Acute Isolated Thalamic Infarcts: Clinical and Imaging Profile in Oman

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Abstract

Introduction— Thalamus is supplied by multiple arteries drawn from both anterior and posterior circulations often with vascular variants, therefore thalamic infarcts can present with a broad spectrum of symptoms and signs that can be challenging both clinically and radiologically.

Objectives and Methods— A retrospective observational hospital-based study. Aiming to identify the clinical profile, arterial territories involved, vascular variations and outcome of patients admitted with acute isolated thalamic infractions. The ability of the imaging modalities used to pick up the thalamic infarcts in the acute phase was also evaluated.

Results— Over a period of 33 months, 1260 acute ischemic stroke cases were admitted. 53 (4.2%) were isolated thalamic infarctions. Hemiparesis and dysarthria were the most common clinical presentation. 29/53 patients underwent head computerized tomography within 24 hours of the symptom onset. The thalamic infarct was only detected in 5/29 (17.2%) of the scans. Computerized tomographic angiography was used to assess the vasculature in 49/53 (92%) of patients. Normal posterior circulation was seen in only 17/49 (35%). Bilaterally absent posterior communicating artery 13/49 (26%) was the commonest variant seen. The majority of the lesions 41/53 (77%) were localized to the ventrolateral thalamus (thalamogeniculate) artery. There was no mortality, and 40/53 (75%) achieved a modified Rankin Scale of ≤2 upon discharge.

Conclusions— Isolated thalamic infarcts represent a broad spectrum of clinical syndromes. In more than 80% of patients, a computerized tomography head can be normal in the first 24 hours. Only less than one-third had a normal posterior circulation vasculature. The ventrolateral (thalamogeniculate) thalamus artery was the most commonly involved vascular distribution.

Keywords— Isolated, Thalamus, Infarct, Clinical presentations, Vascular territory, Variation , Posterior circulation vasculature.

INTRODUCTION

Thalamus plays a major role in cortical activity[1]. It projects to the cortex in a highly organized fashion. Nearly all the pathways that project to the cerebral cortex do so via synaptic relays in the thalamus. Anatomically and functionally four regions can be distinguished in the thalamus; anterior, posterior, medical, and lateral[2].

The complex thalamus anatomy can be divided into the following four main regions. (1) Nonspecific thalamic nuclei which include midline, interlaminar, reticular, and some areas of the ventral anterior nuclei that mediate the general cortical alerting response[3]. These nuclei receive strong projections from mid-brain reticular formations and project back to the mid-brain and to the specific thalamic nuclei. (2) Specific thalamic nuclei mediate sensory response from different parts of the body and project to somatosensory, visual, and auditory cortex areas[4]. It includes the ventral, lateral and

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TABLE 1: The baseline demographic and clinical characteristic of the patients.

Variable	n (%)
Sex	
Men	32 (60.4)
Women	21 (39.6)
Age, mean±SD	60.42±11.62
Hypertension	42 (79.2)
Diabetes Mellitus	26 (49.1)
Dyslipidemia	7 (13.2)
Ischemic Heart Disease	4 (7.5)
Atrial Fibrillation	3 (5.7)
Smoking	5 (9.4)
Chronic Kidney Disease	6 (11.3)
Obesity	0 (0)
Old Stroke	11 (20.8)
Alcoholic	1 (1.9)
Others	9 (17.0)

TABLE 2: Etiology of ischemic stroke.

Variable	n (%)
Earlier stroke TOAST	
No	30 (56.6)
Lacunar	17 (32.1)
CE	6 (11.3)
Hemorrhagic transformation	4 (7.5)
Conservative treatment	53 (100)
TOAST classification – etiology	
Lacune posterior circulation	34 (65.4)
Lacune posterior circulation + other cardio-embolic source	2 (3.8)
Atrial fibrillation	2 (3.8)
Atrial fibrillation + other cardioembolic source	1 (1.9)
Atrial Fibrillation+ Valve disease	1 (1.9)
Endocarditis	1 (1.9)
Intracranial atherosclerotic disease	6 (11.5)
Intracranial atherosclerotic disease+ Lacune posterior circulation	1 (1.9)
Left ventricular thrombus	1(1.9)
Other cardio embolic source	2 (3.8)
Others -mention etiology below	1 (1.9)

basal nuclei (medial geniculate body and lateral geniculate body) which are the relay station for the visual pathway which receives input from retinal region cells through the optic tract and projects to the calcarine cortex through the optic radiations. (3) Limbic nuclei which include the dorsomedial and anterior thalamic nuclear groups. They mediate mood and motivation[5-7]. (4) Effector nuclei ventrolateral (ventro-anterior and ventro-lateral) that mediates language and motor function[8,9]. It receives input from the cerebellum and projects to the precentral cortex or primary motor cortex. (5) Associative nuclei including dorsolateral and posterior area of the thalamus e.g., Pulvinar which contributes to the high level of cognitive function[10].

