

RETICULAR BRAINSTEM MYOCLONUS AS A SYMPTOM POST-CARDIAC ARREST: A CASE REPORT

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ABSTRACT Background: Improvements in cardiac arrest cannot reduce mortality and disability. Neurological disability or sequel that occurs can be in the form of cognitive and motor disorders. **Report:** A 66-year-old man with congested complaints diagnosed Acute Decompensated Heart Failure (ADHF) type C and intubated pneumonia due to respiratory failure. Two days of treatment, the patient experienced Atrial Fibrillation (AF) without a pulse, Ventricle Tachycardia (VT) / Ventricle Fibrillation (VF) asystole then Return of Spontaneous Circulation (ROSC). Post cardiac arrest of patients with Glasgow Coma Scale (GCS) 6 coma awareness accompanied by jerking movements several times, especially when touched in the distal extremity. **Discussion:** Patients with brief systemic circulation stops may experience cerebral ischemia-anoxia. Disorders that occur in the early phase after cardiac arrest is a decrease in severe coma. Anoxic conditions can cause myoclonus. Myoclonus due to lesions in the brain stem includes exaggerated startle, reticular reflex myoclonus and palatal myoclonus or tremor. **Conclusion:** Reticular brainstem myoclonus as a neurological manifestation of post-cardiac arrest due to anoxia.

KEYWORDS myoclonus, brainstem, anoxia, cardiac arrest

Introduction

Cardiac arrest (sudden cardiac arrest) is a condition in which blood flow stops suddenly due to acute failure of the heart to pump blood effectively [1]. There is no exact data on the number of cardiac arrest events in the world. However, data from various countries can provide an overview of the epidemiology.

In the United States, the rate of cardiac arrest reaches more than 100 events per 100,000 population each year [2]. Meanwhile, in Southeast Asia, a region that includes Indonesia, the incidence rate is lower at around 20 events per 100,000 population [3]. However, this number is estimated to be too little due

to the number of cardiac arrest events that unhandled that it is unrecorded.

Improvements that occur in the management of cardiac arrest can not reduce mortality and disability. Although the initial treatment of patients reaches the return of spontaneous circulation (ROSC), most patients still fall into a coma. Of these, more than half never regained consciousness and died [4].

Some patients can regain consciousness, but sufferers are also at high risk of suffering from neurological sequelae. These neurological sequelae can be either cognitive or motor deficit. In the United States, it estimated that there are 12,000 survivors of cardiac arrest with persistent neurological deficits each year with a total prevalence of 50,000 patients [5].

The high mortality and disability after a cardiac arrest is hypoxic heart injury (hypoxic brain injury) — failure to circulate during cardiac arrest results in the inability of brain perfusion. The brain is susceptible to perfusion failure, and the condition of perfusion failure in a short time can cause permanent and semi-permanent damage to the structure and function of the brain [6].

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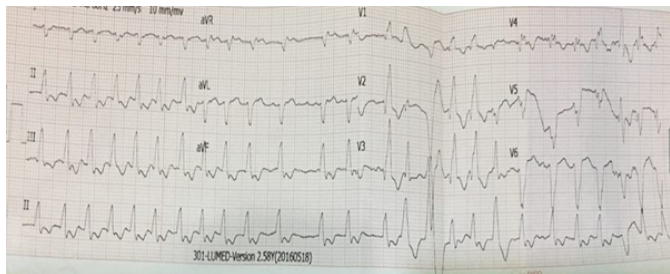


Figure 1: Electrocardiogram pre-hospital. Atrial rhythm, axis RAD, rate 140 times/minutes, P and PR can't be evaluated, QRS wave 120 msec, SV 2 + RV5 <3.5 mV, R / S ratio V1 > 1, PVC (+) couplet, polymorphic. Conclusion: Atrial Fibrillation Rapid Ventricular Response 140 x / min, RBBB, PVC.

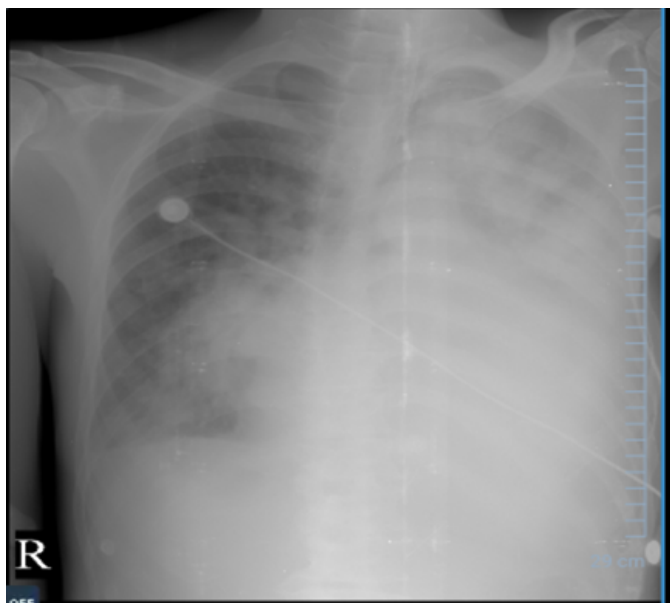


Figure 2: Chest X-ray shows pneumonia with bilateral pleural effusion.

