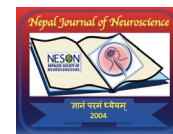


Truncal pulsion in acute ischemic stroke – Clinico-anatomical correlation



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Abstract

Introduction: Truncal pulsion is a compelling sensation of being pulled to one side, in the context of normal motor and sensory functions and normal co-ordination. It is seen in a variety of ischemic strokes. This article aims at identifying ischemic stroke syndromes associated with truncal pulsion, with review of the neural substrate responsible, to help in recognition and reporting.


Methods and Materials: This was a prospective study conducted at Department of Neurology, at Pushpagiri institute of medical sciences and research centre, Thiruvalla, Kerala, India, over a period of 5 years, among patients with acute ischemic stroke. We studied all patients with acute ischemic stroke, who were admitted to our department, over a five-year period. Patients presenting with truncal pulsion were shortlisted and were assessed by investigators independently. All patients underwent MRI brain, which was assessed by investigators 1 and 2, independently. The demographic profile, risk factors, clinical features, neuroimaging findings and outcomes were analysed using SPSSv21.

Results: A total of 1456 patients with acute ischemic strokes were identified, of which 27 with truncal pulsion were included in the study. The common sites of infarction resulting in truncal pulsion were lateral medulla, cerebellum, thalamus, pons and midbrain. One patient had infarct involving anterior cingulate. Truncal pulsion was ipsilateral in infarcts involving medulla and cerebellum and contralateral in infarcts involving the pons, midbrain, mesencephalo-diencephalic junction, thalamus and cingulate.

Conclusions: Truncal pulsion, a compelling sensation of falling, is a disabling symptom occurring in a variety of strokes and it poses significant challenge in neuro-rehabilitation.

Key words: cingulate, ischemic stroke, medulla, midbrain, pons, thalamus, truncal pulsion

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Introduction

Truncal pulsion refers to the compelling sensation of being pulled to one side or falling to one side, that cannot be explained by weakness, sensory loss or ataxia.¹ Truncal ipsi-pulsion refers to falling towards the side of the lesion and it has been reported in lesions of peripheral vestibular system, cerebellum, medulla and lower pons.² Truncal contra-pulsion refers to falling towards the side opposite to the lesion and it is described in lesions of upper pons, midbrain, thalamus and cingulate.^{3,4} Though there have been studies on truncal latero-pulsion in lateral medullary infarcts, detailed studies on truncal pulsion in strokes involving other areas are sparse.

This article aims at identifying ischemic stroke syndromes associated with truncal pulsion, with review of the neural substrate responsible, to aid in recognition and reporting.

Methods and Materials

This was a prospective study conducted at the department of Neurology at Pusphpagiri Institute of

Results

Medical Sciences and Research Centre, Thiruvalla, Kerala, India over a period of five years, from June 2015 to June 2020. We identified 1456 consecutive patients with acute ischemic strokes. Patients presenting with truncal pulsion were included in the study and the neurological findings were independently assessed by investigators. All the patients underwent a standard protocol of investigations, including MRI brain, 12-lead ECG and transthoracic echocardiography. The neuroimaging findings were assessed by investigators 1 and 2, independently. The demographic profile, vascular risk factors such as systemic hypertension, diabetes mellitus, dyslipidaemia, and history of heart disease / chronic kidney disease were recorded. Functional outcome was assessed at discharge and at the end of two weeks. Statistical analysis was done using SPSS v21.

A total of 27 patients with truncal pulsion were identified over this five-year period. Age group ranged from 34 years to 80 years, with a mean age of 62.9 ± 2.6 years. There were 19 males and 8 females. Twenty-two patients were hypertensive, 20 were diabetic and nine patients had dyslipidaemia. Five had pre-existing coronary artery disease and two had atrial fibrillation. Four patients had pre-existing chronic kidney disease.

Truncal pulsion was contralateral in five patients, of which one had infarct involving the midbrain, two had infarcts involving the thalamus, one in the mesencephalo-diencephalic junction and one in the pons. The patient with midbrain lesion had infarct involving the paramedian region and had associated ipsilateral third nerve palsy with contralateral limb ataxia, suggestive of Claude syndrome (Table 1, case 8, Figure 1) Of the two patients