Cerebrovascular disease is the most common cause of discrete thalamic pathology resulting in signs and symptoms of a localizing value. The main risk factors for thalamic strokes include atrial fibrillation, hypertension, atherosclerosis, diabetes, and dyslipidemia. Rarely, it includes factors such as migraine and vasculitis[11]. The mean age at presentation is 48.2 years for thalamic vascular lesions. A study by del Mar Saez and colleagues[11] showed that 67% of patients to be men and 33% were women. The knowledge of the vascular supply of thalamic nuclei helps greatly to understand the so-called thalamic syndromes and localization of thalamic lesions. The thalamic arteries arise from the posterior communicating arteries and form perimensencephalic segment of the posterior cerebral artery[12-15]. This study demonstrates the clinical and vascular variation seen in thalamic infarcts and address the anatomic variations in our cohort.

OBJECTIVES AND METHOD

A retrospective 33 months observational hospital-based study was conducted at the central stroke unit of the Sultanate of Oman. The central stroke unit is the largest and the only comprehensive stroke unit in the country. It receives stroke patients either through the emergency department or as a referral from other stroke units or hospitals in the country.

This study included patients aged 18 and above who were admitted to the unit with acute isolated thalamic infarct from January 2018 to September 2020. The data was retrieved through electronic medical record system. The radiological images were reviewed through the electronic picture archiving and communication system by a radiologist. The arterial territories involved and vascular variations and the ability of the imaging tools used to pick up the thalamic infarcts in the acute phase were also evaluated.

Acute thalamic infarct was defined as an acute onset of focal neurological deficits within 48 hours. The diagnosis of acute stroke was confirmed by detailed neurological examination and brain imaging. Computerized tomography or magnetic resonance imaging were used to confirm the diagnosis. The vascular and arterial territories involved were accessed by computerized tomographic angiography.

Treatment of acute stroke trial (TOAST) classification was used to classify ischemic stroke etiology. Both the national

