

Challenges in Clinical Electrocardiography

ST-Segment Elevation in a Woman With Out-of-Hospital Cardiac Arrest

Chang Kai-Chun, MD; Lin Yi-Syuan, MD; Lin Po-Chih, MD

Case Presentation

A woman in her mid 70s presented to the emergency department following a sudden loss of consciousness during a meal. When paramedics arrived, the patient had a palpable pulse, but an out-of-hospital cardiac arrest (OHCA) developed in the ambulance. Cardiopulmonary resuscitation (CPR) was initiated, achieving return of spontaneous circulation (ROSC) after 19 minutes without a shockable rhythm. The reported medical history included hypertension. The patient underwent an electrocardiogram (ECG) on arrival (Figure, A).

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Questions: What are the ECG findings? How can these findings be interpreted in the given clinical context?

Interpretation

The ECG revealed irregular, narrow atrial fibrillation with rapid ventricular rhythm at a rate around 140 to 150 beats per minute. The ST segment was remarkable for convex ST elevation across lateral leads (V₄ through V₆, I, aVL) with fusion of QRS complex, which formed a triangular morphology known as the same configuration called shark-fin phenomenon and lambda-wave pattern (Figure, B). ST depression in leads II, III, and aVF was considered a reciprocal change of leads I and aVL. Precordial leads V₁ through V₂ also had marked ST depression, which may indicate posterior leads ST elevation.

Clinical Course

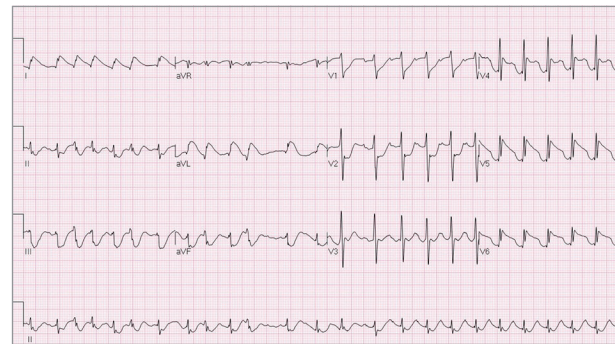
In the emergency department, follow-up ECG showed persistent shark-fin phenomenon. Echocardiography revealed preserved left ventricular ejection fraction without regional wall motion abnormalities. Laboratory analysis indicated mixed metabolic and respiratory acidosis (pH: 7.14, PaCO₂: 68 mm Hg, HCO₃⁻: 22.4 mmol/L, base excess: -6.7 mEq/L [to convert to mmol/L, multiply by 1], lactic acid: 5.37 mmol/L) without hyperkalemia (potassium: 3.54 mEq/L [to convert to mmol/L, multiply by 1]). Cardiac biomarkers were nearly normal (troponin T: 15.91 ng/L [to convert to µg/L, multiply by 1], N-terminal pro-B-type natriuretic peptide: 100.5 pg/mL [to convert to ng/L, multiply by 1]). Due to unconsciousness, an emergent CT was arranged and revealed diffuse subarachnoid hemorrhage without myocardial hypo-enhancement, pulmonary embolism, or pneumothorax. Subarachnoid hemorrhage-related ST-T changes were favored so coronary angiogram was not performed. The patient was admitted to the neurological intensive care unit. Due to poor prognosis, the family opted for palliative care. The patient passed away on the second day of admission.

Discussion

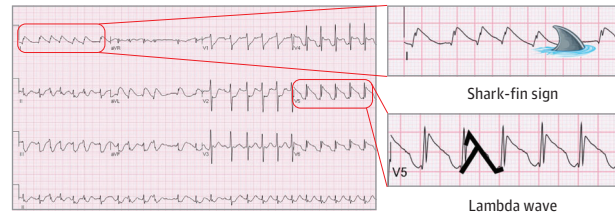
This case presented an ST-segment elevation with a unique presentation of the giant R waves (amplitude ≥1 mV) that merged with markedly elevated ST segments, which formed a triangular mor-

Figure. Electrocardiogram (ECG)

