

ISSN Online: 2164-5337 ISSN Print: 2164-5329

St-Segment Elevation Myocardial Infarction with Multiple Complications: A Case Report

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How to cite this paper: Diop, K.R., Sene, M.A., Beye, S.M., Mingou, J.S., Ngaïdé, A.A., Diouf, Y., Ndiaye, P.G., Diop, C.M.B., Diouf, M.T., Kane, A., Diao, M. and Kane, A. (2023) St-Segment Elevation Myocardial Infarction with Multiple Complications: A Case Report. World Journal of Cardiovascular Diseases, 13, 124-129.

https://doi.org/10.4236/wjcd.2023.133010

Received: January 18, 2023 Accepted: March 27, 2023 Published: March 30, 2023

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Abstract

Introduction: ST-Segment Elevation Myocardial Infarction (STEMI) usually involves complete thrombotic occlusion of a coronary artery and require urgent reperfusion; it is one of the leading cause of global mortality and morbidity worldwide. A variety of mechanical, rhythmic, conductive, embolic or hemodynamic complications can occur following STEMI, especially when the treatment is delayed or inadequate. Clinical presentation: A 58-year-old patient with hypertension was admitted to our department for a circumferential STEMI complicated by an ischemic stroke; received 24 hours after the onset of pain. His blood pressure was 100/60 mmHg, heart rate was 55 beats/min. The examination revealed right central facial paralysis and a slight motor deficit of the right upper limb (muscle strength 4/5). The first electrocardiogram (ECG) showed a significant circumferential ST-segment elevation with Q waves in the same territory, as well as a Luchiani Wenckebach atrio-ventricular block. The first echocardiography performed showed apical akinesia along with the presence of an apical thrombus. Coronarography was not performed because it was not available and the patient was given curative low molecular weight heparin combined with dual antiplatelet therapy, an angiotensin converting enzyme inhibitor and high dose statins. Seventy-two hours later, the ECG showed a complete atrioventricular block with narrow QRS and the average ventricular rate was 51 beats/min. The patient was asymptomatic. Another echocardiography was performed to assess new complications and showed a rupture of the left ventricular wall and a moderate amount of circumferential pericardial effusion, without any sign of cavity compression. No particular therapeutic attitude was adopted apart from close monitoring with daily ECG and echocardiography. Ten days later, spontaneous regression of the AV Bloc was noted. **Conclusion:** ST-Segment Elevation Myocardial Infarction is a major cause of morbidity and mortality worldwide. A variety of complications can occur after myocardial infarction, especially when revascularisation is delayed or inadequate.

Keywords

Myocardial Infarction, Delayed Reperfusion, Complication

1. Introduction

ST elevation myocardial infarction (STEMI) usually involves a complete thrombotic occlusion of the coronary artery and requires urgent reperfusion. It is one of the leading causes of global mortality and morbidity worldwide [1]. A variety of complications: mechanical, rhythmic, conductive, embolic and hemodynamic can occur following STEMI, especially when treatment is delayed or inadequate [1]. We report the case of a 58-year-old patient received 24 hours after the onset of STEMI, with multiple complications; mechanical (ventricular wall rupture), conductive (AtrioVentricular Block) and embolic (apical thrombus with ischemic stroke).

2. Clinical Presentation

This was a 58-year-old patient with uncontrolled hypertension admitted for a STEMI located in the circumferential territory, received 12 h after the thrombolysis delay (24 hours after the pain onset) complicated by an ischemic stroke in the bilateral cerebellar territory.

His blood pressure was 100/60mmHg and the heart rate 55 beats/min.

The physical examination revealed a right central facial paralysis and a slight motor deficit of the right upper limb (muscle strength 4/5).

The first electrocardiogram (ECG) showed a significant ST-segment elevation in the circumferential territory with Q wave in the same territory and a Luchiani Wenckebach Atrio-Ventricular Block (Figure 1).

The echocardiography performed showed an apical akinesia with apical thrombus (Figure 2).

Coronary angiography was not performed because of its unavailability. The patient received low molecular weight heparin combined with dual antiplatelet therapy, a angiotensin converting enzyme inhibitor and high dose statins (80 mg/day).

Seventy-two hours later, the ECG showed complete atrioventricular block with narrow QRS, the mean ventricular rate was 51 cycles/min. (Figure 3). The second echocardiography performed to assess the heart showed a rupture of the left ventricle wall, and a moderate quantity circumferential pericardial effusion, without any sign of cavity compression (Figure 4).

No particular therapeutic attitude was adopted apart from close monitoring with daily ECG and echocardiography.

Ten days later, spontaneous regression of the AV Block was noted (Figure 5).

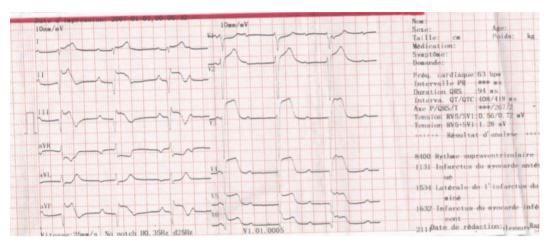


Figure 1. The first ECG showed a significant ST-segment elevation in the circumferential territory with Q wave in the same territory, and Luchiani Wenckebach Atrio-Ventricular block.