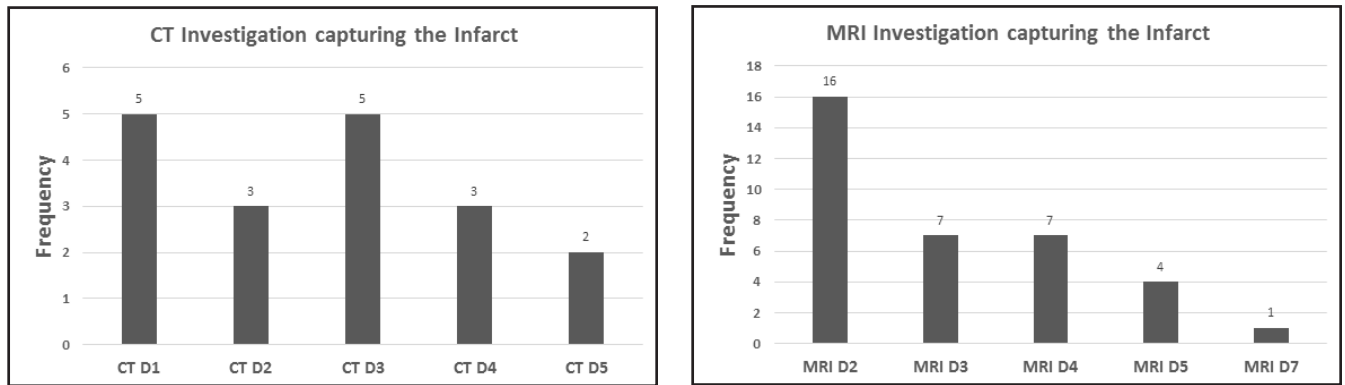**FIGURE 1**: Investigations capturing the infarct.
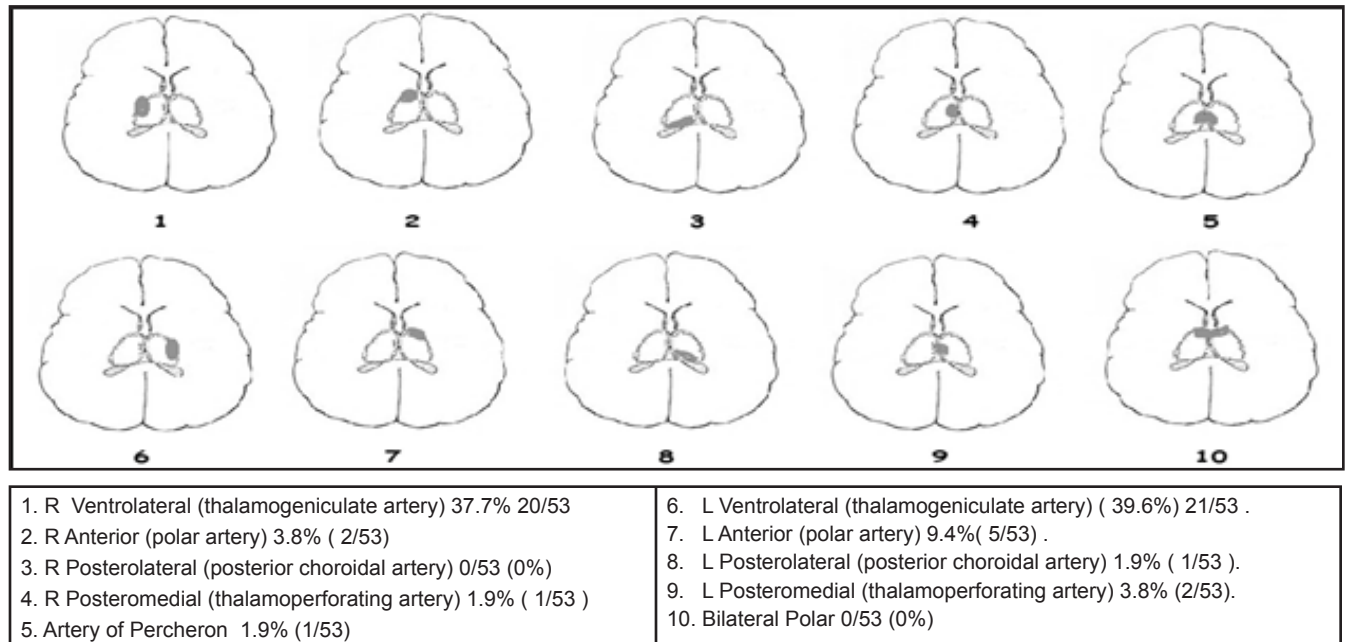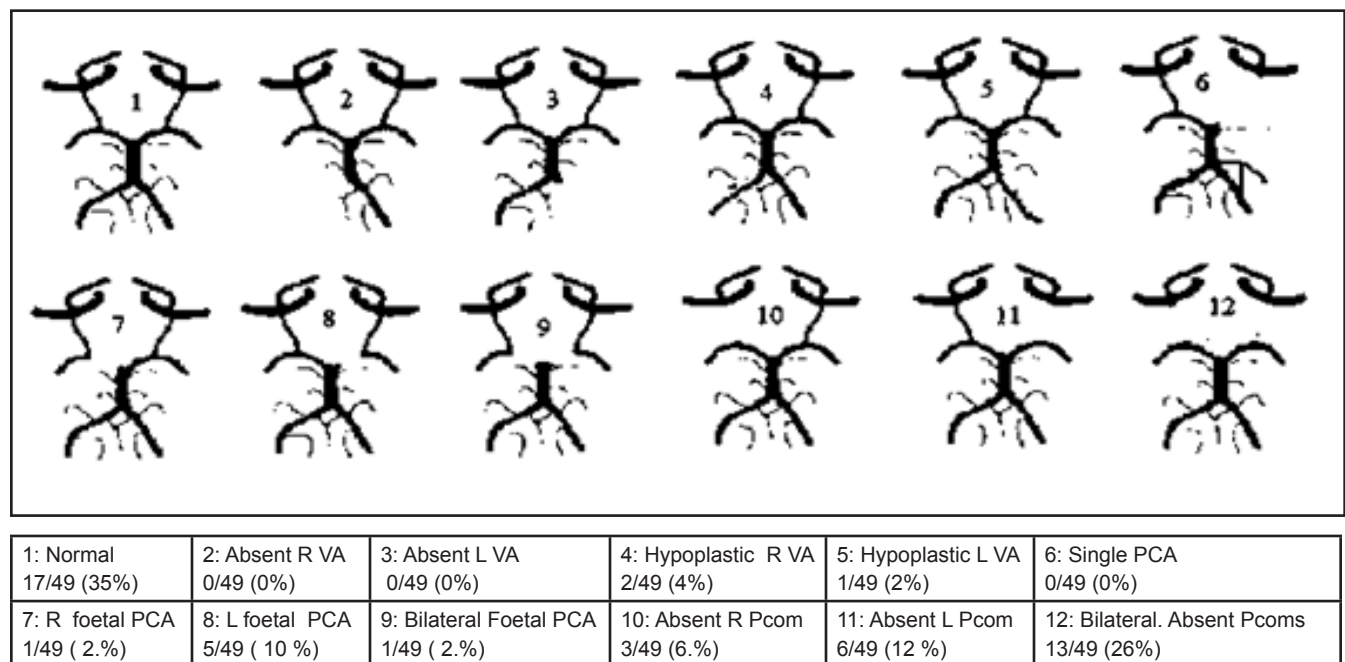

