# "IMAGING PATTERNS AND CLINICAL SPECTRUM IN ARTERY OF PERCHERON INFARCTION"

**Dr. V. BALA MURALI KRISHNA[1] *Dr. V. SRIKANTH[2], Dr. VENKATA SANDHYA NAIDU YARAM[3] Dr. MAHIJA SIRIPURAPU[4], Dr. K.CHANDRASEKHAR[5]**

1. Associate Professor Department of Radio Diagnosis , Dr. Pinnamaneni Siddhartha Institute Of Medical Sciences And Research Foundation, Chinaoutpally, Krishna District ,Vijayawada.
2. Associate Professor Department of Radio Diagnosis , Dr. Pinnamaneni Siddhartha Institute Of Medical Sciences And Research Foundation, Chinaoutpally, Krishna District ,Vijayawada.
3. Resident (3rd year) Department of Radio Diagnosis , Dr. Pinnamaneni Siddhartha Institute Of Medical Sciences And Research Foundation, Chinaoutpally, Krishna District ,Vijayawada.
4. Resident (3rd year) ,Department of Radio Diagnosis , Dr. Pinnamaneni Siddhartha Institute Of Medical Sciences And Research Foundation, Chinaoutpally, Krishna District,Vijayawada.
5. HOD & Professor , Department of Radio Diagnosis , Dr. Pinnamaneni Siddhartha Institute Of Medical Sciences And Research Foundation, Chinaoutpally, Krishna District ,Vijayawada.

***Corresponding Author**: Dr. V. SRIKANTH , Associate Professor Department of Radio Diagnosis , Dr. Pinnamaneni Siddhartha Institute Of Medical Sciences And Research Foundation, Chinaoutpally, Krishna District ,Vijayawada.



## ABSTRACT:

**BACKGROUND:**

Occlusion of the Artery of Percheron leads to a distinctive pattern of ischemia, manifesting as bilateral paramedian thalamic involvement, with or without midbrain involvement. While the classic imaging findings are frequently acknowledged, the literature reveals a scarcity of reported cases, limited mostly to small case series and isolated instances of Artery of Percheron infarction.

**AIM:**

This study aimed to comprehensively characterize the imaging spectrum of Artery of Percheron (AOP) infarction based on cases obtained from our institute.

**MATERIALS AND METHODS:**

Study Design : Hospital based Retrospective study

Study area:, Department of Radio Diagnosis , Dr. Pinnamaneni Siddhartha Institute Of Medical Sciences And Research Foundation, Chinaoutpally, Krishna District Vijayawada.

Study period: 5 years (between 2017 and 2022 )

Sample size: we reviewed the imaging and clinical data of 25 patients diagnosed with Artery of Percheron (AOP) infarction. The primary inclusion criterion for imaging analysis was the presence of abnormal signal intensity on MR imaging, specifically involving the distinct arterial territories of the bilateral paramedian thalami, with or without rostral midbrain involvement. Exclusion criteria encompassed cases with neoplastic, infectious, or inflammatory etiologies.

**RESULTS:** We identified 4 ischemic patterns of AOP infarction:

1) Bilateral paramedian thalamic with midbrain (56%),
2) Bilateral paramedian thalamic without midbrain (28%),
3) Bilateral paramedian thalamic with anterior thalamus and midbrain (12%), and
4) Bilateral paramedian thalamic with anterior thalamus without midbrain (4%).

**CONCLUSION:**

Artery of Percheron (AOP) infarctions should be considered among potential causes for sudden alterations in consciousness. The preferred diagnostic modality is MRI. The identification of four distinct ischemic patterns in our case series enhances the ability to recognize AOP infarction, thereby aiding in the neurologic evaluation and management of patients presenting with thalamic strokes.

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**Introduction:**

Variations in the normal cerebral circulation are frequently observed, strokes resulting from occlusion of these variant arteries are uncommon and can present diagnostic challenges. The artery of Percheron (AOP) is a noteworthy example of such a variant artery. In cases where a patient exhibits altered sensorium without an apparent cause, it is crucial to consider AOP infarction in the differential diagnosis.

