. All eight patients showed thalamic atrophy on follow-up imaging, and one patient showed atrophy of the basal ganglia [Figure 2].

Cognitive screening, screening for depression and functional status

The median time for follow-up cognitive assessment was 5.32 years (range: 15–98 months) after the initial occurrence of CVT. A total of 18 patients out of 29 could be contacted for telephonic follow-up. Eight patients (42.10%) had evidence for cognitive dysfunction on TICS-M. Deficits were mainly observed in the domains of recent memory and attention. Scores of 0–1 on mRS were achieved in all 18 patients. Three (16.66%) patients had evidence of depression on screening with PHQ-9. These findings are depicted in Table 1.

Correlation between cognitive screening, depression, and clinicodemographic characteristics

Correlation was examined between cognitive outcomes (TICS-M scores) and the clinical and radiological variables. No significant difference was found in the TICS-M scores between patients having unilateral versus bilateral lesions. Similarly, no significant difference was found in the TICS-M scores between patients having deep gray matter involvement (caudate and thalamus) as compared to those having additional cortical involvement (frontal, parietal, temporal, or occipital). Additional superficial sinus involvement did not predict cognitive outcomes. Our sample size was inadequate to examine the correlation between PHQ-9 scores and the side and site of lesions.

Neurological features at presentation (headache, vomiting, aphasia, seizures, hemiplegia, extrapyramidal features, altered sensorium) were not found to correlate with cognitive impairment

Table 1: Clinical and Demographic features of patients with deep CVT

Characteristics	Patients with cognitive impairment (*n*=8)	Patients without cognitive impairment (*n*=18)
Demographics		
Age in years, Mean±SD	35.5±11.8	26.6±5.7
Gender, *n* (%)		
Male	3 (37.5)	4 (22.2)
Female	5 (62.5)	14 (77.8)
Symptoms at presentation, *n* (%)		
Headache	8 (100)	16 (88.9)
Altered sensorium	7 (87.5)	17 (94.4)
Vomiting	5 (62.5)	17 (94.4)
Hemiparesis	2 (25.0)	6 (33.3)
Seizures	1 (12.5)	2 (11.1)
Aphasia	2 (25.0)	1 (5.6)
Others	1 (12.5)	
Risk factors, *n* (%)		
Anemia	6 (75.0)	16 (88.9)
Hyperhomocystenemia	4 (50)	6 (33.3)
Vitamin B12 deficiency	3 (37.5)	3 (16.7)
Oral Contraceptive	0	6 (33.3)
Post partum state	2 (25.0)	2 (11.1)
Alcoholism	1 (12.5)	1 (5.6)
Others	2 (25.0)	3 (16.7)
Imaging characteristics, *n* (%)		
Parenchymal lesion	8 (100)	17 (94.4)
a) Subcortical alone	7 (87.5)	15 (83.3)
b) Subcortical and cortical	1 (12.5)	2 (11.1)
Structures involved		
a) Thalami	8 (100)	16 (88.9)
b) Basal ganglia	4 (50)	14 (77.8)
Bilateral lesion	7 (87.5)	14 (77.8)
Sinus involved		
a) Straight sinus	7 (87.5)	17 (94.4)
b) Vein of Galen	7 (87.5)	17 (94.4)
c) Internal cerebral veins	3 (37.5)	15 (83.3)
d) Others	4 (50)	7 (38.9)
Hydrocephalus, *n* (%)	5 (62.5)	17 (94.4)
Follow up imaging, *n* (%)	1 (12.5)	7 (38.9)
M-TICS Score, Mean±SD	39.5±2.62	47.5±1.39
Patients with depression on PHQ-9 Questionnaire, *n* (%)	2 (25.0)	1 (5.6)

and did not differ significantly between patients having unilateral versus bilateral involvement. Presence of thalamic and caudate atrophy (rated by an experienced neuroradiologist via visual inspection) did not correlate with scores on cognitive testing. However, volumetric analysis was not done.

