Acute Percheron Infarction: A Precision Learning

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Abstract

Background: So far, the diagnosis of acute AOP infarction is uncommon. The purpose of our study was to characterize the relationship between the imaging spectrum of acute AOP infarction and its clinical manifestations and prognosis on the basis of 23 cases.

Methods: A total of 23 patients with acute AOP infarction in our institution from 2014 to 2019 were reviewed retrospectively. All cases were evaluated with computed tomography (CT), magnetic resonance imaging (MRI), detailed clinical and evaluated prognosis used a modified Rankin scale (mRs), blood studies, electrocardiogram and transthoracic echocardiography. All standard risk factors were recorded in these patients. mRs scores 90 days after discharge.

Results: We identified 4 various patterns of acute AOP infarction: (1) bilateral paramedian thalamic infarction (BPTI, 52%), (2) bilateral paramedian thalamic with rostral midbrain infarction (BPTRMI, 30%), (3) bilateral paramedian and anterior thalamic infarction (BPATI, 13%), and (4) bilateral paramedian thalamic with red nuclei infarction (BPTRNI, 4%). These patients had consciousness disorder, memory dysfunctions, vertical gaze paresis, mesencephalothalamic syndrome and so on. The 65% patients with BPTI and BPATI who experienced a good functional recovery and could carry out daily life activities (mRS score ≤ 2). However, patients with BPTRMI who have an unfavorable outcome.

Conclusion: Although the clinical feature of patients with AOP infarction is variable, DWI or ADC map can improve the diagnosis of acute AOP infarction patterns. Acute AOP occlusion requires immediate diagnosis and treatment initiation for a more favorable outcome and additional unnecessary procedures.

Background

Thalamic arterial supply is classically categorized into 4 territories: anterior supplied by the polar or thalamo-tuberal arteries, paramedian supplied by the paramedian or thalamo-perforating arteries, inferolateral supplied by the thalamo-geniculate arteries, and posterior supplied by the posterior choroidal arteries[1–4]. The artery of percheron (AOP), first described by Percheron G in 1973, is a solitary arterial trunk of bilateral thalamo-perforating artery originated from P1 segment of one of posterior cerebral artery (PCA), which supplies bilaterally the paramedian thalamic territories[5]. The AOP is an uncommon arterial variant. Meanwhile, rostral midbrain have supplied by the superior mesencephalic or rubral arteries, which can branch separately from P1 segment of the PCA or share a common origin with AOP (Fig. 1). Thus, AOP probably supply the rostral midbrain or red nucleus[6–9]. Acute infarction of AOP is a rare characteristic pattern of ischemia. Acute AOP infarction dramatically affect people's health status, potentially in a life-threatening manner, although a relatively small ischemic lesion in the paramedian bithalami[10]. So, diagnosing the acute AOP infarction is vital to guide the appropriate management and to prevent additional unnecessary procedures[11]. The AOP is very small diameter that cannot be displayed by magnetic resonance angiography (MRA) and digital subtraction angiography (DSA) can't show its presence, stenosis or occlusion, while only a few authors have found AOP obstruction by

DSA[12–15]. MR imaging, especially diffusion-weighted imaging (DWI), plays an important role in the diagnosis of acute infarctions of AOP. At the moment, most neuroimaging literatures only described the relationships between clinical presentation and imaging[6, 16]. However, we not only retrospectively collected 23 cases of AOP infarction aim to summarize the specific imaging patterns and clinical characteristics but also investigated the prognostics.

Materials And Methods

This study was approved by the institutional review board. We retrospectively identified 23 patients with acute AOP infarctions (age range, 23–86 years; mean, 58 ± 14 years; 8 female, 15 male) by searching MR reports database (keywords: bilateral acute paramedian thalamic infarction) from 2012–2019. According to state of an illness, all patients were examined by one or more head CT and routine MR imaging including axial T2-weighted images (T2WI), axial T1-weighted images (T1WI), axial fluid-attenuated inversion-recovery (FLAIR) and DWI sequences. Inclusion criteria: high signal intensity on DWI or low signal intensity on apparent diffusion coefficients (ADC) maps were noted in bilateral paramedian thalami with or without rostral midbrain involvement. Exclusion criteria: the top of the basilar artery syndrome, diffuse midline glioma of thalamus, viral encephalitis, arteriovenous fistula (AF) and wernicke encephalopathy. In addition, MRA in was performed in 20/23 patients. Computed tomography perfusion (CTP) in 5/23; Susceptibility weighted imaging (SWI) in 4/23.

