

CASE REPORT

ST Elevation in Lead aVR with Malperfusion Syndrome: Sign of Severe Aortic Dissection

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ABSTRAK

Kenaikan segmen ST di elektrod aVR elektrokardiografi di dalam kes diseksi aorta mempunyai risiko kematian yang tinggi. Ia melibatkan diseksi yang berlaku di akar aorta. Kami melaporkan kes pesakit lelaki muda yang menghidap penyakit darah tinggi, yang datang ke Jabatan Kecemasan dengan sakit dada dan sakit di bahagian kaki. Pemeriksaan fizikal pada bahagian bawah kiri anggota adalah konsisten dengan iskemia akut. Elektrokardiogram menunjukkan serangan jantung akut di bahagian elektrod anterolateral, dan kenaikan ST segmen di elektrod aVR. Ekokardiografi menunjukkan akar aorta yang berukuran 4.51 cm dan kehadiran flap intimal, meningkatkan kebarangkalian diseksi pada akar aorta dan arteri koronari. Tomography Computed Angiogram menunjukkan terdapat diseksi aorta dari akar aorta termasuk flap intimal berhampiran permulaan arteri koronari kiri, hingga ke arteri iliac utama ke arteri iliac kiri. Malangnya, pesakit memilih untuk tidak menjalani pembedahan dan akhirnya meninggal dunia selepas 48 jam di masukkan ke wad. Kes ini menjelaskan bahawa, dalam kes diseksi aorta yang hadir dengan sindrom 'malperfusion', kenaikan segmen ST pada elektrod aVR akan meningkatkan kebarangkalian terjadinya diseksi aorta yang teruk melibatkan akar aorta dan juga arteri koronari, yang menjadikan prognosis pesakit lebih teruk.

Katakunci: diseksi aorta, kenaikan segmen ST aVR, sindrom malperfusi

ABSTRACT

Aortic dissection presenting with ST elevation in lead aVR of electrocardiogram is strongly associated with mortality. It is also associated with dissection involving the root of aorta and coronary vessels. We report a case of young male with hypertension, who presented with severe chest pain and unilateral lower limb

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pain. Physical examination of the left lower limb was consistent with acute limb ischemia. Electrocardiogram revealed acute anterolateral myocardial infarction together with ST elevation in aVR. Bedside transthoracic echocardiography showed a dilated aortic root measuring 4.51 cm with presence of intimal flap which raised the suspicion of dissection of root of aorta and left coronary artery. Computed tomography angiogram revealed aortic dissection from the root of aorta including the intimal flap near the origin of the left coronary artery, down to common iliac extending to the left iliac artery. Unfortunately, the patient opted for non-surgical intervention and succumbed 48 hours later. This case highlights that in case of aortic dissection, which presents with malperfusion syndrome, the presence of ST segment elevation at lead aVR should raise the suspicion for extensive aortic dissection involving the aortic root and left coronary artery which signifies unfavourable outcome.

Keywords: aortic dissection, malperfusion syndrome, ST elevation aVR

INTRODUCTION

aVR is the most commonly ignored lead among the 12 leads of electrocardiogram (ECG) (Gorgels et al. 2001; Hirata et al. 2010). Nevertheless, it plays significant role in improving accuracy of clinical diagnosis and prognosis of cardiovascular conditions, such as acute coronary syndrome, malignant arrhythmias and aortic dissection (AD) (Ali & Chrissoheris 2008; Kireyev et al. 2010; Riera et al. 2011). Stanford A Aortic Dissection (SAAD) is a life-threatening cardiovascular emergency which may present with ST elevation in ECG. Generally, any SAAD patients with ECG changes tend to have poor outcome and for ST elevation in lead aVR particularly, was associated with dissection involving the main trunk of left coronary artery (Hirata et al. 2010). SAAD patients who present with malperfusion which signifies end organ

damage are also strongly associated with mortality (Berretta et al. 2018). This case illustrates an extensive AD in a young man with hypertension whose ECG changes revealed ST elevation at lead AVR and anterolateral leads. We emphasize the discussion on ECG changes with extension of the AD and the importance of recognizing this ECG feature for management and prognostic value.