FIGURE 2: Pattern of thalamic territories involved.

1. R Ventrolateral (thalamogeniculate artery) 37.7% 20/53	6. L Ventrolateral (thalamogeniculate artery) (39.6%) 21/53 .
2. R Anterior (polar artery) 3.8% (2/53)	7. L Anterior (polar artery) 9.4%(5/53) .
3. R Posterolateral (posterior choroidal artery) 0/53 (0%)	8. L Posterolateral (posterior choroidal artery) 1.9% (1/53).
4. R Posteromedial (thalamoperforating artery) 1.9% (1/53)	9. L Posteromedial (thalamoperforating artery) 3.8% (2/53).
5. Artery of Percheron 1.9% (1/53)	10. Bilateral Polar 0/53 (0%)

FIGURE 3: Variation of post circulation vasculature: (Assessed by CT Angiogram in 49 patients).

1: Normal 17/49 (35%)	2: Absent R VA 0/49 (0%)	3: Absent L VA 0/49 (0%)	4: Hypoplastic R VA 2/49 (4%)	5: Hypoplastic L VA 1/49 (2%)	6: Single PCA 0/49 (0%)
7: R foetal PCA 1/49 (2.%)	8: L foetal PCA 5/49 (10 %)	9: Bilateral Foetal PCA 1/49 (2.%)	10: Absent R Pcom 3/49 (6.%)	11: Absent L Pcom 6/49 (12 %)	12: Bilateral. Absent Pcoms 13/49 (26%)

RVA: right vertebral artery, LVA: left vertebral artery, PCA: posterior cerebral artery, Pcom: posterior Communicating, R : right, L : left

institute of health stroke scale and modified Rankin scale were used to access the stroke severity during admission and discharge.

Statistical Analysis

Data were summarized using descriptive statistics measures. Continuous variables were summarized using mean with stander deviation and median with interquartile range. Categorical variables were summarized using the frequency and percentage. Correlation between National Institute of Health stroke scale at admission and modified Rankin scale at discharge was tested using the non-parametric Spearman's rank correlation coefficient. A p-value less than 0.05 were considered statistically significant. All the analysis was carried out using the international business machines, statistical package for the social science Statistics version 26.0.

RESULTS

During the period of 33 months, the central stroke unit received and treated a total of 1967 stroke patients. Out of these 1260 were acute ischemic stroke cases. Isolated thalamic infarctions were only seen in 53 (4.2%). All patients were evaluated clinically within 48 hours of symptom onset. Men constituted 60.4 % of the cases. The mean age was 60.4 years (range 42-97 years). Hypertension was the most common risk factor seen in 42/53 (79.2%), followed by diabetes mellitus 26/53 (49.1%), dyslipidemia 7/53 (13.2%) and atrial fibrillation 3/53 (5.7%). Table 1: Summarize the characteristic of the cohort group.

Most of the patients presented with multiple symptoms. Hemiparesis (including facial weakness) was the most common complain seen in 41/53 (77%) of patients. Dysarthria was the second most frequent clinical presentation of thalamic infarct seen in 20/53 (37.3%) of patients. Isolated sensory symptoms were reported by 14/53 (26.4%) patients, 10/53 (18.9%) patients had ataxia, and only 5/53 (9.4%) patients had cognitive deficits (memory and language deficits) or visual symptoms 5/53 (9.4%). Loss of consciousness was seen only in 4/53 (7.5%) patients. Upon admission, the median national institute of health stroke scale for the studied group was 4 (2-6). The median modified Rankin Scale at discharge was 1 (1-2.5). Most of the patients' modified Rankin Scale improved through the course of admission. 15.1% (8/53) had achieved modified Rankin scale of 0. 39.6% (21/53) of patients had a modified Rankin Scale of 1, 20.8% (11/53) had a modified Rankin scale of 2, and 18.9% (10/53) had a modified Rankin scale of 3. Only 5.7% (3/53) had modified Rankin Scale of 4. There was no mortality.

