SUPRAVENTRICULAR TACHYCARDIA COMPLICATING DIFFUSE ST DEPRESSION WITH ST ELEVATION IN AVR

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ABSTRACT

Background: Lead aVR is frequently neglected in routine clinical practice. Usually, basal septum receives blood supply from very proximal septal branches of the left anterior descending artery. Transmural infarction of this area usually causes lead ST segment elevation in lead aVR signaling proximal left coronary artery (proximal LAD or left main) occlusion. Ischemia and infarction leads to metabolic and electrophysiological changes that may cause silent and symptomatic life-threatening arrhythmia.

Case Report: We reported 50 years old male patient presented to the ED 15 minutes since the onset of severe pain in the abdomen accompanied by nausea and sweating. With ECG of diffuse ST-segment depression with STE-aVR. The patient was then diagnosed with NSTE-ACS with probable left main coronary artery (LMCA) obstruction with differential diagnosis of cholecystitis/ cholelithiasis with accompanying stable coronary artery disease. Patient felt better and rejected hospitalization. The patient then came 7 hours later with dyspnea and worsening abdominal pain. ECG of PSVT 189x/minute. Troponin was >10 ng/mL. Patient refused cardioversion and adenosine/ ATP was unavailable. Amiodarone 150 mg over 10 minutes was administered. After consideration, patient was then referred to coronary angiography capable center for immediate invasive strategy.

Conclusion: ST elevation in lead aVR may signal a severe proximal left coronary artery disease (LMCA or proximal LAD). Regardless whether it is caused by proximal left coronary artery disease or not, it is also an independent predictor of mortality.

Keywords: supraventricular tachycardia, ST elevation aVR, left main coronary obstruction

Abstrak

Pendahuluan:: Sadapan aVR seringkali diabaikan dalam praktek sehari-hari. Pada umumnya septum basal menerima aliran darah dari cabang proksimal arteri left anterior descending. Infark transmural pada daerah tersebut umumnya menyebabkan elevasi segment-ST pada sadapan aVR, hal tersebut menandakan oklusi arteri koroner kiri proksimal (oklusi LAD atau left main). Iskemia dan infark menyebabkan perubahan metabolic dan elektrofisiologi yang dapat menyebabkan aritmia yang mengancam jiwa.

Laporan kasus: Kami melaporkan pasien laki-laki 50 tahun yang dating ke IGD 15 menit sejak awitan dari nyeri pada abdomen yang diikuti dengan mual dan keringat dingin. Pada EKG ditemukan depresi segmen-ST yang luas dengan elevasi segmen-ST pada sadapan aVR. Pasien tersebut didiagnosa dengan sindrom koroner akut tanpa elevasi segmen ST dengan kemungkinan sumbatan pada arteri koroner kiri utamadengan diagnosis bandingkolesistitis/kolelitiasis dengan angina pectoris stabil. Pasien merasa gejala membaik dan menolak perawatan di rumah sakit. Pasien kemudian datang 7 jam kemudian dengan sesak

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nafas dan nyeri abdomen yang memberat. Elektrokardiogram menunjukan PSVT (Paroxysmal Supraventricular Tachycardia) 189x/menit. Troponin >10 ng/mL. Pasien menolak kardioversi dan adenosine/ATP tidak tersedia. Amiodarone 150 mg dalam 10 menit diberikan. Setelah pertimbangan, pasien dirujuk untuk angiografi koroner untuk strategi invasif .

Kesimpulan: Elevasi segmen-ST pada sadapan aVR dapat menandakan adanya penyempitan pada arteri koronaria sinistra segmen proksimal. Elevasi segmen-ST merupakan prediktor independen dari mortalitas pasien terlepas dari sebab utamanya.

Kata kunci:: takikardia supraventrikel, elevasi segment ST aVR, obstruksi arteri koroner utama kiri

INTRODUCTION

Lead aVR is directionally non-adjacent to any other ECG lead thus frequently neglected in routine clinical practice. It has a frontal plane vector of −150° directly facing the thinner wall of the right ventricular outflow area and through it the basal aspect of the interventricular septum below the aortic and pulmonary valves.[1] Usually, basal septum receives blood supply from very proximal septal branches of the left anterior descending (LAD) artery. Transmural infarction of this area usually causes lead ST segment elevation in lead aVR (STE-aVR) signaling proximal left coronary artery (LCA) occlusion before the first septal artery (proximal LAD or left main).[1] Ischemia and infarction leads to metabolic and electrophysiological changes that may cause silent and symptomatic life-threatening arrhythmia.[2]

CASE REPORT

A 50 years old male patient presented to the emergency department 15 minutes since the onset of severe pain in the epigastrium and right upper quadrant. He was nauseous but denied vomiting and sweating. There was no chest pain nor shortness of breath. His past medical history was significant for hypertension which is managed by amlodipine 5 mg daily. On admission, physical examination showed blood pressure of 130/80 mmHg, heart rate of 114x/minute, temperature 36,5˚C and respiratory rate of 22x/minute. Tenderness in the epigastrium and right upper quadrant were encountered. Murphy sign was negative and cardiorespiratory examination was within normal limits. ECG reveals diffuse ST-segment depression (I, II, aVL, and V4-6) with STE-aVR (Fig.1). Creatinine was increased (1.5 mg/dL), there were also increased ALT and AST but <3 times the upper normal limit.

