Crashed in a Minute on Table

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56 years old Lady Doctor

- Had known history of hypertension for 10 yrs. Recovered from severe with multiple organ failure before 6 years back.
- Osteo-arthritis on self medications (NSAID)
- Sedentary, BMI-26.
- Food-Non Veg, Positive Dyslipidemia.
- Never undergone any cardiac evaluation before.
- Default medications for HTN, DLP

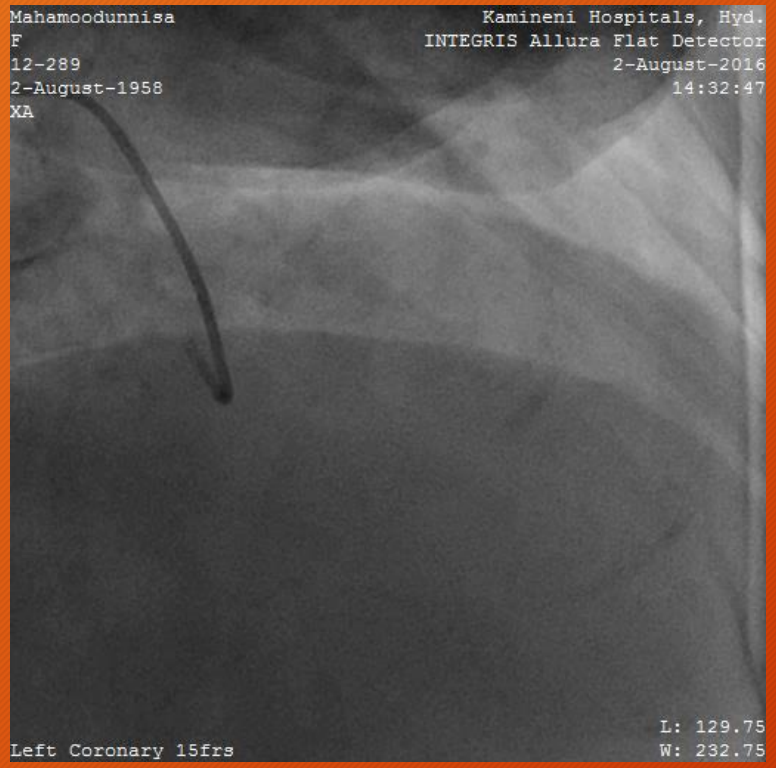
Clinical Evaluation

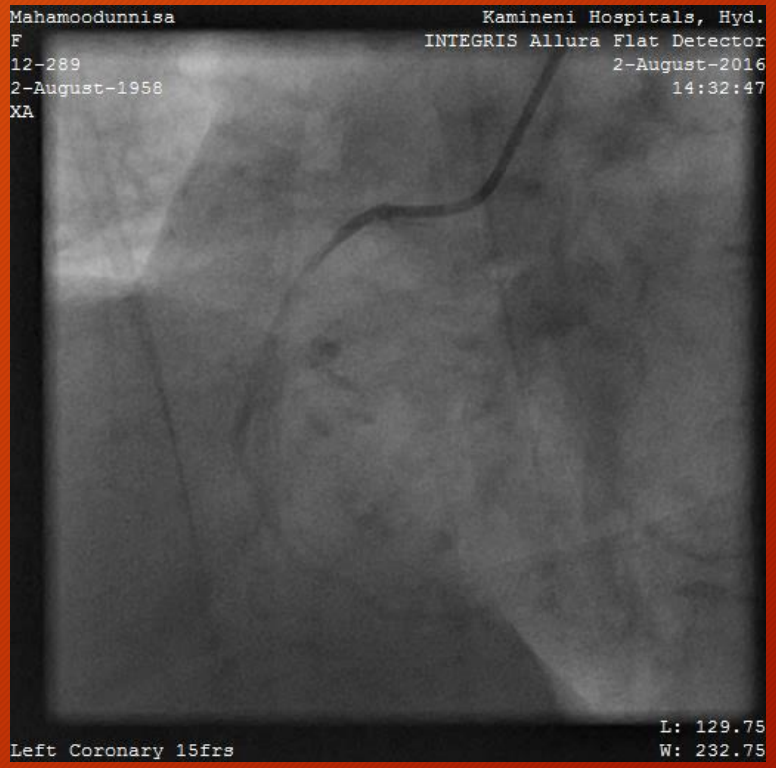
- One day at 7 PM arrived in emergency department with crescendo angina for 36 hrs. took first EKG after 24 hrs at an outside diagnostic center and got reported with AMI. Attended emergency at 36hrs.
- EKG- ST elevation in all chest leads and ST-T changes in other leads.
- Elevated cardiac enzymes, RBS-104 mg, Cr-1.4, CTnI-2600ng/dl, LVEF-35% with severe hypokinetic IVS and Apical LV, CXR-N and USG Abdomen-mild Hepatomegaly and mild renal parenchymal changes.
- BP-120/80, HR-90/min, O2-92%, Skin- wet, Lungs- mild bilateral crepitations.

