*Case Report*

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# Anterior Wall Myocardial Infarction; Challenges in Recognition and Management in an Intellectually-Disabled Patient in the Emergency Department

**Tanmay Kumar Jha[1], Ritu Sabharwal[2], Chintala Venkata Sai Chiranjeevi[3], Sarat Kumar Naidu[4], Dheeraj Bhaskaran Nair[5]**

**Author's Affiliation:**
[1]Resident [2,4]Attending Consultant [3]Senior Resident [5]Head of Department, Department of Emergency Medicine, Max Super Speciality Hospital, Vaishali, Ghaziabad, Uttar Pradesh 201012, India.

**Corresponding Author:**
**Tanmay Kumar Jha**
Resident, Department of Emergency Medicine, Max Super Speciality Hospital, Vaishali, Ghaziabad, Uttar Pradesh 201012, India.
E-mail: drtanmaykumarjha@gmail.com

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**Abstract**

Acute MI would rarely be a first differential diagnosis in a young boy of 19 years old withintellectual disabilitywith non-specific complaints.

We present a case of a 19-year old boywithhistory of seizure disorder, who presented to the Emergency Department with complaint ofnon-specific chest discomfort since1 day.While being examined, he had one episode of seizure after which he suddenly developed cardiac arrest;he was immediately given high quality CPR along with DC shock (initial rhythm = VF) as per ACLS protocol. Post ROSC2D Echo revealed LV dysfunction with LVEF~ 40%. Post ROSC ECG revealed AWMI. Patient was loaded with antiplatelets, statins and anticoagulants. Patient underwent CAG which revealed single vessel disease. Subsequently, he underwent primary PTCA+ Stent (DES) to LAD with good end result. He was discharged in a stable condition after 6 days.

**Keywords:** ST Elevation Myocardial Infarction (STEMI); Anterior Wall Myocardial Infarction (AWMI); Acute Myocardial Infarction (AMI); Left Anterior Descending Artery (LAD); Coronary Angiography (CAG); Primary PTCA + Stent to LAD; Intellectual Disability; Ventricular Fibrillation (VF); Cardiopulmonary Resuscitation (CPR); DC Shock; Seizure Disorder; Generalized Tonic-Clonic Seizure (GTCS).

## Introduction

Acute Myocardial infarction in a mentally disabled and a seizure disorder patient is often missed or misdiagnosed because of the non-specificity of signs and symptoms and non-reliability of history.

Acute coronary syndrome (ACS) is an umbrella term representing a common end result, acute myocardial ischemia: Including ST-segment elevation myocardial infarction (STEMI), non-ST-STEMI (NSTEMI), and unstable angina (UA). In simple terms, ACS is the situation where the blood supplied to the heart muscle is suddenly blocked [2].

ST-segment-elevation myocardial infarction (STEMI) occurs when a coronary artery becomes blocked by a blood clot, causing the heart muscle supplied by the artery to die. It belongs to a group of heart conditions known as acute coronary syndromes [2].

Anterior STEMI results from occlusion of the left anterior descending artery (LAD). Anterior myocardial infarction carries the worst prognosis of all infarct locations, mostly due to larger infarct size.

The ECG findings of an acute anterior myocardial infarction wall include:

1. ST segment elevation in the anterior leads (V3 and V4) at the J point and sometimes in the septal or lateral leads, depending on the extent of the MI. This ST segment elevation is concave downward and frequently overwhelms the T wave. This is called "tombstoning" for obvious

reasons; the shape is similar to that of a tombstone.

2. Reciprocal ST segment depression in the inferior leads (II, III and aVF).
3. According to the American College of Cardiology/ American Heart Association guidelines for STEMI, there must be "new ST segment elevation at the J point in at least two contiguous leads of ≥ 2 mm (0.2 mV) in men or 1.5 mm (0.15 mV) in women in leads V2-V3 and/or of ≥ 1 mm (0.1 mV) in other contiguous chest leads or the limb leads." This means 1 millimeter in any two contiguous leads, except leads V2 or V3, where the elevation must be 2 mm in men or 1.5 mm in women.

The risk factors for AWMI are same as that of other myocardial infarctions like diabetes, hypertension, hyperlipidemia, smoking etc. Typical presentation of MI is easy to diagnose but it becomes challenging if the patient is mentally disabled and has a history of seizure disorder and with very vague and non-specific symptoms.

CHD in young adults is not as well characterized as CHD in older individuals because it occurs less frequently, but this disease can have devastating consequences for young patients and their families.

As in older adults, the majority of coronary events in young adults are related to atherosclerosis, and one or more of the traditional CHD risk factors is typically present. Young patients, however, are more likely than older patients to be smokers, male, obese, and to have a positive family history. Risk factor reduction is thus of major importance in managing young CHD patients.

