#### **CASE REPORT**



# Inferolateral thalamic ischemia secondary to PCA P2 perforator occlusion mimics MCA stroke syndrome

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#### Abstract

Paramedian thalamic strokes following occlusion of the posterior medial (paramedian) thalamic perforators have been previously described in great detail. However, the stroke syndrome associated with occlusion of posterior lateral (inferolateral) thalamic perforators is less commonly known. We present an illustrative case of an inferolateral thalamic perforator stroke mimicking a middle cerebral artery (MCA) syndrome and provide a review of the literature. A 62-year-old male presented with dysarthria, contralateral hemisensory loss, and contralateral weakness, concerning for possible MCA stroke. However, close examination revealed the hemiparesis to be ataxic in nature. Imaging revealed a left PCA P2 segment occlusion and lacunar infarction of the ventral lateral (VL) and ventral posterior (VP) thalamus, the main thalamic destination of cerebellar and sensory pathways. The case is unique because the P1 segment and posterior communicating artery (Pcom) remained patent, resulting in selective occlusion of only the posterior lateral (inferolateral) thalamic perforators at the P2 level. Acute loss of the posterior lateral (inferolateral) thalamic stroke syndrome, as it may be mistaken clinically for an MCA occlusion. The benefit of mechanical thrombectomy for this type of stroke is not well established and should be considered carefully.

**Keywords** Thalamic stroke  $\cdot$  Posterolateral thalamic perforator  $\cdot$  Inferolateral thalamic perforator  $\cdot$  Ventrolateral thalamic nucleus  $\cdot$  Ventral posterior thalamic nucleus

# Introduction

Arterial blood supply to the thalamus is mainly via perforating arteries (perforators) from the posterior and to a lesser degree the anterior circulation. Anterior (polar) thalamic perforators originate from the middle third of the posterior communicating artery (Pcom) [16]. They reach the anterior pole of the thalamus by following the mamillothalamic tract. Posterior thalamic perforators emanate from the posterior cerebral artery (PCA) and basilar artery (BA).

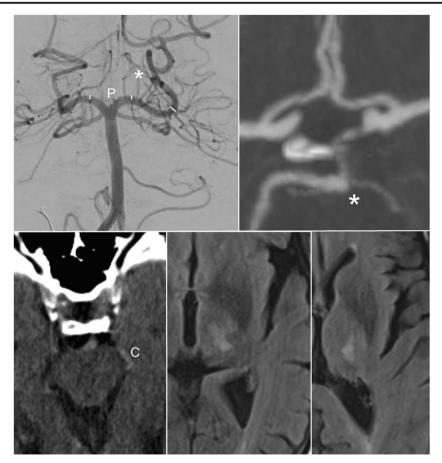
The proximal PCA is divided by the Pcom into the P1 segment (basilar artery to the origin of the Pcom) and the

<sup>2</sup> Department of Neurosurgery, University of Hannover, Hanover, Germany P2A segment (Pcom origin to the posterior margin of the cerebral peduncle; Fig. 1). Accordingly, posterior medial thalamic perforators originate from the basilar artery and the P1 segment. Posterior medial thalamic perforators have been described as paramedian arteries [16], the thalamoperforating [20] or retromamillary pedicle [10] versus the posterior thalamosubthalamic [5] or interpeduncular artery. Historically, injury to those perforators was feared while clipping basilar apex aneurysms. Even now during times of endovascular coiling and occasional flow diversion at the basilar apex, a healthy respect for the paramedian perforators is well advised. The corresponding paramedian thalamic stroke has a well-known presentation with abulia, lack of motivation, memory and personality changes. Bilateral strokes from a common paramedian trunk (artery of Percheron) often manifest as altered mental status, hypersonnolence, and memory disturbance with gaze palsies and can be particularly devastating [6, 17].

