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A DEADLY ELECTROCARDIOGRAPHY SHARK FIN SIGN IN ST ELEVATION MYOCARDIAL INFARCTION: A CASE REPORT

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ABSTRACT

Shark fin ECG pattern, also known as 'giant R waves', or 'triangular QRS-ST-T waveform' is a high-risk ECG pattern associated with ST-elevation myocardial infarction (STEMI). Shark fin ECG is typically accompanied by occlusion of the left main coronary artery and the left anterior descending artery, and it is considered to be related to a high risk of death through cardiac arrest and cardiogenic shock. Objective: This case report aims to present a case study of a patient with acute coronary syndrome st-elevation myocardial infarction with a shark fin ECG pattern. Methods: The method used is a descriptive case report design conducted on one subject. Data were collected by conducting interviews, assessments, and therapy for one patient with a shark fin ECG sign in ST-Elevation myocardial infarction. Data analysis compared the results with the theory and previous study. Results: The result of this study indicated that Mr. X aged 44 years, was in cardiac arrest and unstable hemodynamic, and experienced a return of spontaneous circulation (ROSC) after he was given 45 minutes of cardiopulmonary resuscitation (CPR), and the electrocardiography result showed shark fin sign and extensive anterior st elevation, the patient then underwent primary percutaneous intervention and the coronary catheterization showed total occlusion in left anterior descending artery (LAD), proximal stenosis 20% in the left circumflex artery (LCx), and proximal stenosis 30% in the right coronary artery (RCA). The drug-eluting stent was finally placed in the LAD. Conclusions: Shark fin sign ECG pattern indicates total occlusion and a large area of transmural ischemia, and predicts high mortality, early recognition and prompt aggressive treatment can reduce the risk of more serious complications and mortality.

Keywords: acute coronary syndrome; cardiogenic shock; extensive anterior STEMI; shark fin ECG

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INTRODUCTION

ST-elevation myocardial infarction (STEMI) is a serious form of acute coronary syndrome that involves complete or nearly complete occlusion of one or more coronary arteries. This leads to widespread myocardial ischemia and subsequent damage or death of heart tissue. This condition is typically triggered by plaque rupture and blood clot formation in the coronary arteries, although other factors may also contribute (Akbar *et al.*, 2024). The estimated annual incidence of myocardial infarction in the United States includes 550,000 new cases and 200,000 recurrent cases. In 2013, 116,793 persons in the United States experienced a fatal myocardial infarction, with 57% occurring in men and 43% in women.

Approximately 38% of patients who present to the hospital with ACS have a STEMI (Mozzafarian *et al.*, 2016). There have been notable advancements in treating STEMI over the past few decades, primarily due to the greater use of mechanical and pharmacological reperfusion methods. Despite general advances, some high-risk subsets of STEMI patients have a poor prognosis, such as those with cardiogenic shock (CS) and cardiac arrest (CA).

Based on a previous study (Omer *et al.*, 2020) 8.8% of patients had CS, 11% had CA, and 4.1% had both.

The 12-lead ECG is the primary diagnostic tool for evaluating patients with suspected ACS. ST-segment elevation (measured at the J-point) at least two contiguous leads ≥ 2.5 mm in men < 40 years, ≥2 mm in men ≥ 40 years (Byrne *et al.*, 2023). In addition to the well-known convex STEMI pattern associated with acute occlusive MI, patients may present with uncommon ECG changes such as the shark fin (SF) pattern (Escabi-Mendoza *et al.*, 2023). Shark fin ECG pattern characterized by a "triangular QRS-ST-T waveform" (TW) pattern with positive polarity in the leads exploring the ischemic region. The shark fin ECG pattern has also been associated with a large amount of ischemic myocardium (such as with left main coronary artery or proximal left anterior descending artery occlusion). It was identified as a predictor of poor in-hospital with higher mortality risk from cardiogenic shock and/or ventricular tachycardia (VT)/ ventricular fibrillation (VF) (Escabi-Mendoza *et al.*, 2023).

