REVIEW ARTICLE

Improving the understanding of neural mechanisms and guiding targeted therapy for clinical syndrome after thalamic infarction with advanced neuroimaging

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Abstract: Thalamic infarction can result in a diverse array of symptoms, including motor and sensory deficits, memory and attention difficulties, and alterations in mood and behavior; these symptoms are collectively referred to as thalamic infarction syndrome. The neural mechanisms underlying these symptoms are not yet fully understood, hampering the development of effective and individualized treatments. The current understanding of the pathology of thalamic infarction syndrome is mainly based on conventional computed tomography/magnetic resonance imaging scans, which reveal blood supply to various nuclei groups and clinical features. Thalamic infarction syndrome can be categorized into four groups according to the affected territory and associated vascular syndrome. Recent advancements in neuroimaging techniques, which enable the precise identification of affected pivotal thalamic subnuclei, altered brain structures, white matter pathway integrity, abnormal neural activity, and maladaptive states of brain networks, can enhance our understanding of the clinical mechanisms and inform the development of more effective therapeutic strategies. This review summarizes research on the pathological neural mechanisms of thalamic infarction syndrome and highlights future directions.
Keywords: Thalamic infarction; Clinical syndrome; Neural mechanism; Advanced neuroimaging.

INTRODUCTION

As a subtype of ischemic stroke, which is one of the leading causes of mortality and long-term disability worldwide [Wu *et al.*, 2019], thalamic infarction accounts for 11% of posterior circulation infarcts and 3-4% of cerebral ischemic events [Gurley & Edlow, 2019; Saez de Ocariz *et al.*, 1996] and represents a common and complex clinical entity.

The thalamus, which comprises several functional nuclei, plays an important role in a variety of functions, such as regulating sensory information; controlling motor signals; managing arousal, alertness, and attention; and supporting certain cognitive processes [Annoni *et al.*, 2003; Emmanuel Carrera, 2006; Schmahmann, 2003; Sieveritz & Raghavan, 2021; Van Der Werf *et al.*, 1999]. Its most critical role is serving as a relay center for the exchange of information between the cortex and other parts of the central nervous system (CNS) [H, 1874; Saez de Ocariz *et al.*, 1996; Schmahmann, 2003]. According to conventional neuroimaging techniques (computed tomography angiography; digital subtraction angiography), the thalamus is supplied by four major vascular territories, each preferentially providing blood

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to specific groups of nuclei. This distinct pattern of blood supply to the thalamus has important implications for the development of a vascular syndrome after thalamic infarction [Emmanuel Carrera, 2006; H, 1874; Saez de Ocariz *et al.*, 1996; Schmahmann, 2003]. Depending on the affected supplying arteries and insulted nuclei, damage to the thalamus can cause a variety of clinical syndromes, including hemisensory deficits, hemiparesis, refractory central neuropathic pain, hemispatial neglect, neuro-ophthalmologic defects (such as visual field loss), and cognitive or neuropsychological impairments [Castaigne *et al.*, 1981; Emmanuel Carrera, 2006; Saez de Ocariz *et al.*, 1996; Schmahmann, 2003], all of which can have a profound impact on personal quality of life.

