## Case Report

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## Pseudo-Subarachnoid Hemorrhage; Chronic Subdural Hematoma with an Unruptured Aneurysm Mistaken for Subarachnoid Hemorrhage

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Dookyung Son 🕞, Youngha Kim 💿, Changhyeun Kim, and Sangweon Lee

Department of Neurosurgery, Pusan National University Yangsan Hospital, Yangsan, Korea

## ABSTRACT

Subarachnoid hemorrhage (SAH) usually occurs due to aneurysmal rupture of intracranial arteries and its typical computed tomography (CT) findings are increased attenuation of cisterns and subarachnoid spaces. However, several CT findings mimicking SAH are feasible in diverse conditions. They are so-called as pseudo-SAH, and this report is a case of pseudo-SAH which is misdiagnosed as aneurysm rupture accompanied by bilateral chronic subdural hematoma (cSDH). A 42-year-old male with severe headache visited our institute. Non-contrast brain CT images showed increased attenuation on basal cistern, and cSDH on both fronto-temporo-parietal convexity with midline shifting. Trans-femoral cerebral angiography was done and we confirmed small aneurysm at right M1 portion of middle cerebral artery. Under diagnosis of SAH, we planned an operation in order to clip aneurysmal neck and remove cSDH. cSDH was removed as planned, however, there was no SAH and we also couldn't find the rupture point of aneurysm. Serial follow-up CT showed mild cumulative cSDH recurrence, but the patient was tolerant and had no neurologic deficit during hospitalization. We have checked the patient via out-patient department for 6 months, there are no significant changes in volume and density of cSDH and the patient also have no neurologic complications.

Keywords: Pseudo-subarachnoid hemorrhage; Subarachnoid hemorrhage; Chronic subdural hematoma; Elevated intracranial pressure; Cerebral edema

## **INTRODUCTION**

Subarachnoid hemorrhage (SAH) usually occurs due to aneurysmal rupture of intracranial arteries. In many cases, it may undergo to lethal course because of complications such as rebleeding, vasospasm, cerebral edema and malignant hydrocephalus. Hence rapid and precise diagnosis is necessary, and most of SAH can be diagnosed with non-contrast computed tomography (CT), additional cerebral CT angiography gives more accuracy making diagnosis. Increased attenuation of cerebral cisterns and subarachnoid spaces are distinguishing findings of SAH.<sup>6</sup> However, several radiologic findings mimicking SAH are feasible in diverse conditions, including diffuse cerebral edema, pseudo-tumor cerebri, hypoxic encephalopathy, meningitis, subdural hematoma (SDH) and so on.<sup>5,11,17</sup> In the literatures, these CT findings mimicking of SAH have been called pseudo-SAH.<sup>1,6</sup>

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Address for correspondence: Youngha Kim

Department of Neurosurgery, Pusan National University Yangsan Hospital, 20 Geumo-ro, Yangsan 50612, Korea. E-mail: twins728@hanmail.net

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#### ORCID iDs

Dookyung Son D https://orcid.org/0000-0003-0936-9317 Youngha Kim D https://orcid.org/0000-0003-0477-3169

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#### **Conflict of Interest**

The authors have no financial conflicts of interest.

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While SDH caused by aneurysmal rupture of intracranial arteries have been reported in many literatures, in cases of chronic subdural hematoma (cSDH) are rarely reported; to the best our knowledge, few case of bilateral cSDH mimicking SAH were reported in the literature. In the other hands, standard treatment of cSDH is burr hole trephination for simple drainage, and it have been reported that if craniotomy is performed there is a higher risk of recurrence of cSDH.<sup>19</sup>

We report a patient who was incorrectly diagnosed as SAH of unruptured aneurysm accompanied by bilateral cSDH.

## **CASE REPORT**

A 42-year-old male presented with severe headache for a week visited the emergency room. Past medical history revealed hypertension and gout. He took operation 9 years ago with mitral valve prolapse and was prescribed aspirin, but stopped on his own. Non-contrast brain CT images showed SAH on basal cistern and cSDH on right fronto-temporo-parietal convexity with midline shifting (**FIGURE 1**). There were brain magnetic resonance imaging (MRI) and magnetic resonance (MR) angiography images performed 3 months ago because of his headache, and incidental unruptured right M1 aneurysm was revealed (**FIGURE 2**).