Discussion

In this first-of-its-kind study from India, we evaluated the long-term functional and cognitive outcomes in patients with DCVT during the COVID-19 pandemic by using a telephonic screening instrument (TICS-M) that was adapted to the local language (Kannada).

Longitudinal studies in CVT are few and focus on functional outcomes (mRS), rather than cognitive sequelae, which are often missed.[2,14,15] In a prior study, Koopman *et al.*[14] observed that up to 75% of patients with an apparently good outcome in CVT nevertheless reported long-term sequelae, such as impaired concentration, troublesome headache, depression, and fatigue. Long-term follow-up studies for functional and cognitive outcomes of disorders such as DCVT and predictors of the outcome in DCVT in the Indian setting are lacking, constituting an important knowledge gap, which our study addressed.

Compared to patients with arterial stroke, our cohort was relatively young (mean age: 29.67 ± 8.6 years). Although a female preponderance (72.4%) was seen, which could be explained by factors such as postpartum CVT, this risk factor constituted only a small percentage, and factors such as anemia, hyperhomocysteinemia, and vitamin B12 deficiency

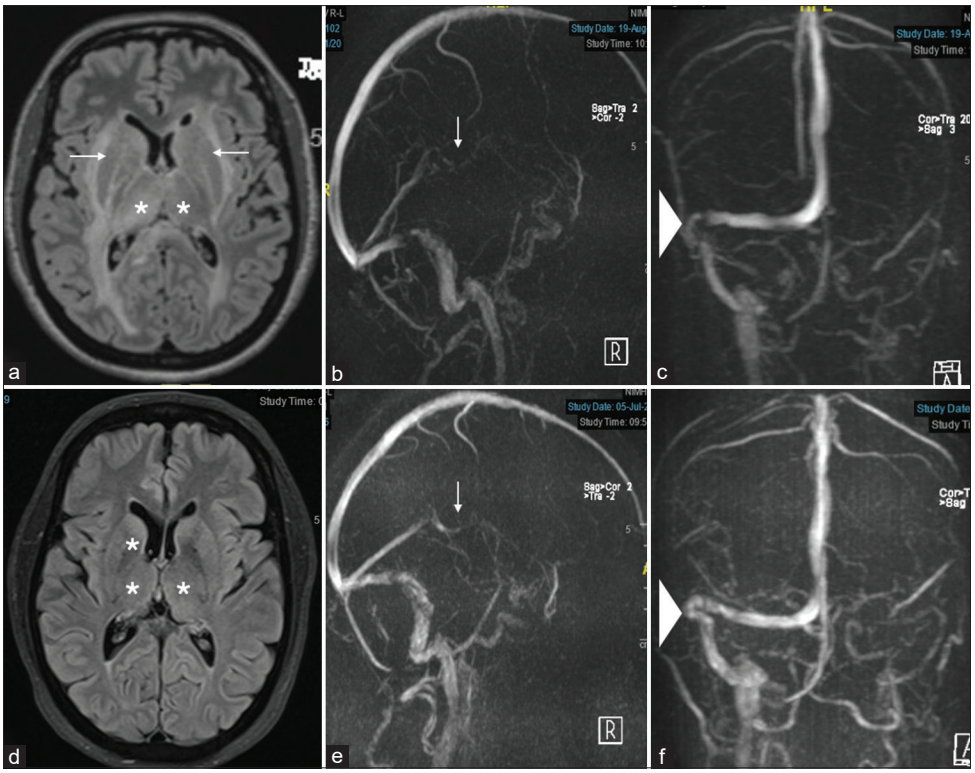

Figure 2: (a) MRI T2 FLAIR images of patient with cognitive impairment prior to treatment showing bilateral basal ganglia (solid white arrow), bilateral thalamus (white asterisk) involvement with periventricular ooze. (b and c) Time-of-flight MRV images show the absence of flow in deep venous sinuses (solid white arrow) and irregular flow at the right transverse sigmoid sinus junction (white arrowhead). Follow-up images after 35 weeks show (d) atrophy of bilateral thalami and right basal ganglia (white asterisk) and (e and f) recanalization of deep sinuses (solid white arrow) and right transverse sigmoid sinus junction (white arrowhead)