The shark fin is an unusual yet high-risk ECG pattern of STEMI that must be identified early and distinguished from other conditions with similar ECG patterns such as wide-complex tachycardia, hyperkalemia (*Escabi-Mendoza et al.*, 2023), and "triangular" configuration that resembles J-waves of Brugada syndrome (Cipriani *et al.*, 2017). This case report aims to highlight the recognition and differentiation of the shark fin sign ECG pattern from other causes of similar ECG changes to prevent the risk of acute complications like cardiogenic shock and ventricular arrhythmias.

METHOD

The method used is a descriptive case report design. The sample consisted of one patient who suffered from ST-elevation myocardial infarction and was admitted to the emergency room of Dr. Suhardi Hardjolukito, Airforce Central Hospital. The inclusion criteria for the sample were patients diagnosed with STEMI and shark fin sign ECG. The data from the subject were collectively obtained from anamnesis, physical examination, and supporting examinations such as ECG, Thorax X-ray, coro angiography, and laboratory results. Evaluation and examination were carried out in June 2022.

Data analysis was performed qualitatively by describing and analyzing the conditions of the patients from the anamnesis, physical examinations, and supporting examinations (ECG, Thorax X-ray, coro angiography, and laboratory results). The results of the analysis were compared to existing theories and other similar case studies from the literature to understand relevant aspects and treatment implications. The ethical principles followed in this study include autonomy, where patients have the right to decide about their care; beneficence, which emphasizes the obligation to act for the good of the patient; and veracity, where honesty and truth in the delivery of information to patients are emphasized.

RESULT

Mr. X, a 44-year-old male came to our emergency room unconscious with an impalpable pulse, and an absence of breath. Two hours prior the patient rode a bicycle from Salatiga to Purworejo for about 90 km, and then the patient complained of sudden typical chest pain, agitation, and diaphoresis for about an hour. He was an active smoker. From the physical examination, the patient had been in cardiac arrest conditions since three minutes ago, then CPR was conducted and the monitor showed ventricular tachycardia. The patient then was given CPR for 45 minutes, seven times defibrillations, six times of 1 mg intravenous epinephrine, and 300 mg and 150 mg of intravenous amiodarone, and the patient then

achieved ROSC. The patient was still in a coma condition (GCS: E1Vx on ventilator M1), his blood pressure was 80/40 mmHg his heart rate was 110 bpm, his respiratory rate was 30 times/minute, the temperature was 36.8 °C, and oxygen saturation was 98% on ventilator. The ECG showed ST elevation in V1-V6, lead I, and aVL with giant R waves and triangular shape (shark fin ECG pattern), ST depression in lead II, III, and aVF. The laboratory results showed elevated creatine kinase myocardial band (CK-MB) 163 (>24), and leukocytes 16.030 (> 10.000).

The patient then was diagnosed with Extensive Anterior STEMI Killip IV (Shark fin sign ECG), Cardiogenic Shock, ROSC Post Cardiac arrest mode VT/VF. We treated the patient with intravenous dobutamine 3 µg/kg/minute for the cardiogenic shock, 320 mg of aspilet, 300 mg of clopidogrel, 80 mg of atorvastatin, intravenous amiodarone 1 mg/ hour, and bolus anticoagulants intravenous heparin 3000 IU and maintenance 600 IU/hour. Our emergency team transferred the patient to the higher center hospital for primary percutaneous coronary intervention (primary PCI).

In the higher center hospital, coronary angiography was done. The result from the coro angiography was total occlusion of the proximal left anterior ascending coronary artery (LAD). The total occlusion of the proximal left anterior descending coronary artery was relieved, and three drug-eluting stents (3 DES) were placed in LAD. After that, the patient's condition improved, and he could do daily activities.

