Introduction: Myocardial bridge (MB) is a congenital anomaly in which a segment of a coronary artery takes a “tunneled” intramuscular course under a “bridge” of overlying myocardium. The pathogenesis occurred primarily at diastolic because systolic contraction obstructs the flow of coronary artery, particularly to sub-endocardium part. Medication is the main choice for preventing myocardial ischemic complication. This case report explains a patient with myocardial bridging with STEMI anterolateral on ECG and ventricular extrasystole (VES).

Methods: A 62-year-old man came to the Siloam hospital emergency room with complaints of seizures. Initially, the patient complained of the abdominal pain continuously felt throughout the abdomen, sore and twisting sensation. The next day the patient got seizure and came to hospital with sudden spasm throughout the body, the hands and feet moved vertically, unconsciously, and was difficult for answering questions. The patient's shortness of breath condition was difficult to be analyzed. Physical examination was normal. The vital signs were BP: 170/100 mmHg; HR: 110x /m; RR: 24x/m; BT: 37.10°C. The ECG showed anterolateral STEMI and VES, trivial heart valve insufficiency with left ventricle systolic function was in good condition. There was increased in CKMB level (34.8) and normal level of troponin T (8,8). The CAG was done and the result was myocardial bridging vein was found in Left anterior Descending (LAD) without coronary artery stenosis.

Result: Myocardial bridge is categorized as normal anatomy variance of coronary artery. The previous study showed the clinical course of MB is generally benign with a five-year survival rate of 97.5%, but a problem was found in this patient in which this case showed a patient in whom myocardial bridging is potentially pathologic. He came in seizure and abdominal pain condition only without any other symptoms of myocardial ischemic or heart attack, but the ECG showed anterolateral STEMI with VES arrhythmia, which reinforces a suspicion of ACS, therefore CAG was done. The CAG result showed a systolic compression (milking) in LAD which confirmed the presence of MB. The patient was treated with amiodarone IV until the ECG returned to normal.

Conclusion: Myocardial bridges have traditionally been considered as benign condition, but this case is a proof that MB can possibly induce arrhythmia, and warrant the recent studies that the clinical complications of MB can be dangerous (acute coronary syndrome, arrhythmia, transient ventricular dysfunction, and sudden death). The prognosis of patients with MB, therefore, is not as benign as it was believed to be in the past. This case report is an example of the clinical effects of myocardial bridge. It is therefore interesting not only because of the rarity of the case, but also because it brings the attention of cardiologists to an anomaly that is often neglected.