SIMulatED

Royal Darwin Hospital Emergency Department

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# Scenario Run Sheet: NIV-beware the bearded man

## Scenario Overview

**Estimated Scenario Run Time:**  15-20 mins

**Estimated Guided Reflection Time:** 30-40 mins

**Target Group:** ED Registrars and Nurses

**Brief Summary:** 48yo man who develops sudden LV failure post thrombolysis of inferior STEMI, requires NIV for APO and aggressive afterload reduction to stabilise prior to transfer for papillary m repair/CABG

## Learning Objectives

**General**

Emergency management of acute dyspnoea and shock post myocardial infarction

**Scenario Specific**

Awareness of causes of shock + acute mechanical complications of myocardial infarction

Critical appraisal of recent management in a deteriorating patient by (verbal handover/document review)

Rationale and delivery of supportive therapies for APO and LV dysfunction in the context of ACS, including therapies influencing preload, afterload, inotropy and positive pressure ventilation

Recognition of severe acute mitral valve dysfunction post MI requiring definitive surgical repair

## Equipment Checklist

**Equipment**

CVC box, art line box, Phillips NIV, intubation checklist, infusion pumps x2

**Medications and Fluids**

Giving set, 0.9% saline, GTN IV, patch, nitroprusside, adrenaline, noradrenaline, dobutamine, metaraminol, frusemide IV

**Documents and Forms**

ED nursing chart and medical notes; intubation checklist; ventilator record

**Diagnostics Available**

VBG, ECG, ultrasound, glucometer, UA (leuk, blood)

**Images for sim presentation**

Green sheet/Nursing obs and drug chart, ECG series, VBG series (type 2 resp failure), CXR series, echocardiogram SX and PSLA views

**Mannequin props**

Beard

## Scenario Preparation/Later Parameters

**Initial Later**

GCS **14** RR 32 P 80 BP 90/50 GCS **15** RR 22 HR 70 BP 110/70

Sats 90% 4L CIG T 36.8 BSL gas SaO2 98% O2 T 36.9

**Mannequin Features**

SimMan 3G

## Participants

**Staff Actors**

ED Registrars x3, Night med reg ED Consultant, ICU, Cardiology, available by phone

Nurses x3 ED

**Instructor Roles**

- Provide the team with clinical signs

## Candidate Instructions/Triage Information

It is 0200h. Resus nurse tells you 48yo man admitted under cardiology in resus 2 has developed respiratory distress and BP 90/50. You are handed the casenotes. You recall he is an inferior STEMI patient who was thrombolysed in ED by the Evening team after 48hours stuttering chest pain; he is still awaiting admission by the med reg. First Troponin of 37,000. Pain free post TNK, aspirin, clexane, clopidogrel.

## Patient Instructions

Extremely breathless, denies palpitations or pain anywhere, feels light headed

**HPC** (casenotes)– Delayed presentation with 48h CP, Inferior STEMI with Q waves, Trop 37, 000, second trop in lab at present. Initial vitals – Dual HS, clear chest, HR 60 and hypertensive 170/90. Show drug and obs charts. HR 60-70 + BP stable at 130/70 and pain free.

**Medical History**: Never sees a GP, no regular meds, heavy smoker, strong FH IHD, no previous stroke or surgery. NKDA. Binge etoh consumption on weekends

**Social** Single, tradesman, lives alone

## Proposed Scenario Progression

* Rapid synthesis of clinical notes and nursing handover of acute deterioration
* Assembles team including medical registrar and coordinates focussed primary survey for predictable complications of delayed presentation of ACS
* Chest exam: widespread crepitations to mid zones; Pulmonary oedema on CXR
* Initiates NIV with BiPAP; requires gel on beard for good seal. Intubation not required
* Hyperdynamic apex beat, Mid systolic murmur at LSE, no thrill
* Repeat/serial ECG’s to exclude dysrhythmia /recurrent ischaemia+reinfarction
* ? expertise to performs EFAST scan – exclude pericardial tamponade (LVFWR); unlikely to see IVS rupture or pap muscle rupture unless expert sonographer; look for global/regional wall motion defect
* Initiates GTN infusion, inotropes (adrenaline +/- dobutamine) to appropriate endpoints, considers diuretics
* Inserts arterial and central venous access with consideration of recent thrombolysis (compressible site)
* Consults with cardiology, ICU and ED consultants, recognising need for ICU/CCU for IABPC/angiogram pending referral to cardiothoracic unit
* Explanation to patient

