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Atrial Fibrillation After Spontaneous Intracerebral Hemorrhage

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ABSTRACT

Introduction: Cardiac arrhythmias often occur during the acute phase of a stroke and can cause hemodynamic instability and sudden cardiac death. Data regarding determinants and the course of arrhythmia onset in the acute phase of stroke are still rare, especially in hemorrhagic stroke.
Case report: A male, 74 years old, came to the emergency department with a complaint of weakness in the left hand and leg that had occurred suddenly 5 hours previously. Weakness is described by the left arm and leg still being able to be lifted a few seconds later and falling again. This complaint was accompanied by lips that appear pursed and severe dysarthria. The patient was said to have had a history of uncontrolled hypertension for 10 years. A previous history of heart disease was denied. A CT scan of the head without contrast showed intracerebral hemorrhage in the basal ganglia and right external capsule with a volume of 5 cc. An electrocardiography (ECG) examination showed sinus rhythm without any indication of heart rhythm disturbances. Further ECG examination after the 4th day of hospitalization showed atrial fibrillation with rapid ventricular response (AFRVR). The patient eventually died one day after the heart rhythm changed.
Discussion: The incidence of AF varies depending on the type of stroke that occurred, the monitoring device used, the interval between initial monitoring and stroke onset, and the duration of cardiac monitoring. AF in patients can occur as a result of a hemorrhagic stroke, but it is also possible because of the presence of previously undetected paroxysmal AF. High NIHSS correlates with system disturbances in autonomous cardiovascular systems. According to current NIHSS measurements, hospital admission is required as a parameter for heart rhythm monitoring for more than 24 hours.
Conclusion: Newly detected AF after ICH greatly influences the clinical outcome of patients. Understanding the mechanism and pathophysiology of new AF after ICH will contribute to a better understanding of the management of the patient.

Keywords: atrial fibrillation, cardiac arrhythmia, case report, stroke, intracerebral hemorrhage

INTRODUCTION

Cardiac arrhythmias often occur during the acute phase of a stroke and can cause instability in hemodynamics and sudden cardiac death. Apart from arrhythmia as a consequence of cardiac comorbidities, interactions in neuro-cardiology and dysfunctional autonomy can also complicate the course of cerebrovascular disorders. Various types of ECG abnormalities and cardiac arrhythmias can occur after an ischemic stroke or hemorrhagic stroke. This pattern of ECG abnormalities varies from abnormal T waves and QT prolongation to

some serious cardiac arrhythmias, such as atrial fibrillation (AF).[1]

Previous research has reported that the frequency of newly detected AF was significantly higher in patients with stroke compared to those without stroke. AF can be found in patients with acute stroke, though there is no proof of previous cardiac arrhythmia. Until this moment, it is known that patients suffering from acute stroke with AF had worse clinical outcomes compared with those without AF. Despite this, the current guidelines for the implementation of strokes with AF are still unable to provide a practical explanation of how long and important cardiac monitoring should be. This happens because data regarding determinants and the course of arrhythmia onset in the acute phase of stroke are still rare, especially in hemorrhagic stroke.[1,2]

CASE REPORT

A man, 74 years old, came to the emergency room (ER) with a complaint of weakness in the left hand and leg, which occurred suddenly 5 hours after eating. Weakness is described by the left arm and leg still being able to be lifted, but a few seconds later they fall again. This complaint was accompanied by lips that appeared pursed and severe dysarthria, so the patient's words were difficult to understand. These complaints persisted until the patient arrived at the hospital. History of headaches, blurred vision, seizures, nausea, vomiting, and fever were denied by the patient and family. The patient had a history of ischemic stroke twice, in 2012 (10 years ago) and 2017 (5 years ago), with weakness on the left side of the body. Although he has experienced two stroke episodes, the patient was said to still be able to carry out daily activities as usual with minimal symptoms. The patient was known to not have regular follow-up after experiencing a second stroke, so he did not get antiplatelet therapy. The patient was said to have had uncontrolled hypertension for 10 years with irregular amlodipine treatment. The patient never experienced symptoms of heart disease such as chest palpitations, chest pain, shortness of breath, and swelling of the extremities.