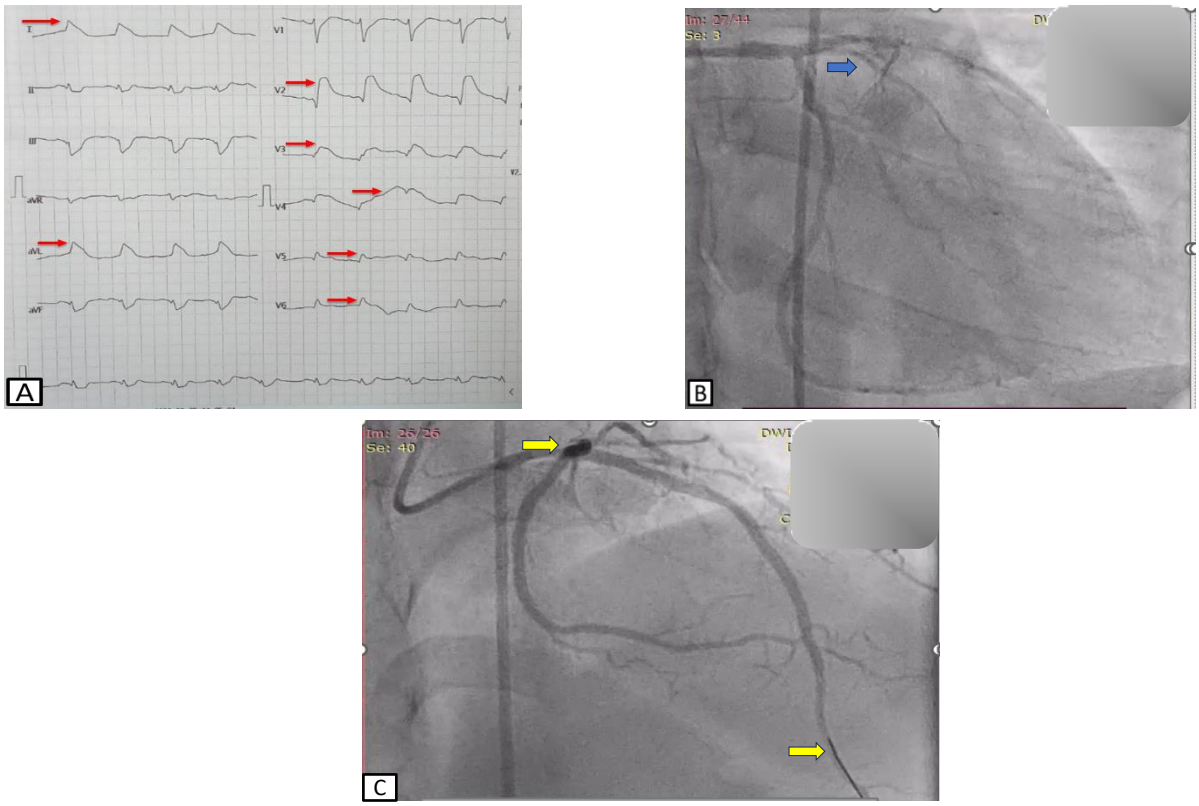


Figure 1. The results of supporting examinations [A] The electrocardiography of the patient at the Hardjolukito ER, the red arrows showed the shark fin sign. [B] The coro angiography of the patient at the higher center hospital, the blue arrow showed the total occlusion at the proximal LAD. [C] The coro angiography of the patient, the yellow arrows showed the (3 DES) and ballooning of the proximal LAD.

DISCUSSION

Acute coronary syndromes (ACS) comprise a spectrum of conditions that include patients presenting with the latest changes in clinical symptoms or signs, with or without changes on 12-lead electrocardiogram (ECG) and with or without acute elevations in cardiac troponin (cTn) concentrations (Byrne *et al.,* 2023). ACS is associated with a broad range of clinical presentations, from patients who have acute chest discomfort to symptom-free with cardiac arrest, electrical/hemodynamic instability, or cardiogenic shock (CS). Acute chest discomfort described as pain, pressure, tightness, heaviness, or burning (> 20 minutes) is the leading symptom that prompts the clinical diagnosis of ACS.