Case report

A 66-year-old male patient comes to the Emergency Department with shortness of breath. The patient complained of shortness of breath since five days of SMRS, weighing since one day of SMRS (before hospitalization). Shortness improves with position changes. Cough and fever for three days of SMRS. History of high blood pressure (-), history of diabetes (-), kidney disease (-), stroke (-) history of heart disease with heart block disorders since 2010. History of heart disease, high blood pressure and diabetes in the family denied. The patient does not smoke and does not consume alcohol. Patient compos mentis with blood pressure 120/72 mmHg (within dobutamine), pulse 138 times/mins irregular with respiratory rate and temperature were normal limits. Oxygen saturation of 88% with hood 10 litres/ minute. JVP: PR \pm 4 cm H₂O. A thoracic examination found Cor: S1 & S2 irregular, murmur (+) systolic blowing grade III / VI in ICS IV PSL Sinistra. Pulmo with vesicular breath accompanied by crackles in the lower right and left lungs. Blood exam shows respiratory acidosis. Chest X-ray shows the presence of pneumonia and bilateral pleural effusion. Patients diagnosed with ADHF profile C and pneumonia with respiratory failure. Mechanical ventilation is done to overcome respiratory failure that

occurs. Two days of treatment in patients with cardiac arrest then hemodynamic conditions return (ROSC). The patient complained of experiencing movements in the limbs suspected of seizures. The actions that arise in the form of jerking movements, especially in the upper arm when the patient touched on the hands, chest, legs but movements do not occur when rubbed on the shoulders and pelvis. The action lasts for \pm 1-2 minutes. This movement takes place in 1 day. The eyes closed, no mouth bubbly, no bedwetting. Patients coma with only gag reflexes, no motoric lateralization, accompanied by myoclonus. The patient was not subjected to electroencephalography (EEG) and electroneuromyography (ENMG). For therapy: Furosemide, warfarin, Meropenem, Amikacin, Methylprednisolone, Nebuliser Combivent, Clonazepam. The patient died ten days later due to multi-organ failure due to sepsis.

Discussion

Case patients experience cardiac arrest and ROSC after receiving resuscitation. Cardiac arrest is a cause of metabolic and structural damage to the central nervous system. Patients with short systemic circulation stops may experience mild cerebral ischemia-anoxia representing a reversible metabolic encephalopathy. Coma after ROSC lasts for several hours when a coma occurs. Patients with severe ischemia-anoxia have structural damage to specific areas of the brain.

Cerebral anoxia is a broad term relating to brain disorders in which the condition of blood flow to the brain is decreased or impaired. Ischemic anoxia describes the situation of the vascular supply of the brain experiencing disorders such as after suffering cardiac arrest [7]. Based on this term, the patient with hypoxic encephalopathy caused by anoxic ischemia that occurs after cardiac arrest.

An area with susceptible damage due to anoxia is cortical, hippocampus, cerebellum, and basal ganglia [7]. Areas of the brain have different sensitivity to anoxic conditions. This sensitivity depends on the metabolic activity, the size of the perfusion field and the density of neurons. Areas of the brain with very high metabolic demand, very high perfusion in neurons with high density will tend to suffer damage due to anoxia [7]. Patients in the case showed symptoms of decreased consciousness, accompanied by myoclonus movements after cardiac arrest. The disorder that occurs most often in the early phase after cardiac arrest is a decrease in a severe coma. Severe motor deficits can also occur in patients with multiple severe cognitive impairments after experiencing an improvement in coma conditions. Patients can experience permanent vegetative state in severe anoxic cases [7].

Myoclonus as rapid muscle movement. The movements that arise in the form of brief shock-like involuntary movements due to muscle contractions (positive myoclonus) or interruptions of short contractions of active muscles (negative myoclonus). Myoclonus can be classified based on clinical features, pathophysiology or underlying causes. Based on the clinical picture and supported electrodiagnostic examination can accurately determine the location of the lesions that occur. Myoclonus can occur due to injuries in the cortex, brain stem, spinal cord, and rarely the peripheral nerves. Myoclonus due to damages in the brain stem includes exaggerated startle, reticular reflex myoclonus and palatal tremor [7]. A case report by Beudel et al. is the first case that can clearly describe the anatomy, and an anomaly called the dolichovertebral artery that can trigger myoclonic movements [2].

In some cases, the brain stem reflexes are found to be negative except that the vomiting reflex. Its indicates medulla oblongata is still reasonable. Hyperactivity situation so that it can trigger myoclonus.

Reticular reflex myoclonus is different from startle syndrome based on several clinical features. Both of these myoclonus movements occur with sensory stimulation, but auditory stimulation is effortless to give rise to startle syndrome. Patients with very sensitive hyperreflexia appear myoclonus movements when touched on the mantle area (head, upper chest and back). Reticular reflex myoclonus is very sensitive arising when a touch on the distal extremity. The existence of uncontrol jerking induced burst on electromyography (EMG). Duration 10-30 ms supports the diagnosis of reticular reflex myoclonus, but if the period is more than 75 ms indicates the occurrence of startle syndrome [8, 9]. The patient shows the possibility of reticular reflex myoclonus based on clinical features of jerking movements [10]. Jerking arises when a distal touch on the hands and feet. ENMG examination undid because the patient's condition is not stable, and the ENMG device is available cannot be moved. Case patients receive clonazepam therapy to treat myoclonus movements that occur in addition to managing their cardiac arrest. Benzodiazepines have beneficial therapeutic effects, especially clonazepam in myoclonus jerking [11,12].

Conclusion

Reticular myoclonus brainstem as a neurological manifestation in post-cardiac arrest patients due to cerebral anoxic ischemia that occurs.

Disclosure Statement

There were no financial support or relationships between the authors and any organization or professional bodies that could pose any conflict of interests.

Competing Interests

Written informed consent obtained from the patient for publication of this case report and any accompanying images.

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