In contrast, the inferolateral thalamic stroke associated with occlusion of the posterior lateral thalamic perforators off the

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**Fig. 1** The lateral posterior thalamic perforators and the corresponding vascular territory are depicted. Top left: digital subtraction angiography, right vertebral artery injection, anterior-posterior (ap) view, healthy subject, normal anatomy. The small vertical white lines mark the origins of the posterior communicating arteries (Pcom), dividing the posterior cerebral artery (PCA) segments P1 and P2A. The small oblique white line on the left PCA marks the origin of the middle inferior temporal artery, the distal end of the P2A segment. The letter "P" marks a large medial posterior thalamic perforator irrigating both sides—the artery of Percheron. The asterisk "\*" marks a similarly large lateral posterior thalamic perforator on the left side, just distal to the Pcom origin. Top right: computed

tomography angiography (CTA) of the patient presented. The circle of Willis can be recognized easily. The site of the PCA occlusion is marked by the asterisk "\*." As the left P2 is not opacified, the left superior cerebellar artery (SCA) appears to be more prominent. A large Pcom is present on the left side, taking off immediately prior to the P2 occlusion. The hyperdense structure in the center represents the dorsum sellae with the posterior clinoids. Bottom left: CT on admission, axial scans. The density ("C") likely resembles the embolic clot. Bottom center and right: brain MRI FLAIR sequence, axial slices. The extent of the ventral lateral and ventral posterior thalamic stroke after occlusion of the proximal P2 with its posterior lateral thalamic perforators is recognizable

P2A segment is less recognized. The clinical presentation of an inferolateral thalamic stroke shares some features with the much more common MCA stroke and will therefore be encountered by stroke interventionalists with request for thrombectomy. In the following, we present an illustrative case and review the literature regarding the posterolateral also known as inferolateral thalamic perforator strokes.

# **Case presentation**

A 62-year-old right-handed male and former smoker presented to an outside hospital with new onset right-sided weakness, numbness and difficulty speaking 10 h after last known normal. He was outside the window for tPA. He was subsequently transferred for consideration of thrombectomy for suspected middle cerebral artery occlusion. On exam, he displayed right-sided nasolabial fold flattening, weakness, and ataxia as well as hemisensory loss and extinction to touch and temperature. He had full visual fields by confrontation. His reflexes were symmetrical 2+. His NIH Stroke Scale score was 7. Computed tomography (CT) demonstrated no hemorrhage, ASPECTS 10 with a 7 mm hyperdensity in the area of the P2A segment, suggestive of thrombus (Fig. 1). CT angiography (CTA) confirmed focal occlusion of the proximal left P2 segment with minimal distal reconstitution via collaterals. The P1 segment and Pcom remained patent. Brain MRI showed FLAIR and diffusion hyperintensity in the left ventrolateral and ventral posterior thalamus, consistent with acute infarction [24]. He was admitted for supportive care and workup for etiology of his stroke. Nine days after admission, he was discharged on dual antiplatelet therapy with a modified Rankin Score (mRS) of 4. At 3-month follow-up visit, the patient endorsed residual right hand and face numbness with resolution of his other neurological symptoms.

# Literature review

Four classical thalamic stroke territories have been identified in the literature based on the territories' arterial supply [5, 8, 10, 16, 20, 21, 23].

- 1. The anterior thalamic territory includes the reticular nucleus, ventral anterior nucleus (VA), rostral part of the ventrolateral nucleus (VL), ventral pole of the medial dorsal nucleus (MD), ventral part of the internal medullary lamina, and anterior thalamic nuclei. This territory is mainly part of the limbic system. Arterial supply is by means of the anterior thalamic perforators, which have been described as the thalamic pedicle of the anterior internal optic artery [8], tuberothalamic [2], premamillary [10], anterior thalamosubthalamic paramedian [5], or polar artery [16]. These perforators follow the mamillothalamic tract anatomically. They are absent in about one third of cases, and the anterior territory is then supplied by the paramedian arteries [16, 20]. Clinical stroke features include predominantly cognitive impairment and decreased executive function and vigilance as well as transient facial paralysis and occasionally dsyphasia if left-sided [5].
  - 2. The paramedian thalamic territory is composed of the medio-dorsal nucleus (MD), internal medullary lamina, and intralaminar nuclei. This territory is supplied by the paramedian arteries (also known as posterior medical thalamic perforators, the thalamoperforate pedicle [18] or the posterior thalamosubthalamic paramedian versus retromamillary artery [10]). Reduced level of consciousness is the hallmark of the paramedian thalamic stroke but the syndrome may also include cognitive impairment and vertical gaze palsy [5, 7]. There can be some contralateral hemiataxia or tremor secondary to damage of the red nucleus.
  - 3. The inferolateral thalamic territory comprises the ventro-lateral and ventro-posterior nuclei. It is supplied by the lateral posterior thalamic perforators described as thalamogeniculate [10], inferolateral [16], or inferior external optic arteries [8], which enter the thalamus between the geniculate bodies. As illustrated in this