Chest pain descriptors should be classified as cardiac, possibly cardiac, and likely non-cardiac. Chest pain-equivalent symptoms include dyspnoea, epigastric pain, and pain in the left or right arm or neck/jaw. Misdiagnosis or delayed diagnosis is sometimes due to an incomplete history or difficulty in eliciting symptoms from the patient. To understand the complexity of ACS-related symptomatology, careful history-taking and comprehensive interaction with the patient are crucial and may help to facilitate an early and accurate diagnosis (Byrne *et al.,* 2023). In our case, our patient complained of sudden typical chest pain, agitation, and diaphoresis for about an hour, and three minutes before he came to our emergency room, he was in a cardiac arrest condition (impalpable pulse and absent of breath).

Patients presenting with suspected ACS are frequently categorized according on their ECG for initial management. Following that, patients can be classified based on the presence or absence of cardiac troponin increase (once the findings are available). While they are closely connected, it is vital to note that ACS is not synonymous with myocardial infarction (MI). MI is defined as cardiomyocyte necrosis in the presence of acute myocardial ischemia. The term ACS refers to unstable angina, non-ST elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI) (Byrne *et al.,* 2023). STEMI is a severe acute coronary syndrome characterized by complete or near-complete coronary artery occlusion of one or more coronary arteries, causing transmural myocardial ischemia and subsequent myocardial injury or necrosis, resulting in prolonged myocardial ischemia and rupture of the heart muscle caused by plaque rupture and thrombus development in the coronary artery (Akbar *et al.,* 2024).

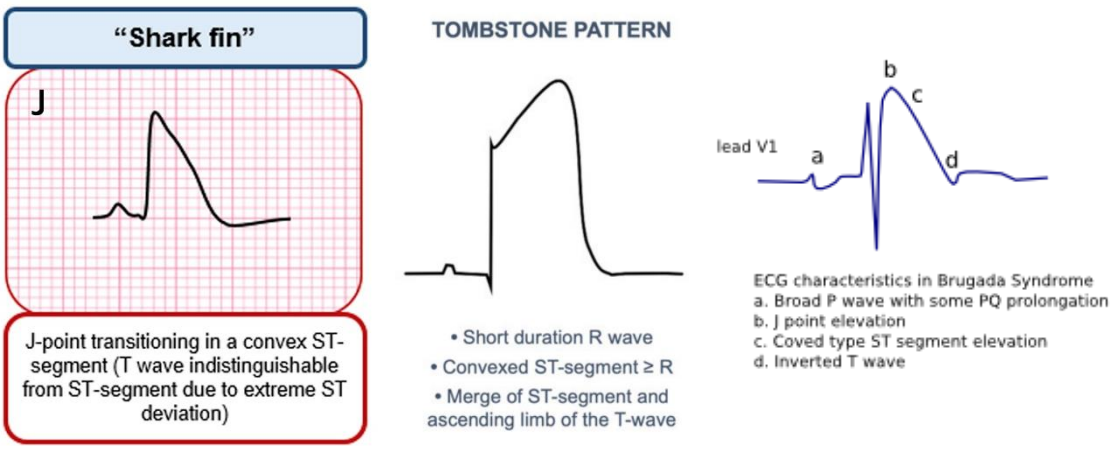
The 12-lead ECG is the primary diagnostic tool for assessing patients with suspected ACS. ST-segment elevation (measured at the J-point) at least two contiguous leads ≥ 2.5 mm in men < 40 years, ≥2 mm in men ≥ 40 years, or ≥ 1.5 mm in women regardless of age in leads V2-V3, and/or ≥ 1 mm in the other leads (in the absence of left ventricular hypertrophy or left bundle branch block [LBBB]) (Byrne *et al.,* 2023). The ECG patterns of STEMI patients upon admission may reveal varied durations of R waves, morphology of ST-segment elevation, T wave changes, and the presence or absence of Q and S waves, depending on the location, severity, and duration of ischemia injury (Cipriani *et al.,* 2017).

