

# Pseudo-subarachnoid hemorrhage in a patient with acute cerebellar infarction

Vivek Misra\*, Romy Hoque\*, Eduardo Gonzalez-Toledo<sup>†</sup>, Roger E. Kelley\* and Alireza Minagar\*

\*Department of Neurology and <sup>†</sup>Department of Radiology, Louisiana State University Health Sciences Center at Shreveport, Shreveport, LA, USA

**Objective:** Hyperdensity in the basal cisterns on computed tomography (CT) brain scan is a diagnostic feature in subarachnoid hemorrhage. However, false enhancement of the subarachnoid space on CT scan may be seen in diffuse neurological conditions with acute presentation. This is the first reported case of a 'focal lesion' (acute cerebellar infarct) producing a 'pseudo-subarachnoid hemorrhage' secondary to increased intracranial pressure.

**Clinical presentation:** A 42-year-old man presented with acute onset of left hemiataxia and neck pain. CT brain scan showed a left cerebellar infarct. During the admission, he had sudden onset of altered sensorium, and an emergency CT scan showed diffuse cerebral edema and hyperdensity of the basal cisterns. The patient then developed sudden cardiac arrest and all resuscitative measures failed. Autopsy showed no evidence of subarachnoid hemorrhage.

**Conclusion:** Pseudo-subarachnoid hemorrhage is a rare clinical entity. It should be considered as a possibility in the evaluation and management of patients presenting acutely with CT brain scan showing hyperdensity of the basal cisterns and vascular imaging revealing absence of any aneurysm or vascular malformation. [Neurol Res 2008; 30: 813–815]

**Keywords:** Cerebellar infarction; pseudo-subarachnoid hemorrhage; Hounsfield units

## INTRODUCTION

Subarachnoid hemorrhage is diagnosed by the presence of hyperdensity in the subarachnoid space and basal cisterns on computed tomography (CT) brain scan. In fact, CT has 95–98% sensitivity in patients with subarachnoid hemorrhage presenting within 24 hours of onset<sup>1</sup>. However, other diffuse neurological conditions presenting with acute altered sensorium may rarely produce attenuation of basal cisterns on CT brain scan mimicking subarachnoid hemorrhage. We report a case of focal cerebellar ischemia causing diffuse cerebral edema and producing a 'pseudo-subarachnoid hemorrhage' appearance on CT.

## CASE PRESENTATION

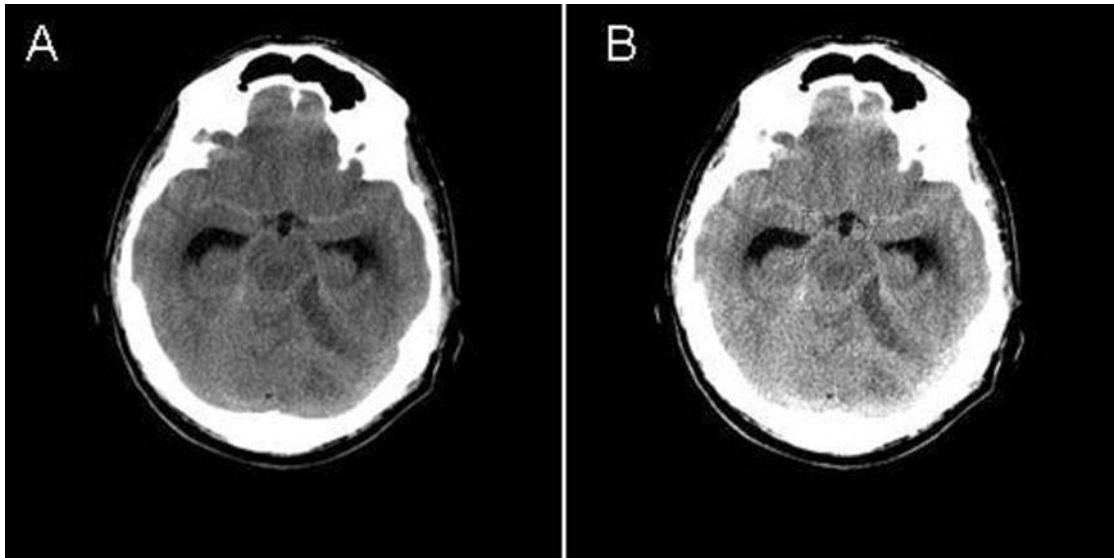
A 42-year-old man with a history of uncontrolled hypertension presented with acute onset of left hemiataxia and neck pain. The neurological examination revealed left finger-to-nose dysmetria and hemiataxia with no clinical evidence of weakness, numbness, cranial nerve involvement, neck rigidity or papilloedema. The CT head, on presentation, revealed hypodensity in the left cerebellar hemisphere consistent with acute ischemia (Figure 1A,B). Magnetic resonance imaging (MRI) of the head (Figure 2A–E) revealed a single