On physical examination, the patient was aware with the Glasgow Coma Scale (GCS) 15 with a blood pressure of 160/100 mmHg, pulse rate of 90 times per minute, a respiratory rate of 18 times per minute, and a temperature of 36.5 °C. Neurological examination showed NIHSS 8 with right grade 3 flaccid hemiparesis in the left upper and left lower extremities. Babinski reflex was found in the left extremity. On neurological examination of the cranial nerves, supranuclear paresis of the left VII and XII nerves was found. No meningeal signs were found. The patient's language function, orientation, memory, emotions, and cognition are not impaired. Laboratory examinations, including complete blood count, blood sugar, lipid profile, uric acid, liver function, kidney function, and electrolytes, remained within normal limits.

When arriving at the ER, the patient had an ECG examination, and the results showed no heart rhythm disturbances. A chest x-ray examination revealed cardiomegaly with increased pulmonary vascularity and aortosclerosis. A head CT scan without contrast shows intracerebral hemorrhage in the basal ganglia and right external capsule, with a volume of 5 cc (Figure 1).

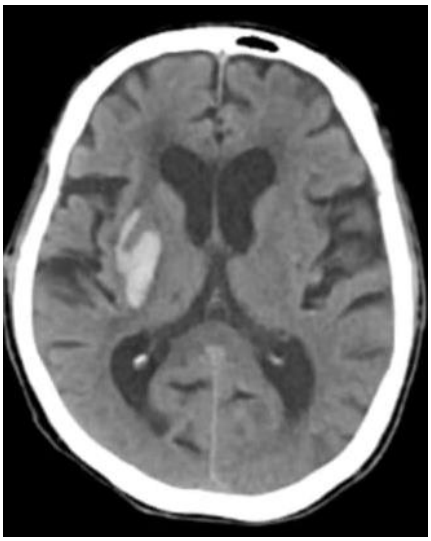
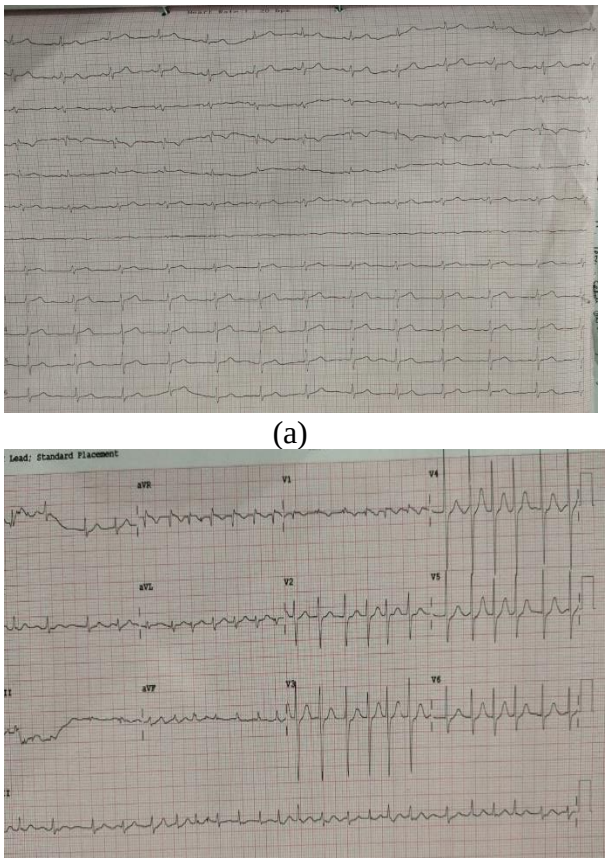


Figure 1. Head CT scan without contrast with intracerebral hemorrhage in the basal ganglia and right external capsule.

The patient was hospitalized in the high-care unit for close monitoring of vital signs and heart rhythm. During treatment, his systolic blood pressure rose to 200 mmHg, and he was given amlodipine 10 mg and bisoprolol 5 mg once a day. The patient also got intracranial pressure-lowering therapy using mannitol.



(a)

(b)

Figure 2. (a) Initial ECG at hospital admission and (b) ECG after 4 days of hospitalization.

On the fourth day of treatment, there was an increase in pulse to 150 times per minute with changes in irregular heart rhythm. ECG recording shows atrial fibrillation with rapid ventricular response (AFRVR). The patient was then given digoxin therapy to control the heart rhythm. After digoxin therapy, the patient's pulse rate appears to be back to normal but still irregular. The patient's consciousness decreased with GCS E2V2M4. A repeated head CT was carried out, which showed no significant changes compared with the previous results. The patient then experienced a seizure with a clonic pattern throughout the body, which was stopped after a diazepam injection. After the seizure stopped, the patient's consciousness returned to GCS E2V2M4. The patient's blood pressure then continued to decline, and he eventually died one day later.

