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

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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

thrombosis, vasospasm or tako-tsubo syndrome. In this case, this gentleman was diagnosed as TSTEMI based on the significant risk factors, history, ECG changes and raised troponin. Spontaneous reperfusion was achieved rapidly due to endogenous fibrinolysis and the presence of recruitable collateral vessels. Regarding the management, should this gentleman receive thrombolysis? The definitive treatment for TSTEMI remains unclear. However, according to a previous study by Meisel et all, data suggest immediate medical therapy with an early angiogram is an appropriate approach. Thrombolysis is not indicated as there is complete resolution of ST elevation.

CONCLUSION

Albeit there is no evidence of ongoing ischemia, patients with TSTEMI are at high risk of reocclusion. Hospital admission with continuous ECG monitoring is required. Intense medical therapy must be initiated if there is unavailability of a PCI centre.

KEY WORDS

Transient ST elevation, Myocardial Infarction

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OCCASIONALLY, I SWELL – A CASE REPORT OF DELAYED-ONSET ACE INHIBITORS-ASSOCIATED ANGIOEDEMA

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INTRODUCTION

Angiotensin Converting Enzyme (ACE) inhibitors-associated angioedema is a non-allergic, drug induced complication related to bradykinin accumulation. It usually affects the lips, tongue, face and can lead to death due to airway obstruction. Urticaria is absent. The incidence is rare between 0.1%- 0.2%, but the widespread use of ACE inhibitors mandates a special awareness by all clinicians.

CASE REPORT

62 year-old lady with no known allergy history, presented with progressive tongue and lips swelling throughout the day. Further history, few self-limiting milder episodes developed 4 months after perindopril was prescribed. Clinically, there was no urticaria, her airway was still patent. Standard treatment for anaphylaxis was initiated but progress of resolution was poor. Fortunately, symptoms resolved eventually and Perindopril was withheld. Patient has been symptom free until perindopril was restarted back 3 months after discharge due to uncontrolled hypertension. She was readmitted for another attack of angioedema.

DISCUSSION

This patient was on ACE inhibitor, developed episodic, non-urticarial angioedema over the lips and tongue. Symptoms free after discontinuation of perindopril confirmed the diagnosis of ACE inhibitors-associated angioedema. This complication usually begins as mild self-resolved episodic attacks even without discontinuation of ACE inhibitors. But the crescendo nature of severity will ultimately lead to life-threatening airway obstruction. The onset of angioedema in this patient was 4 months after initiation of perindopril. Although ACE inhibitors-associated angioedema typically occurs within 2 weeks of treatment but delayed-onset of angioedema up till to years had been reported. Unlike allergic angioedema, ACE inhibitors-associated angioedema