Emergency Treatment

- Loading dose clopidogrel, ASA and Bolus reopro given. Half dose of TPA inhibitor given at night. But Patient was experiencing intermittent chest pain whole night.
- Next took the patient for CAG with Noradrenalin.
- CAG-LAD proximal to mid 90-96% type C lesion, RCA- 80-90% lesion at mid level.
- We kept additional artery and venous lines for supporting devices.

CAG through Right Radial Route



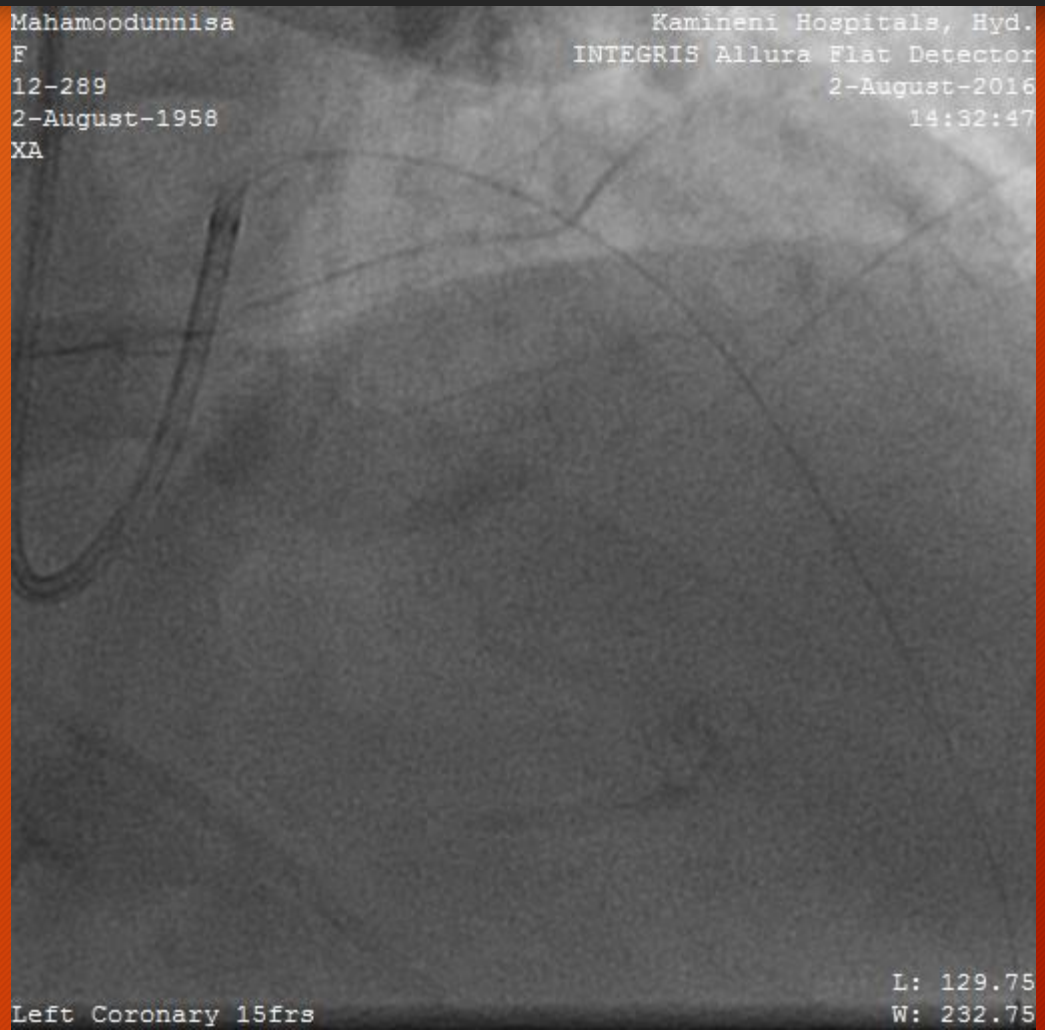


After CAG in 10 min developed Hypotension

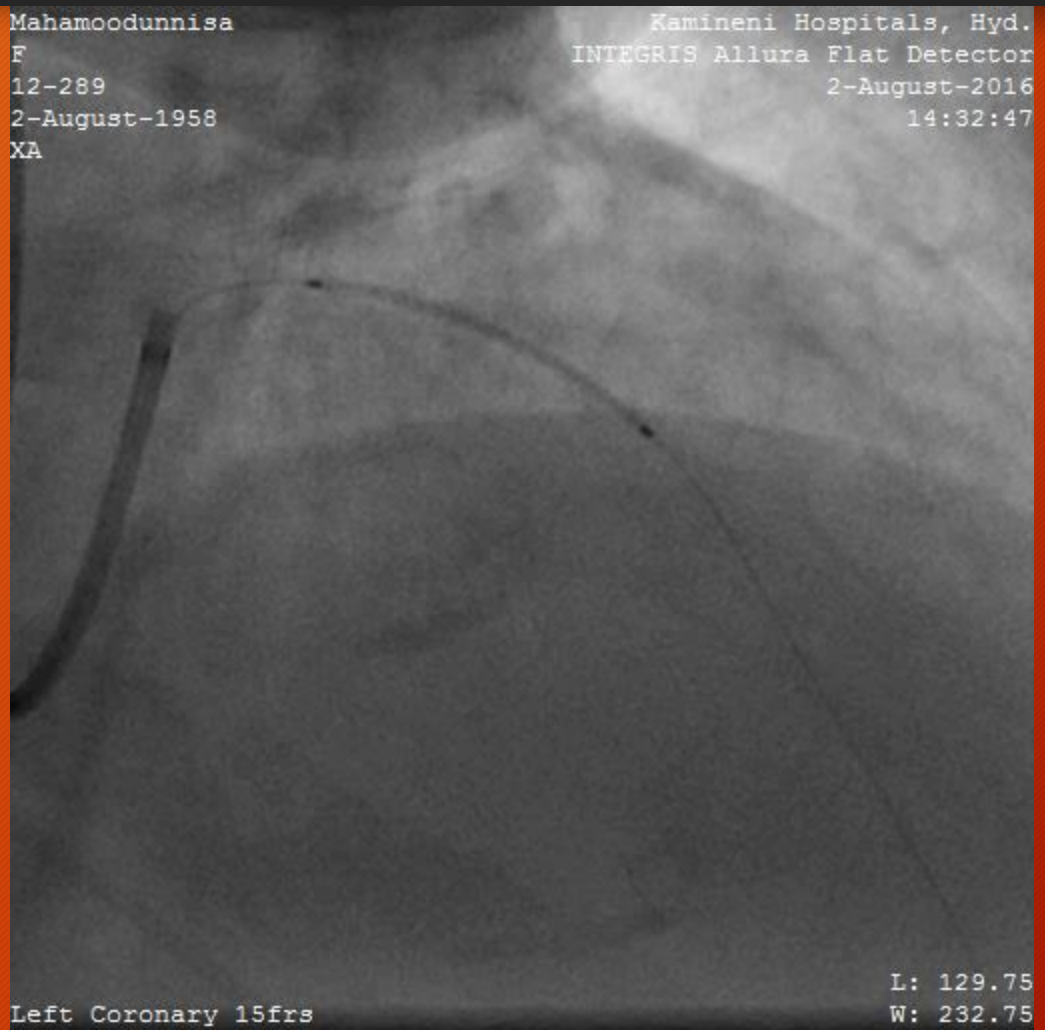
Patient was on Noradrenalin and Dobutamin. Suddenly developed hypotension while discussing with relatives. Increased the dose to maximum when hypotension.

