To the Editor: In the literature, there are few reports about unilateral isolated basal vein thrombosis. Owler et al.[1] reported the first case with imaging evidence in 2004.

Six days before admission, an otherwise healthy 53-year-old male developed sudden hemihyposthesia and hemiparesis of the right limb. Over several days, his right-sided hemiplegia progressively worsened with the development of diplopia and a lisp. The patient was then admitted to the hospital.

On admission, neurologic physical examination showed dysarthria, bilateral miosis, slow pupillary light reflex, weakened pharyngeal reflex, reduction in the right limb strength (right arm strength grade 4/5, right leg strength grade 2/5), and hemihypesthesia of the right limb.

Magnetic resonance imaging (MRI) revealed a unilateral well-defined lesion involving left thalamus, basal ganglia, mesencephalon, and pons. On T1-weighted imaging, the area of the lesion showed low signal and mild enhancement after intravenous gadolinium. On T2-weighted imaging, the lesion had high signal intensity. There was mass effect due to swelling of these areas. In addition, there were multiple punctiform hyperintense lesions in the left ambient cistern. Left basal vein of Rosenthal was not exhibited in contrast-enhanced magnetic resonance venography [Figure 1].

The initial pressure of lumbar puncture was 260 mm H\textsubscript{2}O (1 mm H\textsubscript{2}O = 0.0098 kPa). Cerebrospinal fluid examination showed that red blood cell count (160 × 10\textsuperscript{6}/L), protein (46.3 mg/dl), and glucose (7.55 mmol/L) were elevated. Electrocardiography and laboratory analyses of the serum were all within normal ranges.

The patient was diagnosed with unilateral isolated basal vein thrombosis. After 8 days on low-molecular-weight heparin treatment, the patient’s symptoms gradually improved. Right limb strength increased (right arm strength grade 4+/5, right leg strength grade 4+/5). He was continued on anticoagulant therapy with warfarin for the next 3 months. The patient refused reexamination at 3-month follow-up, because his symptoms had disappeared completely at the time.

Many etiology causes, such as pregnancy, oral contraceptives, protein C and S alterations, malignancy, dehydration, and malnutrition, are associated with cerebral venous thrombosis. No etiological factor can be identified in this patient.

Several of the patient’s cerebral structures including the thalami, basal ganglia, mesencephalon, and pons were affected. Vascular lesions, mainly arterial infarctions, tumors such as gliomas, and tumefactive demyelinating lesions need to be considered in the differential diagnosis. Diffusion-weighted imaging can be used to differentiate arterial infarction from venous infarction. Venous edema is due to its vasogenic edema, which is characterized by increased diffusion.[2] However, arterial infarction is due to cytotoxic edema, which is due to restricted diffusion.[2] With venous infarction, cytotoxic edema coexists with vasogenic edema and manifests as patchy areas of increased and decreased diffusion.[1] MRI of gliomas can present as masses with compression of adjacent structures, but these imaging changes are not specific. Spectroscopy shows an increase in the choline peak. Tumefactive demyelinating lesions are characterized as mass-like lesions with or without ring enhancement on contrast-enhanced MRI. None of these features were visualized in this patient’s imaging, thereby supporting the diagnosis of basal venous thrombus.

Particular imaging techniques and attention to specific features may be helpful in establishing the correct diagnosis. Hyperintensity on MR T1-weighted imaging in the intracranial venous sinus is the important evidence of venous sinus thrombosis. Stripe-shaped hyperintensity on MR T1-weighted imaging is a sign of cerebral cortical venous thrombosis.[3] In this case, the high punctiform signal in the left ambient cisterna on MR T1-weighted imaging indicated a diagnosis of venous thrombosis, which was further supported by the absence of the left basal vein in magnetic resonance venography and a good response to anticoagulant therapy.

Declaration of patient consent

The authors certify that they have obtained patient consent forms.

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In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest
There are no conflicts of interest.

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Figure 1: (a‑c) Magnetic resonance T1‑weighted imaging showed a hypointense lesion; (d) magnetic resonance diffusion‑weighted imaging showed a focal hyperintense lesion; (e‑g) magnetic resonance T2‑weighted imaging showed a hyperintense lesion; (h) contrast‑enhanced magnetic resonance T1‑weighted imaging showed mild enhancement (arrow). In addition, there were punctiform hyperintense signals in the left ambient cistern (b‑c, arrows). Preliminary maximum intensity projection imaging (i) and enlarged maximum intensity projection image (j) showed that left basal vein was not exhibited (narrow arrows). The right basal vein was clearly exhibited in contrast‑enhanced magnetic resonance venography (bold arrow).