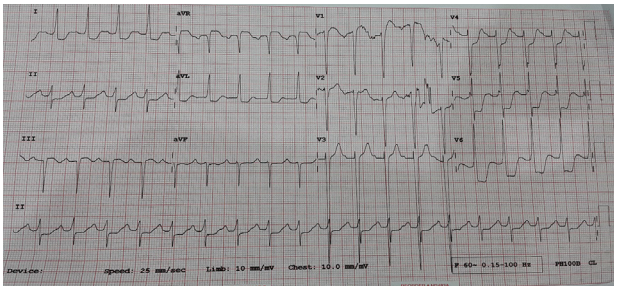

Figure 1. ECG at first admission

Based on these findings, he was diagnosed with NSTE-ACS Non ST-segment Elevation Acute Coronary Syndrome) with probable left main coronary artery (LMCA) obstruction with differential diagnosis of cholecystitis/cholelithiasis with accompanying stable coronary artery disease. The patient was given aspirin 160 mg sublingual, clopidogrel 300 mg per oral, ISDN 5 mg sublingual (twice), antacid per oral, omeprazole 40 mg IV and ondansetron 8 mg IV. He experienced improvement in symptoms and rejected hospitalization afterward. He then came 7 hours later with dyspnea and worsening abdominal pain. Blood pressure 134/70 mmHg, heart rate 189x/minute, respiratory rate 28x/minute and temperature 37˚C. On physical examination, there were rales in both bases of the lung. ECG showed PSVT 189x/minute (Fig 2). Troponin was >10 ng/mL. He refused cardioversion and adenosine/ATP was unavailable. After carotid sinus massage was avoided because of myocardial infarction (a concern of the atherosclerotic state of carotid arteries), amiodarone 150 mg over 10 minutes was administered considering non-dihydropyridine calcium channel blocker may further depress the ventricular function if PSVT does not convert into sinus rhythm. Digoxin was

less preferred since it may worsen ischemia in NSTE-ACS (. After consideration, he was then referred to the coronary angiography capable center for immediate invasive strategy.

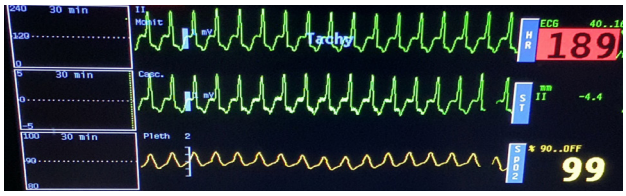

Figure 2. ECG at second admission

DISCUSSION

ST elevation in lead aVR may signals a severe proximal LCA disease (before the first septal artery) as the basal septum (which the lead aVR faces) receives blood supply either from the proximal septal branches of LAD or from the PDA branch of RCA in those with prior proximal LCA occlusions.[1] Classical ECG pattern highly predictive of left main coronary occlusion/3-vessel disease includes STE in lead aVR + extensive ST depression (most prominent in leads I, II, and V_{4-6}) with the STE in aVR ≥ V_1.[3-5] A retrospective study by Kosuge, et al (310 patients) reported that STE-aVR has a sensitivity of 78%, specificity of 86%, positive predictive value (PPV) of 57%, negative predictive value (NPV) of 95% and predictive accuracy of 84% for predicting LMCA or 3VD with NSTE-ACS.[6] Diffuse ST depression plus ST elevation in lead aVR was useful in identifying LMCA disease and mortality reflected by a multivariable analysis that showed an OR 4,72 (95% CI 2,31 - 9,64; p< 0,001) for this combined pattern to predict LMCA occlusion.[7] A study stated that presence of a diffuse ST depression in eight or more leads combined with ST elevation in aVR and V_1 during an episode of ischemic chest pain has a 75% predictive accuracy for LMCA or 3VD.[8]

In this patient, however, the ST depression was only in 6 leads without V_1. Kosuge et al studied 572 patients with NSTEMI and found that degree of STE in aVR was the strongest independent predictor of severe LMCA occlusion/3VD requiring CABG with OR 29,1, followed by positive troponin T level OR 1.27. STE ≥ 1.0 mm in aVR identified severe LMCA occlusion /3VD with 80% sensitivity, 93% specificity, 56% PPV, and 98% NPV.[9] STE-aVR ≥0,5 mm was also an independent predictor of mortality (in-hospital mortality was 19% vs 5%).[10]