Approximately 20% of CHD in young adults, however, is related to non-atherosclerotic factors, such as coronary abnormalities, connective tissue disorders, and autoimmune diseases. Cocaine and other illicit drug use have been increasingly associated with acute myocardial infarction and accelerated atherosclerosis.

The differences in etiologies and risk profiles of younger and older CHD patients result in differences in disease progression, prognosis, and treatment [3].

AMI in young patients presents distinct clinical characteristics, a different treatment, management and outcome with respect to the older group [4].

Hence, it is important to identify the major risk factors and clinical profile of ACS in young adults so that future preventive measures can be taken in the form of lifestyle modification and pharmacotherapy. According to recent epidemiological studies, more than half of the worldwide cardiovascular disease burden will be borne by the Indian subcontinent in the next decade [1].

**Case Study**

A19-year old boyof Indian origin with intellectual disability and history of seizure disorderwas brought to the emergency department at around 05:00pm by his motherwith complaints ofchest discomfort and palpitations since one day.

The mother alsomentioned that the patientwas intellectually disabled for which he was under treatment. The patient and his mother thought the symptoms were dueto the stress from his ongoing exams in school.

He also had history of seizure disorder for which he was on oral Cap. Gabapentin 300 mg OD, Tab. Oxcarbazepine 300 mg OD.

There was no history of nausea, vomiting, sweating episodes, radiation of chest pain, syncope.

He had multiple ED visits in the past due to similar complaints but all investigations were insignificant and a probable diagnosis of anxiety neurosis was made in the past.

Physical examination revealed the child was conscious and cooperative. His respiratory rate was18/min. His pulse rate was 100/min, BP was 100/60mmHg, RBS was 114mg/dL, His $SpO_2$ was 98% on room air.

He didn't have any pallor, icterus, cyanosis, jugular venous distention, pedal edema or lymphadenopathy.

Neurological, cardiovascular, respiratory and per abdominal examinations were insignificant.

He was attached to a cardiac monitor. A large IV. cannula was inserted in left cubital vein and samples were taken for ABG, cardiac enzymes and other routine investigations like CBC, LFT, KFT.

While being examined, he had one episode of GTCS and became unresponsive. Initially it was thought to be a post-ictal episode but carotid pulses were not palpable and the defibrillator monitor showed VF.

High quality CPR was immediately started as per ACLS protocolwith AMBU bag ventilation with the ratio of 30 compressions to 2 effective breaths.

Biphasic Defibrillator was charged to maximum dose (200 J) immediately and after application of jelly to the defibrillator pads,shock was delivered after clearing the patient and switching compressor roles.

High quality CPR was continued for 2 minutes after shock. Defibrillator rhythm was analyzed

which showed normal sinus rhythm. Carotid pulse was immediately checked which was palpable and there was visible chest rise which was then confirmed with auscultation.

ROSC was achieved after 6 cycle of CPR, with patient's GCS returning to 15/15 immediately (E4V5M6).

He started talking and following commands. Nasal cannula was applied to the patient and the oxygen flow titrated to keep $SpO_2$ >94%.

His systolic BP was calculated to be 94 mm Hg, so no I.V. bolus of crystalloids or inotrope infusion was started in Emergency Department.12-lead ECG showed ST elevation suggestive of Anterior wall MI.

Since the patient was already following commands, Targeted Temperature management wasn't considered. Phenytoin infusion was also started for his seizures and a neurologist consult was sought.

After stabilization in the ED the patient was immediately shifted for coronary angiography along with ED personnel and cardiologist with defibrillator attached, pulse oximeter and a ready crash cart to the catheterization laboratory. The door to balloon time in this case was 47 minutes.

**Course in the Hospital and Outcome**

Aprovisional diagnosis of Acute Anterior Wall Myocardial Infarction, post CPR, LV dysfunction (LVEF~40-45%) was made and the patient shifted immediately tocatheterization laboratory.

Patient underwent Coronary Angiography immediately which showed mid thrombotic lesion with 90% stenosis in LAD (Single Vessel Disease) and thus confirming the diagnosis.

Subsequently he underwent primary PTCA + Stent (DES) to LAD

Post procedure patient developed hypotension, for which patient was put on inotropic support (Noradrenaline).

In view of deranged LFT, Gastroenterology opinion was taken and advice incorporated. Patients cardiac enzymes were elevated beyond the normal range which just added to the already confirmed diagnosis.

Subsequently, patient's clinical condition improved, inotropic supports were gradually tapered-off and stopped. The patient was discharged in a stable condition after 6 days.