Clinical workup included assessment of cardiovascular risk factors, such as arterial hypertension, hypercholesterolemia and diabetes mellitus, 24-hour dynamic electrocardiogram (DCG) and transesophageal or transthoracic echocardiography, transcranial Doppler and colour-coded duplex ultrasound sonography sonography. Hypercoagulopathies include protein C, protein S, activated protein C resistance and factor V Leiden mutation.

Each patient was assigned to one or more of the following etiologies on the basis of clinical risk factors including cardioembolism, large artery atherosclerosis, small artery occlusion and so on. During hospitalization, neurological examination was performed and the patients were followed up for 6 months. Three months after discharge, modified Rankin Scale (mRS) score ≤ 2 was good prognosis ; score ≥ 3 was poor prognosis.

Image Analysis

Images of all patients with acute AOP infarction were reviewed by 2 senior neuroradiologists and consistent conclusions were reached. The acute ischemic territories involved in each patients were recorded under the following categories: only paramedian bithalamus (asymmetric or symmetric), paramedian bithalamic with rostral midbrain, paramedian bithalamic with anterior thalamus (right, left or bilateral), paramedian bithalamus and anterior thalamic with rostral midbrain and paramedian bithalamic with red nucleus (right, left or bilateral). Acute infarction was defined as hyperintense in DWI and hypointense in ADC map.

Results

Clinical syndromes

Table 1 (detailed in Supplementary Material) shows the risk factors and neurological symptoms for all cases. The most common syndromes was altered mental status in 22 cases (96%), which include 11 cases of hypersomnia (48%), 7 cases of coma (30%) and 4 cases of drowsiness (17%). Other characteristic include: vertical gaze palsy in 8 cases (35%), hypomnesia in 7 cases (30%), oculomotor nerve palsy in 7 cases (30%), disorientation in 5 cases (22%), baryllalia and aphasia in 6 cases (26%), movement disorders in 9 cases (39%), ataxia in 5 cases (22%) and hemiplegia in 2 cases (9%).

Imaging findings

Head CT of 22 patients with acute AOP infarctions performed 2 hours after the onset of symptoms showed no acute hemorrhage or ischemia, only one patient had bilateral parathalamic and rostral midbrain hypodensity (Fig. 2). One patient had bilateral paramedian thalamus and left anterior thalamus hypodensity at 24 hours after onset (Fig. 3). MR imaging showed characteristic symmetrical bilateral paramedian thalamic in 30% (7/23) and symmetrical in 22% (5/23). Acute infarctions of paramedian thalamic and the rostral midbrain was present in 30% of patients (7/23), a distinct pattern of V-shaped hyperintensity on axial DWI along the pial surface of the midbrain in the interpeduncular fossa (Fig. 2) was found in 17% cases (4/23). Bilateral paramedian thalamic ischemia with anterolateral paramedian territory (Additional Fig. 1) in 13% of patients (3/23). Bilateral paramedian thalamic infarction with left red nucleus (Additional Fig. 2) in 4% of patient (1/23). None of the cases had a paramedian bithalamus and anterior thalamic with rostral midbrain. Stenosis or occlusion of the vertebral artery, basilar artery or P1-segments of PCA was observed in 5 patients, whereas in 15 patients MRA was normal. CT perfusion image demonstrated symmetrically increased mean transit time (MTT) and decreased mean cerebral blood flow (CBF) and mean cerebral blood volume (CBV) in the bilateral paramedian thalami in the in 4 patients (Additional Fig. 3). No bleeding signal was found in SWI of 4 patients.

Treatment and prognosis

All patients were classified as ischemic cerebrovascular disease after admission treatment, therapeutic schedule included antiplatelet/anticoagulation, plaque stabilization and collateral circulation improvement. Patient No.4/8/11/14/16/19 was given alteplase in the time window (time window < 4.5h-6h). Heparin has also been used post-recanalization after thrombolysis. Low-molecular-weight heparins calcium injection (4000IU/12h) by subcutaneous was given to patients No.5/7/9/12/13/17 due to cardioembolism. Patients No.1/2/6/15 were given aspirin 200mg/d orally. Patients No.3/16/18/20/21/22/23 were treated with aspirin 200mg/d combined with clopidogrel 7.5 mg/d.