SN	Age	Sex	Truncal pulsion	Ocular pulsion	Limb ataxia	MRI brain – location of infarct
1	48	F	I/L	I/L	+	Lateral medulla
2	78	M	I/L	I/L	+	Lateral medulla
3	65	M	I/L	I/L	+	Lateral medulla
4	48	M	I/L	I/L	+	Cerebellum
5	46	M	I/L	I/L	+	Cerebellum
6	80	M	I/L	I/L	+	Cerebellum
7	35	M	C/L	-	-	Mesencephalo-diencephalic junction
8	34	M	C/L	-	+	Midbrain
9	64	F	C/L	-	-	Thalamus
10	65	F	Retropulsion	-	-	Cingulate
11	80	M	Retropulsion	-	-	Bilateral Cerebellar tonsils
12	62	F	I/L	I/L	+	Cerebellum
13	71	M	I/L	I/L	+	Cerebellum
14	64	F	I/L	I/L	+	Cerebellum
15	68	F	C/L	-	-	Thalamus
16	73	F	I/L	I/L	+	Lateral medulla
17	68	M	I/L	I/L	+	Lateral medulla
18	63	M	I/L	I/L	+	Lateral medulla
19	72	M	I/L	I/L	+	Lateral medulla
20	63	M	I/L	I/L	+	Lateral medulla
21	65	M	I/L	I/L	+	Lateral medulla
22	68	M	I/L	I/L	+	Lateral medulla
23	71	M	I/L	I/L	+	Lateral medulla
24	65	M	I/L	I/L	+	Lateral medulla
25	62	M	I/L	I/L	+	Lateral medulla
26	58	F	I/L	I/L	+	Lateral medulla
27	58	M	C/L	-	+	Pons

Table 1: Clinical details of patients with truncal pulsion, with site of lesion on MRI. C/L – contralateral, I/L- ipsilateral, F- female, M- male

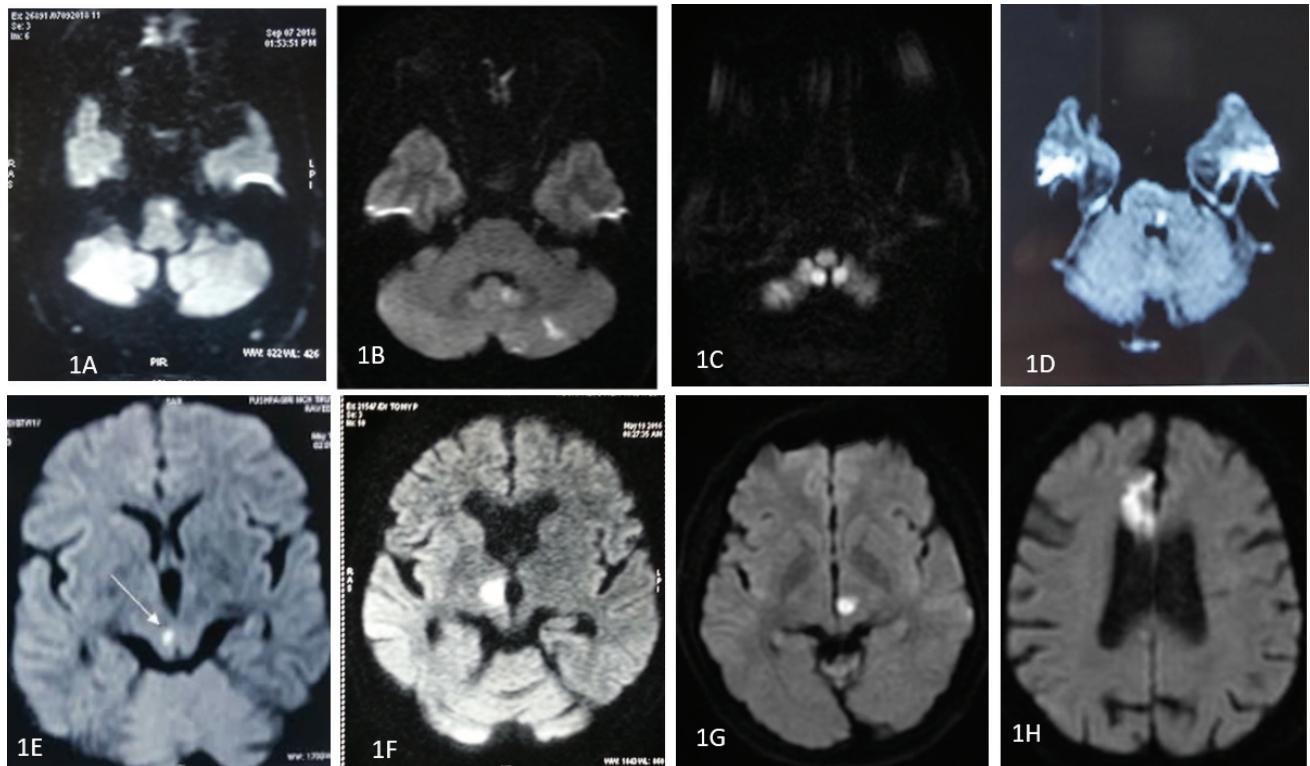


Figure 1: Diffusion weighted MR images in 8 representative patients showing acute infarcts in: 1A: left lateral medulla, 1B: left vermis and left cerebellar hemisphere, 1C: bilateral cerebellar tonsils, 1D: left pons, 1E: right midbrain, 1F: right mesencephalo-diencephalic junction, 1G: left thalamus, 1H: right anterior cingulate

