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Early Left Ventricular Thrombus Following Ventricular Fibrillation/ Ventricular Tachycardia Electrical Storm

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ABSTRACT

Left ventricular thrombus (LVT) formation is a serious clinical complication of low-flow states that may be seen in an ischaemic, arrhythmic heart. While LVT formation has a poor prognosis, in the setting of myocardial infarction it is usually a result of post-infarct sequelae such as left ventricle aneurysms, and inflammatory changes from damaged tissue, with the LVT taking several days to form. Arrythmias such as ventricular tachycardia (VT) or ventricular fibrillation (VF) may also lead to thrombus formation, as they contribute to stasis due to decreased cardiac output. Large anterolateral myocardial infarctions can cause electrical or arrhythmic storm, characterized by more than three episodes of VT or VF in a 24-hour period. This prolonged state of dyskinesis further increases the risk of thrombosis, creating a compounding effect. Here, we report the case of a patient who had a VF cardiac arrest with electrical storm secondary to anterolateral myocardial infarction complicated with LVT formation found on echocardiogram after the cardiac arrest, which was absent on presentation. This thrombus formation occurred particularly early during the course of the patient's arrest, possibly due to the compounding factors increasing the risk of thrombosis. Herein, we discuss in detail the risk factors for LVT formation, its mechanism and management options. A review of the literature also shows that LVT formation in the acute phase of arrest, as seen in our patient, is rare.

KEYWORDS

Left ventricular thrombus, ventricular fibrillation, ventricular tachycardia, electrical storm

LEARNING POINTS

- Left ventricular thrombus (LVT) formation occurs 3–14 days after myocardial infarction, but in the setting of concomitant ventricular fibrillation arrest, may occur within the first 24 hours.
- Risk factors for LVT formation include a large infarct, anterior/anterior apical infarction, decreased ejection fraction (particularly <30–35%), left ventricular aneurysm, and delayed time to revascularization.
- Although diagnosis is generally made on transthoracic echocardiography with intravenous contrast, cardiac MRI with contrast has better sensitivity and specificity.
- Treatment consists of anticoagulation with a vitamin K antagonist or heparin for 3–6 months with a repeat echocardiogram to confirm the thrombus has organized or resolved. Further trials are needed to assess the efficacy of direct oral anticoagulants.

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INTRODUCTION

Electrical storm (ES) is a life-threatening medical emergency that is defined as ≥3 episodes of ventricular tachycardia (VT) or ventricular fibrillation (VF) or appropriate implantable cardioverter-defibrillator (ICD) shocks occurring within 24 hours and requiring intervention. Triggers for ES are myocardial ischaemia, acute heart failure, electrolyte disorders, hypoxia, drug-related arrhythmogenicity and thyrotoxicosis. Left ventricular thrombus (LVT) is a serious complication of acute myocardial infarction and non-ischaemic cardiomyopathies, and occurs due to a low-flow state. LVT has a poor outcome and is associated with high mortality. Although ES is a low-flow state, very few cases have been reported showing an association between ES and LVT.

CASE DESCRIPTION

Our patient was 55-year-old man with a medical history of HIV, polysubstance abuse, COPD, hypertension and diabetes mellitus, who presented to the emergency department with complaints of shortness of breath and epigastric abdominal pain for 3 days. He denied chest pain, palpitations or any other significant symptoms.

His vital signs showed a heart rate of 137, respiratory rate of 27, blood pressure of 156/102 mmHg, temperature of 36.9°C, and O2 saturation of 93%. The initial physical examination was significant for bilateral diffuse rhonchi and epigastric abdominal tenderness without distention or guarding. Lab work was consistent with high anion gap metabolic acidosis in the setting of hyperglycaemia and ketosis, urine drug screening was positive for cocaine and opiates, and chest x-ray showed extensive infiltrate involving the majority of the left lung. An initial electrocardiogram (EKG) showed left ventricular hypertrophy, and the initial echo showed an LVEF of 55–60% with normal systolic function, and mild left ventricular hypertrophy. No left ventricular thrombus was noted at this time.

The patient was transferred to the ICU where an insulin infusion was started for diabetic ketoacidosis management and vancomycin for pneumonia, with initial improvement, later complicated with ventricular fibrillation requiring advanced cardiovascular life support and mechanical ventilation. The return of spontaneous circulation was achieved after 90 minutes of downtime and the patient was later stabilized with the assistance of three vasopressors. A repeat EKG showed an accelerated idioventricular rhythm and non-specific intraventricular block with an anterolateral injury pattern. The echocardiogram showed a 35×15 mm apical thrombus, but given the bleeding diathesis secondary to ischaemic hepatitis, anticoagulation was deferred at this time.

