Case Report
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Status Epilepticus Due to Basıllar Artery Occlusion: Case Report

Seyma Nur CALISIR[1], Oğuzhan ABANOZ[2], Basar CANDER[1]
[1]Department of Emergency Medicine, Bezmialem Vakıf University, Istanbul, Turkey
[2]Department of Cardiology Bezmialem Vakıf University, Istanbul, Turkey

Abstract

We present this case to highlight the rapid and effective multidisciplinary intervention involving emergency medicine, neurology, cardiology, and radiology departments for a young patient initially brought to the emergency department with suspected head trauma, who was later diagnosed with status epilepticus. A 42-year-old male patient experienced syncope on the street, followed by a generalized tonic-clonic seizure lasting about one to two minutes. Bystanders called an ambulance. The patient had a known diagnosis of atrial fibrillation and had a pacemaker implanted three years ago. Brain CTA showed embolic occlusion extending from the proximal to the apex of the basilar artery. With a preliminary diagnosis of status epilepticus, neurology and anesthesia clinics were consulted. Due to basilar artery occlusion, interventional radiology was contacted, and thrombectomy was planned. The patient was transferred to the neurology ward with a GCS of 15 and no neurological sequelae. Basilar artery occlusion (BAO) accounts for about 10% of large vessel occlusion (LVO) strokes. It is associated with higher morbidity and mortality compared to anterior circulation strokes, with a mortality rate of ≥40% Early diagnosis is lifesaving due to the ambiguous course of symptoms and high mortality. The goal is to reduce mortality with the help of an experienced neuroradiologist and comprehensive diagnostic and therapeutic approaches.

Introduction

Basilar artery occlusion (BAO) ranges from mild transient symptoms to highly fatal and morbid devastating strokes (1). Non-specific prodromal symptoms such as dizziness or headache may indicate BAO, followed by distinguishing features of BAO including alterations in consciousness, quadriparesis, pupillary and oculomotor abnormalities, dysarthria, and dysphagia (1,2,3). When encountering a patient exhibiting brainstem dysfunction, BAO must be rapidly confirmed or excluded. Early recognition of BAO allows for intravenous thrombolysis or endovascular treatment (4).

Case

A 42-year-old male patient experienced syncope in the street, followed by a generalized tonic-clonic seizure lasting approximately one to two minutes. Bystanders called an ambulance. Approximately ten minutes later, the patient had another seizure, prompting the paramedic team to administer 5 mg of midazolam, which was repeated with the same dose due to the continuation of the seizure. The patient was brought to the emergency department with active epileptic seizures. He was placed in the red area within a safety perimeter, his airway was secured, and he was transported by ambulance. Eight milligrams of diazepam were administered, and the dose was repeated due to ongoing seizures. A loading dose of 2 g levetiracetam was given. The patient, presenting with status epilepticus, was intubated in a controlled manner to secure the airway. Upon arrival, his Glasgow Coma Scale (GCS) was assessed as 4 (E1V1M2). He was not cooperative in orientation. Pupillary reflexes were absent on the right side. Both arms were in bilateral extensor positions, and the Babinski sign was positive on the right. The patient had a known diagnosis of atrial fibrillation. Three years prior, a pacemaker was implanted, and twenty-seven years ago, he underwent surgery for an atrial septal defect. His medications included metoprolol and rivaroxaban. There was no history of alcohol or substance use, although he was a smoker. Initial venous blood gas analysis showed pH 7.19, HCO_3^- 20 mmol/L, and lactate 9 mmol/L. The electrocardiogram (EKG) revealed left bundle branch block (LBBB) (Figure 1). After cessation of the seizure, a brain computed tomography and angiography (CT/CTA) were performed due to a history of head trauma and to evaluate the etiology of the seizure and potential bleeding. The brain CTA showed an embolic occlusion extending from the proximal part of the basilar artery to the top segment.With a preliminary diagnosis of status epilepticus, consultations were made with the