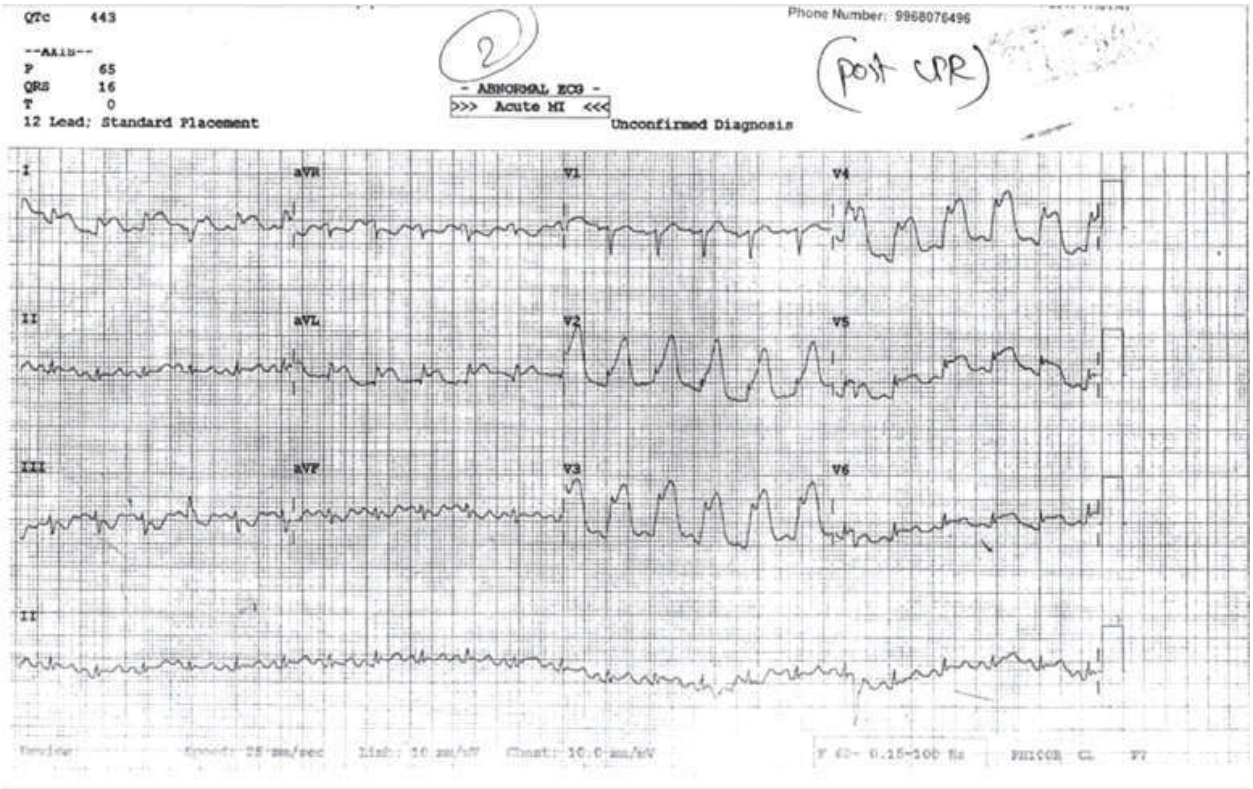


**Fig. 1:**

**Discussion and Therapeutic Considerations**

A good and immediate clinical history is one of the hallmarks of diagnosing a case of Acute Coronary Syndrome (ACS). This scenario becomes very difficult when the patient (intellectually disabled in this case) or attendants can't give accurate history.

The most common symptom of myocardial ischemia and myocardial infarction is retrosternal chest discomfort. The patient may perceive this discomfort more as a pressure or tightness than actual pain.

*Symptoms suggestive of ACS may also include*

- Uncomfortable pressure, fullness, squeezing or pain in the center of the chest lasting more than several minutes.
- Chest discomfort spreading to the shoulders, neck, one or both arms, or jaw.
- Chest discomfort spreading into the back or between the shoulder blades.
- Chest discomfort with light-headedness, dizziness, fainting, sweating, nausea or vomiting.
- Unexplained sudden shortness of breath, which may occur with or without chest discomfort.

In our case, it was difficult to suspect ACS with his given history and symptoms because he had multiple similar episodes and also his symptoms were not very typical.Also in a mentally-challenged young person, it is not very common to suspect ACS as the first differential diagnosis.

However due to strong vigilance and quick response in dealing with the patient's condition,it was possible for us to revive the patient in a timely manner.

When the patient initially had GTCS he was given Inj. Lorazepam 4 mg i.v. stat

After ROSC was achieved, and with the patient fully conscious, alert and the 12-lead ECG was suggestive of AWMI the patient was given Tab. Ecosprin 325 mg, Tab. Ticagrelor180 mg, Tab Atorvastatin 80 mg and Inj.Heparin 5000 IU i.v.

An Emergency physician can't think of a diagnosis of ACS or myocardial ischemia in a young patient without proper history and order a 12-lead ECG without evaluating the patient. In the patients or attendants who can't give proper history as in this case report it becomes very challenging for us to diagnose, treat and refer.