We initially suspected SAH due to rupture of previously found incidental right M1 aneurysm, urgent cerebral angiography was done. We confirmed small aneurysm at right anterior temporal branch origin of middle cerebral artery M1 portion, and its depth was 2.5 mm and

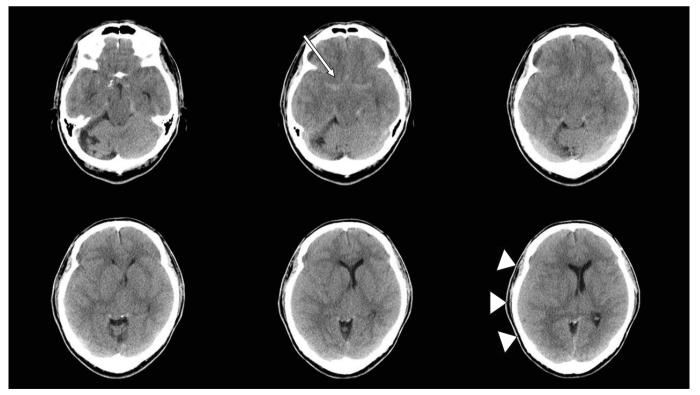
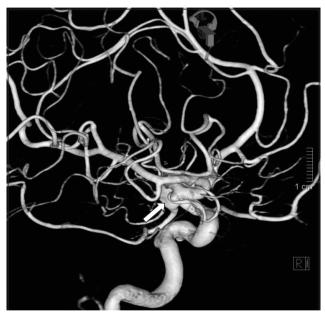


FIGURE 1. Initial non-contrast brain computed tomography findings showed increased attenuation at basal cistern (arrow) and fluid collection along the right fronto-parietal convexity (triangles) with midline shifting.





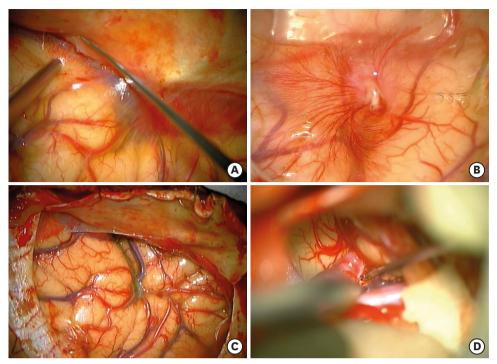
FIGURE 2. Previous magnetic resonance angiography showed unruptured aneurysm at right M1 (arrow).



**FIGURE 3.** Trans-femoral cerebral angiography was performed, and then small aneurysm at right anterior temporal branch origin of middle cerebral artery M1 portion was confirmed (arrow).

neck diameter was 2.3 mm (**FIGURE 3**). Under diagnosis of SAH, we performed ipsilateral craniotomy in order to remove cSDH and clip of aneurysmal neck. When the dura was opened, we found vascularized inner membrane of cSDH which looks like arachnoid adhesion and neovascularization of inner membrane of cSDH (**FIGURE 4A & B**). After resection of inner membrane of cSDH, normal arachnoid membrane was observed (**FIGURE 4C**). However, there was no SAH at the arachnoid space and we also couldn't find a rupture point of aneurysm (**FIGURE 4D**). Aneurysmal neck clipping was done as routine manner and no complication was occurred during the operation. Non-contrast brain CT was performed immediately after operation, and it was also done on 4th, 8th and 12th days after operation. Serial follow-up CT showed mild cumulative cSDH recurrence, but the patient was tolerant and had no neurologic deficit during hospitalization. He discharged on 14th day after operation. We had checked the

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**FIGURE 4.** (A) Vascularized inner membrane of cSDH looks like arachnoid adhesion. (B) Neovascularization of inner membrane of cSDH was also found. (C) After resection of inner membrane of cSDH, normal arachnoid membrane was observed. (D) No subarachnoid hemorrhage was observed during sylvian fissure dissection. cSDH: chronic subdural hematoma.

patient via our out-patient department for 6 months, the remnant cSDH was almost absorbed and the patient still had no neurologic complications.