The thalamus, primarily receives its blood supply from multiple small vessels originating from the posterior communicating artery (PcomA) and the P1 and P2 segments of the posterior cerebral arteries (PCAs)(1).The vascular supply to the thalamus is classified into four territories: anterior, paramedian, inferolateral, and posterior (refer to Fig 1).(Fig 2)

(1) Anterior region: Supplied by the polar (or thalamotuberal) arteries arising from the posterior communicating artery (PcomA); [3][4]

(2) Paramedian region: Supplied by the paramedian (or thalamoperforating) arteries which are branches from the P1 segment of the posterior cerebral artery (PCA);

(3) Inferolateral region: Supplied by thalamogeniculate arteries arising from the P2 segment of the PCA; and,

(4) Posterior region: Supplied by the posterior choroidal arteries from the P2 segment of the PCA[5] [6]. (Table 1)

Artery of Percheron Infarction (API) is a rare cause of bilateral thalamic infarction, accounting for less than 1% of all thalamic strokes. The artery of Percheron is a single arterial trunk that arises from one of the proximal branches of the posterior cerebral artery (PCA) and supplies the bilateral paramedian thalamus, the rostral midbrain, and the posterior hypothalamus.[2]

Artery of Percheron Infarction (API) can manifest with a spectrum of neurological symptoms, including altered consciousness, memory impairment, behavioral changes, ocular movement abnormalities, and vertical gaze palsy.[7][8]

The role of imaging is paramount in diagnosing API, and a comprehensive understanding of the distinct imaging patterns is essential for accurate diagnosis and effective management of the condition. Timely and accurate diagnosis of an Artery of Percheron (AOP) infarction is crucial for directing appropriate, time-sensitive management and preventing unnecessary additional procedures. This paper aims to review the specific imaging patterns observed in API and their clinical significance.

The imaging differential of bithalamic lesions is broad and includes arterial and venous occlusion, infiltrative neoplasms, infectious and inflammatory lesions.

**MATERIALS AND METHODS:**

Imaging and clinical data of 25 patients with AOP infarction from 2022 to 2017 were reviewed retrospectively. This study was conducted in the Department of RADIO-DIAGNOSIS at Dr.Pinnamaneni Siddhartha Institute and Medical Sciences. Patients with AOP infarction over a period of 5-years (2017-2022) were identified from the computerized discharge summaries and the data was collected from the inpatient records and the stroke clinic where the patients were being followed up. Radiological images were reviewed. The diagnosis of AOP infarction was based on magnetic resonance imaging (MRI) and magnetic resonance angiography.

**INCLUSION CRITERIA:**

The primary imaging criterion for inclusion was abnormal signal intensity on MR imaging involving the bilateral paramedian thalami with or without rostral midbrain involvement. The abnormal signal intensity was defined as a hyperintense T2 or FLAIR signal intensity on MR imaging with or without restricted diffusion or postcontrast enhancement, within a specific bilateral paramedian thalamic distribution, corresponding to a distinct arterial territory. A compatible clinical presentation (eg: rapid onset of altered mental status and ophthalmoplegia) was also used to support inclusion, when clinical data were available.

The primary imaging criterion for inclusion was the presence of abnormal signal intensity on MR imaging, specifically involving the bilateral paramedian thalami with or without rostral midbrain involvement. Abnormal signal intensity was defined as a hyperintense T2 or FLAIR signal intensity on MR imaging, with or without evidence of restricted diffusion or postcontrast enhancement, within a distinct bilateral paramedian thalamic distribution aligned with a specific arterial territory. Inclusion was further supported by a compatible clinical presentation, such as the rapid onset of altered mental status and ophthalmoplegia, when clinical data were available."

**EXCLUSION CRITERIA:**

Patients presenting with conditions that could mimic AOP infarction, such as deep venous system infarction or tumors, were excluded following comprehensive evaluation with appropriate imaging and tests. Additionally, individuals with top-of-the-basilar artery occlusions accompanied by bilateral thalamic infarcts were excluded. Furthermore, patients with pathology-proven direct thalamic involvement by neoplasm, which could mimic ischemia on contrast-enhanced imaging were also excluded.



**RESULTS:**

All patients were evaluated by MRI performed on Philips 1.5Tesla .

IMAGING FEATURES:

Bilateral paramedian thalamic infarctions were present in all patients (25/25).

