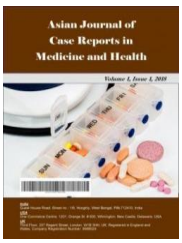

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Type 2 Myocardial Infarction- A Case Report

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Author's contribution

The sole author designed, analysed, interpreted and prepared the manuscript.

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Case Study

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ABSTRACT

Type 2 MI (T2MI) was defined as myocardial infarction other than due to coronary artery disease (CAD), produced by disparity between supply and demand of oxygen .Cases were reported in literature where T2 MI was diagnosed even in presence of even 90 % block in coronaries, where recent ischemic changes were shown to be not due to the CAD but to disparity between supply and demand of oxygen. There was considerable overlap with the classical type1 NSTE MI and T2 MI and distinguishing between the two was considered challenging. Though T2 MI constituted about 25% of all cases of MI, the centres reporting Type 2 MI ranged between 0-13% only. Type 2 MI, the new heterogeneous group, was officially recognised by the task force on the universal definition of MI in 2007. The scope and criteria were changing since it was defined in 2007. Further, it appeared that -coronary cause of MI was more important than disparity between supply and demand of myocardial oxygen supply. For instance Sepsis, one of the important causes of T2 MI, could cause Type 2 MI by myocardial depression even in presence of normal oxygen perfusion.

A case reported initially as NSTEMI, was retrospectively considered the possibility of T2MI. The reasons there of and the intricacies in the diagnosis of T2 MI are discussed in this article.

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Keywords: Type 2 MI; Myocardial depression; Low EF; Sepsis as a cause of T2 MI.

1. INTRODUCTION

Type 2 MI was defined as cardiomyocyte necrosis caused by conditions other than atherosclerotic coronary artery disease (CAD) and secondary to decrease in oxygen supply (e.g. hypoxemia, anaemia, hypotension, and endothelial dysfunction) and / or increased demand (e.g. tachycardia, arrhythmia, and sepsis) [1]. The main causes were anaemia, followed by sepsis, arrhythmia and post-operation. Sepsis as a cause of type-II MI was more common among patients presenting with STEMI compared those presenting with NSTEMI (40.7% vs. 19.2%, This was explained by the fact that NSTEMI and unstable angina were caused by partial (incomplete) coronary artery occlusion. A partial occlusion results in a reduction of coronary blood flow and this causes sub endocardial ischemia. Based on the current guidelines, differentiating between patients with type 2 myocardial infarction and acute myocardial injury was challenging as there remained overlap between these two clinical entities, and classification was therefore inconsistent in clinical practice [2]. The International Classification of Diseases (ICD) coding system does not recognize type 2 myocardial infarction (T2 MI) as a separate entity; therefore, patients with type 2 MI continue to be categorized under the general umbrella of non–ST-segment–elevation myocardial infarction (NSTEMI).

2. THE CASE REPORT

A 69 year old patient, presented with rest angina and exertional dyspnoea of 24 hrs duration. For four days prior to this, he suffered a pan-sinusitis associated with fever, not responding to cefpodoxime and NSAIDS. Few days before, he visited a hospital to attend on a relative for five days. The unremitting sinus pain and unresponsiveness to cefpodoxime was thought to be due to hospital- acquired infection. At the cardiology OPD his oxygen saturation was found to be 86 %, pulse rate was 140 bpm, regular and low volume and his BP was 90 / 70 mm Hg. There was severe LVF and lungs were congested. The ECG showed findings consistsnt with ACS -Unstable Angina. He was admitted immediately in ICU. The ECG showed anterior and inferior wall ischemia, RBBB and LPHB with sinus tachycardia (HR 140 bpm) The ECG reports at admission (Fig. 1A) and at review after one year were presented (Fig. 1B). The 2D echo (Fig. 2A, Fig. 2B) showed low EF of 30% with severe systolic dysfunction, hyporkinesia of the anterior and inferior walls and mild MR and serial improvement in EF was shown in Table 1 .The angiogram (Fig. 3) showed diffuse TVD (triple vessel disease)with 90 % block in LCF and 70 % block distally and 50 to 60% block proximally. In LAD, with Trop T (troponin -T) becoming positive when tested in ICU, the initial diagnosis of unstable angina was revised to. CAD- Anterior wall NSTEMI, with old inferior wall MI showing fresh ischemic changes. Past h/o of DM 2 for nearly three decades but not hypertensive. In 2004 he had inferior wall MI and was treated with primary angioplasty and stent to RCA.