All patients were followed up for 3 months upon discharge, mRS score are shown in Table 1. There were 65% cases with a favorable outcome (mRS score ≤ 2) who experienced a good functional recovery and could carry out daily life activities. Patient partially recovered normal mental status. Patient No.4 was full patient recovery. Patient No.1/2/6/10/13/15/18/19/20 of vertical ophthalmoplegia recovered in various

degree. Patient *No.3/10/19/23* had memory loss and patient *No.5/11/19* had obvious recent memory decline. Patient *No.7/8/16/22* had unclear articulation. Patient *No.2/6/8/9/10/13/17/18/20/22* had dyskinesia, the patients with rostral midbrain infarction were dependent and unable to walk without assistance. Patient *No.5/7/9/10/13/18/20* still had disorientation or mild ataxia symptoms.

Discussion

Etiology and risk factors

Acute ischemia of AOP are uncommon, which accounted for 0.1% – 0.3% of all cerebral infarction[6, 17]. However, this is likely a conservative estimate due to the insufficient knowledge of it and limitations in their inclusion criteria[6, 18]. AOP arising from P1 segments of the PCA supply predominantly paramedian bithalami with variable contribution to the rostral midbrain or anterolateral thalami[6, 18]. In our studies, hypertension, smoking, diabetes mellitus, and hyperlipemia are risk factors for acute Percheron infarction. Small vessel disease and arteriosclerosis was the cause of acute AOP infarcts in 14 patients (61%), which is consistent with the previous reports[16, 19]. Cruz-Cosme et al[16, 19] indicated that cardioembolism was the most common cause, while only 7 cases (30%) had a potential source of cardiac embolism in our series. In addition, basilar artery dissection was seen in patient *No.21* and *22*. Atherosclerosis and small vessel disease are the main causes of acute AOP infarction.

Relationship imaging diagnosis and clinical features

Acute AOP ischemia has great variability with respect to symmetry, size, and territory, which is mainly due to the thalamic arteries vary between individuals with respect to the parent vessel from which each branch arises, the number and position of the arteries and their tributaries[1, 2, 16, 20, 21]. Paramedian bithalami of the 23 patients showed high signal intensity on FLAIR with restricted diffusion and hypointense in the ADC map (positive in 100%). Valentina Francioni et al[22]. show a DWI/FLAIR mismatch typical of hyperacute paramedian bithalami ischemia, which points out the DWI and ADC map is very important for the differentiation and therapy in acute AOP infarction. Although head CT is not sensitive to acute AOP infarction, it is indispensable to exclude cerebral hemorrhage at admission. Adam MacLellan et al[23]. identified a case describing AOP ischemic on acute CT perfusion imaging for the first time, however, thrombolysis was not performed in that symmetrical perfusion abnormalities were not recognized. Most interestingly, we found that patient *No.4/8/11/14* show corresponding bithalamic ischemia changes on acute CT perfusion imaging and was given thrombolysis to avoid deterioration. DSA rarely show the presence of AOP, more often than not, the AOP are too small to be visualized by angiography. Similarly, although the AOP is rarely visualized with conventional MRA, it is very valuable to observe the stenosis of BA and PCA by MRA. Obstruction of the AOP oftentimes leads to ischemia of the midbrain (57%) and/or the anterior thalamus (19%)[6] while we found that acute infarction of the rostral midbrain was present in 30% and anterior thalamus in 13%. The polar artery is absent in 30% – 60% of the population in that PcomA is highly variable (absent or hypoplastic)[24]. Then, paramedian arteries not only may supply the paramedian but also the anterior thalamic territories, especially when the polar artery