Put stent 2.75/23 at 12 atm to LAD proximal

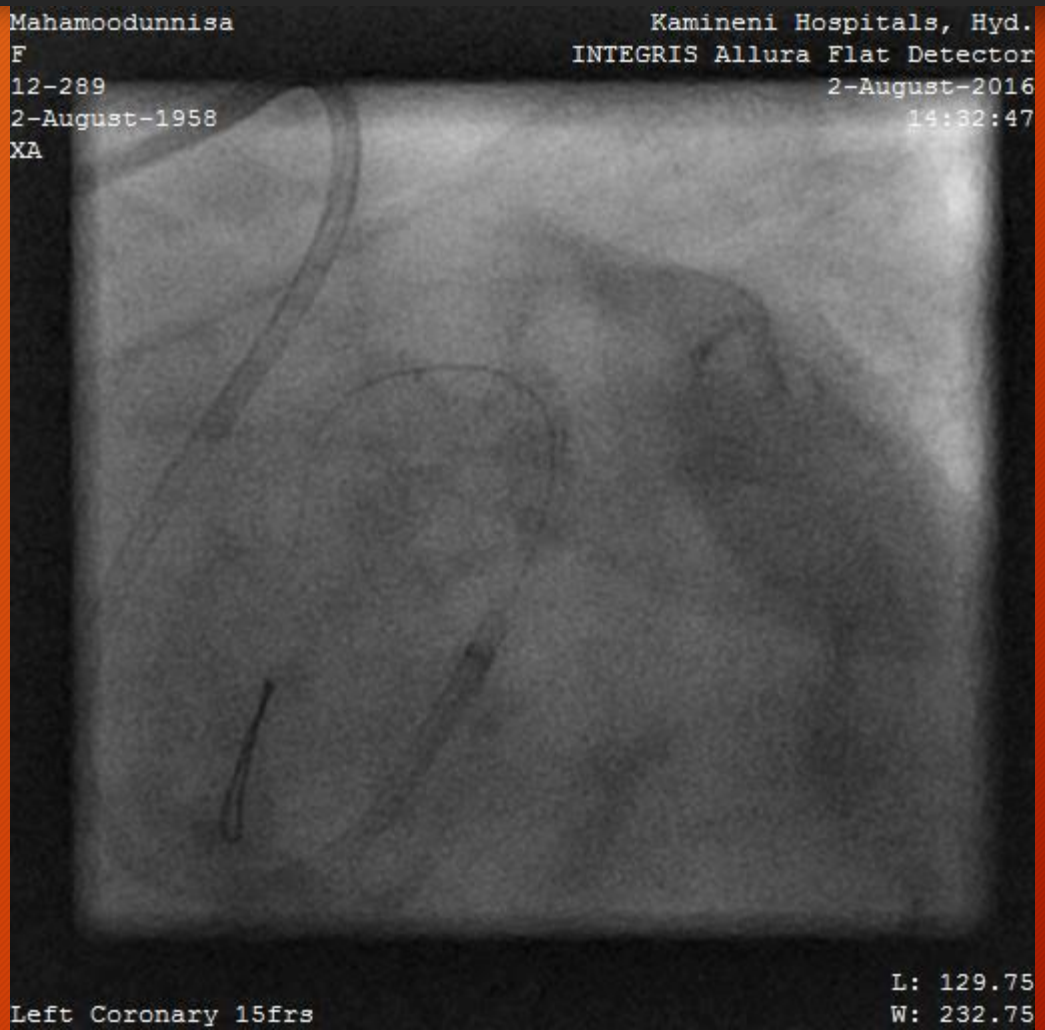
There is no damping in LAD. 6 Fr Guide



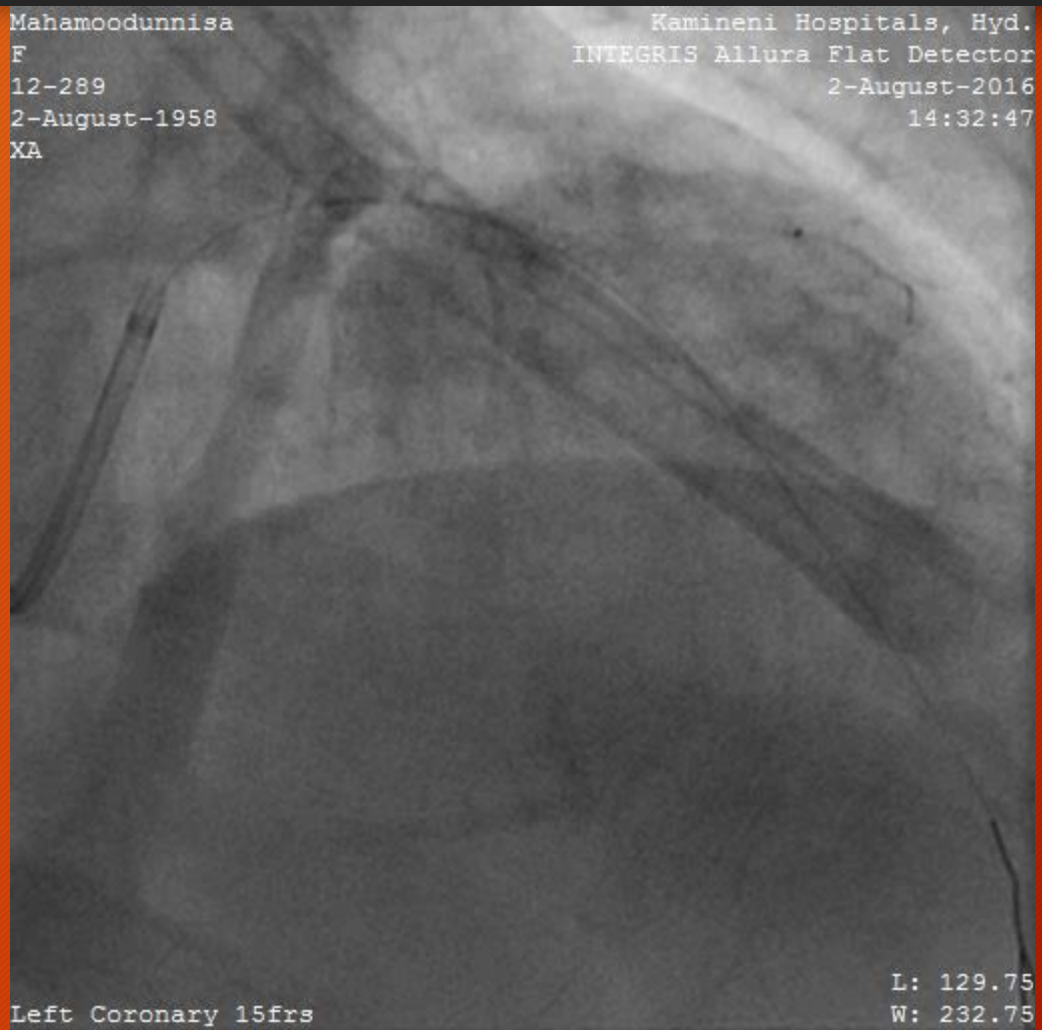
Stent deployment. Xience-Exp 2.75/28mm



No Dissection and Damping



A minute of Complete cardiac arrest



There is no time for Device management

- All essential measure have been taken. But nothing were achieved.
- The reasons were lot to think and discuss