CASE REPORT

A 33-year-old male with underlying hypertension and history of stroke in the preceding year, presented to the Emergency Department (ED) with sudden onset of severe chest pain and diaphoresis which progressed to severe left lower limb pain. Upon assessment, he was fully conscious but in distress due to pain. Initial blood pressure (BP) was 207/80; pulse rate 70 beats/minute with respiratory rate of 20

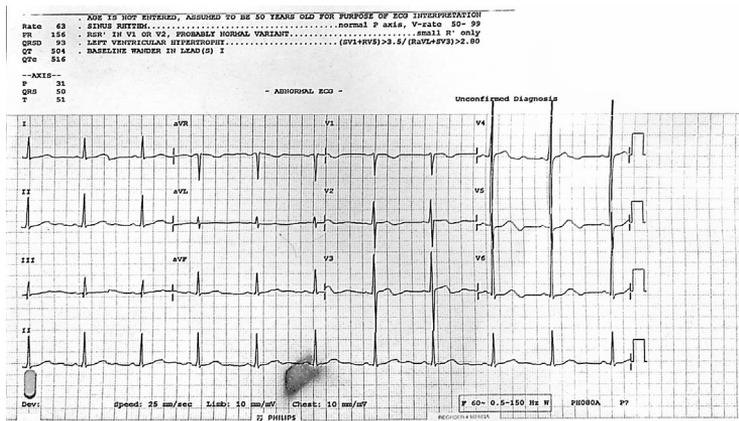


Figure 1: Initial ECG at presentation showing features of Left Ventricular Hypertrophy. Absence of any ST elevation at the precordial or limb leads.

breaths/minute and oxygen saturation of 100% on room air. Cardiovascular examination revealed end diastolic murmur at the aortic region. The left lower limb was cold and pale with absence of the popliteal, posterior tibialis and dorsalis pedis pulses. Other pulses were normal.

Initial ECG showed sinus rhythm with left ventricular hypertrophy (LVH) features without any ischemic changes (Figure 1). Subsequent ECG after 15 minutes, revealed ST elevation

involving all the precordial leads and lead 1, aVL and aVR (Figure 2). Bedside transthoracic echocardiography showed a dilated aortic root measuring 4.51 cm with presence of intimal flap (Figure 3). Abdominal aortic ultrasound also revealed the presence of intimal flap with aortic diameter of 2.6 cm. Computed tomographic angiography (CTA) confirmed SAAD of the aortic root, with the intimal flap involving the origin of the right and left coronary arteries (Figure 4). Dissection also

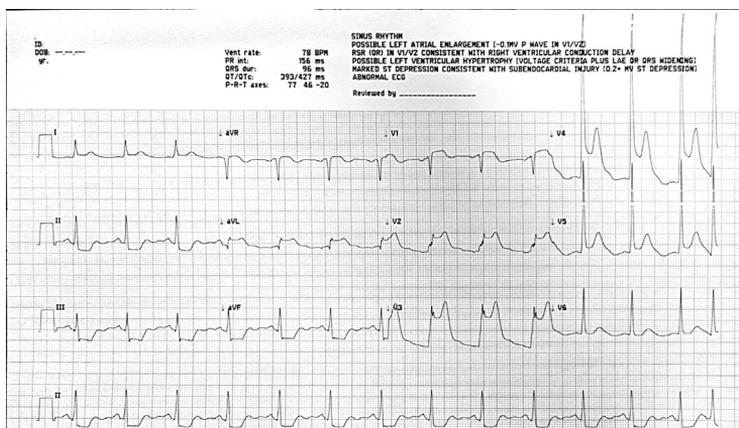


Figure 2: ECG showing ST elevation Lead I, aVL, aVR and from V1 to V5, along with reciprocal ST depression in inferior leads

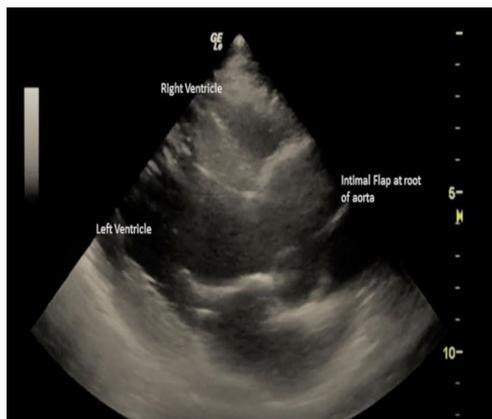


Figure 3: Bedside transthoracic echocardiography parasternal long axis view showing dilated root of aorta with presence of intimal flap.