with thalamic infarcts, one had truncal contra-pulsion as the sole manifestation. The location of these infarcts was in the postero-lateral thalamus (Table 1, case 9, Figure 1) The patient with infarct in the mesencephalon-diencephalic junction also had truncal contra-pulsion as the sole manifestation (Table 1, case 7, Figure 1). The patient with pontine infarction had truncal contra-pulsion. Twenty patients had truncal ipsi-pulsion, of which six had infarcts involving cerebellum and 14 had infarcts involving the lateral medulla (Table 1). All the six patients with infarcts involving the cerebellum, who presented with truncal ipsi-pulsion had associated ocular ipsi-pulsion, gaze evoked nystagmus and ipsilateral limb ataxia. Truncal ipsi-pulsion in all the 14 patients with lateral medullary infarcts was associated with ocular ipsi-pulsion, gaze evoked nystagmus and ipsilateral limb ataxia (Table 1). One patient with bilateral cerebellar tonsil infarct had truncal retropulsion. One patient with anterior cingulate infarct also presented with truncal retropulsion (Video 1). At follow up, 2 weeks later, 20 patients had a mRS score (modified Rankin score) of 4 (moderately severe disability; unable to walk without assistance of another individual), 5 had a score of 3 (moderate disability; requiring some external help but able to walk without the assistance of another individual) and 2 had a score of 0 (no residual symptoms).

Discussion

Truncal latero-pulsion or body latero-pulsion is a disabling symptom noted in various ischemic strokes. Though it has been described commonly in lateral medullary syndromes, the presence of the same in strokes involving other areas of the brain have not been well characterised. The neural circuitry responsible for the same is elusive and intricate.

Truncal balance is maintained primarily by vestibular function and proprioceptive function. One of the mechanisms of truncal latero-pulsion is thought to be due to vestibular dysfunction in the roll plane of the vestibulo-ocular reflex.⁵ The ascending graviceptive input (vestibulo-thalamic pathway) from the otoliths at the level of the vestibular nuclei are passed on to the oculomotor nuclei in the brainstem, thalamus and cortical centers for control of body position and perception of verticality. The graviceptive pathway is crossed, with the medial longitudinal fasciculus.⁵ Hence, involvement of the ascending fibres of the vestibular and cerebellar pathway in the brainstem can result in body pulsion. The vestibulo-thalamic pathway (ascending graviceptive pathway), the dentatorubrothalamic pathway, the fastigio-thalamic pathway and the thalamocortical fascicle play important roles in the maintenance of body posture and stability, and

a lesion affecting these pathways can cause body latero-pulsion.

Truncal ipsi-pulsion is commonly described with lateral medullary infarcts and the neural structures responsible for the same at this level are the vestibular nucleus, descending lateral vestibulospinal tract [LVST] or the ascending dorsal spinocerebellar tract [DSCT])² (Figure 2). Thomke et al have proposed that truncal ipsi-pulsion with limb ataxia results due to loss of proprioceptive information, due to involvement of DSCT, whereas ipsi-pulsion without limb ataxia results due to impaired vestibulospinal posture control due to lesion in the LVST.² In our study, truncal ipsi-pulsion was noted in 14 patients with lateral medullary infarcts and it was associated with ipsilateral limb ataxia.

In the study by Ye et al in patients with isolated cerebellar infarctions, latero-pulsion was observed in approximately 85% of the patients.⁶ The cerebellum regulates posture control by acting on the cerebral cortex via the thalamocortical projection and via the connections to the brainstem. Signals from the labyrinth ascend the vestibular nerve to the flocculus and vermis of the cerebellum. The fastigial nucleus has excitatory connections to the contralateral vestibular nucleus. The caudal vermis has an inhibitory influence on the ipsilateral fastigial nucleus. Therefore, lesions of the caudal vermis disinhibit the fastigial nucleus, resulting in stimulation of the contralateral vestibular nucleus, resulting in ipsi-pulsion.⁶ Isolated latero-pulsion has been reported in a patient with a lesion in the rostral vermis, the explanation being that it receives the spinocerebellar tract.⁶ A lesion

involving the nodulus results in contralateral pulsion. In our study, six patients with infarcts in the cerebellar vermis had truncal ipsi-pulsion. Infarction of the cerebellar tonsil is rare, it has been reported that tonsillar infarction can result in truncal contra-pulsion. It is proposed that the tonsil exerts inhibitory effects on the vestibular nucleus on the ipsilateral side, similar to nodulus. Thus, a lesion in the nodulus disinhibits the ipsilateral vestibular nucleus, resulting in truncal contra-pulsion.⁷ However, in our patient with infarction in bilateral cerebellar tonsil, truncal retropulsion was noted. Truncal retropulsion in cerebellar tonsillar infarction has not been reported previously.