Given the unsatisfactory effects of conventional drug therapy on clinical symptoms and the lack of more effective individualized therapeutic methods, elucidating the neural mechanisms underlying these syndromes is crucial for developing more effective and targeted treatments [Bogousslavsky *et al.*, 1988; Hosomi *et al.*, 2015; Klit *et al.*, 2009; Wu *et al.*, 2019; C. Ye *et al.*, 2022; Chen Ye *et al.*, 2022]. Advanced neuroimaging techniques, such as functional magnetic resonance imaging (fMRI) [Bonkhoff *et al.*, 2020, 2021; He *et al.*, 2021; Salvalaggio *et al.*, 2020; Wang *et al.*, 2021], diffusion tensor imaging (DTI) [Chen *et al.*, 2019; Jang *et al.*, 2019; Li *et al.*, 2011; Nemati *et al.*, 2022] and positron emission tomography (PET) [Dieterich *et al.*, 2005; Dieterich & Brandt, 2008; Kopelman, 2015; Rudolphi-Solero *et al.*, 2022; Stenset *et al.*, 2007; Weder *et al.*, 1994; Willoch *et al.*, 2004], have made important contributions to our understanding of these mechanisms. Thalamic infarction can lead to structural and functional changes in the brain, including altered white matter integrity [Chuo Li, 2011; Krause *et al.*, 2012], cortical reorganization [He *et al.*, 2021; Krause *et al.*, 2014; Wang *et al.*, 2021], and even brain network dysfunction [Favaretto *et al.*, 2022; He *et al.*, 2021; Hosomi *et al.*, 2015; Wang *et al.*, 2021]. These changes can affect both adjacent and distant brain regions, such as the basal ganglia (motor pathways) [Favaretto *et al.*, 2022; Wang *et al.*, 2021], prefrontal cortex [Krause *et al.*, 2014], sensory pathways [He *et al.*, 2021; Wang *et al.*, 2021] and even vision pathways [Holly Bridge, 2011; Millington *et al.*, 2014; Chen Ye *et al.*, 2022], all of which may result in a wide range of clinical symptoms following thalamic infarction. As a result, there has been growing interest in developing neuroimaging-based targeted therapies for individuals affected by thalamic infarction [Elias *et al.*, 2020; Guo *et al.*, 2022; Hosomi *et al.*, 2015]. One promising approach is the use of neuromodulatory techniques to enhance the function of undamaged brain regions and to promote plasticity in damaged regions, particularly in central poststroke pain patients after thalamic infarction [Elias *et al.*, 2020; Guo *et al.*, 2022; Hosomi *et al.*, 2015; Urits *et al.*, 2020].

In this review, we briefly summarize the patterns of blood supply to the thalamus and corresponding vascular syndromes according to conventional brain imaging techniques and outline progress in understanding the pathological mechanisms of clinical syndromes and the neurobiology underlying stroke recovery after thalamic infarction using advanced neuroimaging techniques. Neural mechanism-based targeted therapeutic options and future directions for studying thalamic infarction are proposed.

THALAMIC VASCULAR SYNDROME BY CONVENTIONAL NEUROIMAGING TECHNIQUES

As first reported by Duret [H, 1874] and Foix *et al.*, [1925], the blood supply to the thalamus mainly comprises four major arteries [Bogousslavsky *et al.*, 1986, 1988; Castaigne *et al.*, 1981; Clarke *et al.*, 1994; Ghika-Schmid & Bogousslavsky, 2000; Graff-Radford *et al.*, 1984, 1985; Tatu *et al.*, 1998; von Cramon *et al.*, 1985], including the tuberothalamic artery, inferolateral artery, paramedian artery, and posterior choroidal artery (Figure 1A and B). As each vessel supplies specific nuclei groups, combining clinical symptoms and lesion locations (according to conventional brain scanning, CT, or MRI) has been proposed as a method of diagnosing thalamic infarction syndrome in clinical practice [Schmahmann, 2003]. These distinct supply patterns can be summarized as four standard territories (Figure 1C) and three variants as follows [Emmanuel Carrera, 2006; Schmahmann, 2003]:

1. ANTERIOR TERRITORY INFARCTION

The anterior portion of the thalamus is supplied by the tuberothalamic artery, which is also known as the anterior thalamoperforating artery [Castaigne *et al.*, 1981; Graff-Radford *et al.*, 1984] or polar artery [G, 1976; Percheron, 1973]. The tuberothalamic artery arises from the middle third of the posterior communicating artery (PComm), and the

clinical features of this infarction are characterized by severe and widespread neuropsychological abnormalities. During the early stages of infarction in this area, one may observe varying degrees of consciousness and silence. As the condition progresses, persistent personality changes can occur, including disorientation in time and space, apathy, and lack of self-awareness. Emotional disruptions may also be present. Other common symptoms of anterior thalamic infarction include difficulty [Foix, 1925] forming new memories, language impairment (difficulty speaking and articulating words), and dysarthria, which is related to the connection with the mammillary bodies in the hippocampus.

