Artery of Percheron: basic knowledge and tips on how to diagnose an ischemic infarction of this usually overlooked neurovascular variant in emergency settings

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Learning objectives

The purpose of this poster is:

• to learn the normal neurovascular anatomy and common variants of the blood supply of the thalami and midbrain;

• to describe the clinical presentations and key imaging findings of the infarction of the artery of Percheron;

• to present tips for adjusting imaging protocols in emergency settings.

Background

1. Overview of the neurovascular anatomy of the thalami and midbrain

The thalami and midbrain have a complex blood supply with a great number of feeding arteries [1, 2]. The arterial supply is provided by perforating branches from the posterior cerebral artery (PCA) and the posterior communicating artery (PcomA) [3]. Although there is a significant variation and overlap, thalamic vascular supply is classically categorized into 4 territories [4-6] (Fig. 1):

• Anterior,
• Paramedian,
• Inferolateral,
• Posterior.

The anterior territory is supplied by the polar artery or thalamotuberal arteries, which arise from the PcomA [4, 5]. The inferolateral territory is supplied by the thalamogeniculate arteries, which arise from the P2 segment of the PCA [4, 6]. The posterior territory is supplied by the posterior choroidal arteries, which also arise from the P2 segment of the PCA [5, 6]. The paramedian territory of the thalami is supplied by the perforating thalamic arteries, also called paramedian arteries [4-6]. The paramedian thalamic arteries arise from the P1 segment of the PCA and may share a common origin with the superior paramedian mesencephalic arteries that supply the medial areas of the upper brainstem [3, 6, 7] (Fig. 2).

2. Normal variants of the paramedian thalamic arteries
Regarding the arterial configuration of the paramedian arteries, there are several normal variants [1, 3], which have great variability with respect to number, size, and territorial contribution [6, 8]. According to Percheron there are four normal variants of the neurovascular anatomy of the thalami and midbrain [4]:

1. **Variant I** is most common, where each perforating artery arises from each left and right PCA [4, 5] (Fig. 3a).

2. **Variant IIa** is a less common, asymmetrical variant, where perforating arteries arise directly from the proximal segment of one of the PCAs [3-5] (Fig. 3b).

3. In **variant IIb**, the bilateral perforating thalamic arteries arise from a single arterial trunk called the artery of Percheron, which arises from the P1 segment of one PCA. It supplies the paramedian thalami and the rostral midbrain bilaterally [2-5] (Fig. 3c).

4. **Variant III** is an arcade variant with several small perforating branches arising from a single arterial arc that bridges the P1 segments of both PCAs [3, 5] (Fig. 3d).

In addition to the medial thalamic nuclei, these paramedian thalamic arteries supply the interpeduncular nucleus, the decussation of the superior cerebellar peduncles, the medial part of the red nucleus, the third and fourth cranial nerve nuclei and the anterior portion of the periaqueductal grey matter [3]. Consequently, occlusion of the artery of Percheron causes a bilateral paramedian thalamic infarctions with or without midbrain infarction [2, 4, 5] (Fig. 4). Additional involvement of the anterior thalamus is uncommon [5].

**As strokes in these territories are infrequently diagnosed, we have to wonder if they are really rare or highly underdiagnosed** [1]. The great number of variants of the blood supply in the posterior cranial fossa, especially the high variability of presence and size of P1 segments, which give rise to the paramedian arteries, may be a clue, that the **Artery of Percheron is not such an infrequent variant** [1, 6].

The prevalence of the artery of Percheron is unknown [1, 6]. In one study Hiratsuka et al. evaluated 134 patients (with some neurological symptoms) using 3D TOF MRA on 3T MRI and concluded that 20,5 % of the patients in their sample had an artery of Percheron [9]. In other studies the characteristic Artery of Percheron infarct pattern was estimated to occur from 0,1 to 2 % of all ischemic strokes and from 4 % to 18 % of all thalamic strokes [6].
3. Clinical presentation of the artery of Percheron infarction

The complex anatomy and function of the human thalamus and its variable vascular supply are responsible for the extremely variable clinical features when this structure is damaged by an ischemic infarction; in addition, the vascular overlap with the underlying midbrain will extend the spectrum of these clinical features to include midbrain signs [5, 10].