Each case was found to fit into 1 of 4 patterns (Table 2)

Bilateral paramedian thalamic with midbrain in 56% ( 14 /25), -Pattern 1 (fig 3)

Bilateral paramedian thalamic without midbrain 28% ( 7/25), - Pattern 2 (fig 4)

Bilateral paramedian thalamic with anterior thalamus and midbrain 12 % ( 3 /25), -Pattern 3 (fig 5 ) and

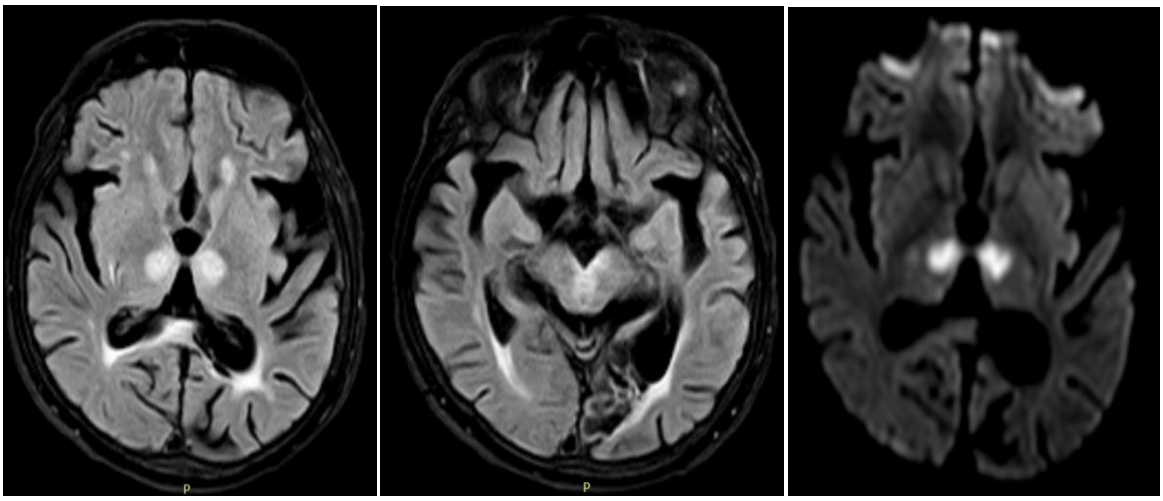
Bilateral paramedian thalamic with anterior thalamus without midbrain 5% (1 /25).-Pattern 4 (fig 6)

A distinctive V-shaped hyperintensity was observed on axial FLAIR and/or DWI along the pial surface of the midbrain, adjacent to the interpeduncular fossa, in patients with midbrain involvement."

Table 2:

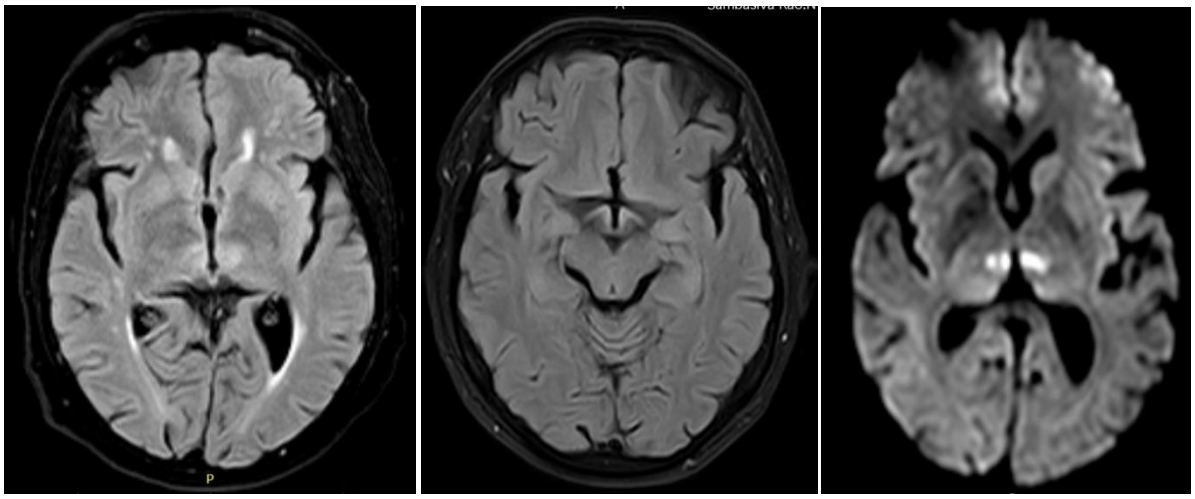
| PATTERNS OF AOP INFARCTION | | |
|---|---|---|
| Bilateral paramedian thalami with midbrain | 14/25 | 56% |
| Bilateral paramedian thalami without | 7/25 | 28% |

