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A Rare Presentation of Prinzemetal Angina in Makurdi Nigeria: Case Report and Review of The Literature

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Abstract

Background: ***Prinzemetal angina is characterised by episodic chest pain at rest due to coronary artery vasospasm with transient ST segment elevation on the electrocardiogram and angiographically normal coronary arteries. In more severe cases, ST elevation may be followed by T-wave inversion for hours or days which eventually resolve. Rare complications of Prinzemetal angina include life threatening ventricular tachyarrhythmias, cardiogenic shock, severe heart failure and myocardial infarction.*** ***Case presentation:*** ***We report a case of a 41-year-old man who presented to the emergency department with severe chest pain, severe breathlessness and cardiogenic shock. Chest pain resolved following administration of nitrates. Result of electrocardiogram carried out after chest pain had subsided revealed Q wave in lead V1 and T-wave inversion in leads V1 – V4. Coronary angiography revealed normal coronary arteries. He was treated with losartan, antiplatelets, and statins with good results. He was discharged and followed up on above medications and has remained asymptomatic.*** ***Conclusion:*** ***Rare and life-threatening presentations of Prinzmetal angina require immediate therapy. Further attacks of angina can be prevented using nitrates, calcium channel blockers, renin-angiotensin system inhibitors and statins.***

Keywords: ***Prinzemetal angina, Coronary vasospasm, Cardiogenic shock.***

1. Introduction

In 1959, Prinzemetal et al [1] described a variant form of angina pectoris which was not precipitated by exertion and was associated with ST segment elevation on the electrocardiogram (ECG). It differed from the typical angina in that these patients characteristically had normal exercise tolerance with episodes of angina occurring during the late evening or early morning hours in individuals who are at rest or during sleep .It was later concluded that the angina was due to episodic vasospasm of coronary arteries that were not occluded by pathological processes such as

atherosclerosis, emboli, or spontaneous dissection [1-3]. This syndrome became known as Prinzmetal angina, variant angina or vasospastic angina.

The exact pathophysiologic mechanisms leading to coronary artery vasospasm are not yet completely understood. Patients with Prinzemetal angina exhibit few traditional cardiovascular risk factors but may have a history of smoking [4].

Atypical cases of Prinzemetal or variant angina do exist. Individuals exhibiting angina symptoms that are associated with ST-segment depressions on the ECG, that are triggered by exertion, and /or who have atherosclerotic coronary artery disease are still considered to have variant angina if their symptoms are caused by coronary artery vasospasm [2].

Prinzemetal angina may present with complications such as ventricular tachyarrhythmias or syncope [5,6]. Other rare but severe complications include myocardial infarction, cardiogenic shock and severe congestive heart failure. We hereby report a case of Prinzemetal angina in a 41-year-old man who presented with severe chest pain, breathlessness and cardiogenic shock resembling acute myocardial infarction.

2. Case Presentation

A 41-year-old male was rushed into the Emergency Department (ED) of a mission hospital around 6 am in the morning with a history of severe chest pain, palpitations, severe breathlessness and sudden collapse. This was triggered suddenly by a near head on collision with an oncoming vehicle from which he had quickly avoided. He was not a known hypertensive or diabetic patient but suffered from peptic ulcer disease. He had no significant alcohol or smoking history. He had a similar attack of the same illness one month prior to presentation and was treated in the same centre with full recovery. Three years earlier, he had undergone ECG which revealed features suggestive of myocardial infarction following an attack of mild chest pain.

On examination he was fully conscious with profuse sweating. Urgent random blood sugar was 123mg/dl. Cardiovascular examination revealed absent pulses with hypotension (blood pressure of 80/50 mmHg). He was immediately commenced on Intravenous normal saline infusion and 300mg aspirin was administered to be chewed. Sublingual nitroglycerin 500µg was given. Intramuscular adrenalin 1:10,000 at a dose of 1mg was administered. Vital signs were monitored hourly and immediate consult by a cardiologist was sought. Pulse and BP improved following administration of adrenalin and remained stable at a pulse rate of 95 beats per minute (bpm) and BP of 100/70mmHg nine hours later.

The cardiologist on arrival reviewed the patient and made an assessment of cardiogenic shock from repeat myocardial infarction. She added clopidogrel 75 mg daily to the aspirin 75 mg daily, sublingual nitroglycerin 500 µg as required, isosorbide dinitrate 5 mg daily, rosuvastatin 20 mg daily and rabeprazole 20mg twice daily. She requested for investigations to be carried out immediately.

Result of ECG revealed heart rate of 91bpm, sinus arrhythmia with T wave inversion in leads V1 to V4 and Q waves in lead V1 (Fig. 1). Cardiac troponin and creatine kinase MB could not be carried out within the locality. Echocardiography revealed normal sized cardiac chambers with interventricular septal wall motion abnormality and normal ejection fraction of 57.3%. Result of fasting lipid profile, fasting plasma glucose and kidney function tests were within normal limits. Patient was referred outside the town to Abuja for coronary angiography by the cardiologist as facilities were not available within the town.