An ischemic stroke in the territory of an artery of Percheron usually presents with three main symptoms that are found in patients with bilateral paramedian thalamic strokes [4, 6, 11] (Fig. 5):

- **Vertical gaze palsy** (65 % of patients),
- **Memory impairment** (58 % of patients),
- **Coma** (42 % of patients).

Bilateral paramedian thalamic lesions are often accompanied by rostral midbrain lesions, producing a "mesencephalothalamic" or "thalamopeduncular" syndrome. In addition to the mentioned triad, the syndrome is characterized by [6, 11]:

- other oculomotor disturbances,
- hemiplegia,
- cerebellar ataxia,
- movement disorders.

An ischemic stroke in the territory of an artery of Percheron usually presents with confusion, somnolence, loss of consciousness or coma [3, 6], and the patients recover partially with variable degree of remaining neurological deficits [1]. The altered mental status is explained by involvement of reticular activating system and the disrupted connections between the thalamus and the anterior, orbitofrontal and medial prefrontal cortices [12].

Altered mental status is frequently followed by eye movement disturbances such as vertical gaze palsy [3], which is due to disruption of the cortical input that traverses the thalamus to reach the rostral interstitial medial longitudinal fasciculus [12]. If the nucleus of the third cranial nerve is involved [1] patients present with oculomotor nerve palsy [12]. Few cases with motor or sensory deficits, like hemiplegia or hemisensory loss, have been reported [1, 3]. Most prevalent long-term consequences are orientation and
concentration deficits, behavioral disturbances and memory impairment [3, 5, 6]. Memory impairment is present when lesions affect the mammillothalamic tract, anterior nucleus and dorsomedial nucleus [6].

Images for this section:

**Fig. 1:** Territories of thalamic vascular supply in relationship to thalamic nuclei. The nuclei have been marked with the following initials: lateral dorsal nucleus (LD), lateral posterior nucleus (LP), ventral anterior nucleus (VA), ventral lateral nucleus (VL), ventral posterior nucleus (VP), ventral intermediat nucleus (VI), ventral posterolateral (VPL), and ventral posteromedial nucleus (VPM).
**Fig. 2:** Thalamic nutrition - branches of the PCA. The branches have been numbered according to the following key: internal carotid artery (1), middle cerebral artery (2), PcomA (3), thalamotuberal arteries (4), basilar artery (5), P1 segment of the PCA (6), P2 segment of PCA (7), P3 segment of PCA (8), paramedian thalamic arteries (9), posterior choroidal arteries (10), thalamogeniculate arteries (11). The thalamus is marked by number 12.
**Fig. 3:** Anatomic variations of the arterial supply to the paramedian thalamic-mesencephalic region as described by Percheron. Variant I (a) - common variation, many small perforating vessels arising from the P1 segments of both PCAs. Variant IIa (b) - perforating arteries arise from one of the PCAs. Variant IIb (c) - the artery of Percheron is a single perforating blood vessel arising from one P1 segment. Variant III (d) - an arcade of perforating branches arising from an artery bridging the P1 segments of both PCAs. The vessels marked by initials: thalamic perforators (TP), midbrain perforators (MP), posterior cerebral artery (PCA), superior cerebellar artery (SCA), basilar artery (BA), anterior inferior cerebellar artery (AICA) and artery of Percheron (AOP).
Fig. 4: Anatomy and expected territory of infarction in the presence of occlusion of artery of Pecheron. Transverse section through the level of thalami and basal ganglia with expected area of infarction - both paramedian thalami (white star) (a). Transverse section through the midbrain and the expected area of the involved midbrain (white triangle). V-shaped hypodense area along the pial surface of the midbrain adjacent to the interpeduncular fossa (the "V" sign) (b).
Fig. 5: The three most common symptoms accompanying artery of Percheron infarction. Passive examination of the oculocephalic reflex (a): a1-a5: Natural response in normal patients, referred to as doll’s eye sign (the gaze deviates contralaterally in the opposite direction of where the head is turned). a6-a10: Vertical gaze palsy, the eyes remain fixed on the vertical axis during the passive movement of the head. Memory impairment (b). Coma (c).
Findings and procedure details