3. DISCUSSION

Significant CAD was demonstrated in 75% of T2 MI patients as opposed to 55% reported in the literature. One third of the T2 MI patients had in fact angiographic diagnosis of T1 MI, with plaque rupture. In T2 MI patients, the element of inflammation and hyper-coagulation cause acute progression of pre-existing coronary lesions (T1MI), on top of the oxygen mismatch myocardial damage (T2 MI) [3].

The pros and cons in favour of and against T1 MI or T2 MI are discussed below.

3.1 Type 1 (NSTEMI) or Type 2 MI?

The global task force, reviewed the universal definition of myocardial infarction and recognised the need to provide clearer diagnostic criteria and guidance [4]. Based on the current guidelines, differentiating between patients with type 2 myocardial infarction and acute myocardial injury is challenging as there remains overlap between these two clinical entities, and classification is therefore inconsistent in clinical practice [5]. The International Classification of Diseases (ICD) coding system does not recognize type 2 myocardial infarction (MI) as a separate entity; therefore, patients with type 2 MI continued to be categorized under the general umbrella of non–ST-segment–elevation myocardial infarction (NSTEMI).

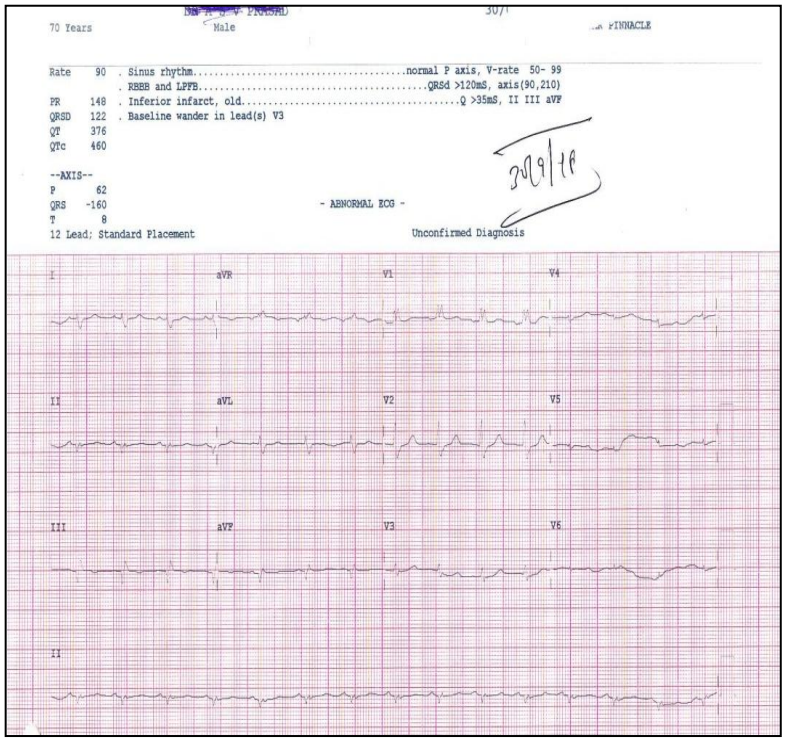

70 Years
Male
PINNACLE
Rate 90
PR 148
QRSD 122
QT 376
QTc 460
. Sinus rhythm.....normal P axis, V-rate 50- 99
. RBBB and LPFB.....QRSd >120mS, axis(90,210)
. Inferior infarct, old.....Q >35mS, II III aVF
. Baseline wander in lead(s) V3
--AXIS--
P 62
QRS -160
T 8
12 Lead; Standard Placement
- ABNORMAL ECG -
Unconfirmed Diagnosis
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