## DISCUSSION

Pseudo-SAH has been reported in many literatures. This finding can be observed in various conditions including diffuse cerebral edema, pseudo-tumor cerebri, hypoxic encephalopathy, meningitis, infarction, SDH and intrathecally administered contrast material, spontaneous intracranial hypotension, after myelography and polycythemia.<sup>6-8,10</sup> Although the exact mechanisms remain unknown, cerebral edema might be related to pseudo-SAH. Increased volume in the supratentorial compartment compress the cerebral hemispheres and cause diffuse effacement of the cortical sulci, with a decrease in the size of cerebrospinal fluid (CSF) space.<sup>1,7</sup> The resultant subarachnoid space becomes relatively devoid of CSF and is instead filled with a larger fraction of meninges and blood vessels than in the normal state, and it may lead to increase attenuation of CT.<sup>1,7)</sup> In meningitis, it is assumed that there is a different mechanism. Toxins formed by invaded microbes break the blood-barin barrier allowing proteinaceous material to leak into the subarachnoid space, mixing with purulent material and leading to increased signal attenuation.<sup>2,3)</sup> In our case, the patient had bilateral cSDH and this disease entity might contribute to outbreak of increased intracranial pressure. Rabinstein et al.<sup>13)</sup> reported their experience of pseudo-SAH in bilateral subdural hematoma with 2 cases, authors also concluded that this disease entity produced effacement of sulci and basilar cisterns and these changes are able to result in increased density on CT by combining with the ensuring vascular congestion.

In our case, the patient was incidentally diagnosed having an unrupture aneurysm, and patient's symptoms are similar to typical SAH hence we planned pterional craniotomy for aneurysmal neck clipping. However, it is commonly accepted that open craniotomy treating cSDH has potentially risk of recurrence of cSDH. Yadav et al.<sup>19)</sup> summarized the indication of craniotomy for removal cSDH in their review article; significant membrane, multiloculated, organized and calcified or ossified cSDH candidate for craniotomy.<sup>4,9,14)</sup> The author also mentioned the reason why craniotomy often does not reach to successful result that it is hard to obtain brain re-expansion after craniotomy and the residual rigid inner and outer membranes facilitate dead space and hematoma recurrence. The patient was inevitably operated with craniotomy, and regrettably recurred cSDH. If the diagnosis was made precisely, it is assumed that he had not experienced recurrence.

Because of high spatial resolution and soft tissue contrast, MR image is superior to CT in the diagnosis of brain disease. To date, many investigators have reported that MR fluid attenuated inversion recovery (FLAIR) and susceptibility weighted imaging (SWI) are superior to CT in precisely detecting SAH. Noguchi et al.<sup>12)</sup> proved theoretically through equations that using MR FLAIR can subtly detect SAH in early phase. Verma et al.<sup>18)</sup> declared by combining SWI and FLAIR, MRI yield a distinctly higher detection rate for SAH than CT alone. Rabinstein et al.<sup>13)</sup> mentioned that proper MRI study can prevent unnecessary cerebral angiography. In our case, if additional MR scan was done in the emergency room, we might be able to get more information of patient and treat rationally.

In addition to MRI, methods and cautions for accurate diagnosis of pseudo-SAH have been mentioned by several authors. Generally, xanthochromic CSF is diagnostic clue of SAH but performing lumbar puncture in patients with pronounced mass effect from SDH may be potentially dangerous.<sup>8,13)</sup> Hasan et al.<sup>8)</sup> summarized the clinical and radiological characteristics of typical aneurysmal SAH and pseudo-SAH according to the specific disease situation and suggested distinguishing factors. They also emphasized reasonable judgment of causative relations by listening to detailed personnel medical history. There were attempts to identify pseudo-SAH using Hounsfield unit (HU). Given et al.<sup>7)</sup> reports that HU of SAH (60–70 HU) is greater than that of normal gray matter (30–40 HU), white matter (20–30 HU) and pseudo-SAH (29–33 HU). Senthilkumaran et al.<sup>15,16)</sup> argued that if the HU values were low, it would be likely to be pseudo-SAH and therefore more invasive diagnostic test should be obviated. In our case, the mean value of HU measured by irrelevant neurosurgeon at 10 locations in the basal cistern was 37.7. However, using the HU is not generally accepted at present, hence further precise verification of this subject should be required.

## CONCLUSION

The possibility of pseudo-SAH should not be overlooked even in patient with unruptured aneurysm. Neurosurgeons need to acquaint the clinical and radiological features of pseudo-SAH in order to make accurate diagnosis and obviate unnecessary invasive examination.

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