1. Basics of imaging techniques

The infarction of artery of Percheron presents itself as abnormal signal on computer tomography (CT) and/or magnetic resonance imaging (MRI) involving the bilateral paramedian thalami with or without rostral midbrain involvement:

- **On CT** we see hypoattenuation, as a region of decreased attenuation (compared with gray and white matter) representing edema in a specific bilateral paramedian thalamic distribution [6] (Figs. 6, 7, 9, 11).

- **On MRI** the signal is hyperintense on T2 or FLAIR sequences with or without restricted diffusion or postcontrast enhancement, within a specific bilateral paramedian thalamic distribution, corresponding to a distinct arterial territory [6]. Early diagnosis is best made by a diffusion-weighted imaging (DWI) sequence using MRI [12]. MRI normally allows visualization of the initial infarct in cases of acute cerebral ischemia and is usually used in stroke centers as the primary or early secondly imaging modality [4].

- The artery of Percheron is rarely visualized with conventional angiography, and to our knowledge, only four other authors have successfully demonstrated this variant [5, 6]. It is also too small to be visualized by CTA (Figs. 8, 10) or MRA [12].

Lazzaro et al. [6] identified four patterns of ischemic infarctions when Percheron artery is occluded:

1. **43 %** of patients demonstrated damage to both paramedian thalami and midbrain (this was the most common pattern).

2. **38 %** of patients had ischemic damage to paramedian thalami only, without midbrain involvement.

3. In **14 %** of patients the damage involved the anterior thalamic nuclei in addition to paramedian thalami and upper midbrain.

4. In the least common pattern (**5 %**), there was ischemic damage of bilateral paramedian and anterior thalami; the midbrain was spared in these cases [6].
They also found that a previously unreported finding (the "V" sign) on FLAIR and DWI sequences was identified in 67% of cases of Artery of Percheron infarction with midbrain involvement and this sign supports the diagnosis when present [5, 6](Fig. 4). The "V" sign is represented by a distinct pattern of V-shaped hyperintensity on axial FLAIR and/or DWI along the pial surface of the midbrain adjacent to the interpeduncular fossa [6].

The imaging differential of bithalamic lesions is broad and includes [6]:

- arterial and venous occlusion,
- infiltrative neoplasm,
- infectious and inflammatory lesions,
- a single large embolus at the basilar tip could result in a similar infarct pattern, especially in combined paramedian thalamic and midbrain infarcts. However, this would typically manifest as the "top of the basilar" syndrome with additional characteristic posterior circulation infarcts.

2. Tips for adjusting imaging protocols in emergency settings

It is difficult to suspect bithalamic paramedian infarcts because of the complex anatomy causing large clinical variability [4]. Diagnosing an artery of Percheron infarction is critical to direct the appropriate time sensitive management and to prevent additional unnecessary procedures. **The appropriate diagnosis has to be made at the earliest possible stage** if the patient is still within the time limits for thrombolytic therapy as the efficiency decreases rapidly with time [1, 4]. The diagnosis is often made many hours or even days after the clinical onset. At this stage, thrombolytic therapy is ineffective and dangerous [3, 4].

In the emergency settings we suggest the following considerations:

- As in other locations in the brain, the combination of pathologic DWI and normal findings on T2-weighted images suggest an acute stroke. If the lesions are already visible on T2, the time window for thrombolysis is over [1, 4].

- With a brain MRI showing an acute stroke, intravenous thrombolysis can be performed, if the deadline for achieving it is not exceeded (4 hours and 30 minutes after the onset symptoms) [4].