American Heart Association/ACCF/HRS recommendation for interpreting STE-aVR as representing "ischemia due to multivessel or LMCA obstruction," and should be referred for urgent coronary angiography.[11] Atrial tachyarrhythmias (supraventricular) are uncommon during the early phase of myocardial infarction (MI) and are often transient and might result from augmented sympathetic stimulation. PSVT complicating possible LMCA occlusion in this patient hinders an already compromised coronary perfusion with addition myocardial workload. This patient has unstable hemodynamic and needs urgent electrical cardioversion. Since patient rejected electrical cardioversion and adenosine/ATP is unavailable, we move to next alternative. Carotid sinus massage was avoided because of myocardial infarction in which there is a concern of the atherosclerotic state of carotid arteries (CSM was contraindicated in those with past history of myocardial infarction). In this least ideal situation another AV nodal blocking agents such as non-dihydropyridine CCB (verapamil and diltiazem) may be an alternative, however, we fear that it may further depress the ventricular function if PSVT does not convert into sinus rhythm. Digoxin was less preferred since it may worsen ischemia in NSTE-ACS. Amiodarone 150 mg over 10 minutes was administered with maintenance was given in hope of converting to sinus rhythm without increasing myocardial oxygen consumption demand or depression of LV function.[2] With positive troponin, relatively unstable hemodynamic condition and the possibility of LMCA occlusion the patient was referred to coronary angiography capable hospital for immediate invasive strategy.

CONCLUSION

ST elevation in lead aVR may signal a severe proximal left coronary artery disease (LMCA or proximal LAD). Regardless whether it is caused by proximal left coronary artery disease or not, it is also an independent predictor

of mortality. Anterior/lateral AMI had a higher incidence of tachyarrhythmia and was more likely to die during than patients with inferior/ posterior AMI (11,3% vs 7,7%). The former was supplied by LCA, in which obstruction of its main artery might be evidenced by STE-aVR. Arrhythmias in LMCA obstruction leads to even more oxygen supply and demand imbalance.

REFERENCES

1. Wong C, Gao W, Stewart RAH, Benatar J, French JK, Aylward PEG, et al. aVR ST elevation: An important but neglected sign in ST-elevation acute myocardial infarction. Eur Heart J 2010;31(15):1845–53.
2. Das B, Mishra TK. Prevention and management of arrhythmias in acute myocardial infarction. International Journal of Contemporary Medical Research 2016;3(5):1401-1405.
3. Gorgels AP, Engelen DJ, Wellens HJ. Lead aVR, a mostly ignored but very valuable lead in clinical electrocardiography. J Am Coll Cardiol 2001;38:1355–6.
4. Gorgels AP, Vos MA, Mulleneers R, de Zwaan C, Bar FW, Wellens HJ. Value of the electrocardiogram in diagnosing the number of severely narrowed coronary arteries in rest angina pectoris. Am J Cardiol 1993;72:999–1003.
5. Yamaji H, Iwasaki K, Kusachi S, Murakami T, Hirami R, Hamamoto A, et al. Prediction of acute left main coronary artery obstruction by 12-lead electrocardiography. ST-segment elevation in lead aVR with less ST-segment elevation in lead V(1). J Am Coll Cardiol 2001;38:1348–1354.
6. Kosuge M, Kimura K, Ishikawa T, Ebina T, Shimizu T, Hibi K, et al. Predictors of left main or three-vessel disease in patients who have acute coronary syndromes with non-ST-segment elevation. Am J Cardiol 2005;95:1366.
7. Nikus KC, Eskola MJ, Virtanen VK, Vikman S, Niemelä KO, Huhtala H, et al. ST-depression with negative T waves in leads V4–v5 – a marker of severe coronary artery disease in non-ST elevation acute coronary syndrome: A prospective study of angina at rest, with troponin, clinical, electrocardiographic, and angiographic correlation. Ann Noninvasive Electrocardiol 2004;9:207.
8. Hanna EB, Glancy DL. ST-segment depression and T-wave inversion: Classification, differential diagnosis, and caveats. Cleve Clin J Med 2011;78:404.
9. Kosuge M, Ebina T, Hibi K, Morita S, Endo M, Maejima N, et al. An early and simple predictor of severe left main and/ or three-vessel disease in patients with non-ST-segment elevation acute coronary syndrome. Am J Cardiol 2011;107(4):495–500.
10. Aygul N, Ozdemir K, Tokac M, Aygul MU, Duzenli MA, Abaci A, et al. Value of lead aVR in predicting acute occlusion of proximal left anterior descending coronary artery and in-hospital outcome in ST-elevation myocardial infarction: an electrocardiographic predictor of poor prognosis. J Electrocardiol 2008;41(4):335–41.
11. Knotts RJ, Wilson JM, Kim E, Huang BD, Birnbaum Y. Diffuse ST depression with ST elevation in aVR: Is this pattern specific for global ischemia due to left main coronary artery disease? J Electrocardiol 2013;46(3):240–48.