2. PARAMEDIAN TERRITORY INFARCTION

This area is supplied by the paramedian artery, which is also known as the thalamoperforating pedicle or thalamoperforating artery [Foix, 1925]. It branches off from the P1 segment of the posterior cerebral artery (PCA) or the interpeduncular segment and passes through the posterior perforated substance. Approximately 1/3 of infarcts in these territories occur bilaterally, as the left and right vessel branches originate from the same trunk, referred to as type B artery of Percheron. The paramedian region encompasses parts of the anterior thalamus, medial and ventral thalamus, and part of the interlamellar area. The nuclei supplied by this artery include the reticular, limbic, effector, conjunctive, specific sensory, and lamella nuclei. Given the numerous nuclei in this region, the symptoms of infarction can include visual impairment, hemiparesis, memory loss, and dyskinetic mutism caused by ischemia of the midbrain reticular formation, posterior hypothalamus, mammillary body, and/or anteromedial nuclei of the thalamus.

3. INFEROLATERAL TERRITORY INFARCTION

The thalamic geniculate artery, which arises from the P2 branch of the PCA, supplies this region and is located posterior to the PComm. Infarction of the lateral hypothalamic artery can result in thalamic pain syndrome, which is primarily characterized by intractable pain that is not alleviated by pain medications, potentially due to the dissociation of thalamic and cortical inhibition. This pain can have an immediate or delayed onset and affects approximately 80% of patients with infarctions in this area. Other symptoms of thalamic pain syndrome, include sensory loss and mild hemiparesis (sensorimotor stroke). Sensory disturbances can encompass all modalities, and pure sensory strokes can also occur.

4. POSTERIOR TERRITORY INFARCTION

This region is supplied by the posterior choroidal artery, which arises from the P2 segment of the PCA and comprises a group of small vessels. Reports on infarctions in this area are scarce. According to the existing data, the most common symptom is quadrant blindness, which can be accompanied by loss of lateral sensation, aphasia, and memory impairment. A distinctive manifestation of posterior choroidal artery infarction is the ocular motor disturbance, which is relatively rare and not specific to thalamic infarction. Patients may also experience a complex hyperkinetic syndrome with a delayed onset, which can include ataxia, tremor, dystonia, myoclonus, sensory loss, and pain.

5. THE OTHER THREE VARIANTS

The other three variant thalamic territories are the anteromedial, central, and posterolateral regions. Infarctions in the anteromedial region, which is located in the posterior part of the anterior region and the anterior part of the paramedian region, often result in symptoms such as vertical gaze palsy, anterograde amnesia, aphasia, lack of motivation, and apathy. The central area represents the intersection of the four standard thalamic territories, and infarcts in this area are rare. When present, patients may experience cognitive loss, vertical gaze palsy, loss of arousal, and ataxia. The posterolateral thalamus is located in the anterior part of the posterior region and the posterior part of the inferior lateral region. Infarctions in this area manifest as aphasia, ataxia, and impairments in executive functions.

ADVANCED NEUROIMAGING FINDINGS ON THALAMIC INFARCTION SYNDROME

After a thalamic infarction, the resulting behavioral symptoms can be diverse and can mimic many "cortical" disorders or brainstem syndromes [Emmanuel Carrera, 2006]. Additionally, behavioral disorders associated with anterior and paramedian damage to the thalamus can be difficult to distinguish from primary psychiatric conditions, particularly