hyperintensity on diffusion-weighted imaging (DWI) of the left cerebellar hemisphere, which was also hypointense on apparent diffusion coefficient maps. Magnetic resonance angiogram (MRA; Figure 3A–C) revealed absence of any aneurysm or vascular abnormality. The next day, the patient had sudden deterioration in the level of consciousness and an emergent CT brain scan revealed diffuse cerebral edema and hyperdensity in the basal cisterns. Shortly after the CT scan, the patient developed sudden cardiac arrest and all resuscitative measures failed. Autopsy showed the presence of diffuse cerebral edema, left-sided cerebellar infarction and the absence of subarachnoid hemorrhage or any aneurysm or vascular malformation.

## DISCUSSION

Pseudo-subarachnoid hemorrhage is the rare appearance of hyperdensity on the CT scan in the absence of any blood in either the basal cisterns or the subarachnoid space. The absence of subarachnoid hemorrhage can be concluded in these patients on the basis of lumbar puncture findings or discovered at autopsy as in this case. Given *et al.*<sup>2</sup> described seven cases of pseudo-subarachnoid hemorrhage attributed to diffuse cerebral edema. The patients in their series had cerebral edema secondary to hyponatremia, anoxic injury, pseudo-tumor cerebri, septic shock, diabetic ketoacidosis, sudden infant death syndrome and an unknown metabolic disorder (possibly Leigh disease).

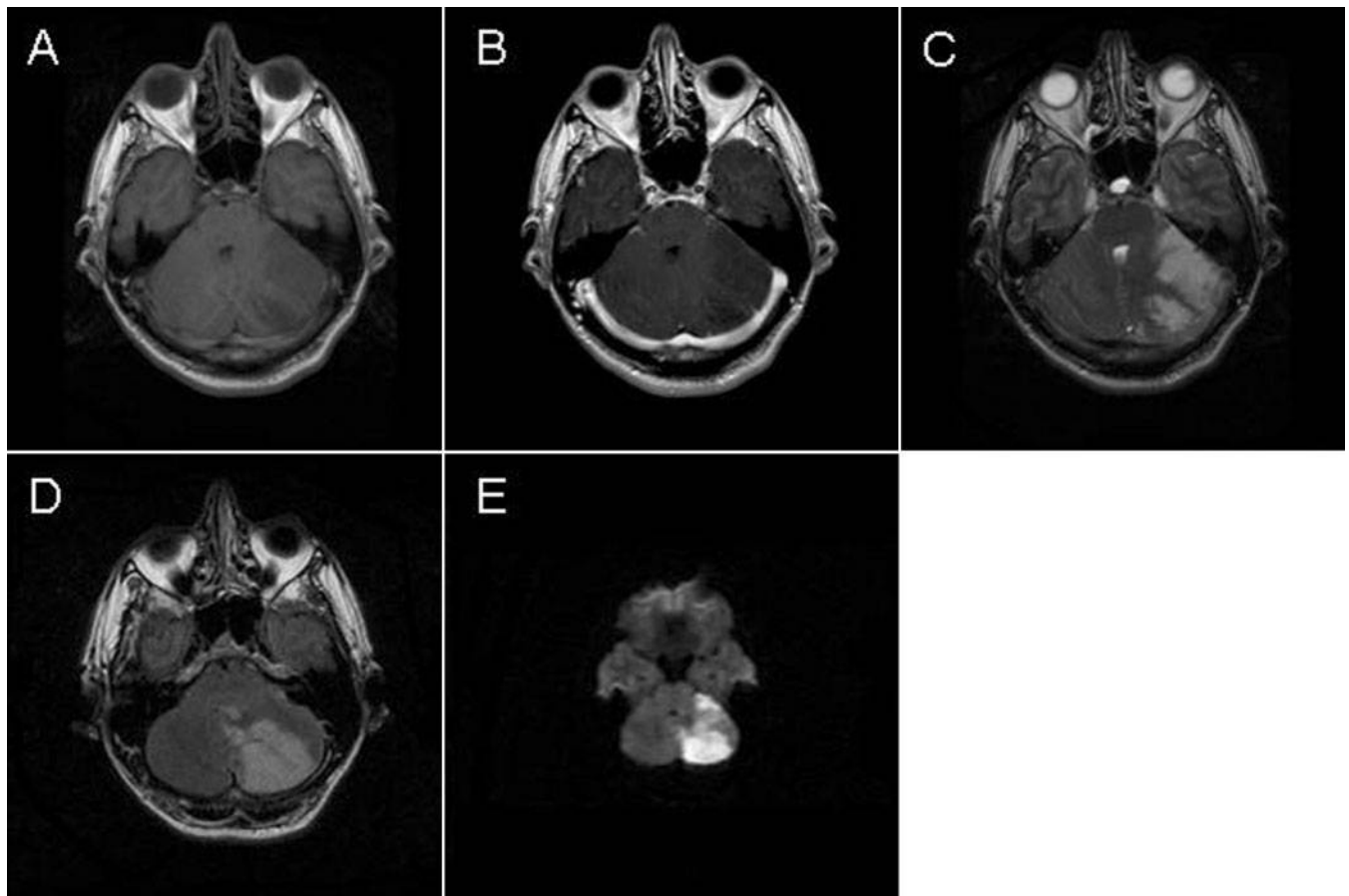
Correspondence and reprint requests to: A. Minagar, Department of Neurology, Louisiana State University Health Sciences Center at Shreveport, 1501 Kings Highway, Shreveport, LA 71130, USA. [aminag@lsuhsc.edu]