| | | |
|---|---|---|
| midbrain | | |
| Bilateral paramedian and anterior thalami with midbrain | 3/25 | 12% |
| Bilateral paramedian and anterior thalami without midbrain | 1/25 | 5% |

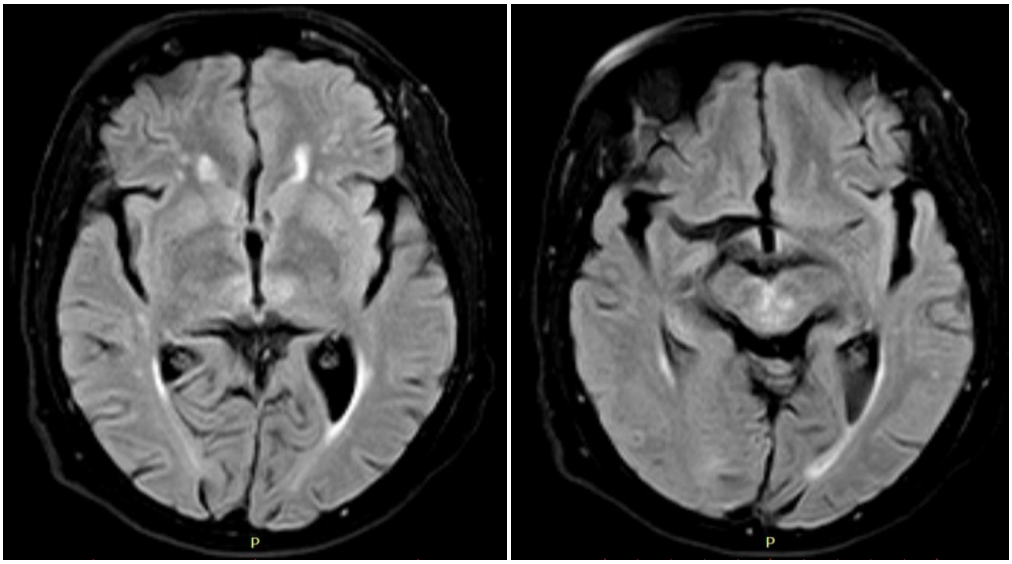


FLAIR sequence showing hyperintense signal in bilateral paramedian thalami and showing midbrain peduncular "V" hyperintensity. DWI image showing hyperintense signal in bilateral paramedian thalami (Pattern 1) (fig:3)



