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ONE DESTINATION
CARDIAC SCIENCE
MARENGO CIMS HOSPITAL

Marengo CIMS Hospital

HEALTHY HEART

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Marengo CIMS Hospital

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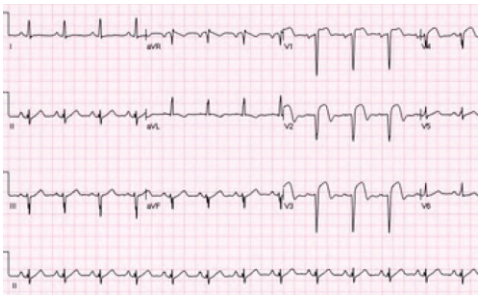
Left Ventricular Thrombus: A Clinical case Based Discussion

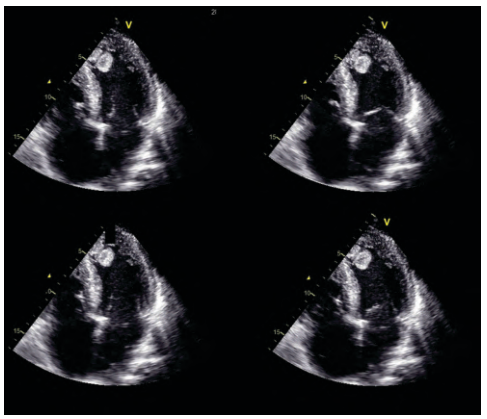
1. History, Examination and Tests:

- We had a patient: Mr A C P, M/61 years, weighing 64 kg, having DM for last 8 years, normotensive, with a habit of smoking 10 bidies per day
- He developed Anterior Wall ST Elevation Myocardial Infarction 5 days before, but presented late after 24 hours of symptom onset and so was not offered fibrinolytic therapy.
- He was referred to our hospital with following treatment:
- Aspirin: 150 mg, Prasugrel 10 mg, Atorvastatin 40 mg
- Metoprolol 25 mg, Ramipril 2.5 mg, Frusemide 20 mg
- Glimeperide 3 mg, Metformin 1 gm
- Now has Dyspnoea grade II and a sense of fatigue
- On examination: Heart Rate: 100/Min, BP: 116/72 mm Hg, SpO: 96% on room air
- CBC, SGPT, GhbA1c were within acceptable range. S Creatinine was 1.5 mg, (eGFR of 53 ml),S. Potassium was 4.4 mEq/L

ECG: Established AWMI, QS in V1 – V4, ST Elevation Persistent



Echo: Apical dyskinesia, LVEF: 20-25%, Clot at LV Apex



2. Clinical Dilemma:

As a clinician, we had few questions

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to answer to ourselves:

A) Would only DAPT with Prasugrel be sufficient?

B) Do we need to add anticoagulation?

- What are the options and which one is the best? : UFH, LMWH, VKA, DOAC
- How long do we need to give anticoagulation?

C) If we add an anticoagulation, do • • we need to change DAPT?

- Would SAPT be enough?
- Which one antiplatelet would be the safest and most effective? : Aspirin, Clopidogrel, Prasugrel or Ticagrelor?
- What should be the dose?

D) Would our choice of anti heart failure treatment matter?

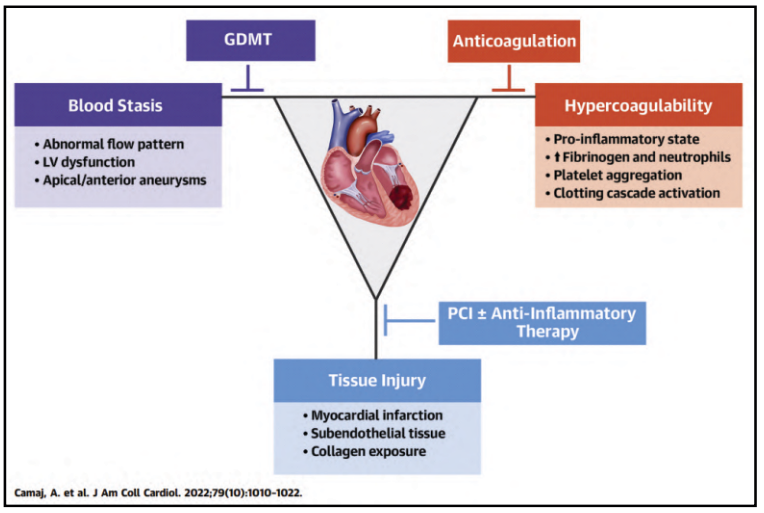
E) Do the LV clots dissolve or persist?