Fig. 1 (A)

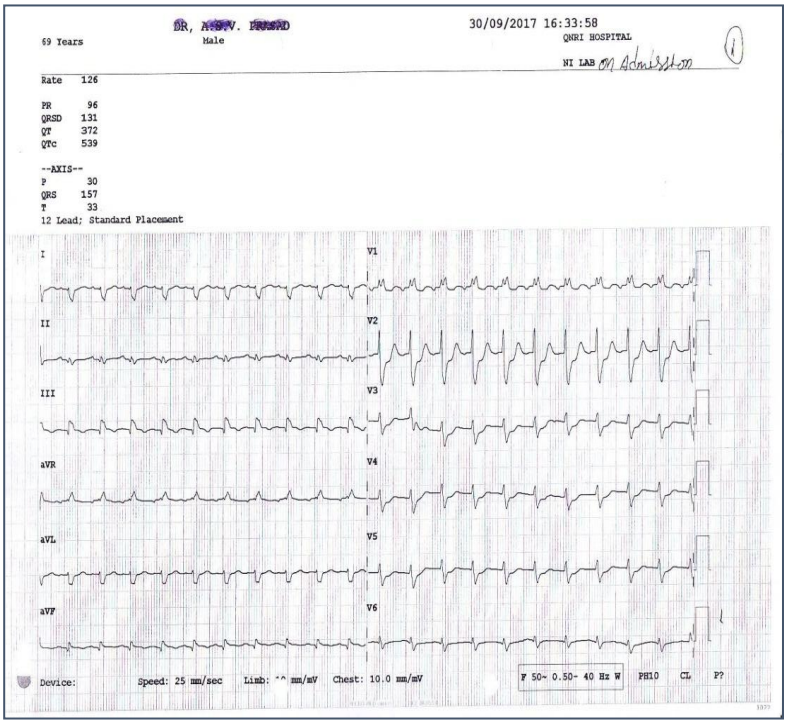
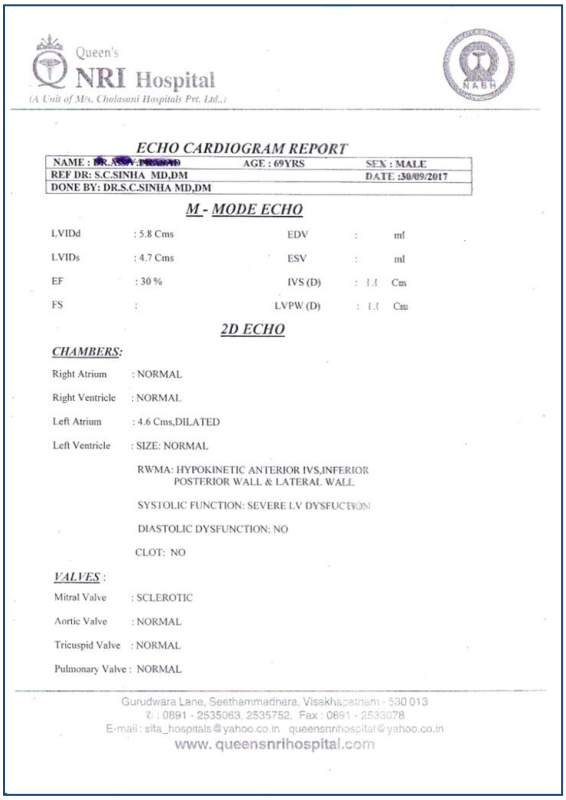

69 Years
Male
30/09/2017 16:33:58
QNRI HOSPITAL
NI LAB on Admission
Rate 126
PR 96
QRSD 131
QT 372
QTc 539
--AXIS--
P 30
QRS 157
T 33
12 Lead; Standard Placement
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
Device:
Speed: 25 mm/sec
Limb: ^^ mm/mV
Chest: 10.0 mm/mV
F 50~ 0.50~ 40 Hz W
PH10
CL
P?