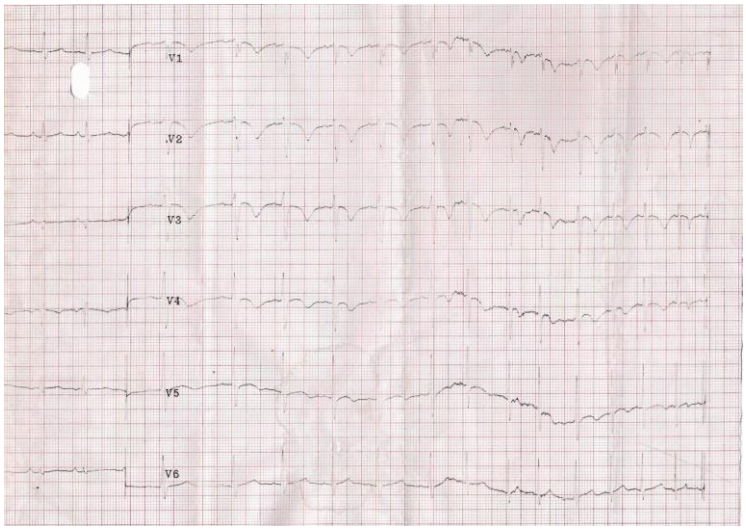

Fig. 1. ECG on day 2 following resolution of chest pain shows Q waves in lead V1 and T- wave inversion in leads V1 – V4.

On the second day chest pain had subsided and vital signs were stable but he had dyspnoea on mild exertion and became intolerant of the nitrates. Nitrates were discontinued and he was then placed on losartan 12.5 mg daily, intravenous frusemide 20 mg daily, anticoagulants, antiplatelets and statin therapy. He improved remarkedly and travelled to Abuja for coronary angiography.

Result of coronary angiography revealed normal coronary arteries (Figs. 4 and 5). He was discharged with a diagnosis of Prinzemetal angina on losartan, antiplatelets and statin therapy to be followed up as an outpatient. Amlodipine was not commenced at this point to avoid precipitating heart failure symptoms. ECG carried out on discharge revealed that T wave inversions in V1 – V4 were still present but Q waves had disappeared (Fig. 2).

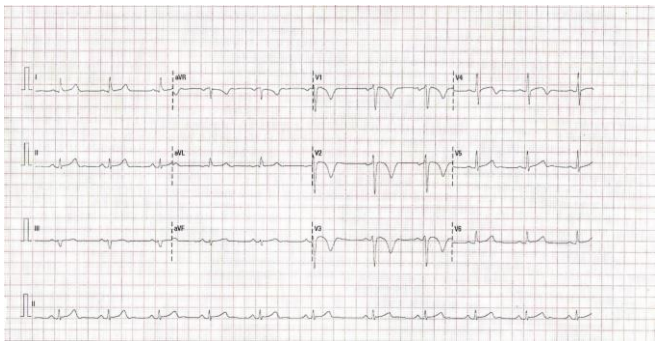

Fig. 2. ECG on day 16 showing absent Q waves in lead V1 and T wave inversion in leads V1 – V4.

Repeat ECG done 10 months later revealed normal findings. He had remained asymptomatic since discharge on losartan, antiplatelets and statins. He was lost to follow up till 21 months later when he reported for a checkup having remained asymptomatic. Repeat ECG done at 21 months revealed normal findings (Fig. 3). Amlodipine 2.5mg was then added to his regimen.

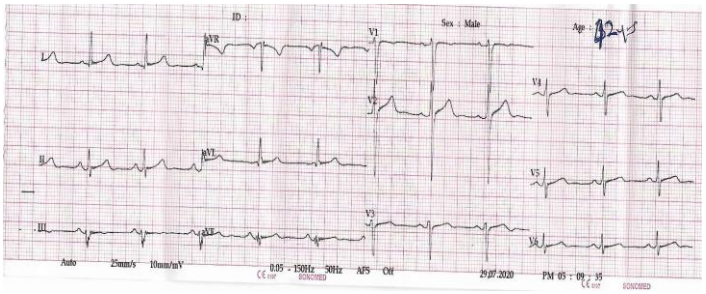

Fig. 3. ECG at 21 months showing normal findings in V1 – V4.

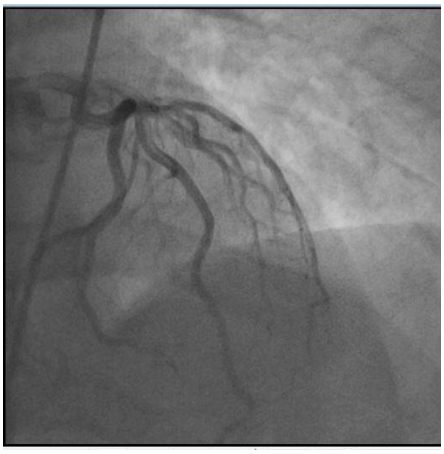

Fig. 4. Normal coronary angiography.