In practice, however, the whole spectrum of electrocardiography (ECG) abnormalities indicating acute coronary ischemia or occlusion has been described, including multiple high-risk ECG patterns associated with acute myocardial ischemia caused by severe stenosis or coronary artery blockage. One of them is hyperacute T waves, which frequently precede noticeable ST-segment elevation, and the "shark fin" sign (Asatryan *et al.,* 2019). The shark fin ECG pattern is distinguished by a “triangular QRS-ST-T waveform” (TW), defined as a unique, large wave (amplitude ≥1 mV) resulting from the fusion of the QRS complex, ST-segment, and T-wave, displaying a “triangular” morphology with positive polarity in the leads

determining the ischemic region (Cipriani *et al.*, 2017). The etiology of shark fin ECG has yet to be understood. Theoretically, it might be formed by a unique spread of ventricular activation caused by local ischemia injury, and it reformed extended conduction (slowing conduction) when the electrical current travels through the ischemia area, directly lengthening and amplifying the R wave. It can be concluded that during acute myocardial ischemia, the slowing of transmural electrical conduction results from changes in biochemical processes and ion concentrations, including potassium loss, a drop in pH, calcium overload, and the inactivation of sodium channels (Cipriani *et al.*, 2017). This ECG finding may be mistaken for wide complex tachycardia, ECG changes associated with myopericarditis, or hyperkalemia. Additionally, it can be confused with the tombstoning ECG pattern of STEMI, which occurs in 10-15% of patients. The tombstoning ECG pattern differs from the Shark Fin pattern in that it features an elevated, upward-convex ST segment that links a small, short R wave with a larger, broader T wave (Jaiswal and Shah, 2021).

The Shark Fin pattern also resembles Brugada syndrome type 1, which is marked by coved ST segment elevation in leads V1-V3 followed by an inverted T wave (*Liroff et al.*, 2016). Both share a predisposition to VF due to the R-on-T phenomenon from phase-2 re-entry, as seen in Brugada syndrome. Shark Fin represents severe LV myocardial ischemia, leading to mechanical paralysis and cardiogenic shock (Cipriani *et al.*, 2017). It also shares similarities with sine wave hyperkalemia, this ECG is described as a merger of the widened QRS complex with the elevated ST segment and T wave and loss of the P wave (Loubser *et al.*, 2023).

There have been cases where the Shark Fin pattern was initially misdiagnosed as VT. One such case, reported by Escabi-Mendoza et al. (2023), involved a Shark Fin ECG mistaken for VT due to its wide triangular waveform, which did not respond to defibrillation or intravenous amiodarone. The patient was urgently referred to the cardiology team, where further ECG revealed the Shark Fin pattern associated with extensive anterolateral MI. Successful PCI was carried out, revealing and treating a proximal LAD occlusion with thrombectomy and stenting. Another case reported by Andreou et al. (2021) involved a patient with a Shark Fin pattern caused by a left main occlusion, which was mistakenly diagnosed as VT, resulting in delayed reperfusion and a poor outcome after revascularization. It is essential for frontline physicians to recognize the Shark Fin ECG pattern and distinguish it from other similar patterns to prevent delays in reperfusion therapy, thereby improving prognosis and outcomes.