when there are limited indications of neurological dysfunction [Emmanuel Carrera, 2006; Schmahmann, 2003]. Advanced imaging techniques, such as high-resolution MRI, can provide more precise diagnoses of damaged thalamic nuclei and their related clinical features [Emmanuel Carrera, 2006; Rondina *et al.*, 2016; Vogels *et al.*, 2021; Xu *et al.*, 2018]. Tractography with diffusion MRI [Behrens *et al.*, 2003] and functional imaging methods, such as fMRI [Johansen-Berg *et al.*, 2005; Puig *et al.*, 2018] and PET [Dieterich *et al.*, 2005; Dieterich & Brandt, 2008; Kopelman, 2015; Rudolphi-Solero *et al.*, 2022; Stenset *et al.*, 2007], can also shed light on the impact of the thalamic injury on the CNS and provide valuable insights into the altered connectivity underlying behavioral deficits after thalamic infarction [Emmanuel Carrera, 2006; Ward, 2023].

1. MORE PRECISE THALAMIC LESION LOCALIZATION

High-resolution MRI is one of the most commonly used neuroimaging techniques in the past few decades to evaluate nuclei after thalamic infarcts. This technique provides detailed information about the anatomy of the thalamus and surrounding structures, which can help to identify the location and extent of the infarct [Rondina *et al.*, 2016; Xu *et al.*, 2018].

High-resolution MRI is particularly useful for the evaluation of microinfarcts [Tao *et al.*, 2022] and small vessel diseases [Jiang *et al.*, 2021; Y. Yan *et al.*, 2022], which can be difficult to detect with conventional CT or MRI. In addition, a combination of high-resolution MRI and the voxel-based lesion-symptom mapping (VLSM) technique [Bates *et al.*, 2003; Chris Rorden, 2007] provides better insight into the association between the thalamic lesion and the symptom, allowing precision diagnosis and treatment. Sprenger *et al.*, [2012] identified the ventral-posterior pulvinar nuclei (VPL) as the most high-risk subregion for infarcts leading to central pain of thalamic origin, with an odds ratio of 81, through lesion-symptom mapping on high-resolution T1-weighted images. Furthermore, Krause and collogues precisely located the key subregions for the occurrence of pain as belonging to more posterior, inferior, and lateral parts of the VPL [Krause *et al.*, 2012]. In a study including all post-stroke pain types, they also found that VPL and the pulvinar subdivisions of the thalamus were significantly associated with clinical symptoms [Delboni Lemos *et al.*, 2022]. In a cohort study evaluating multidimensional neuropsychological impairments in thalamic infarction patients, verbal memory deficits were correlated with anterior thalamic lesions [Scharf *et al.*, 2022]. Recently, in a study focusing on thalamic infarction syndrome inducing thalamic aphasia, the lesioned left mediodorsal thalamic nucleus was identified as significantly associated with language impairments via VLSM [Stockert *et al.*, 2022]. These results indicate that the occurrence of specific thalamic infarctions, particularly central post-stroke pain (CPSP) of thalamic origin, is spatially specific, and lesions in different thalamic subregions are highly linked to the occurrence of specific symptoms, providing a theoretical basis for early and precise identification of such syndromes and improvement in individualized interventions.

2. STRUCTURAL ALTERATIONS IN ADJACENT AND REMOTE BRAIN REGIONS

The thalamus is a relay station for information in the CNS [Salt, 2001], and accumulating neuroimaging evidence indicates alterations in a wide range of CNS regions after thalamic infarction, as shown in Figure 2.

2.1. Adjacent structures

Adjacent structures closely connected to the thalamus, such as the basal ganglia, hippocampus, and white matter between the thalamus and cortex, are affected by thalamic damage, and these impacts are correlated with certain clinical symptoms [Aggleton *et al.*, 2010]. Damage to the thalamus leads to changes in the activity and morphology of the basal ganglia, which can result in various motor and sensory deficits [Kalia & Lang, 2015; Meyer *et al.*, 2016]. fMRI data indicated abnormal dynamic functional states in the basal ganglia and their connections with the thalamus [Favaretto *et al.*, 2022], which indicates a crucial impact of this region on recovery from neurological impairment and long-term outcomes. Li *et al.* found that thalamic infarction led to abnormalities in subfields of the hippocampus that were associated with impairments in memory. The underlying neural mechanisms may involve interactions between the thalamus and the hippocampus [Aggleton *et al.*, 2010]. Moreover, thalamoinsular opercular white matter was found to significantly correlate with central pain syndrome in thalamic stroke patients [Delboni Lemos *et al.*, 2022].