- To our knowledge there is only a single report of a symptomatic patient presenting an acute Percheron stroke with normal early brain MRI in the literature [1]. We suggest
that, upon the presentation of acute rostral brain stem stroke, accompanied by a nonconclusive brain MRI, we should perform a new brain imaging by MRI within therapeutic times or should consider doing interventional explorations focused on the vertebrobasilar territory [4].

- We have to consider, that artery of Percheron is rarely visualized with conventional angiography [6]. Performing conventional angiography may not be indicated, because lack of visualization of the artery does not exclude its presence (because it is occluded) [2]. Although it is unlikely we would visualize the artery of Percheron, maybe if perform conventional angiography, it can show other clues which would make the final diagnosis easier (e.g. filling defect of a PCA segment, which would influence our decision making process). Conventional cerebral angiography should not be used routinely to diagnose Percheron artery occlusion [5].

3. Learning point

Considering all the given information, we suggest that in emergency settings, where the severity of the clinical features (coma and vertical gaze palsy) does not correlate with the imaging findings (e.g. negative head CT and CTA), we have to take into consideration the possibility of the artery of Percheron infarction and perform a follow-up head CT or MRI within therapeutic times, to make the correct diagnosis when treatment with thrombolysis is still possible. If the diagnosis is made in the shortest time possible the thrombolytic therapy can still be done and the outcome can be fairly good [3].

Images for this section:
**Fig. 6:** Non-contrast brain CT. A 69 year-old Caucasian woman, last seen awake 10 hours earlier, was found unresponsive in front of her apartment. Brain CT scan performed on the day of admission was normal, in particular without early signs of ischemia at the level of both thalami. There was only a small old lacunar infarction present in the left thalamus (a). A new brain CT scan was performed 24 hours later and showed symmetrical ill-defined areas of hypodensity in the medial part of both thalami corresponding to the occlusion of the artery of Percheron (white arrows) (b).
Fig. 7: Non-contrast brain CT of same patient as in Fig. 6. Brain CT scan performed on the day of admission did not show any early signs of ischemia at the level of the mesencephalon (a). The hypodense area in the right thalamus extended into the anterior part of the mesencephalon and cerebral peduncle. The hypodense area in the left thalamus extended only into the anterior part of the mesencephalon (white arrow) (b).
**Fig. 8:** CTA of the same patient as in Fig. 6. The basilar artery and the left PCA (black arrow) were both transient (a). At first glance the right PCA appears to be fully opacified. Detailed examination of the CTA images revealed a filling defect of P1 segment of the right PCA (white arrow) and another rare anatomic variant: duplication of the right superior cerebellar artery (black arrow), which could have been mistaken for a transient right PCA. These findings demonstrate that when we suspect an artery of Percheron, we should also consider the possibility, that other rare anatomic variants of the posterior circulation may be present (b).
Fig. 9: Non-contrast brain CT of a 61 year-old Caucasian woman, which presented with a severe headache and a dilated pupil on the left. Brain CT scan showed a subarachnoid hemorrhage in the perimesencephalic region, ventricles and Sylvian fissure. There were no early signs of ischemia at the level of thalami (a) and the mesencephalon (b). Obtained DSA images showed the source of the subarachnoid hemorrhage.

Fig. 10: CTA of the same patient as in Fig. 9 shows transient P1 segments of the right and left PCA (white arrows) before endovascular treatment.
**Fig. 11**: Non-contrast brain CT of the same patient as in Fig. 9. One day after stenting of the basilar artery and the left PCA. Non-contrast brain CT showed signs of ischemia in paramedian regions of both thalami (white arrows) (a) and mesencephalon (black arrow) (b). We presume that during the endovascular treatment an embolus occluded an artery of Percheron.
Conclusion

When bilateral medial thalamic infarcts are found, occlusion of the artery of Percheron should be considered as the main diagnosis [2, 3]. We proposed that in emergency settings, in cases where the main clinical features (e.g. coma and vertical gaze palsy) are severe and the primary imaging modalities are unremarkable, we should consider the possibility of the artery of Percheron infarction. Anytime the initial imaging is normal, new brain imaging by MRI or CT scan should be performed at least within the first 48 hours [4].

Personal information

References