A ECG on arrival at emergency department



B Shark-fin sign and lambda wave



A, The ECG of the patient on arrival in the emergency department revealed atrial fibrillation with rapid ventricular rhythm at a rate around 140 to 150 beats per minute. The ST segment was remarkable for convex ST elevation across lateral leads (V₄ through V₆, I, aVL) with fusion of QRS complex. B, There are shark-fin sign over leads I and aVL and lambda wave over leads V₄ through V₆, showing merged R wave and elevated ST segment.

phology, the so-called shark-fin sign, giant R wave, triangular QRS-ST-T waveform, or lambda-wave pattern. The shark-fin sign is associated with poor outcome in patients with ST-segment elevation myocardial infarction (STEMI). However, the patient experienced acute-onset consciousness loss without evidence of chest discomfort or shockable rhythm, and echocardiography revealed no evidence of regional wall motion abnormality. ST-segment elevation with cause other than myocardial infarction was suspected and was later confirmed to be subarachnoid hemorrhage related.

The ST-segment elevation raised concerns about acute coronary syndrome (ACS). ACS is the leading cause of OHCA, and guidelines recommend a primary percutaneous coronary intervention (PPCI) strategy for patients with ROSC and persistent ST-segment elevation.¹ Nevertheless, other potential causes should be considered within the clinical context. The differential diagnosis for ST-segment elevation in a patient with cardiac arrest encompasses benign early repolarization, acute myopericarditis, stress cardiomyopathy, Brugada syndrome, pulmonary embolism, hyperkalemia, left bundle branch block, left ventricular aneurysm, or intracranial hemorrhage.

The shark-fin sign was first characterized by Ekmekci et al² in 1961 and was later reported to be found in patients with STEMI, takotsubo cardiomyopathy, and type 2 myocardial infarction from coronary spasm and hemorrhagic shock. In patients with STEMI presenting with shark-fin sign, the sign was usually associated with a large area of myocardial ischemia from occluded left main or proximal left anterior descending arteries. Recent studies revealed shark-fin sign in STEMI as a predictor of high risk of cardiogenic shock, ventricular fibrillation, and in-hospital mortality and was reported to present in 1.9% of all patients with STEMI in a study group.³ The mechanism of R wave merging with the down-sloping ST segment remained unclear and was speculated to be associated with the significant slowing of transmural electrical conduction, due to biochemical and ion concentration changes during large areas of myocardial injury.⁴

To our knowledge, it was the first case of shark-fin sign with the cause of subarachnoid hemorrhage. The association between intracranial hemorrhage and ECG changes is well documented, with investigations suggesting sympathetic overstimulation as the possible underlying mechanism. ECG changes observed in intracranial hemorrhage include the presence of U waves, T wave abnormalities, QTc prolongation, high R wave, ST depression, and bradycardia.⁵

In cases where intracranial hemorrhage leads to OHCA with concurrent ST-segment elevation, opting for a PPCI strategy may exacerbate the hemorrhage and compromise the patient's prognosis. This case underscores the significance of meticulous clinical assessment for ST-segment changes and the inclusion of intracranial pathology in the differential diagnosis. Clinical clues that suggest intracranial pathology in the context of ST-segment changes encompass the absence of chest pain before events, abrupt consciousness changes without recovery, the absence of a shockable rhythm during CPR, and incompatible LV regional wall motions of echocardiography results.

Take-Home Points

- In patients presenting OHCA with ROSC and a persistent ST-segment elevation, differential diagnosis other than acute myocardial infarction should be kept in mind and measurements need to be excluded.
- The shark-fin phenomenon is mostly associated with myocardial infarction or takotsubo cardiomyopathy but could be rarely found in intracranial hemorrhage.
- ECG changes in intracranial hemorrhage could mimic lots of ECG changes in ACS.

ARTICLE INFORMATION

Author Affiliations: Division of Cardiology, Department of Internal Medicine, National Taiwan University Hospital, Taipei, Taiwan.

Corresponding Authors: Po-Chih, Lin, MD, Division of Cardiology, Department of Internal Medicine, National Taiwan University Hospital, No. 7 Chung-Shan S Rd, Taipei, Taiwan (juipeter@gmail.com).

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