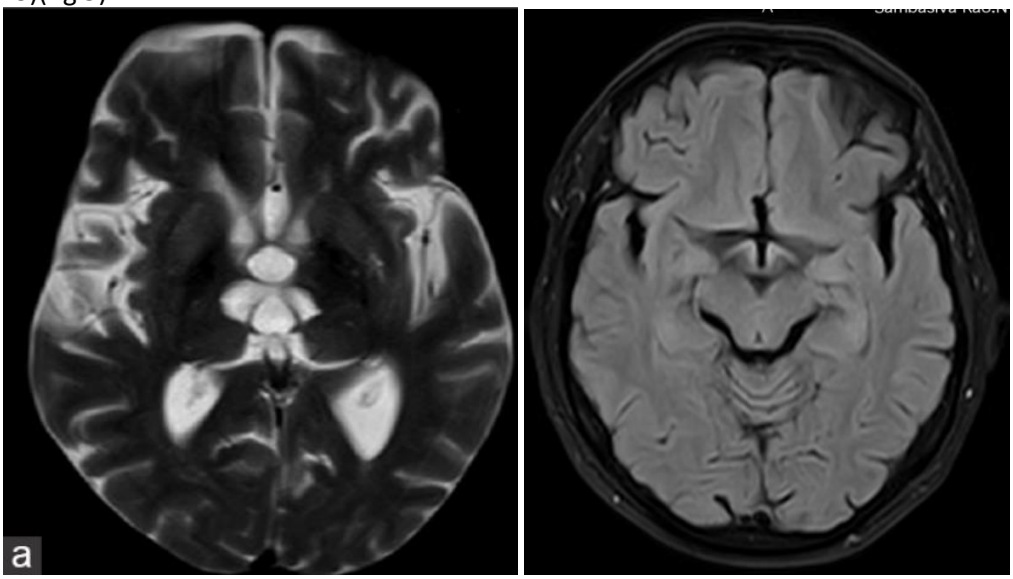


Subtle FLAIR hyperintensities in bilateral paramedian thalami without midbrain invovement. DWI image showing hyperintense signal in bilateral paramedian thalami (Pattern 2) (fig :4)



Subtle FLAIR hyperintensities in bilateral paramedian thalami, anterior thalami and rostral midbrain . ( Pattern 3)(fig 5)






T2 axial sequence showing bilateral paramedian thalamic and anterior thalamic infarcts without midbrain (Pattern 4) (fig:6)

CLINICAL FEATURES:

- The most common presenting symptom was the association with disorders of consciousness, observed in 24 out of 25 patients (94%).
- Sudden loss of consciousness within varying intervals, ranging from 3 to 24 hours, was evident in 21 out of 25 patients (70.5%).
- Fluctuations in consciousness were a presenting symptom in 11 out of 25 patients (33.3%).
- Disorders of gaze were observed in 12 out of 25 patients.
- Apathy, muteness, and hypersomnolence were seen in 5 out of 17 patients (29%).
- Memory impairment (anterograde amnesia) was noted in 4 out of 17 patients (24%), all of whom exhibited involvement of the anterior thalamus on imaging.
- Headache was a prominent presenting complaint in 3 out of 17 patients (18%).

**DISCUSSION:**

At our institution, we identified 25 patients with AOP infarction during a 5-year period.
We identified 4 ischemic patterns of AOP infarction:
1) Bilateral paramedian thalamic with midbrain involvement (56%),
2) Bilateral paramedian thalamic without midbrain involvement (28%),
3) Bilateral paramedian thalamic with anterior thalamus and midbrain involvement (12%) &
4) Bilateral paramedian thalamic with anterior thalamus without midbrain involvement(4%).

These 4 distinct patterns are consistent with known variations in the paramedian artery. Another distinctive imaging finding was a V-shaped hyperintense signal intensity on axial FLAIR and DWI images along the pial surface of the midbrain in the interpeduncular fossa.
MRI with diffusion-weighted imaging (DWI) sequence is best to pick up early ischemic changes caused by AOP infarction. Very rarely, visualization of the acute infarction may be absent on the initial DWI, and if the index of suspicion is high, a repeat imaging should be done.

The most common presenting features in AOP infarcts include disorders of consciousness and gaze. Bilateral paramedian thalamic infarction can lead to a reduction in consciousness, ranging from somnolence to coma, typically of a transient nature.Lesions in the paramedian region of the thalamus, responsible for reward learning[7], can induce apathy and a lack of motivation[8]. Some reported manifestations include presleep behavior and a propensity to assume sleep-inducing body postures[9]. In cases where the anterior thalamus is involved, memory impairment may arise due to the impact on the mammillothalamic tract, the inferior thalamic pedicle, as well as anterior and dorsomedial nuclear confabulations, which can be frequently associated.[10][11][12]