**Figure 1:** Non-contrast CT of the head showing (A) hyperattenuation of the basal cisterns with acute infarction in the left cerebellar hemisphere and (B) attenuation values in Hounsfield units

Anoxic ischemic encephalopathy has been the most common cause of 'pseudo-subarachnoid hemorrhage' reported so far<sup>2-5</sup>. Other reported causes also include

gliomatosis cerebri<sup>6</sup>, bilateral subdural hematomas<sup>7,8</sup>, secondary polycythemia<sup>9</sup>, subarachnoid space enhancement following administration of high-dose



**Figure 2:** Magnetic resonance imaging (MRI) T<sub>1</sub>-weighted (A) and T<sub>1</sub>-weighted with gadolinium (B) showing hypointensity consistent with infarction in the left cerebellar hemisphere. This also appears hyperintense on T<sub>2</sub>-weighted (C) and hyperintense on fluid attenuated inversion recovery (FLAIR) sequences (D). The lesion appears hyperintense on diffusion weighted imaging (DWI) consistent with acute left cerebellar ischemia (E)



**Figure 3:** Magnetic resonance angiograms (MRA) showing the left internal carotid (A), the right internal carotid (B) and the vertebrobasilar circulations (C). No vascular anomalies are seen

contrast material during spinal angiography<sup>10</sup>, acute purulent leptomeningitis<sup>11</sup>, idiopathic intracranial hypertension and viral meningoencephalitis<sup>12</sup>. All these conditions cause diffuse intracranial involvement resulting in diffuse cerebral edema and increased intracranial pressure.

Our patient presented with focal left cerebellar ischemic infarction. The focal cerebellar edema led to obliteration of the basal cisterns, as seen on CT brain and eventually resulted in diffuse cerebral edema. This is different from the previously reported cases with either diffuse cerebral insult or bilateral cerebral lesions at the onset. The proposed mechanism for 'pseudo-subarachnoid hemorrhage' in the clinical setting of diffuse cerebral edema is compression of the subarachnoid space by the expanding brain tissue resulting in displacement of the cerebrospinal fluid (CSF), which is hypodense on CT, and dilatation and engorgement of the pial veins. This, in turn, results in relative hyperdensity of the basal cisterns on CT<sup>2,12</sup>. Cerebral edema also results in decreased attenuation of the brain tissue, which contributes to the appearance of relative hyperdensity of the basal cisterns as seen on brain CT in contrast to the surrounding brain tissue<sup>2</sup>.

In our patient, the cytotoxic cerebellar edema resulted in compression of the ambient cistern with subsequent brain stem and cerebral edema. The resulting venous engorgement (hyperattenuation on CT) and surrounding edema (hypoattenuation) produced the false enhancement of the basal cisterns. However, the attenuation values in Hounsfield units (HU) in the basal cisterns in pseudo-subarachnoid hemorrhage is significantly lower (29–33 HU) compared with those in true subarachnoid hemorrhage (60–70 HU)<sup>2</sup>.

Pseudo-subarachnoid hemorrhage has also been reported in four patients with 'spontaneous intracranial hypotension' with subdural fluid collections. These patients also presented with headache. The brain CT findings were caused by obliteration of the basal cisterns due to brain sagging and resolved after successful treatment of the underlying CSF leak<sup>13</sup>.

Neurological conditions producing 'pseudo-subarachnoid hemorrhage' on CT may also mimic subarachnoid hemorrhage with their acute presentation.

Lumbar puncture must be considered, if no obvious contraindication, in patients presenting acutely with hyperdensity of basal cisterns on CT and with no evidence of aneurysm or vascular malformation on angiography. The absence of xanthochromia in the CSF of these patients should prompt clinical recognition and management of neurological conditions that may produce 'pseudo-subarachnoid hemorrhage' appearance on CT.

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