Larger amount of myocardium insult

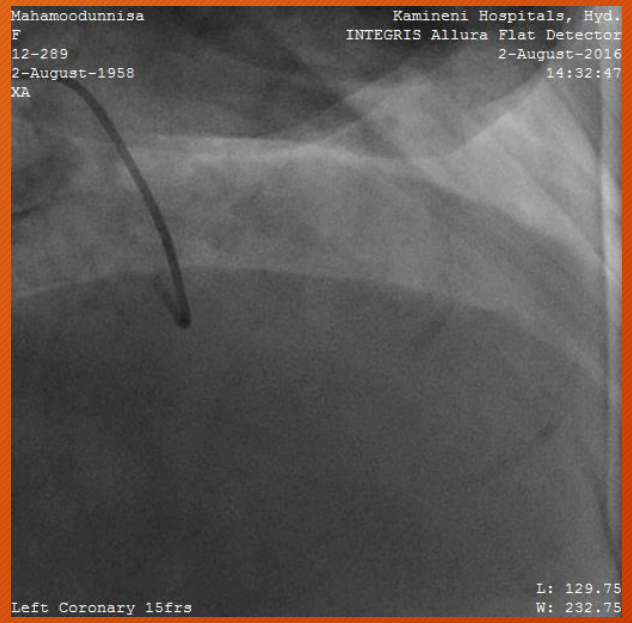
- Larger volume of myocardial apoptosis is the main reason for slow flow, no flow and sudden cardiac arrest in side the hospital and out side the hospital.
- In Massive apoptosis no procedure, measures, devices and management will work.
- So far we have no any commercially available bio marker to assess massive apoptosis is called heavenly closure of Myocytes function.

Timing of Apoptosis in AMI and Markers

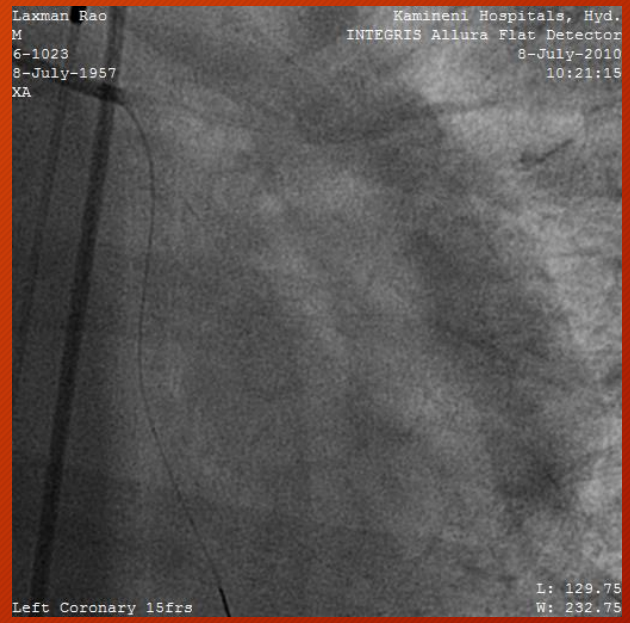
- 6-120 hrs. after MI- TUNEL + DNA laddering
- 3-14 hrs. after MI – TUNEL + DNA laddering
- 3.5-22 hrs. after MI – Annexin-V
- Some other markers like; Bcl-2, Bax, sFas, P53.
- JAK/STAT- Janus Kinase/ Singnal Transducer and Activator of Transcription
- SAPK- Stress Activated Protein Kinase.
- sFas- an inhibitor of apoptosis, sFas ligand –an inducer of apoptosis.
- TUNEL- Terminal deoxynucleotidyl Transferase mediated dUTP nick end Labelling

Identifying Radiolucency by Fluoroscope

Ex. AWMI. WP-3 days



Evolving ILMI. WP-4 days. IABP support



What I have found from my Researches

- Those who recovered from multiple organ failure will have poor result in ACS.
- Window period more than 36 hrs. Crescendo angina and highly elevated CtnI.
- Sedentary life style
- Female always comes under high risk
- Finally important and only one pathological cause is Apoptosis of large volume of myocytes. Massive apoptosis happen in above said risk factors.