is absent (Fig. 2B)[1, 3, 12, 25, 26]. Therefore, an infarction of the paramedian bithalamus and the anterior thalamus may be explained by occlusion of a single AOP, rather than synchronous occlusions of an AOP and the polar artery. In addition, patient *No.7* showed bilateral paramedian thalamic infarction with only left red nucleus. Similar to previous studies[9, 10], we also analysis the relationship between AOP infarction region and clinical presentation. Based on previous report[6, 9], we found 4 different AOP infarction patterns: (1) bilateral paramedian thalamic infarction (BPTI, 52%), (2) bilateral paramedian thalamic with rostral midbrain infarction (BPTRMI, 30%), (3) bilateral paramedian and anterior thalamic infarction (BPATI, 13%), and (4) bilateral paramedian thalamic with red nuclei infarction (BPTRNI, 4%). No case involving bilateral paramedian and anterior thalamic infarction with midbrain involvement was found. The thalami contain reticular and intralaminar nuclei, associative nuclei, effector nuclei, sensory nuclei and limbic nuclei. Acute infarction of AOP destroy these nuclei in different combinations and result in complex syndromes depending on which nuclei are involved[1]. The typical clinical symptoms are "altered mental status, vertical gaze palsy, and memory impairment". Other symptoms include: disorientation, hemiplegia, cerebellar ataxia, movement disorders and dysarthria[1, 6]. Disorder of consciousness in 22 cases, except patient *No.15*, may be due to involvement of the reticular activating system. Excitedly, recent evidences suggest that the paraventricular nuclei supplied the AOP is a key wakefulness-controlling nucleus in the thalamus[27]. The vertical gaze palsy observed in patient *No.2/10/13/18/20* with BPTRMI is due to an associated involvement of the midbrain tegmentum, which including the interstitial nucleus of Cajal and rostral interstitial nucleus medial longitudinal fasciculus (RINMLF). Meanwhile, vertical gaze palsy in patient *No.1/15/19* with BPTI were also reported, which is because the thalami having a vital role in cortical input processing to the RINMLF[27]. Memory dysfunctions were reported in patient *No.3/5/9/10/11/19/23* cases (BPATI in patient *No.5/11/19*). The dorsomedial nucleus located in paramedian territory had supplied by the AOP, the mammillothalamic tract and anterior nucleus located in anterior territory had supplied by polar artery or AOP, which are commonly implicated in memory function[29, 30]. When the anterior thalamic territory is also involved, memory impairment is typically more severe[29, 30]. In our series, patient *No.5/11/19* with BPATI still had serve memory deficit after 90 days. Oculomotor nerve palsy including ptosis, ocular movement disorders and ocular abnormal reflexes in patient *No.2/6/10/13/18/20* with BPTRMI were related to acute ischemia of the oculomotor nucleus located in near midline of mesencephalic. The mesencephalothalamic or thalamopeduncular syndrome included movement disorders, hemiplegia and cerebellar ataxia occurs in patients with BPTRMI[8, 31], which is have a bearing on midbrain involvement. The superior mesencephalic or rubral artery branched separately from P1 segment of the PCA or share a common origin with AOP have supplied blood to the interpeduncular nucleus, medial part of the red nucleus, nucleus of cranial nerve III and anterior part of the periaqueductal gray matter located in dorsal midbrain[1, 7, 9, 31]. Similar to polar artery, a single AOP may result in BPTRMI, rather than synchronous occlusions of an AOP and the superior mesencephalic or rubral artery.

Treatment and prognosis

In this study, 65% of the patients who had a favorable outcome. Complete recovery from bilateral paramedian thalamic infarction has occasionally been reported[32]. Current, objectives of a treatment

regimen for acute AOP occlusion are to promote recanalization[33]. Patient No.4/8/11/14/16/19 were treated with thrombolytic therapy without cerebral hemorrhage or edema found within 6 hour present on head CT, patient No.4 was full patient recovery with residual absence of cognitive impairment (mRs score=0), as well as patient No.8/11/14/16/19 was fully conscious with partial memory deficit (mRs score=1). At the moment, thrombolytic therapy (time window<4.5h-6h) is the most effective treatment for acute AOP occlusion[11], our result is consistent with previous studies[13]. Patients with outside 6h/contraindicated of thrombolysis were treated with antiplatelet and heparin. We found a favorable outcome (mRs score=1 or 2, except patient No.7) in patient No.1/3/5/12/15/17/21/22/23 with BPTI. Meanwhile, an unfavorable outcome (mRs score=3 or 4) was present in patient No.2/6/9/13/18/20 with BPTRMI, which was consistent with previous studies[10, 16]. Cassourret G et al. have reported a cases with unconsciousness that have marked cognitive and motor improvement but still with partial memory deficit by intravenous heparin on day 2 after onset. Oral anticoagulants have been prescribed to 23 patients to prevent future episodes. Based on reported literature and our cases, we present evidence for a treatment algorithm for acute AOP infarction once linical characteristics is classical or has been confirmed DWI and ADC map (Fig. 3).