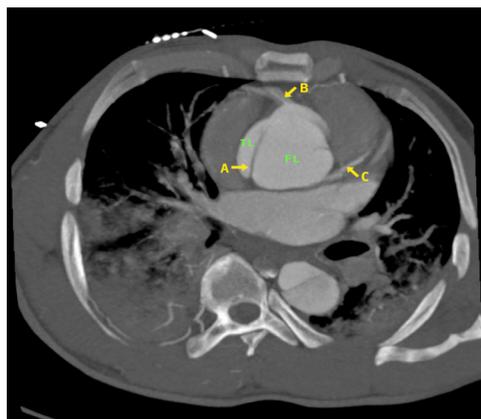


Figure 4: Computed Tomography Angiography showing proximal ascending aorta with intimal flap (A) at the root of the aorta separating the false lumen (FL) from the true lumen (TL) involving the right coronary artery (B) and left coronary artery (C).

extended up the ascending aorta, arch (Figure 5), carotid arteries, descending thoracic aorta, abdominal aorta down to the left common and external iliac artery.

Intravenous labetalol was commenced immediately with the dose of 10 mg boluses followed by infusion and subsequently intravenous nitroglycerin and nitroprusside was added to optimise his blood pressure. The patient was advised for emergency operative intervention by the cardiothoracic team. However, he declined despite detailed explanation of the high risk of death without surgical intervention. He was then admitted to the Surgical High Dependency Unit for medical management. Unfortunately, he developed left middle-cerebral-artery infarction complicated with acute kidney injury and malignant arrhythmias. He succumbed to death 48 hours later.

DISCUSSION



Figure 5: Intimal flap separating the false lumen (FL) from the true lumen (TL) at the arch of the aorta.

2000), whilst the incidence among the young patient is infrequently reported (Campillo et al. 2019). However, the characteristics and risk factors among young patients may differ from older patients such as congenital heart disease, Marfan syndrome and cocaine abuse (Campillo et al. 2019; Zhu et al. 2019). Hypertension as depicted in this case, is the most common and important underlying comorbidity for development of AD. Similarly based on a study on young AD in China, hypertension was reported in majority of the cases (Zhu et al. 2019). Atherosclerotic disease, bicuspid valves and connective tissue disease are among other risk factors (Chenkin 2017).

AD patients have myriad of non-specific manifestations hence being called "great masquerade" and it demands Emergency Physicians (EP) to always maintain high index of suspicion to avoid missing this lethal condition in ED (Elefteriades et al. 2008). The most common symptom is abrupt onset of chest pain (Hagan et al. 2000). However, patients may also manifest symptoms depending on the affected vessel branch which include malperfusion symptoms such as stroke like features, acute paraplegia, acute renal failure and acute limb ischemia as reflected in our case. Dissection involving aortic bifurcation, iliac arteries, femoral arteries and subclavian artery may manifest as acute limb ischemia with pulse deficit causing malperfusion syndrome. Due to the myriad of clinical presentations, one in every four patients with AD can be misdiagnosed (Pourafkari et

al. 2017). The management strategy and prognosis may also differ between Stanford Type A and B of AD. Stanford A involves the ascending aorta requiring operative repair whilst Stanford B is confined to descending aorta and may only require medical therapy (Hiratzka et al. 2010). Hence, early identification of SAAD during initial evaluation in the ED is important for prognostication and management strategy.

Based on International Registry of Acute Aortic Dissection Registry (IRAD) in 2005, patients with signs of myocardial ischemia in preoperative ECG or new sign infarction had poor outcome (Trimarchi et al. 2005). These changes were seen due to pre-existing atherosclerosis or dissection which extended to the coronary artery ostium or coronary ostia being covered by the flap occlusion at the coronary sinuses (Trimarchi et al. 2005). Previous studies have reported that 70% of patients with SAAD have ECG changes that include ST elevation, ST depression, T inversions, bundle branch block and many others (Biagini et al. 2007; Chien et al. 2012; Hirata et al. 2010). AD with ECG changes tend to be more severe with higher risk of death and in-hospital complications (Costin et al. 2018). Lead aVR is an augmented unipolar limb lead with the positive electrode being on the right arm. Despite often being ignored during electrocardiogram interpretation, changes in the complexes and segment such as P-QRS-ST-T in lead aVR is often characteristic in many clinical conditions and may improve accuracy of clinical diagnosis and

