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DENGUE PERIMYOCARDITIS; HYPOVOLEMIC VS CARDIOGENIC SHOCK?

Iskasymar Ismail[1], Tengku Suriani Tengku Ismail[2], Norzian Ismail[1]
[1]Universiti Putra Malaysia, Serdang, Selangor, Malaysia
[2]Hospital Serdang, Serdang, Selangor, Malaysia

INTRODUCTION

Cardiac involvement such as functional myocardial impairment, arrhythmia and myocarditis is not uncommon in dengue infections but it may have been frequently under-reported because it usually manifests as a mild and self-limiting condition. Dengue perimyocarditis is rare with only few cases reported worldwide. Nevertheless, more cases of dengue with cardiac involvement, which progress to acute heart failure, cardiogenic shock and death have been increasingly described.

CASE REPORT

A 16 years old boy, diagnosed dengue in defervescence phase with compensated shock, was transferred to emergency department (ED) from a primary care clinic. Upon arrival to ED, he had just completed 10ml/kg/hour normal saline infusion. In ED, patient was still in shock and blood gas analysis showed metabolic acidosis. Hence, 20ml/kg/hour gelafusine infusion was initiated. 12 lead ECG was performed upon noticing ST elevation in lead II on cardiac monitor and revealed saddle shape ST elevation in lead I, II, aVL, V2-V6. Bedside echocardiography showed poor cardiac contractility and presence of pericardial effusion. Patient had a cardiac arrest an hour after arrival in ED. Initial CPR (for 8 minutes) was successful but second cardiac arrest began 20 minutes later. Prior to second cardiac arrest, triple inotropes support was commenced. During second CPR, blood transfusion was started and intravenous dexamethasone was given. Despite the resuscitation efforts, patient was pronounced dead after two hours upon arrival in ED.

DISCUSSION AND CONCLUSION

In addition to circulatory shock due to plasma leakage and occult bleeding, the persistent shock in the above patient can be attributable to cardiogenic shock caused by myocarditis or pericarditis. Therefore, care should be taken not to cause iatrogenic fluid overload. Using echocardiography in dengue patient with features of heart failure and ECG changes, may help clinician to guide fluid resuscitation, identify patients at risk of fluid overload and allow early use of inotropes or use of other alternative therapies.

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HEART STOPPER!

J Ding[1], R Rahmat, S A Cha[1]
[1]Hospital Sultan Ismail, Johor Bahru, Johor, Malaysia

INTRODUCTION

Brugada syndrome is a cardiac sodium channelopathy associated with one of several ECG patterns characterized by incomplete right bundle-branch block and ST elevations in the anterior precordial leads. Autosomal dominant inheritance is found in 50% of case. It affects young males with structurally normal hearts who are prone to develop ventricular tachyarrhythmias leading to syncope, cardiac arrest, or sudden cardiac death.

CASE REPORT

An apparently heathy 35 year-old man suddenly collapses after complaining of dyspnoea. There was no prior history of chest pain, palpitations or syncope. He arrived in the emergency department with asystole. He was resuscitated as per ALS guidelines, ventilated, started on chest compressions and boluses of IV adrenaline. He then develop ventricular fibrillation, was defibrillated multiple times and given IV amiodarone. A return of spontaneous circulation was achieved after 40 mins. An ECG done showed ST elevation in aVR and widespread ST depression and the cause of his cardiac arrest was attributed to acute coronary syndrome. However an ECG done 2 hours later revealed a coved ST elevation V1-V2 followed by negative T wave, rearing the "ugly head" of Brugada syndrome. The further episodes of ventricular tachyarrhythmias and hemodynamic instability precluded transfer to a tertiary center. He succumbed the following day.

DISCUSSION

Brugada syndrome is diagnosed by both ECG and clinical criteria. There are 3 types of ECG abnormality but only type I (Brugada sign) is potentially diagnostic as in this case. One clinical criterion must also be fulfilled: Documented VF or VT, Family history of sudden cardiac death at <45 years old, Coved-type ECGs in family members, Inducibility of VT with programmed electrical stimulation, syncope or nocturnal agonal respiration. The only proven therapy is an implantable cardioverter-defibrillator. Quinidine is a possible alternative.

CONCLUSION

Diagnosis and early intervention of Brugada syndrome is key to preventing sudden death in affected young adults.

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THE MYSTIFYING DISAPPEARANCE OF MYOCARDIAL INFARCTION

Ruzaini R[1], Ng S G[1]
[1]Emergency and Trauma Department, Sarikei Hospital, Sarawak, Malaysia

INTRODUCTION

Transient ST elevation myocardial infarction (TSTEMI) is not classified in the universal definition of MI. Its existence is not well known hence we illustrated a case to share our experience, whereby we were mystified by the sudden disappearance of myocardial infarction.

CASE

A 40 year old gentleman, known case of hypertension, active smoker, presented with a sharp right sided chest pain. Physical examination: unremarkable. Initial ECG showed ST elevation in inferior leads, reciprocal changes in lead AVL, first degree heart block with no right sided heart involvement. Ten minutes later, there was no ST elevation seen in lead II on the cardiac monitor. A repeated ECG showed complete resolution of ST elevation. Troponin I: 11.38(raised). He received aspirin, clopidogrel and S/C clexane without thrombolysis. He remained clinically stable throughout admission. He was discharged well and an angiogram appointment was given.

DISCUSSION

Transient ST segment elevation is not a nosologic entity but rather a clinical sign that can be attributed by various conditions such as coronary