Corresponding Author: Şeyma Nur CALISIR **e-mail:** seymacalisirr@gmail.com
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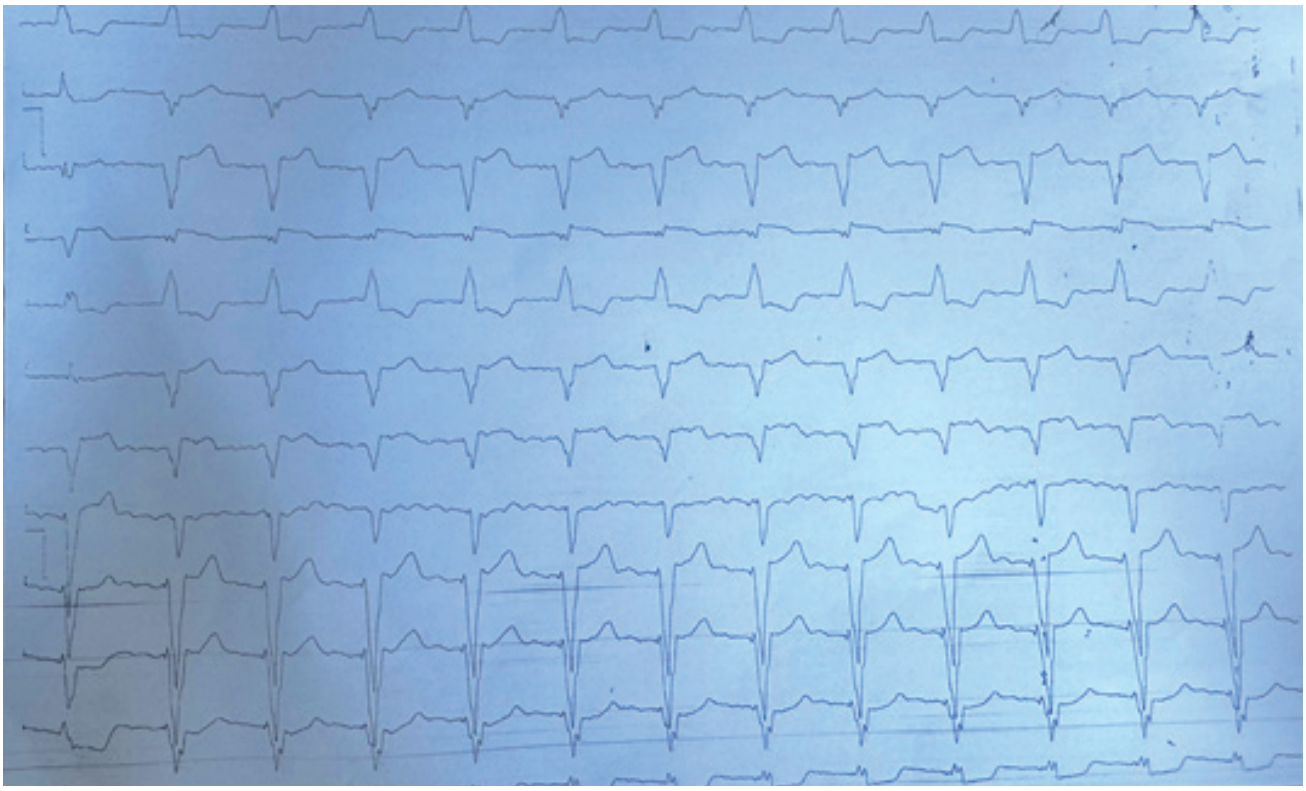

Figure 1: Routine blood tests performed in the emergency department did not reveal pathological conditions.

neurology and anesthesiology departments. Due to the basilar artery occlusion, interventional radiology was contacted, and thrombectomy was planned. Thrombectomy was performed within four hours of presentation, and the patient was monitored in the intensive care unit. A young stroke panel was implemented. Blood tests revealed a homocysteine level of 21 µmol/L. Bedside echocardiography showed an ejection fraction (EF) of 60%, left atrial size of 48 mm, right atrial size of 38 mm, left ventricular dimensions of 45/26 mm, pulmonary artery pressure (PAP) of 30 mmHg, and mild pulmonary insufficiency. Velosity was 0.7 m/s, and the inferior vena cava (IVC) measured 17 mm. No mass vegetations were observed. Transesophageal echocardiography revealed bilateral atrial dilation. In the left atrial appendage, grade 2-3 spontaneous echo contrast (SEC) was observed. On the second day of intensive care follow-up, the patient was transferred to the neurology service without any neurological sequelae (GCS 15). After three days of monitoring in the neurology service, the patient was discharged with prescriptions and recommendations. At the three-month follow-up, the patient reported no active complaints.

Conclusion and Discussion

Basilar artery occlusion (BAO) accounts for approximately 10% of large vessel occlusion (LVO) strokes. Compared to anterior circulation strokes, BAO is associated with higher morbidity and mortality, with a mortality rate of ≥40% (1). Due to the often ambiguous presentation of symptoms and high mortality rate, early diagnosis is life-saving. The involvement of an experienced neurointerventionalist and a comprehensive diagnostic and therapeutic approach aim to reduce mortality.

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