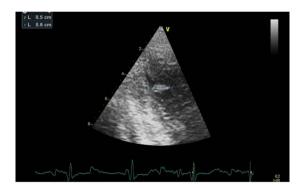


Figure 2. The echocardiography showed a small apical thrombus.

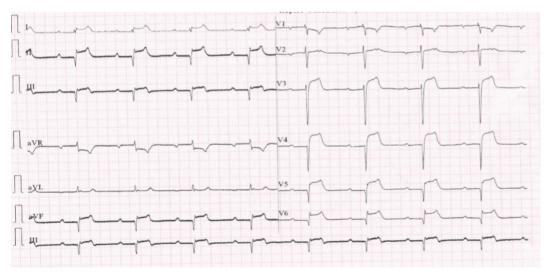


Figure 3. The ECG performed seventy-two hours later, showed complete atrioventricular block with narrow QRS, the mean ventricular rate was 51 cycles/min.

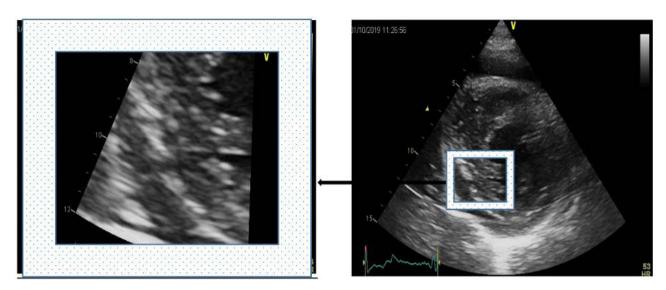


Figure 4. The echocardiography showed the rupture of the heart wall with pericardial effusion.

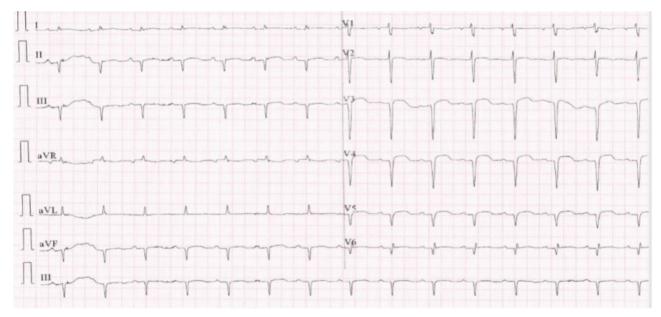


Figure 5. ECG performed ten days later, showed spontaneous regression of the AV block.

3. Discussion

Coronary artery disease is one of the leading causes of death worldwide [2]. The advent of early reperfusion therapy has significantly reduced in-hospital mortality and improved outcomes in survivors of ST-Segment Elevation Myocardial Infarction (STEMI). A variety of complications can occur after STEMI, particularly when reperfusion is delayed or inadequate. Post-STEMI complications can be grouped into five categories: mechanical, rhythmic, conductive, embolic and hemodynamic complications [3].

Thrombus formation is an embolic complication and usually occurs post-STEMI, it has a prevalence ranging from 20% of patients to less than 1.6% with early and appropriate treatment. Thrombus forms after stagnant blood flow may

be the result of ventricle wall motion abnormalities or left ventricle (LV) aneurysms [4] [5]. Other known risk factors for LV thrombus formation include delayed reperfusion, large infarction, previous infarction and low LV ejection fraction. The presence of thrombus can lead to embolization in structures remote from the heart and embolic events usually occur within 10 days of an acute myocardial infarction (MI) [6] [7].

Another mechanical complication is the rupture of the heart wall which, clinical presentations may range from a small pericardial effusion to sudden acute cardiovascular collapse and risk factors for free wall rupture include advanced age, female gender, hypertension and a first episode of MI [8].

Complete atrioventricular block (AVB) is a frequent complication of STEMI with an increased risk in STEMI patients compared to NSTEMI patients [9]. Its exact mechanism remains unclear, but in STEMI located in anterior territory, it is thought to be related to extensive myocardial necrosis due to the interruption of septal perfusion [6] [7]. Spontaneous regression of complete AV Block was noted in our case. However, we could not find an explanation regarding the spontaneous evolution of the complete AV Block in this particular case. The same question is raised by Andrew E et al, also without answer [10].

In our patient, the absence of early and appropriate reperfusion therapy could be considered the root of their multiple complications. This lack of reperfusion explaining the necrosis and the wall motion abnormalities would likely be the origin of the thrombus formation, which would cause the cerebral ischemic stroke. The lack of reperfusion also explains the complete AV Block due to the septal necrosis. Hypertension and a first episode of STEMI in this patient along with a lack of reperfusion could explain the rupture of the left ventricular free wall.

4. Conclusion

This case shows how ST-Segment Elevation Myocardial Infarction can be very dangerous, especially when early and appropriate reperfusion is not achieved. The challenge remains to perform an early appropriate reperfusion therapy and increase public awareness.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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