Fig. 1 (B)



Queen's
NRI Hospital
(A Unit of M/s. Chalasani Hospitals Pvt. Ltd.,)

ECHO CARDIOGRAM REPORT

NAME : [redacted]	AGE : 69YRS	SEX : MALE
REF DR: S.C.SINHA MD,DM		DATE :30/09/2017
DONE BY: DR.S.C.SINHA MD,DM		

M - MODE ECHO

LVIDd	: 5.8 Cms	EDV	:	ml
LVIDs	: 4.7 Cms	ESV	:	ml
EF	: 30 %	IVS (D)	: 1.1	Cm
FS	:	LVPW (D)	: 1.1	Cm

2D ECHO

CHAMBERS:

Right Atrium : NORMAL
Right Ventricle : NORMAL
Left Atrium : 4.6 Cms,DILATED
Left Ventricle : SIZE: NORMAL

RWMA: HYPOKINETIC ANTERIOR IVS,INFERIOR POSTERIOR WALL & LATERAL WALL

SYSTOLIC FUNCTION: SEVERE LV DYSFUCTION

DIASTOLIC DYSFUNCTION: NO

CLOT: NO

VALVES :

Mitral Valve : SCLEROTIC
Aortic Valve : NORMAL
Tricuspid Valve : NORMAL
Pulmonary Valve : NORMAL

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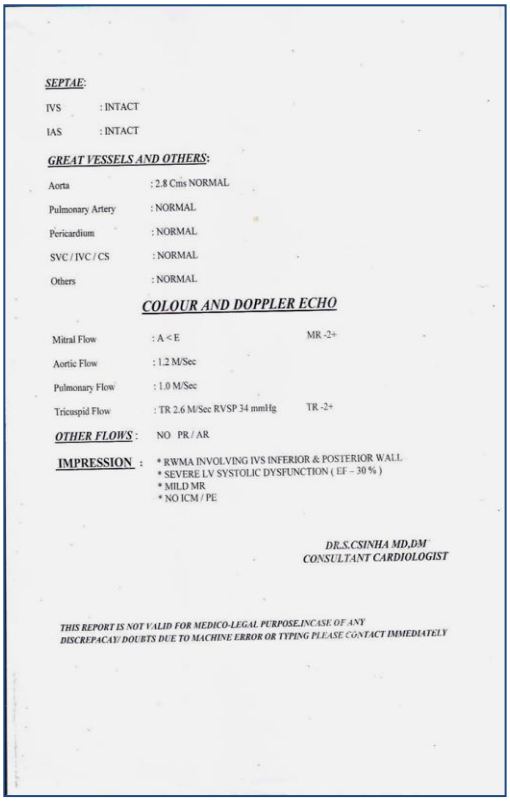
Fig. 2 (A)



SEPTAE:

IVS : INTACT
IAS : INTACT

GREAT VESSELS AND OTHERS:

Aorta : 2.8 Cms NORMAL
Pulmonary Artery : NORMAL
Pericardium : NORMAL
SVC / IVC / CS : NORMAL
Others : NORMAL

COLOUR AND DOPPLER ECHO

Mitral Flow : A < E MR -2+
Aortic Flow : 1.2 M/Sec
Pulmonary Flow : 1.0 M/Sec
Tricuspid Flow : TR 2.6 M/Sec RVSP 34 mmHg TR -2+

OTHER FLOWS : NO PR / AR

IMPRESSION :
* RWMA INVOLVING IVS INFERIOR & POSTERIOR WALL
* SEVERE LV SYSTOLIC DYSFUNCTION (EF - 30 %)
* MILD MR
* NO ICM / PE