Truncal pulsion is reported in brainstem lesions (Figure 2). The vestibular input, i.e., the graviceptive pathway ascends through the brainstem, via the contralateral medial longitudinal fasciculus, to reach the oculomotor nuclei, trochlear nuclei, the thalamus and the cortex. It is proposed that it crosses the midline at the pontine level, hence, infarcts involving the caudal pons, before the crossing result in truncal ipsi-pulsion, whereas rostral pontine lesions cause contra-pulsion.⁴ Truncal contra-pulsion was noted in one of our patients with pontine infarct. Lesions of the midbrain also cause contra-pulsion. It has been reported that the vestibulothalamic, dentatorubrothalamic, and fastigiotalamic fibers join the thalamic fascicle adjacent to the red nucleus and that interruption of any of these fibers, in a mesencephalic infarction can cause latero-pulsion. In mesodiencephalic lesion, truncal contra-pulsion results due to involvement of the crossed cerebellothalamic tract.^{5,8} However, infarction in the region of the superior cerebellar artery results in

Site	Neural structure	Pathways
Peripheral labyrinth	Vestibular nerve	
Cerebellum	Fastigial nucleus Vermis Nodulus	Fastigio-thalamic tract Dentato-rubro-thalamic tract
Medulla	Vestibular nucleus	Lateral vestibulo-spinal tract Dorsal spinocerebellar tract
Pons	MLF	Ascending graviceptive pathway
Midbrain		Vestibulothalamic pathway Dentato-rubro-thalamic Fastigio-thalamic pathway
Thalamus	Ventro-lateral nucleus	Thalamo-cortical pathway Vestibulothalamic pathway Dentato-rubro-thalamic Fastigio-thalamic pathway
Cingulate		Thalamo-cortical fascicle
Supplementary motor area		Thalamo-cortical fascicle

Figure 2: Neural circuitry and structures mediating truncal pulsion

ipsi-pulsion, due to involvement of the same tract before crossing.⁹ Our patient with infarct involving the midbrain, presented with truncal contra-pulsion and features of Claude syndrome.

Pulsion in thalamic lesions can be attributed to the disruption of the fastigio-thalamic projections and the thalamo-cortical projections from the ventrolateral thalamus to the precentral gyrus.¹⁰ This has been previously reported as 'pusher syndrome', described as a disrupted orientation of the body in space and an altered perception of vertical, which leads to shifting the body weight across midline, and pushing with non-paretic extremities towards the impaired side. Pusher syndrome is described with lesions of the posterior thalamus.¹¹ One of our patients with thalamic infarct had truncal contra-pulsion as the sole manifestation.

Apart from the lesion of the above-mentioned deep structures, the involvement of the cerebral cortex is rarely reported in association with truncal pulsion. The infarction of the posterior cingulate can result in truncal pulsion and it has been postulated that the disruption of connection between the cingulate motor area and the vestibulocerebellar system through the thalamus might be responsible for the same.¹² Rarely, truncal contra-pulsion results from lesions of the supplementary motor area (Figure 2).

SMA is connected to the vestibulocerebellar system through the ventrolateral nucleus of the thalamus, which explains the pulsion.¹³

Truncal retropulsion is a less researched entity. It impairs postural control in the sagittal plane, thereby causing a backward shift of the centre of mass.¹⁴ Hence, patients orient their body towards an impaired subjective verticality and resist passive correction of this posture. It is characterized by a spontaneous posterior body tilt and active backward pushing, and resistance against passive correction. In our study, truncal retropulsion has been reported in a patient with anterior cingulate infarction, and bilateral cerebellar tonsil infarction, which has not been reported so far in the literature.

Truncal latero-pulsion can result due to lesions in the flocculonodular lobe, lateral medulla, midbrain, pons, cerebellar peduncles, thalamus, cingulate cortex and supplementary motor area (Figure 2). In most cases, it is associated with an impaired subjective sense of verticality and there is no attempt on part of the subject to correct the posture. This is hence a disabling symptom, posing a great challenge to neuro-rehabilitation.

Conclusion

Truncal pulsion is an interesting clinical phenomenon, seen in a variety of strokes, with an intricate neural

circuitry. Studies elaborating the clinico-anatomical correlation of truncal pulsion in acute ischemic strokes are sparse. Knowledge about this clinical symptom and neural circuitry responsible for the same can help the treating physician in recognition and reporting of the same. It is a disabling symptom, posing a great challenge to neuro-rehabilitation.

Conflict of Interest: None

Source(s) of support: None

Video 1: click on <https://youtu.be/LHWYYBUlx0E>

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