prognostication of cardiovascular conditions such as AD (Gorgels et al. 2001; Kireyev et al. 2010). SAAD patients who present with ST elevation in aVR carries poor prognosis and has been strongly linked with complications such as cardiogenic shock, pericardial tamponade, coronary ostial involvement especially the left coronary artery and increased in-hospital mortality (Kosuge et al. 2015). Pertaining to this case, the ECG findings showed significant ST elevation in precordial leads (Figure 2), and at limb leads aVR and aVL. Although studies have reported ST elevation in inferior leads being the common site for myocardial infarction (MI) in SAAD, ST elevation involving the anterior leads and lead aVR carries poorer prognosis (Chien et al. 2012; Hirata et al. 2010). This was supported by a study on SAAD patients in Japan that compared association of ST segment elevation (STE) in aVR lead and other ECG changes such as hypertrophy of the left ventricle, bundle branch block and ST changes in leads other than aVR with mortality (Kosuge et al. 2011). Among patients with STE in aVR who did not undergo surgical intervention, the mortality rate was reported to be as high as 83.3% (Kosuge et al. 2011). Furthermore, comparison among patients who underwent urgent surgical repair, patients with ST elevation in lead aVR had five times higher risk of in-hospital mortality than those of similar condition without aVR elevation (Kosuge et al. 2011).

In acute coronary syndrome (ACS) setting, ST elevation in aVR can be caused by occlusion to the left main stem

artery or left anterior descending artery leading to transmural ischemia, or may reflect ischemia of the left ventricle involving the subendocardial layer in association with severe three vessel disease or left main stem stenosis (Czerny et al. 2014). In the setting of SAAD, although the mechanism is still not fully understood, ST elevation in aVR is seen in complete obstruction of the left coronary artery ostium leading to ischemia involving the endocardial, myocardial and pericardial layer at the basal septum (Biagini et al. 2007). In the present case, CTA showed a dissection of the left coronary artery at the origin involving the ascending aorta just above the aortic valve, which concludes the hallmark of severe pathology with poor prognosis.

The second most common fatal complication of acute AD after rupture is malperfusion. Malperfusion occurs due to obstruction caused by the dissection that leads to absent or insufficient blood supply reaching the arterial branches (Hiratzka et al. 2010). Coronary malperfusion found to complicate 10-15% of SAAD cases (Hirata et al. 1995; Meszaros et al. 2000; Miller et al. 1979; Yagdi et al. 2006). Coronary malperfusion can be attributed to the severity of the dissection which extend into the ostium of the coronary artery, occlusion of the coronary sinuses secondary to the dissection flap, pre-existing diseased coronaries or a mixed of these factors. History and physical examination added with ECG features of ST elevation in lead aVR in this patient, should raise suspicion of coronary and lower abdominal aortic malperfusion

involving the myocardium and the lower limb. Pulse deficit is a marker of malperfusion and an independent predictor of early mortality among patients with AD (Trimarchi et al. 2005). There is a substantial variation in hospital mortality according to the number of vessels affected. About one in every four AD patients with no pulse deficit died in hospital whereas the mortality is around 56% among patients with three vessels involvement (Tamura 2014). Risk of operative mortality is increased with myocardial malperfusion, hence restoring the perfusion of the coronary is the only lifesaving option in this particular patient. Emergency percutaneous coronary intervention (PCI) provides treatment option as a bridging treatment although surgical aortic repair still remains the treatment of choice in this setting (Yagdi et al. 2006).

Nonetheless, in the present case, patient presented with malperfusion syndrome with high risk ECG features, but the operative intervention was declined, hence the detrimental outcome was expected.

CONCLUSION

In conclusion, Type A AD presenting with malperfusion syndrome such as pulse deficit, myocardial ischemia and acute limb ischemia indicate poor outcome. ECG findings such as ST elevation in lead aVR have clinically important diagnostic and prognostic implications in patients with type A AD as it signifies the possibility of left coronary artery ostial involvement.

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