DR.S.CSINHA MD,DM
CONSULTANT CARDIOLOGIST

THIS REPORT IS NOT VALID FOR MEDICO-LEGAL PURPOSE,INCASE OF ANY DISCREPACAY/ DOUBTS DUE TO MACHINE ERROR OR TYPING PLEASE CONTACT IMMEDIATELY

Fig. 2 (B)

Queen's
NRI Hospital
(A Unit of M/s. Chalasani Hospitals Pvt. Ltd.,)

CORONARY ANGIOGRAM REPORT

Name	[redacted]	IP:1712237
Age/sex	69/M	
Date/cath no	02.10.2017/7852	
Diagnosis	CAD – Old Inferior MI, ACS – NSTEMI, Unstable Angina with severe LVD, CCF, Type II DM	
Indication	ACS – NSTEMI	
Done by	DR.S.C.SINHA MD,DM,FACC,FSCAI	
Referred by	TTK	
Anesthesia	Local	
Contrast	Contrapaque 50ml	
Approach	Right Radial Artery 6F	
Catheters	5F Tig	
Left main	Normal	
LAD	Type III Vessel, Proximal 50-60% lesion, distal long 75% lesion	
Diagonals	Diffuse disease	
Left circumflex	Co-dominant, Proximal 90% lesion, distal occlusion	
OMs	OM1 Diffuse disease	
Right coronary	Co-dominant, ISR 50% lesion, distal diffusely disease	
PDA PLVB	Diffusely diseased	
Impression	CAD – Diffuse TVD	
Advice	Medical Management Vs CABG	

Dr.S.C.Sinha MD,DM,FACC,FSCAI
Consultant Interventional Cardiologist

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Fig. 3

Though T2 MI constituted about 25% of all cases of MI, the centres reporting Type 2 MI ranged between 0-13% only. This could be due to lack of awareness or inherent difficulties in establishing the diagnosis of a type 2 MI.

3.2 Type 1 NSTEMI was considered Initially Because

- Presentation as unstable angina.
- positive Trop-T. However there are causes other than MI to account for the same likewise Trop T positive always doesn't mean infarction. Other cardiac causes like myocarditis, cardiomyopathy, congestive heart failure and non cardiac diseases like renal [6].
- Ischemic changes in anterior and inferior wall with ST depression
- RWMA (raw wall motion abnormality) in anterior and inferior walls.
- EF 30%
- severe LVF / pulmonary oedema
- Hypoxemia (86 % oxygen saturation on admission).
- Angiographic evidence of varying blocks in circumflex and anterior descending branches of left coronary artery. Fresh ischemic changes in posterior descending branch with previous stent in it. There was no evidence of plaque rupture.

3.3 Retrospective consideration of Type 2 MI

- Preceding unstable angina
- The patient attended in a hospital on an occult sepsis case .
- Severe sinusitis in 4 days before the presentation of unstable angina days unresponsive to cefpodoxime and NSAIDS
- Raised CRP.
- control of infection with IV Tazobactum-Piperacillin after hospitalisation
- Tachycardia of 140 bpm.
- Hypoxia (initial 86%) improved by oxygen mask ventilation, in spite of low EF remaining same, suggesting a pulmonary cause than a cardiac cause
- The pulmonary oedema was not due to elevated EDVP (end diastolic ventricular pressure) of left ventricle which is normal on 2D echo .
- Normalisation of EF in subsequent 6 months.

The improvement in EF is presented in Table 1.