## Debriefing/Guided Reflection Overview

**General Opening Questions**

* Let’s get X to recap what happened in the scenario

**Scenario Specific Questions**

* What approach did you take in assessing the sudden deteriorating in this patient? (DDX)
* How did the clinical examination assist you?
* Were any specific tests helpful? What else might you have done?
* I noticed that you gave XYZ - can you talk us through your rationale for these?
* The patient had been recently thrombolysed – how did this impact upon your management?
* If he did not improve, what other therapies might be considered at RDH? What challenges did you face in delivering ideal care in this scenario (access to acute PCI, time of night, transfer for definitive care)
* What nursing challenges were there in delivering the requested treatment? (infusions)
* Human factors

**General Wrap-Up Questions**

* What did you find most beneficial about this scenario?
* What was the most challenging point in this scenario?
* What would you do differently next time?

NOTES

\*Papillary m rupture

Causes 5% deaths from MI, More common with RCA occlusion (inferior MI) thus Posteromed pap m rupture 6-12x more common.

Occurs in both STEMI and nSTEMI, usually 2-5 days post MI; usually first MI (no collaterals formed)

Associated with delayed admission and recurrent angina before presentation

Due to single vessel supply (PDA) c.f anterolat pap (LAD+LCx supply).

Occurs 2-7 days post infarct; complete or partial rupture, varying severity of LVF

Clinically causes acute APO and shock; thrill and variable intensity mid or late or holo-systolic murmur

Rx: Rx APO + aggressive afterload reduction with nitrates/vasodilators, diuretics, IABPC, emergent repair + CABG

Repair mortality rates 25%; higher if female, poor LV fn, older age

\*IVS rupture

Clinically causes loud holosystolic murmur and thrill, biventricular R>L) failure and shock

LAD occlusion especially wraparound LAD type (1/3 people LAD wraps around apex to supply lower IVS instead of RCA supplying this: thus has inferior STE and QW in addition to classic anterior STEMI changes)

Rx:Stabilise cardiogenic shock with afterload reduction to reduce L-R shunt (vasodilators, diuretics); inotropes and IABPC for LV systolic dysfunction; NIV for APO, definitive repair and CABG

\*Death from Rupture of LV free wall (tamponade)

<1% STEMI; occurs in 12% given fibrinolysis UTD: Am Heart J. 2006;151(2):316.

Increased risk with large transmural AUC (CK), first MI (no collaterals), persistent STE or CP (incomplete reperfusion), age>70, females, anterior MI: Am Heart J. 1989;117(4):809. MILIS study

Reduced risk with beta blockade ISIS-1: Lancet. 1988;1(8591):921.

Later complication of MI: occurs 5/7 post MI in 50% cases; by 2 weeks in 90% cases

Reduced risk with early reperfusion (TIMI) except if age >75: GISSI Gruppo Italiano per lo Studio della Streptochinasi nell'Infarto Miocardico ; J Am Coll Cardiol. 1990;16(2):359 ; PPRIMM75 Eur Heart J. 2005;26(17):1705.

Pulmonary Artery catheter to diagnose cardiogenic shock: hT + low Cardiac Index <2L/min/m-2 + high PCWP > 15mmHg

Rx: ALS and pericardiocentesis for tamponade, fluids, inotropes, vasopressors, IABPC + urgent repair and CCBG

Causes of cardiogenic shock (UTD):