case, hemisensory loss, contralateral ataxia, choroathetoid movements, contralateral hemiparesis, and aphasia can be observed [3–5]. The ataxia stems from damage to the cerebellar projections, and choreoathetoid movements arise from damage to the basal ganglia projections, both of which pass through the ventrolateral thalamic nucleus.

 The posterior territory (pulvinar, geniculate body) arterial supply is from the posterior choroidal arteries [14]. Associated strokes cause visual deficits (sector anopia) and sensory loss (hypoesthesia).

Pure anterior or posterior thalamic territory strokes are extremely rare and usually part of strokes involving multiple territories. The paramedian stroke syndrome though is common among thalamic strokes; it has been previously welldescribed [5, 7, 19, 22]. It is caused by an occlusion of the paramedian arterial trunk, typically the largest thalamic perforator. Injury to this perforator is a serious risk during clipping of basilar apex aneurysms [12]. In contrast, isolated inferolateral thalamic strokes are less well-described, although they occur nearly as frequently as the paramedian stroke. In a series of 71 patients with thalamic strokes, nearly 1/3 of patients (19) had an inferolateral stroke, the same as an isolated paramedian stroke or mixed stroke [5]. Thus, inferolateral strokes need to be recognized accordingly.

Originally, Dejerine and Roussy described the "syndrome thalamique" in 1907, essentially a stroke of the inferolateral thalamus [19]. They already noted the associated cerebellar-like ataxia and sensory loss resulting in discoordination of the limbs. Today, the "Dejerine-Roussy" syndrome has become synonymous with chronic pain after stroke as a long-term sequela of infarction of the ventral posterior nuclei. However, pain is typically not part of the acute presentation of the inferolateral thalamic stroke. The ataxic hemiparesis with sensory loss is the main feature of an acute inferolateral stroke [2, 4, 9, 11, 13].

### **Practical implication**

If a large vessel occlusion is present as in our illustrative case (P2 occlusion), stroke intervention via catheter thrombectomy could be considered. Particularly, with involvement of the lateral posterior (inferolateral) thalamic perforators, the NIHSS score with a PCA stroke may be significant, supporting intervention. Nevertheless, the procedure has to be considered carefully as PCA thrombectomy carries risk of cross embolization at the basilar apex. Futhermore, as the perforators are terminal vessels, the "therapeutic window" may be short. In the presented case, we used MRI to determine presence or absence of a diffusion mismatch indicating viable brain tissue [15]. CT perfusion imaging is limited regarding

evaluation of the thalamus and brainstem [1]. If a diffusion mismatch is confirmed by MRI in the presence of a large vessel occlusion, thrombectomy may be indicated. In the illustrative case, the thalamus was already infarcted as documented by the FLAIR MRI (Fig. 1). The risk of an intervention outweighed the benefits.

# Conclusion

Loss of the posterior lateral (inferolateral) thalamic perforators at the proximal P2 segment results in a ventral lateral and ventral posterior thalamic stroke. The clinical impact is significant with contralateral hemisensory loss, contralateral weakness/ataxia, and dysarthria. In select cases of PCA occlusion and timely presentation, endovascular thrombectomy could be considered. MRI screening for salvageable brain (diffusion mismatch) is recommended.

### **Compliance with ethical standards**

**Conflict of interest** The authors declare that they have no conflict of interest.

**Ethical approval** Not applicable. A case report or retrospective chart review with three (3) or fewer (an n < 3) patients not presented as a systematic investigation designed to contribute to generalizable knowledge does not require IRB approval.

Informed consent Informed consent was obtained from the patient.

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