2.2. Distant areas

In addition to areas adjacent to the thalamus, there is increasing interest in alterations of remote regions and their potential relationship with specific clinical syndromes in thalamic infarction [Koh *et al.*, 2021; Weishaupt *et al.*, 2015]. On the one hand, one mechanism underlying this impact in remote regions might be the concept of disconnection, or diaschisis, which has been proposed for decades and is used to explain a series of behavioral abnormalities after stroke [Carrera & Tononi, 2014; Corbetta *et al.*, 2015; Griffis *et al.*, 2019; Langen *et al.*, 2018; Salvalaggio *et al.*, 2020; Xia *et al.*, 2021]. From this perspective, damage to the thalamus, the central hub of the brain, may result in a wide range of effects on connected (yet remote) brain regions. Several pieces of evidence support this proposed mechanism. First, a neuroimaging study combining voxel-based morphometry analysis and tract-based spatial statistics revealed changes in volume and white matter integrity in remote regions, including the brain stem, medial frontal gyrus, precentral gyrus, superior temporal gyrus, supplementary motor area, and postcentral gyrus [Yin *et al.*, 2013], after thalamic infarction. Second, a longitudinal study found significant white matter volume loss in various brain regions in patients after thalamic infarction [Conrad, Habs, Ruehl, Bogle, *et al.*, 2022]. Third, a study observed abnormal gray matter morphometry in the transverse temporal gyrus and superior temporal gyrus that significantly correlated with cognitive dysfunction [S. Yan *et al.*, 2022]. Fourth, remote changes, characterized by secondary impairment of central thalamic radiation and atrophied ipsilesional primary somatosensory cortex, were found in thalamic infarction patients with sensory deficits [Chen *et al.*, 2019]. Moreover, a recent study using advanced diffusion MRI techniques showed that structural connectivity (SC) to regions remote from the lesions (such as the superior longitudinal fasciculus and corpus callosum to the sensorimotor portion) was affected, correlating with somatosensory deficits [Koh *et al.*, 2021].

On the other hand, accumulating evidence has suggested that neurological damage can lead to various neuronal microenvironments, such as neural excitotoxicity, neurovascular structural changes, neurotransmitter abnormalities, and inflammatory responses, which can trigger increased neuronal excitability [Bee & Dickenson, 2008; Finnerup, 2008; Klit *et al.*, 2009; Vestergaard *et al.*, 1995]; in addition, abnormal changes such as hyperresponsiveness, hypoperfusion, and burst electrical activity have also been found in other sites secondary to thalamic vascular damage [Elias *et al.*, 2020]. These mechanisms may lead to alterations in the distal septum, causing specific clinical features through central sensitization and reorganization of the distal cortex [Klit *et al.*, 2009], ultimately leading to the occurrence of specific syndromes.

Thus, these findings, obtained using advanced neuroimaging techniques, demonstrate that thalamic infarction results in changes to the structure of both nearby and distant regions of the brain. These results not only provide a theoretical basis for early and precise identification and individualized intervention for these diseases but also propose a new explanation for the neural pathogenesis of specific clinical syndromes after thalamic infarction.

3. DISORDER OF BRAIN NETWORK REORGANIZATION

Neuroimaging research, with a graph theoretical approach, has demonstrated that the human brain is an integrated and complex network of structurally and functionally interconnected regions, which can be divided into nodes or modules with information communicated among components [Bullmore & Sporns, 2009, 2012].