Over the following days, vasopressor support was down-titrated as tolerated. However, the patient developed right lower extremity deep venous thrombosis, so anticoagulation with heparin infusion was initiated. He was weaned from ventilatory support and self-extubated but during the third week of hospitalization became unresponsive. Computed tomography of the head showed bilateral occipital subacute infarcts followed shortly afterwards by dyspnoea and cardiac arrest. Cardiorespiratory resuscitation was unsuccessful and the patient unfortunately died.

DISCUSSION

Electrical storm is defined as the occurrence of three or more episodes of VT or VF occurring within a 24-hour period. Myocardial ischaemia is a common cause of ES; other causes include electrolyte abnormalities or drug toxicity. Management is by following the ACLS protocol for the management of arrhythmias in addition to the use of antiarrhythmic medications, amiodarone being the first line, while lidocaine and procainamide can also be used [1]. Supportive measures to counteract sympathetic drive include the use of beta-blockers, adequate sedation and analgesia, and avoidance of medications that can perpetuate adrenergic activity. Options for refractory ES are stellate ganglion block, catheter ablation or ECMO [2]. Coronary reperfusion is indicated when myocardial ischaemia is felt to be the cause.

The process of LVT formation is explained by Virchow's triad: endothelial dysfunction, hypercoagulability and stasis. In the setting of myocardial infarction-induced cardiac arrest, ischaemia first leads to endothelial injury and dysfunction. LV dyskinesia and akinesia then lead to stagnation of blood, or stasis. Furthermore, prolonged cardiac arrest will contribute to a low-flow state in the patient, which further perpetuates stasis. In addition, myocardial infarction and cardiac arrest create a hypercoagulable state, the third component of the triad [3]. Risk factors for LVT formation include a large infarct, anterior/anterior apical infarction, decreased ejection fraction (particularly <30–35%), left ventricular aneurysm, and delayed time to revascularization [3].

LVT formation usually occurs 3 days after myocardial infarction and is commonly associated with LV aneurysm formation, with the risk persisting for the next 2 weeks. Before coronary revascularisation became common practice, left ventricular thrombi following anterior wall myocardial infarction occurred in 27% patients within the first 24 hours. However, many were missed as echocardiography was not generally employed. Echocardiography is now more common and it is clear that better and quicker myocardial infarction treatment has reduced the incidence of thrombi [4]. The extremely low flow state in our patient might explain the occurrence of LVT within hours of VF cardiac arrest because he had a total downtime of 90 minutes. A study by Budhram et al. showed that LVT develops early in the natural history of VF arrest and resolves quickly once forward flow is re-established by chest compressions in swine with sonographic evidence of thrombus formation

and resolution once compressions started [5,6]. Coagulofibrinolytic changes following cardiac arrest play a role in creating a hypercoagulable state favouring thrombus formation [7].

Patients with LVT are considered at high risk of thromboembolism, including cerebral embolic stroke and peripheral arterial ischaemia. LVT is also associated with higher in-hospital mortality and increased length of stay. Factors associated with an elevated risk of embolization include a ball-like thrombus which is mobile and pedunculated [8]. The mobility of an LV apical thrombus was found to be the most important predictive factor of early thrombus resolution in a study by Oh et al. [9].

Diagnosis is usually made by transthoracic echocardiography. Intravenous contrast is used for better visualization and assessment of the thrombus [10]. Cardiac MRI with contrast has the advantage of accurately assessing the thrombus and infarct in a single imaging examination. It has higher sensitivity and specificity and is now considered the gold standard in diagnosis.

Management is by initiating anticoagulation. The anticoagulant of choice is a vitamin K antagonist (VKA) with IV heparin. Direct oral anticoagulants (DOACs) appear to be at least as effective and safe as a VKA for stroke prevention in patients with LVT and might be considered an option in such patients based on several retrospective trials [11,12]. However, currently, there are no randomized prospective trials comparing DOACs and VKA. Concerning the duration of treatment, guidelines suggest 3–6 months of anticoagulation [12], but there are no prospective data to support this recommendation. Consequently, a case-by-case decision depending on bleeding risks needs to be made, especially for patients on concomitant dual antiplatelet therapy. Repeat imaging following a period of anticoagulation is recommended to assess for resolution. An organized thrombus is associated with a lower rate of embolization, allowing the safe withdrawal of anticoagulation.

Our patient likely suffered a VF cardiac arrest secondary to anterolateral myocardial infarction. He was unstable and therefore could not be taken for revascularization. Initiating anticoagulation in the patient was also challenging as he was coagulopathic and at high risk of bleeding with thrombocytopenia, an elevated INR, and dropping haemoglobin. Although he was eventually extubated, he suffered acute limb ischaemia likely secondary to embolization from the LVT. Unfortunately, his limb was not salvageable.

LVT has a poor outcome and is associated with high mortality [13]. A review of the literature revealed that Kikuchi et al. reported a case of ES secondary to myocardial ischaemia complicated by a free-floating LVT a few days after the patient was placed on ECMO [14]. The case we presented demonstrates unusual early formation of an LVT a few hours after cardiac arrest and ES.

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