Differential imaging

There are some other etiologies to consider, such as the “top of the basilar” syndrome, deep venous infarction by thrombosis, wernicke's encephalopathy and diffuse midline gliomas, which should not be confused with AOP infarction[34] (Fig. 4). A single large embolus at the basilar artery tip could mimic acute AOP infarction pattern, but it often with additional characteristic cerebellar, brainstem and occipital lobe infarcts. Deep venous infarction by thrombosis and diffuse midline gliomas do not involve the specific blood-supply territory of the AOP but rather involve multiple arterial regions. Wernicke's encephalopathy affects not only the thalamus but also the mammillary bodies, tectal plate, and periaqueductal area. Diffuse midline gliomas in bilateral thalamus with space-occupying effect are hypointense on T1WI and hyperintense on T2WI or FLAIR as well as isointensity on DWI.

Limitation

There are some limitations in the present study. Firstly, 23 patients were retrospectively enrolled from a single institution, and the data were limited sample size. A large, multicenter study is necessitated in the future. Secondly, although many author suggest that thrombolysis are effective firstline treatment options for acute AOP infarcts, documented treated cases of AOP occlusion is rare. Only 6 cases were treated with thrombolytic therapy in our study. The development of treatment options needs to be further improved via systematic case reports and clinical trials of higher power[11].

Conclusion

AOP is a rare unique arterial anatomical variant. Angiography usually does not permit obstruction of the AOP because of its small size[14], thus, angiography should not be used in the diagnosis of AOP acute infarction. DWI and ADC map is best for diagnosis of acute AOP infarction, meanwhile, an understand of

the relationship between imaging and clinical presentation would increase recognition of acute AOP infarction and help guide us in the diagnosis and treatment.

Declarations

Ethics approval and consent to participate

Ethical approval was provided by the first hospital of Jilin University (Ethical approval reference number: 2021-362). Each author certifies that all the investigations were conducted in conformity with ethical principles. Informed consent was written obtained when patients were admitted to hospital.

Consent for publication

Not applicable.

Availability of data and material

The datasets supporting the conclusions of this article are included within the article. The raw data are not publicly available due it involves the privacy of patients but are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Author's contributions

ZB is in charge of result analysis and thesis writing; WXX and CG is in charge of original data collection; WJP is in charge of guiding scheme design and paper revision. All authors have read and approved the manuscript, and ensure that this is the case.

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Tables

Table1. Characteristics in patients with acute AOP infarction					
Patient No./ Age(y)/Sex	Location	Risk factors	Aetiologies	Symptoms	mRS after 90 days
1/65/F	paramedian bithalami	Cho, DM, HTN	Arteriosclerosis of right PCA	Hypersomnia Vertical gaze palsy	2
2/71/F	paramedian bithalami rostral midbrain	Smoke, DM, HTN,CAD	Small vessel disease	Coma Vertical gaze palsy Oculomotor nerve palsy Movement disorders	3
3/68/M	paramedian bithalami	Smoke, Cho, DM, HTN,CAD	Arteriosclerosis of BA	Hypersomnia Movement disorders Aphasia Hypomnesia	2
4/66/M	paramedian bithalami	DM, HTN, CAD	Arteriosclerosis of left PCA	Coma Movement disorders	0
5/63/M	paramedian bithalami anterolateral thalami	Smoke, Drinking, HTN	Sinus bradycardia	Hypersomnia Hypomnesia Disorientation Movement disorders Corticospinal tract signs	2
6/69/M	paramedian bithalami rostral midbrain	Cho, DM, HTN,CAD	Arteriosclerosis of left PCA	Hypersomnia Oculomotor nerve palsy Movement disorders	4
7/45/M	paramedian bithalami	Cho, DM, HTN	Atrial fibrillation	Hypersomnia	3