DISCUSSION

Cardiac arrhythmia is a frequent and potential complication for worse clinical outcomes in patients with acute cerebrovascular disease. Sinus tachycardia, *isolated premature ventricular*, and AF are types of significant cardiac arrhythmia that often appear in patients with acute stroke. The incidence of AF varies depending on the type of stroke that occurred, the monitoring device used, the interval between initial monitoring and stroke onset, and the duration of cardiac monitoring. More than 70% of new AF cases are discovered within 3 days after the patient is hospitalized.[3,4] In a large-scale epidemiology study from 2006 to 2017, it was found that 1 in 6 patients with intracerebral hemorrhage has AF. Although part-time patients with ICH have been known to have AF previously, the proportion of new AF after ICH increased by 2-fold, namely 16.1%, 8.6%, and 16.1%, with an average duration of 2–3 days.[5]

In this case, the patient had a hemorrhagic stroke with newly detected AF after 4 days of treatment in the hospital. AF that occurs in the patient can occur as a result of a hemorrhagic stroke, but it is also possible because of the presence of paroxysmal AF, which has not been detected until hospital admission. Patients with acute strokes may experience an imbalance in the autonomic nervous system. Excessive sympathetic activation is the main pathophysiological mechanism of AF. The imbalanced autonomic nervous system can change the subsequent structure of the atria and heart electricity which will further increase the risk of AF occurrence.[6]

The location of the acute stroke lesion also plays a role in the occurrence of AF, especially if the lesion is located on the

insula. The insula and sub-insula cortex which are vascularized by medial cerebral arteries have a function that is important in regulating the autonomic nervous system. The role of damage to the insula cortex significantly carries a 7-fold risk of the occurrence of AF after stroke.[7] Apart from the insula cortex, other parts of the cerebrum, such as the anterior cingulate cortex, amygdala, hypothalamus, and *periaqueductal gray matter*, are also involved in regulating central system autonomy and modulating heart function.[8]

Ischemic and hemorrhage stroke can also cause damage to the heart muscle, though without previous history of coronary heart disease. As many as 34% of patients with acute stroke can experience increased cardiac enzymes in plasma, which is significantly associated with the occurrence of AF after stroke.[9] *Catecholamine surge* is the reason for damage to heart muscle in patients with acute stroke. In patients with acute stroke, discharge occurs with substances such as interleukin (IL)-1, TNF-a, and IL-6, which stimulate the hypothalamic paraventricular nucleus to synthesize *corticotropin-releasing factor* (CRF). Increased CRF increases sympathetic activity and ultimately increases catecholamine neurotransmitters.[10] Previous research found that arrhythmia more often occurs in patients who have preexisting cardiac comorbidities and vascular risk factors such as older age, hypertension, and diabetes mellitus. The severity degree of neurological deficits with NIHSS scores of more than 5 is also a significant predictor of arrhythmia. High NIHSS correlates with cardiovascular autonomic system disturbances, such as loss of autonomic modulation, decreased parasympathetic activity with hyperactivity of sympathetic, and impaired baroreflex sensitivity. According to current NIHSS measurements, hospital admission is required as a parameter for heart rhythm monitoring for more than 24 hours. ECG monitoring for 72 hours or more after a stroke may increase the detection of undiagnosed AF, as in paroxysmal AF.[2,11,12] Patients with ICH and AF have worse functional and vital clinical outcomes compared to those without AF. These results persist even after adjustment for age, type, gender, ICH location, vascular risk factors, and comorbidities. Premorbid use of anticoagulants is also a risk factor in the study on poor clinical outcomes in ICH patients with AF. A large-scale multinational observational study suggested that initiation of anticoagulant should be given 7-8 weeks after ICH to maximize profit and minimize the risk of bleeding.[5]

CONCLUSION

Newly detected AF after ICH has a major impact on patient's clinical outcomes. Understanding the mechanism and pathophysiology of new AF after ICH will contribute greatly to the management of the patient, such as choosing the appropriate cardiac monitoring strategy and timing of oral anticoagulant initiation for secondary prevention of stroke.

Declaration by Authors

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