*Considerations for the use of PCI remain the following*

1. PCI is the treatment of choice for the management of STEMI when it can be performed effectively with a door-to-balloon time of less than 90 minutes from first medical contact by a skilled medical provider at a skilled PCI facility.
2. Primary PCI may also be offered to patients presenting to non-PCI-capable centers if PCI can be initiated promptly within 120 minutes from the first medical contact.
3. PCI is also preferred in patients with contraindications to fibrinolytics and is indicated in patients with cardiogenic shock or heart failure complicating MI [5].

The treatment of choice for the management of ST-elevation myocardial infarction is coronary angioplasty with or without stent placement. The goal of health care providers in managing patients with this type of acute coronary syndrome (ACS) is to treat within the first hours of the onset of symptoms.Percutaneous coronary intervention (PCI) can restore flow of blood into the myocardium in more than 90% of patients if performed by a skilled provider at a proficient PCI facility with a "door-to-balloon" time of less than 90 minutes [6].

Unstable angina and acute non-ST elevation myocardial infarction (NSTEMI) are medical emergency sub-types of acute coronary syndrome.

The patients that develop hemodynamic instability, heart failure, persistent angina at rest despite intensive medical therapy, mitral regurgitation, formation of a new ventricular septal defect, or sustained ventricular arrhythmias are at extremely high risk. Either way, both of these types of patients should immediately undergo coronary arteriography and revascularization. Fibrinolysis should be avoided in NSTEMI or unstable angina.The choice of revascularization procedure after angiography is dependent on the size and location of the blockage.

PCI is most often performed on appropriate lesions, while coronary artery bypass grafting (CABG) is preferred in left main or equivalent disease, such as two or three vessel disease involving the left anterior descending artery with left ventricular dysfunction [6].

Some issues relate to the performance of PCI and these include the development of bare metal stents (BMS), which have surpassed balloon angioplasty in the management of coronary artery disease because of their ability to prevent restenosis by suppressing arterial recoil and contraction.

However, 10% to 20% of patients may still experience restenosis after one year due to excessive growth of a neointima. These have been reduced due to the emergence of drug eluting stents (DES), which have the ability to reduce revascularization rates by as much as 70%. For this reason, DES is preferred in the majority of PCI procedures [6].

Coronary angiography and percutaneous intervention rates significantly decreased with age. The rate of Coronary artery bypass graft (CABG) surgery was highest among patients aged 65-74 years (8.1%) and 55-64 years (7.7%), but reduced in the youngest (4.7%) and oldest (2.7%) groups.

Major bleeding rates were 2-3% among patients aged < 65 years, and > 6% in those > or = 85 years. Hospital-mortality rates, adjusted for baseline risk differences, increased with age (odds ratio: 15.7 in patients > or = 85 years compared with those < 45 years) [7].

In patients who achieve ROSC after OHCA, morbidity and mortality remain significant in part because of the development of a specific post cardiac arrest syndrome. To achieve improved survival and improved neurological outcomes, it will be necessary to develop and adopt a systematic approach to all elements of the pathophysiological process.

Treatment strategies focusing on both prehospital and postresuscitative care are vital to improving patient outcomes, and may be further optimized with the development of regional systems of care.

Specifically, emphasis should be placed on the development of specialist centers that offer goal-directed therapiesearly coronary angiography, and temporary circulatory support when appropriate, together with comprehensive neurological assessment and therapy [8].

Patients with resuscitated cardiac arrest should be included in existing "STEMI networks" with direct transportation to the specialized "cardiac arrest centers" of excellence. Because of critical role of immediate CAG and PCI, interventional cardiologists should be an essential member of post-resuscitation team.

However, when treating post CA patients we should avoid futility. In unfavorable settings of cardiac arrest (unwitnessed arrest, long delays to pre-hospital team arrival, no BLS, "non-shockable" first rhythm, long ACLS, recurrent arrest) or severe pre-arrest comorbidities, aggressive post-resuscitation treatment is not likely to result in quality survival [9].

## Conclusion

Diagnosing an anterior wall STEMI is not difficult if there is a typical history from a intellectually-sound patient. But it becomes not only difficult but also the history becomes unreliable if the patient is mentally not sound and if such a patient had multiple ED visits with similar complaints without a definitive diagnosis as seen in our case report.

Therefore we as ED physicians must be well aware of such a critrical lifethreatening diagnosis in any patient visiting the ED. More strict monitoring is required in a mentally-challenged patient even if the symptoms are non-specific and even if the patient had multiple ED visits without a conclusive diagnosis.

Myocardial infarction can develop in any person irrespective of age and irrespective of mental capacity. Therefore proper evaluation is of utmost importance and a high degree of suspicion is required to diagnose it early.

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