Table 1. Gradual improvement in EF

Date.	EF
30/9/17.	30%
29/12/17.	42%
23/3/18.	47%
29/6/18.	55%
30/9/18.	57%

Improvement in left ventricular function was defined as an improvement in ejection fraction (LVEF) of ≥10% on echocardiography. Return of EF to normal was defined as an improvement of LVEF to ≥50 % ischemic features , clinical ; ECG , echocardiographic and biochemical changes ,can occur in myocardial depression, apart from MI , with even reversal of cardiac dysfunction . In other words, where a clinical diagnosis of CAD was made (STEMI as well as NSTEMI, where in due course there is some improvement in EF but does not return. In a study, Improvement in LVEF was observed in 44.3% of patients and return to normal systolic function in 10.9. 43% of patients had persistent EF ≤35%, 31% had an EF of 36-49%, and 26% had an EF ≥50% [7] In a prospective study of 42 HF patients whose EF had normalized, the aggregate initial EF was 26%. It increased to ≥40%, with an absolute increase in EF ≥10%.. [8] The improvement of ECG, 2D echo including EF was reinterpreted as being possibly due to myocardial depression by sepsis rather than necrosis (infarction) of cardiac muscle.

Though fixed blocks were seen on angiogram, there was no obvious evidence of plaque rupture causing ischemic changes. Conversely if coronary occlusion causing M I were to be the cause the documented improvements in parameters noted above might not have been possible, as functional recovery of dead cardiac muscle is not possible.

3.4 The Sepsis Back Ground

The prolonged time taken for the recovery, the presence of fever before development of unstable angina, pan sinusitis, tachycardia of 140 bpm and raised CRP, all these suggested that depressed myocardial function rather than myocardial necrosis might be the possible cause. Sepsis could cause myocardial depression and cause T2 MI. The normalisation of EF which is complete and usual in cases of sepsis is another pointer towards its causing T2MI , in this case. even though the disparity between supply and demand of oxygen to myocardium may not be

demonstrable beyond doubt as T2 MI, the possible ways in which sepsis can cause myocardial depression are:

- LV systolic dysfunction is common in septic patients and potentially reversible in survivors.
- A major mechanism of direct cardiac depression in sepsis is the attenuation of the adrenergic response at the cardiomyocyte level due to down-regulation of β-adrenergic receptors and depression of post-receptor signalling pathways. These changes seem to be mediated by many substances, such as cytokines and nitric oxide.
- Another mechanism of direct cardiac depression in sepsis is cardiomyocyte injury or death, which can be induced by toxins, complements, DAMPs, and as-yet-unidentified myocardial depressants.
- The adequate O2 supply in sepsis suggested that myocardial depression is not related to tissue hypo perfusion but rather to circulating depressant factors or other mechanisms.
- The potential candidates responsible for sepsis induced myocardial depression are summarised in Table 2.

Table 2. Potential candidates responsible for septic myocardial depression

1) PAMPs
- (Pathogen-associated molecular patterns)
- 2)Extracellular histones
- 3) DAMPs. (damage-associated molecular patterns)..
- 4) Endotoxin
- 5) TLRs (Tol like receptors)
- 6) Myocardial depressant factor.
- 7) NO (Nitric Oxide)
- 8) Oxidative Stress.
- 9) Autonomic dysregulation and calcium flux
- 10) Inflammatory mediators. interleukins (IL-2, IL-4, IL-6, IL-8, and IL-10), gamma interferon (IFN-γ), TNF-α, IL-1β,andC5a.

4. CONCLUSION

The heterogeneity of T2 MI, the overlap with the spectrum of NSTEMI, the need to establish clear diagnostic criteria for T2 MI and the coexistence of T2 MI in presence of stable CAD were all well accepted. The need to revise the criteria was also felt by the scientific community. Focused the need to consider other parameters than time honoured disparity between demand and supply of myocardial oxygen supply , especially when sepsis was suspected to be the cause of T2 MI. The various other ways the sepsis can cause T2 MI were briefly discussed. The article is intended to create awareness on intricacies and implications in diagnosing T2 MI.

CONSENT AND ETHICAL APPROVAL

As per university standard guideline participant consent and ethical approval has been collected and preserved by the authors.

COMPETING INTERESTS

Author has declared that no competing interests exist.

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