	red nucleus			Barylalia Ataxia Dysarthria	
8/64/M	paramedian bithalami	Smoking, Cho, DM, HTN	Small vessel disease	Hypersomnia Barylalia Movement disorders	1
9/60/M	paramedian bithalami rostral midbrain	Cho, DM, HTN	Atrial fibrillation	Hypersomnia Disorientation Hypomnesia Movement disorders	3
10/67/M	paramedian bithalami rostral midbrain	Smoke, Cho, DM, HTN	Small vessel disease	Coma Vertical gaze palsy Hypomnesia Disorientation Oculomotor nerve palsy Movement disorders	4
11/29/F	paramedian bithalami anterolateral thalami	Smoke	Small vessel disease	Coma Hypomnesia Disorientation	1
12/43/F	paramedian bithalami	Cho, DM, HTN	Endocarditis	Coma Hemiplegia	1
13/77/M	paramedian bithalami rostral midbrain	Smoking, DM, HTN	Atrial fibrillation	Coma Vertical gaze palsy Disorientation Oculomotor nerve palsy Movement disorders	4

14/55/F	paramedian bithalami	Cho, DM, HTN	Small vessel disease	Cerebellar ataxia Barylalia	2
15/60/M	paramedian bithalami	Smoking, Cho, HTN	Small vessel disease	Drowsiness Vertical gaze palsy	2
16/67/M	paramedian bithalami	Cho, DM, HTN	Small vessel disease	Hypersomnia Barylalia	2
17/53/M	paramedian bithalami	Smoke	Atrial myxoma	Drowsiness Movement disorders	1
18/64/F	paramedian bithalami rostral midbrain	Cho, DM, HTN	Arteriosclerosis of right PCA	Drowsiness Cerebellar ataxia Vertical gaze palsy Oculomotor nerve palsy Movement disorders	4
19/66/M	paramedian bithalami anterolateral thalami	Cho, DM	Small vessel disease	Hypersomnia Hypomnesia Vertical gaze palsy	1
20/75/F	paramedian bithalami rostral midbrain	Cho, DM, HTN	Atrial myxoma	Drowsiness Cerebellar ataxia Vertical gaze palsy Oculomotor nerve palsy Movement disorders	5
21/23/M	paramedian bithalami	Smoke	Basilar artery dissection	Drowsiness	2
22/30/F	paramedian bithalami	—	Basilar artery dissection	Coma Aphasia	2

					Movement disorders	
23/65/M	paramedian bithalami	Smoking, Drinking,Cho,DM	Small vessel disease		Hypersomnia	1
					Hypomnesia	
					Hemiplegia	
Abbreviations: M, male; F, female; Cho, hypercholesterolemia; DM, diabetes mellitus; HTN, arterial hypertension; PCA, posterior cerebral artery; BA, basilar artery; AF, atrial fibrillation;CAD, coronary artery disease.						

Figures

Figure 1

Diagrams show the different anatomic regions of the thalamus and its arterial supply. AOP is a solitary arterial trunk of bilateral thalamo-perforating artery originated from P1 segment of one of PCA, which

supplies bilaterally the paramedian thalamic territories(A). When the polar artery is absent, AOP not only may supply the paramedian but also the anterior thalamic territories(B). The rostral midbrain have supplied by the superior mesencephalic artery, which share a common origin with AOP(C). The red nucleus have supplied by the rubral artery, which share a common origin with AOP(D). AOP, artery of percheron; PCA, posterior choroidal artery; BA, basilar artery; AN, anterior nucleus; LN, lateral nuclei; MDN, mediodorsal nuclei; PUL, pulvinar; RN, red nucleus.

Figure 2

Case 2. Axial head CT at 2 hours after onset (A and B) show hypodensity in the bilateral paramedian thalamus and midbrain involvement (white arrow). Axial DWI and ADC maps (C to F) show acute symmetrical infarctions in the bilateral paramedian thalamus and show a V-shaped acute infarction at the interpeduncular fossa of midbrain.

Figure 3

Algorithm for the treatment of artery of Percheron (AOP) occlusion.

Figure 4

Differential diagnosis of AOP infarction. Basilar artery tip syndrome not only could lead to thalamic infarction but also to additional characteristic cerebellar, brainstem and occipital lobe infarcts(A1 and A2). Deep venous infarction by thrombosis show hyperdensity of bilateral internal cerebral veins at head CT(B1, black arrow) and FLAIR show vasogenic edema involved multiple arterial regions of thalamus(B2). Wernicke's encephalopathy often involves the pulvinar(C1) and the mammillary bodies manifested enhancement(C2,white arrow). Diffuse midline glioma in bilateral thalamus with mass effect are hyperintense on FLAIR and isointensity on DWI(D1 and D2).

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