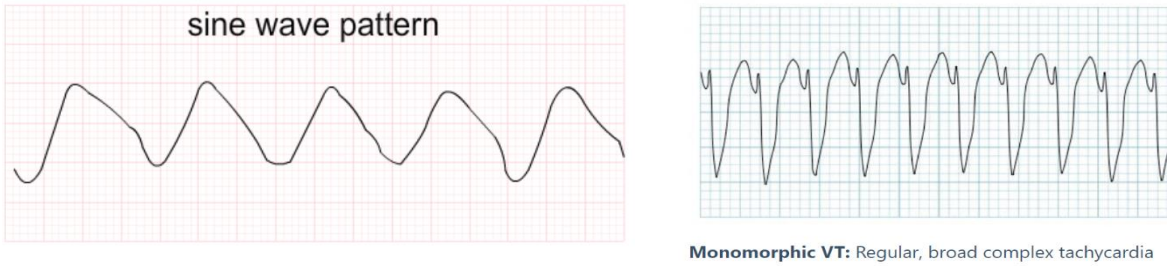


Figure 2. ECGs similar to the Shark Fin pattern

The Shark Fin ECG pattern was most commonly seen in the anterior leads (V1-V4) in 38.1% of cases, followed by the inferior leads (II/aVF/III) in 35.4% (Cipriani *et al.,* 2017). In our patient, the Shark Fin pattern appeared in leads I, aVL, V2-V6, with ST depression in leads II, aVF, and III, indicating extensive anterior ischemia. This ECG pattern is associated with significant ischemic myocardium, such as left main coronary artery (LMCA) or proximal left anterior descending artery (LAD) occlusions. Previous cases have linked Shark Fin ECG to severe LMCA stenosis (Miranda *et al.,* 2019) and critical LAD occlusion, which was treated with thrombectomy and stent placement (Jaiswal and Shah, 2021). In a cohort study by Cipriani et al. (2017), LAD occlusion was the most common cause (40%), followed by RCA (33.8%), with left main occlusion being the least common (1.1%). Similar to our case, coronary angiography revealed complete occlusion in the proximal LAD. Additionally, the Shark Fin pattern has been identified as a predictor of poor in-hospital outcomes, with a higher risk of mortality due to cardiogenic shock and/or ventricular tachycardia (VT) or ventricular fibrillation (VF) (Escabi-Mendoza *et al.,* 2023). In Cipriani et al. (2017) study of 367 patients, 5 patients (1.4%) had the Shark Fin ECG, which is rare, with an annual incidence of 0.7% in consecutive STEMI patients. Among these, 50% had left main coronary artery occlusion, 80% developed cardiogenic shock, 100% had VF, and 40% died during hospitalization. Compared to other ST-elevation ECG patterns, the Shark Fin pattern was strongly associated with the development of VF during the acute phase of myocardial infarction (MI) (Kukla *et al.,* 2008).

A retrospective study by Aizawa et al. (2012) found that triangular "coved" ST-T patterns, including the Shark Fin ECG pattern, were observed in 61.7% of patients who developed VF, compared to only 9.4% of those who did not. These patterns were highly specific in predicting VF, with a specificity of over 95%. This study supports our initial observations that the Shark Fin ECG pattern is a risk factor for VF during the acute phase of MI. Timely coronary revascularization is essential to address these ECG patterns and prevent VF recurrence. Revascularization can be achieved through thrombolytic therapy or primary PCI (PPCI). Previous studies have shown that primary PCI was the most common treatment (88.8%), while thrombolysis and urgent CABG were the least common (0.3%). However, there have been no studies comparing the outcomes of PCI versus thrombolysis in STEMI patients with the Shark Fin pattern.

According to Byrne et al (2023), a PPCI strategy is recommended for patients who have been resuscitated from cardiac arrest and present with persistent ST-segment elevation (or its equivalents) on the ECG (Class I B recommendation). Immediate coronary angiography and PCI are also recommended for patients with ACS and cardiogenic shock (Class I B recommendation). Fibrinolysis should be considered for STEMI patients in cardiogenic shock

if a PPCI strategy is unavailable within 120 minutes of STEMI diagnosis and mechanical complications have been excluded (Class II A, C recommendation). If timely PPCI (<120 min) cannot be performed in patients with a suspected STEMI diagnosis, fibrinolytic therapy is recommended within 12 hours of symptom onset for patients without contraindications (Class I A recommendation). In our case, the patient was resuscitated from cardiac arrest with persistent ST-elevation and was in cardiogenic shock condition. The PPCI center was accessible within 120 minutes of the STEMI diagnosis and the patient was still within the appropriate ischemic time window. Then our emergency team, under the supervision of cardiologists, decided to transfer the patient to the hospital with a PCI center for PPCI.

CONCLUSION

The shark fin pattern is an uncommon but high-risk ECG pattern of STEMI which should be identified promptly and distinguished from other conditions that can cause similar patterns, like wide-complex tachycardia (VT), hyperkalemia, and tombstone appearance. This pattern is linked to a high risk of VT and VF, cardiogenic shock, and death. Therefore, it demands aggressive treatment, including mechanical circulatory support, to prevent complications and improve survival rates.

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