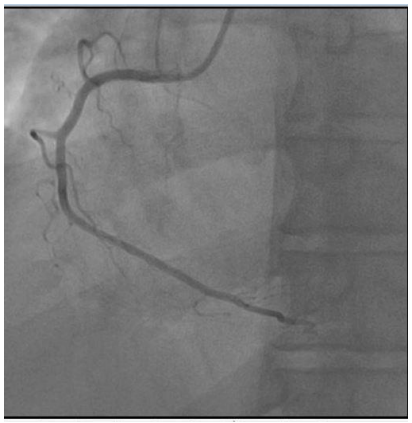

Fig. 5. Normal coronary angiography.

3. Discussion

Prinzemetal angina was first described by Prinzemetal et al [1] in a series of 32 cases presenting with nonexertional chest pain leading to ST segment elevation rather depression during attacks of pain. The angina episodes occur often at

the same time frequently either during the late evening or early morning hours and awakens the patient from sleep. The angina may be associated with syncope or arrhythmias or progress to myocardial infarction [1].

The case presented depicts a typical case of cardiogenic shock, transient myocardial injury and heart failure from coronary vasospasm. It was triggered by sudden fright while dodging an oncoming vehicle. In many cases, coronary artery vasospasm can occur spontaneously without an identifiable cause. Known triggers of spasm in susceptible patients include hyperventilation, cocaine, histamine, catecholamine-like stimulants, tobacco use, and administration of provocative agents such as acetylcholine, ergonovine, histamine, or serotonin [7].

The pathophysiologic mechanism leading to coronary vasospasm is not entirely clear but there are three relevant hypothesis: The first is enhanced contractility of coronary vascular smooth muscle due to reduced nitric oxide (NO) bioavailability caused by a defect in the endothelial NO synthetase enzyme which leads to endothelial function abnormalities [8,9]. The second is that thromboxane A2, serotonin, histamine, and endothelin which are vasoconstrictors released by activated platelets can be released due to abnormal platelet activation leading to coronary vasospasm [8,10]. The third is increased alpha-adrenergic receptor activity in epicardial coronary arteries or the excessive release of the 'flight' or 'fight' catecholamines (e.g. nor adrenalin) that activate these receptors may lead to coronary vasospasm [8,11].

The absence of significant angiographic evidence of coronary artery disease (CAD) in a patient presenting with angina at rest and transient ST-segment elevation is highly suggestive of Prinzemetal angina. The ST-segment elevation is usually only present during attacks of pain and tends to resolve within minutes. It may be followed by T-wave inversion lasting for hours or days in more severe cases [12]. This was probably the case in our patient who had angina at rest with transient T-wave inversion and normal coronary arteries leading to the diagnosis of Prinzemetal angina. In this group of patients, transient ST-segment elevation and non-sustained arrythmias may be detected using Holter monitoring.

Following diagnosis of Prinzemetal angina, calcium channel blockers (CCBs) and long acting nitrates are regarded as first line treatment for the prevention of further attacks of angina pectoris. Nifedipine, amlodipine, verapamil and diltiazem are considered effective. However, amlodipine may be preferable because of its long half-life [13].

Our patient was given losartan instead of amlodipine due to heart failure symptoms and he remained asymptomatic even when the heart failure symptoms resolved. Coronary vasospasm can lead to endothelial dysfunction and the renin-angiotensin system (RAS) has been linked to endothelial dysfunction. Choi et al investigated the impact of RAS inhibitor on long term clinical outcome in patients with vasospastic angina. A total of 3349 patients were divided into two groups based on the use of RAS inhibitor or not. After propensity score matching (PSM), two matched groups were generated and their baseline characteristics balanced. During the 5-year clinical follow up, the RAS inhibitor group showed a lower incidence of recurrent angina, total death, and total major adverse cardiovascular events (MACE) than the non-RAS inhibitor group [14].

It has been reported that the administration of the endothelin antagonist bosetan [15] can lead to complete resolution of symptoms of variant angina. Prospective studies have also shown that addition of 30mg daily of fluvastatin to CCBs for six months significantly reduced the occurrence of coronary vasospasm in patients with vasospastic angina [16]. The case presented remained asymptomatic on a combination of losartan, rosuvastatin and antiplatelet agents.

Severe and rare complications of variant angina require immediate therapy and usually present to the ED. These include life threatening ventricular arrhythmias, atrioventricular block, cardiogenic shock, severe heart failure, and myocardial infarction. Sudden cardiac death (SCD) has also been reported in a few case reports [17,18]. Such patients may require implantation of an internal defibrillator and /or cardiac pace maker to stop such arrhythmias and restore normal sinus rhythm [19]. In all these emergency cases, percutaneous coronary intervention to stent areas with evidence of spasm is only useful in individuals with concomitant coronary atherosclerosis [2].

4. Conclusion

Prinzemetal angina is angina at rest due to vasospasm of normal coronary arteries associated with transient ECG changes. They are usually stabilized on calcium channel blockers and/or long-acting nitrates. Some cases may also be stabilized on RAS inhibitors and statins like the case presented. Rare and life-threatening complications may present to the ED and require emergency treatment and sometimes, interventional therapy.

5. Conflict of Interest

None declared

6. Ethical Approval

Not applicable

7. Informed Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

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