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# Hemiparesis Due to Subarachnoid Hemorrhage

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**Abstract:** An 85-year-old female was found in coma state in a toilet at a public spa area. Her Glasgow Coma Scale in total was 6 and she had isocoric reactive pupil. She had right hemiparesis. The CT examination demonstrated a subarachnoid hemorrhage. Especially subarahnoid hemorrhage at left silvian fissure was thicker than that of right, but no formation of hematoma. The CT angiography revealed a cerebral aneurysm at left internal carotid-posterior communicating artery junction. It was approximately 4 mm in size. She underwent an aneurysmal neck clipping. She complicated cerebral infarction at left putamen and internal capsule due to vasospasm, and right hemiparesis did not improve. Immediate transient vasospastic changes following a hemodynamic insufficient blood supply may have caused the hemiparesis in this case. Some patients with subarachnoid hemorrhage may demonstrate neurological deficits, like those occurring during an ischemic stroke or intracerebral hematoma, so physicians should pay attention to treat such patients gently to avoid the re-rupture of a cerebral aneurysm, especially if the patient has consciousness disturbance.
**Keywords:** subarachnoid hemorrhage; neurological deficit; focal sign.

## INTRODUCTION

The main symptoms of a ruptured cerebral aneurysm are a sudden onset headache or unconsciousness after headache, without any neurological deficit. Approximately one-third of patients with a ruptured cerebral aneurysm demonstrate a neurological deficit based on complications associated with an intracerebral hematoma or localization of subarachnoid hematoma [1]. We herein report a case of a hemiparesis due to the subarachnoid hemorrhage by a ruptured cerebral aneurysm in acute phase and discuss the mechanism underlying this phenomenon.

## CASE PRESENTATION

An 85-year-old female was found in coma state in a toilet at a public spa area. She had no specific past or family history. She was transported by physician-staffed emergency helicopter. On arrival, she remained unconscious. Her Glasgow Coma Scale in total was 6 and she had isocoric reactive pupil. Her vital signs were: blood pressure, 120/86 mmHg; heart rate, 70 rate/min and percutaneous oxygen saturation on 100% under 10L/minute of oxygen delivered via a mask. She had right hemiparesis. The CT examination, which demonstrated a subarachnoid hemorrhage. Especially subarahnoid hemorrhage at left silvian fissure was thicker than that of right, but no formation of hematoma (Figure 1).

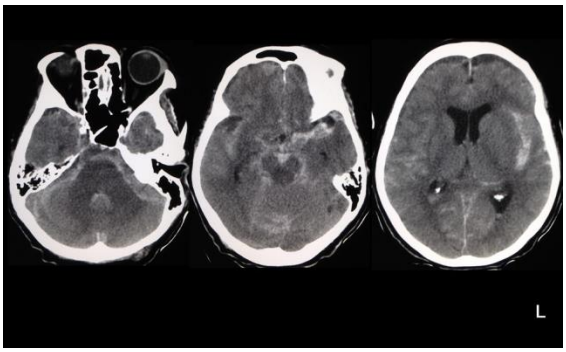


**Fig-1: Head CT on arrival**

The head CT on arrival demonstrates diffuse subarachnoid hemorrhage. Subarachnoid hemorrhage at left silvian fissure is thicker than that of left, but not formation of hematoma.

The CT angiography revealed a cerebral aneurysm at left internal carotid-posterior communicating artery junction (Figure 2). It was approximately 4 mm in size. She immediately underwent tracheal intubation to secure her airway after being given a sedative and muscle relaxant. A biochemical blood examination revealed no specific changes. On the same day, she underwent an aneurysmal neck clipping and extra-ventricular drainage. She complicated cerebral infarction at left putamen and internal capsule, and right hemiparesis did not improve. The patient was transferred to a nursing home on the 45th hospital day with a sequela of right hemiparesis and persistent disturbance of consciousness.

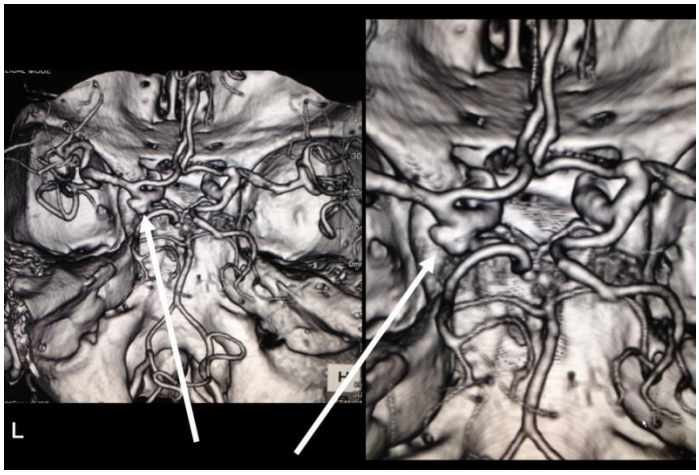


**Fig-2: Computed tomographic angiogram on the 2nd day**

The computed tomographic angiogram demonstrates cerebral aneurysm at left internal carotid-posterior communicating artery junction (arrow).

## DISCUSSION

The present case demonstrated coma state and immediate right hemiparesis due to rupture of cerebral aneurysm at right internal carotid-posterior communicating artery junction without formation of hematoma. The one possible mechanism causing the neurological deficit was subsequent distal embolization or parent vessel occlusion due to local extension from an intrasaccular aneurysmal thrombosis [2]. The factors associated with aneurysmal thrombosis are the size, and particularly, the ratio of the chamber volume to the orifice area, blood stagnation, slow flow and increased blood viscosity [2]. The aneurysmal size of the present case was less than 10 mm and the neck was narrow, so this condition was considered to be unlikely. Another possible mechanism was ischemia based on hemodynamic insufficiency due to vasospasm induced by a subarachnoid hemorrhage [3]. In an animal study, the cerebral artery can exhibit spastic changes induced by subarachnoid hemorrhage immediately after aneurysmal rupture during the subacute phase. Immediate transient vasospastic changes following a hemodynamic insufficient blood supply, which was rapidly resolved by hypertension, may have caused the hemiparesis in this case. Unfortunately, the present case complicated cerebral infarction and hemiparesis did not improve. Previous clinical report demonstrated that transient hemiparesis due to a ruptured middle cerebral aneurysm in the prehospital setting [4].

Some patients with subarachnoid hemorrhage may demonstrate neurological deficits, like those occurring during an ischemic stroke or intracerebral hematoma, so emergency medical technicians and physicians should pay attention to treat such patients gently to avoid the re-rupture of a cerebral aneurysm, especially if the patient has consciousness disturbance.

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