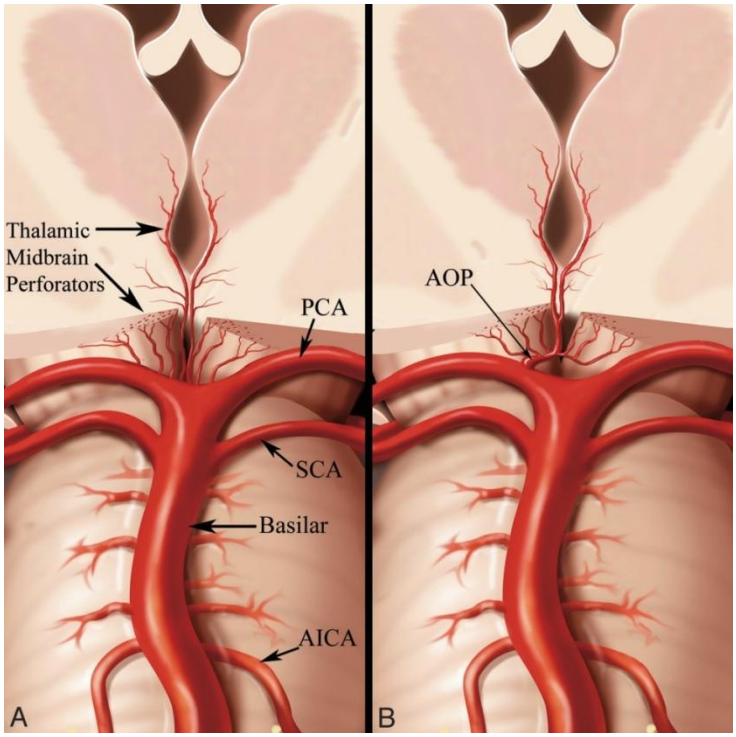



Figure 1A.Conventional anatomy demonstrating paired thalamic and midbrain perforating arteries.
*B* .AOP arising as a single unpaired trunk from P1 supplying the bilateral paramedianthalami and rostral midbrain.

Table 1:Areas that can be affected by AOP Infarction

| Thalamus | Outside Thalamus **(if paramesencephalic arteries also arise from AOP)** |
|---|---|
| Medial thalamic nuclei<br>Anterior thalamus (if polar artery is absent) | Inter peduncular fossa<br>Decussation of superior cerebellar peduncles<br>Medial part of red nucleus<br>3 rd & 4 th cranial nerve nuclei<br>Anterior portion of periaqueductal gray matter |

Lazzaro et al. ]demonstrated 4 distinct patterns of AOP infarction.[13]These include bilateral paramedian thalamic with rostral midbrain (43%); bilateral paramedian thalamic without midbrain (38%); bilateral paramedian and anterior thalamic with midbrain (14%); and, bilateral paramedian and anterior thalamic without midbrain (5%) infarctions. Comparing our series, we found the similar frequency in the patterns detected, the rarest being bilateral paramedian and anterior thalamic without midbrain infarction Furthermore, with AOP infarcts, the midbrain "V" sign can be seen. [13]
Other smaller studies have demonstrated the AOP ischemic pattern in 2% of all ischemic strokes[24] and from 4% to 18% of all thalamic strokes.Several clinical stroke patterns potentially involving the AOP have been described, including bilateral paramedian thalamic, paramedian and polar thalamic, and paramedian thalamic and mesencephalic.
Awareness of the imaging patterns in artery of Percheron infarction can aid in the timely diagnosis and management of this condition.
In the acute setting, the imaging differentials will include a deep venous system thrombosis [14][15].Susceptibility weighted imaging sequences on the MRI can be helpful in demonstrating the thrombosed veins.[16]
If the distal basilar artery gets occluded, both the thalamoperforate arteries (paramedian arteries) and the paramedian mesencephalic arteries on both sides may also get occluded mimicking an AOP occlusion. Clinically, these patients will usually have other features of 'top-of-the-basilar' syndrome.[17]
Wernicke encephalopathy may present with the classical triad of ataxia, altered consciousness, and abnormal eye movements. T2-weighted MR changes will be seen in the mammillary bodies, tectal plate, periaqueductal gray matter, dorsal medulla, and the medial aspects of thalami. There may be restriction of diffusion in these areas mimicking an arterial infarction. [17][18][19]

**LIMITATIONS:**

The retrospective nature of this imaging study led to incomplete clinical data, with some patients' information being unavailable. Additionally, it's worth noting that a single large embolus at the basilar tip could potentially produce a similar infarct pattern, particularly in cases involving combined paramedian thalamic and midbrain infarcts.

**CONCLUSION:**

The identification of four distinct ischemic patterns, coupled with the midbrain "V" sign, in our case series will improve the recognition of Artery of Percheron (AOP) infarction. This improved recognition assist to facilitate neurological evaluation and management for patients with thalamic strokes. These patterns align with previously described clinical syndromes and variations in thalamic vascular supply. Notably, AOP infarcts can manifest with sudden alterations in sensorium, and the presence of gaze disorders may provide valuable diagnostic clues. Suspected cases should undergo MRI with DWI/ADC for a more accurate diagnosis.

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