predominated, indicating a pattern shift in traditional risk factors (postpartum) for CVT.[16] Factors such as improved awareness, improved antepartum and postpartum care, and avoidance of postpartum water restriction may be responsible for this shift. A similar change in the trend of CVT epidemiology has been observed in other studies across the globe.[17,18] Headache was the most common symptom, consistent with previous reports;[19] however, the higher incidence of altered sensorium in our cohort may be due to the involvement of the thalamus, which has an important role in consciousness, sleep, and arousal. Bilateral thalamic involvement leading to obstructive hydrocephalus at the level of the third ventricle[20] may have contributed to encephalopathy.

Our results revealed possible long-term residual cognitive deficits in a significant proportion of patients. Pathological sequelae of impaired venous circulation due to CVT can lead to cognitive dysfunction in several ways. These include blood–brain barrier disruption, neuroinflammation, exacerbation of neurodegeneration, development of cerebral microhemorrhages of venous origin, altered production of cerebrospinal fluid, impaired function of the glymphatic system, dysregulation of cerebral blood flow, and ischemic neuronal dysfunction and damage.[21] Delayed recanalization of deep cerebral veins and thereby delayed restoration of deep venous circulation may underlie thalamic atrophy and long-term cognitive sequelae.[22] It is known that the thalamus as a whole and the pulvinar nuclei, in particular, are involved in cognitive functions such as attention, speed of information processing, and memory.[23,24] Apart from the thalamus, other subcortical anatomical structures involved in cognitive processing such as the caudate nucleus and the cerebellum were involved in some of the patients. Although we did not find a significant correlation between thalamic or caudate atrophy and TICS-M scores, the small sample size and lack of quantitative volumetric analysis preclude definitive conclusions. Differences in cognitive trajectories between patients may also stem from differences in baseline cognitive reserve and cognitive remediation strategies.[25]

It has been previously reported that 30% of patients with CVT had depression at a follow-up after 2.1 years, in the absence of cognitive impairment.[26] As depression can mimic signs of cognitive dysfunction, we assessed for depression separately by using the PHQ-9.[27] It is unclear whether the presence of depression in the three patients in our cohort of patients could be attributed to the antecedent event of DCVT, but a link due to the involvement of the thalamofrontal pathways is plausible.

DCVT is known to carry a higher mortality rate due to the development of complications such as hydrocephalus and status epilepticus.[28] However, in patients who survived the acute stage of illness, we noted that long-term functional outcome was very good, and all the patients attained an mRS

of 0–1. These findings confirm observations from a previous longitudinal follow-up study of CVT, in which an mRS score of 2 or less was found in 81% of patients at a median follow-up duration of 3.8 years.[15] Therefore, acute stage management of DCVT is extremely crucial, and long-term functional outcome tends to be much better than that of arterial stroke.

In addition to the novel demonstration of long-term cognitive sequelae of DCVT, the other highlight of our study was the successful regional language adaptation and application of telephonic cognitive assessment. This is important in culturally and linguistically diverse settings like India, where English fluency may impact the results of cognitive assessment. Although TICS-M has been well validated for use in patients with vascular dementia, ours was a pilot attempt to explore its utility in DCVT patients. We found that the TICS-M Kannada version was simple to apply telephonically. Several cognitive domains could be reliably tested, and a screening of cognitive deficits could be done with the convenience of telemedicine.

Limitations of the study: Although cognitive screening was done using TICS-M (Kannada), detailed neuropsychological evaluation of these patients to ascertain the nature and degree of cognitive impairment could not be done due to the COVID-19 pandemic. Moreover, adaptation into other regional languages will have to be explored to ensure wider application of telephonic cognitive screening.

Conclusion

Survivors of acute DCVT can potentially have long-term cognitive sequelae. Screening for cognitive dysfunction, depression, and functional status can be effectively done using telephonically applied scales that are adapted to the local language. Neuropsychological evaluation and early cognitive rehabilitation may be initiated for patients in whom deficits are identified on cognitive screening.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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