As discussed above, the damage to the thalamus and subsequent disconnection of the central hub of the brain [Guillery, 1995] suggest that clinical syndromes after thalamic infarction are brain network disorders. On the one hand, recent fMRI studies [Chen *et al.*, 2019; He *et al.*, 2021; Wang *et al.*, 2021] have shown altered functional connectivity among certain brain regions in thalamic infarction patients with central somatosensory deficits, indicating secondary changes such as dysrhythmic or hyperexcitable patterns of neural activity, which have been suggested to cause various chronic pain symptoms [Baliki *et al.*, 2014; Ducreux *et al.*, 2006; Seghier *et al.*, 2005]. On the other hand, alterations in SC revealed by diffusion imaging have also been observed in patients with thalamic or spinothalamic pathway lesions and are related to certain symptoms, such as somatosensory deficits and even central poststroke pain [Chen *et al.*, 2019; Chuo Li, 2011; Jang *et al.*, 2017, 2018; Kishi *et al.*, 2009; Li *et al.*, 2011]. More importantly, some reports have shown that structural changes may induce cortical alterations and reorganization of functional

networks in thalamic infarction patients [Conrad, Habs, Ruehl, Boegle, *et al.*, 2022; Stockert *et al.*, 2022]. We propose that these observations denote a model of clinical syndromes after thalamic infarction, in which the brain undergoes a complex process of network reorganization rather than simple recovery from damage to a group of focal nuclei or vascular incidents, as shown in Figure 2.

POTENTIAL TARGETED THERAPEUTIC OPTIONS

Hyperacute therapies (thrombolysis and endovascular therapy) have improved dramatically over the last few decades for ischemic stroke patients; however, a considerable number of patients do not receive reperfusion treatments or experience sequelae after these therapies [Wu *et al.*, 2019], particularly in the thalamic infarction population. A comprehensive exploration of possibilities for neural repair via drugs or neuromodulatory therapy based on the neural mechanisms underlying stroke-induced brain damage is needed, with future clinical studies using advanced neuroimaging techniques [Bonkhoff *et al.*, 2020, 2021; Puig *et al.*, 2018].

Advanced neuroimaging techniques have the potential to greatly enhance our understanding of the neural mechanisms underlying clinical syndromes after thalamic infarction and guide the development of future therapeutic options. The following are ways in which advanced neuroimaging techniques can guide therapeutic options:

1. Facilitate the early identification and accurate prediction of individuals at high risk of developing specific clinical syndromes after thalamic infarction, enabling early intervention to prevent the occurrence of symptoms. For example, in acute thalamic VPL infarct patients with somatosensory deficits, anti-neuropathic pain treatment such as pregabalin can be administered and might help to prevent the development of central poststroke pain. The next step is to conduct interventional studies in acute thalamic infarction patients before the onset of symptoms, based on advanced neuroimaging findings that suggest crucial lesions or other pathological changes in other regions in the pre-symptomatic stage. The disease course of most of these clinical syndromes, especially neuropathic pain syndromes and cognitive disorders, is highly heterogeneous, with most of them occurring weeks to months after the onset of infarction. Therefore, for most patients, there is a sufficient time window to initiate prophylactic treatment.
2. Identify the specific neural mechanisms that are disrupted in clinical syndromes after thalamic stroke. This information can be used to develop and improve existing therapies, such as anticonvulsants and analgesic drugs for central poststroke pain.
3. Monitor the effectiveness of neuromodulatory therapies with more accurate targets, such as repetitive transcranial magnetic stimulation and transcranial direct current stimulation. Moreover, advanced neuroimaging techniques can be used to monitor the effectiveness of different therapies over time and measure brain changes after stimulation therapy.
4. Guide the development of rehabilitation techniques. For example, DTI can be used to identify disruptions in white matter pathways, and fMRI can be used to identify brain regions that are activated during rehabilitation exercises. These imaging techniques can help to optimize rehabilitation techniques and ensure that they target the appropriate neural mechanisms.
5. Facilitate the development of personalized treatment approaches. Advanced neuroimaging data can be used to identify individuals who are likely to respond best to a particular therapy and to develop customized rehabilitation programs for each patient.