A history of the previous stroke was seen in 30 patients (56.6%). The majority of these strokes as assessed by the TOAST classification was lacunar and seen in 34/53 (64 %). Cardioembolic stroke was seen in 8/53(15%) of patients. Only six patients 6/53(11%) had large artery disease.

INVESTIGATIONS: VASCULAR IMAGING

The diagnosis of the thalamic infarct was established either by plane head computerized tomography or magnetic resonance imaging. Computerized tomographic angiography was used to assess the vasculature in 49/53 (92%). The involvement of the left thalamus was seen in 30/53 (56%). Only 1/53 patients had bilateral thalamic involvement. The chance of the plain computerized tomography detecting the thalamic infarcts increased when the imaging was done beyond 48 hours. 29/53 (55%) patients had head computerized tomography within 24 hours of the onset of symptoms of which only 5/29 (17.2%) detected the thalamic infarct. 10/53 computerized tomography head done between 24 to 48 hours of the onset of symptoms of which 3/10 (30%) detected the infarction. In all cases where the computerized tomography head did not show the infarction, magnetic resonance imaging was done to confirm the diagnosis. Fig 1: Investigation capturing the infarct.

The ventrolateral (thalamogeniculate) thalamus artery was the most common arterial territory involved followed by the polar arteries. Fig 2: Pattern of thalamic territories involved. A normal posterior circulation was seen in only 17/49 (35%). Bilaterally absent posterior communication artery 13/49 (26%) was the most variant seen. Fig 3: Variation in posterior circulation vasculature: (Assessed by computerized tomography angiogram in 49 patients)

All patients received conservative treatments single or dual antiplatelet therapy. Hemorrhagic transformation was seen in 4/53 (7.5%) of the patients. It happened spontaneously in three patients and after receiving intravenous recombinant tissue plasminogen activator (rt-PA) in one patient. One patient had fever and gastrointestinal bleeding. Another patient had urinary tract infection. A third patient required developed respiratory failure that required tracheostomy.

DISCUSSION

Ischemic strokes are the most common etiology of discrete thalamic pathology resulting in signs and symptoms of localizing value[16]. In 1874 the detailed vascularization of the thalamus was first studied by Duret[17]. This was then followed by other groups of researchers[16-19]. This included the study of the artery of Percheron[12-14,20] followed by further studies to better understand the clinical anatomical correlation [21-29].

The mean age at presentation of 60.4 years in our study is similar to values from other international studies such as Propokiv et al[30]. Hemiparesis, hemianesthesia, and motor aphasia were the most common symptoms and correlated with the vascular territory of ventrolateral thalamic infarction. Pain is not a common symptom in the acute phase[31] as seen in our study. Recovery in thalamic stroke is found to be good and most patients return to their functional independence[32]. In our study, there was no mortality and 75% achieving modified Rankin Scale of 2 or less by the time of discharge.

Nearly all the pathways that project to the cerebral cortex are via synaptic relays in the thalamus, due to its complex anatomy and blood supply. Therefore, thalamic infarctions can present with a broad spectrum of symptoms and signs. Often these may mislead clinicians to an alternative diagnosis. In such setting, it is important to note that computerized tomographic scans which is the main imaging modality used in acute ischemic stroke settings may not identify these infarction when done within the first 24 hours and whenever thalamic infarcts are suspected, magnetic resonance imaging if available will be a better imaging choice[33]. Apart from the diagnosis of acute infarctions the other differentials include tumor and venous infarcts[34-35] which may also be detected by magnetic resonance imaging. The most common vascular territory involved in our series, constituting 77.3% of thalamic infarctions was the ventrolateral (thalamogeniculate) arteries. This finding is similar to other studies such as the one by Song et al[36], which found that 73% of patients with thalamic infarctions had involvement of ventrolateral (thalamogeniculate) arteries[36].

Unilateral (4-26%) and bilateral foetal posterior communicating artery (2-4%) variants constituted the majority of vascular variants[37,38]. Unlike previous studies[37,38], our study found bilateral foetal posterior communicating artery variants to be part of an overwhelming majority (28.9%) equal to the normal variant. Other common variants include foetal posterior cerebral artery which constitutes 5.6% of variants[39-41].

CONCLUSION

Thalamic infarcts can present with a broad spectrum of clinical syndromes. When acute isolated thalamic infarcts are suspected, brain magnetic resonance imaging should be the imaging of choice. Posterior circulation vasculature and arterial territory variations are frequently seen with thalamic infarctions.

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