CONCLUSIONS AND FUTURE DIRECTIONS

In conclusion, thalamic infarction can result in profound impairments in motor, sensory, and even cognitive/neuropsychological functions. To date, the combination of conventional neuroimaging techniques (CT/MRI) and neurological examination may be unable to meet the precise diagnostic and individualized treatment needs of patients. The use of advanced neuroimaging techniques can enhance the understanding of the neural mechanisms underlying these complex clinical syndromes and can guide specific therapies to target these mechanisms, increasing the chances of favorable outcomes for patients with thalamic infarction.

As the interruption of thalamocortical white matter connections is an important component of thalamic infarction syndromes, future research is

needed to further understand the neural mechanisms of thalamic infarction using advanced neuroimaging techniques and larger sample sizes with longitudinal designs. In addition, most previous studies have employed unimodal or single-dimensional imaging techniques, and the imaging sequences used in some studies have substantial limitations, such as low spatial resolution or the inability to identify the intersection of multiple fibers within a single voxel in DTI models [Kaden *et al.,* 2007]. These limitations may reduce the accuracy of the results and restrict the clinical applications of the findings. Last but not least, these advanced techniques are ineffective without consideration of patients' explicit needs and benefits. Future research should focus on subtle improvements in patients' behavioral deficits with close attention from clinicians and combine this information with neuroimaging data to develop a more effective strategy for mitigating the impact of post-thalamic infarction impairments.

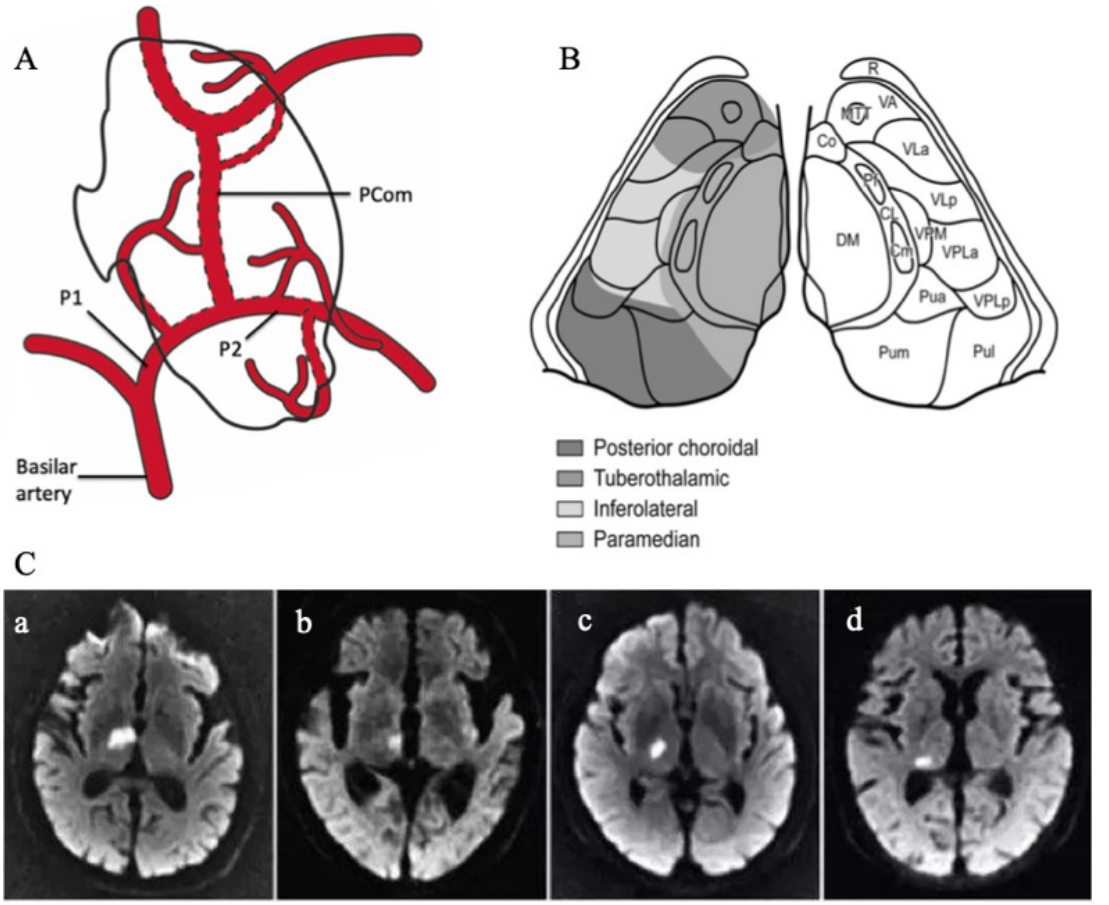

Figure 1. Schematic of the arterial supply of the thalamus and four diffusion-weighted MRI scans of standard infarction types. A, B. Illustration of the major supplying arteries of the thalamus and divisions of thalamic nuclei[Bordes *et al.,* 2020; Emmanuel Carrera, 2006]. C. Diffusion-weighted images from four patients with standard infarction types: a. anterior territory infarction, supplied by the thalamic tuberosity artery; b. paramedian territory infarction, supplied by the paramedian artery; c. inferolateral territory infarction, supplied by the thalamic geniculate artery; and d. posterior territory infarction, supplied by the posterior choroidal artery. PComm: posterior communicating artery; P1: first segment of the posterior cerebral artery; P2: second segment of the posterior cerebral artery; CL: central lateral; CM: centromedial; Co: commissural; Cp: commissural posterior; DM: dorsomedial; MTT: mamillothalamic tract; Pua: pulvinar anterior; Pum: pulvinar medial; Pul: pulvinar lateral; Pf: parafascicularis; R: reticular; VA: ventral anterior; VLa: ventral lateral anterior; VLp: ventral lateral posterior; VPLa: ventroposterolateral anterior; VPLp: ventroposterolateral posterior; VPM: ventroposteromedial.

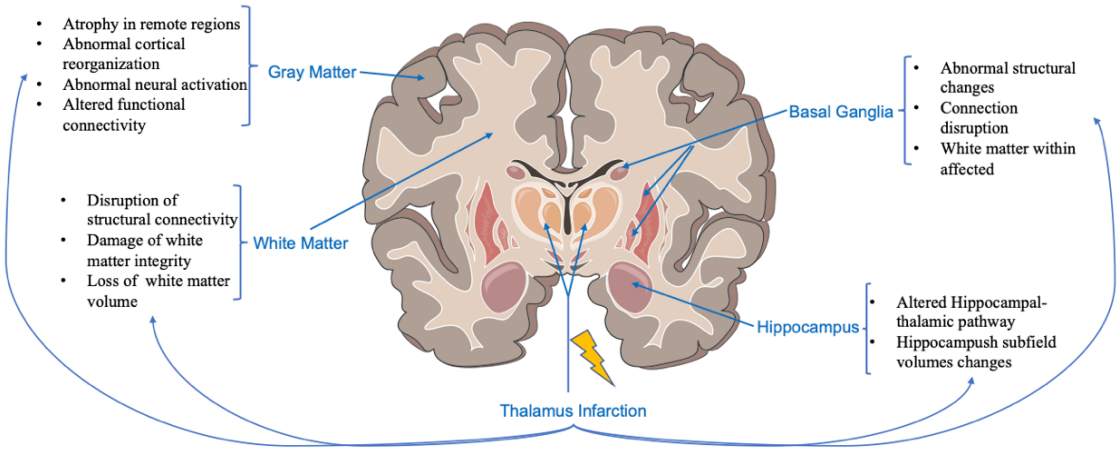

Figure 2. Illustration of affected brain regions after a thalamic infarction.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

C.Y. drafted the manuscript; C.Y. and R.P. performed the literature search; and B.W. supervised the research and revised the manuscript.

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