F) What is the immediate and long-term risk and what is the prognosis?

3. Some Facts about LV Clot after Myocardial Infarction

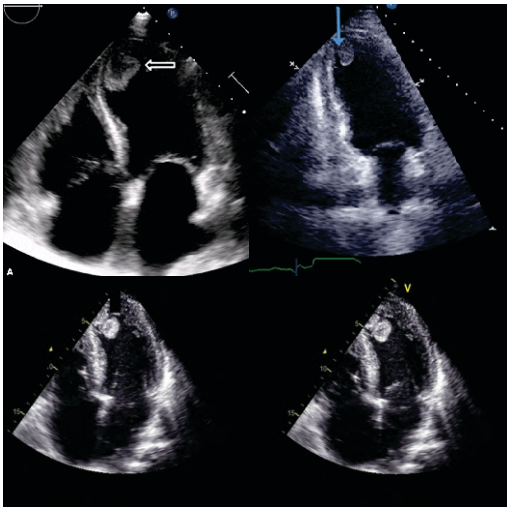
- Incidence of LV thrombus after AMI has declined significantly, mainly due to adoption of standard reperfusion therapy in terms of primary PCI or fibrinolytic.
- In present era LV clot is seen in up to 6.3% of patients with STEMI as a whole and 19.2% of patients with AW STEMI with LVEF <50%.
- The imaging modality impacts the frequency of detection.
- The sensitivity of Trans Thoracic Echocardiography is far less than Cardiac MR imaging for detecting small, laminar LV mural thrombus.
- TTE: Sensitivity - 29%, Specificity - 98% as compared to CMR.
- CMR: Sensitivity - 82-88% Specificity - 100% compared to surgical/pathological confirmation. Especially late gadolinium enhancement improves thrombus detection on CMR.
- Most LV thrombi can be detected by imaging within 2 weeks of AMI. An early echocardiography may not be revealing clot in many cases. So high-risk patients without LV thrombus on early imaging (e.g., within 48 hours after AMI) should be reimaged 2 weeks after the acute event.

4. Pathophysiology:



Virchow's Triad helps us to understand the pathophysiological basis of an LV clot formation during post MI phase and the same will help to define treatment targets. Though the anticoagulation has the pivotal role, efforts directed to minimise or heal the tissue injury and improve global LV function are very important.

5. Risk of LV Thrombus: Devastating systemic embolization



- The risk of systemic embolization is most closely related to
- Thrombus mobility and protrusion as described on imaging
- Size and number clot/clots
- The rate of systemic embolization varies between 3% in patients with consistently therapeutic anticoagulation levels and up to 19% in patients with poorly controlled anticoagulation

6. Treatment of diagnosed LV thrombus:

- Studies have produced conflicting results regarding the safety and efficacy of Vitamin K Antagonists compared to Direct Oral Anticoagulants.

- VKA with a goal PT INR of 2-3 has been a more time tested therapy. At present most of the international guidelines suggest that DOAC should be used if VKA cannot be tolerated.
- In immediate post MI situation, triple therapy that is DAPT with VKA should be used for minimum number of days. This depends upon the ischemic vs bleeding risk. Generally triple therapy is used until hospital discharge or up to a maximum period of 6 weeks.
- For Tripple therapy the principles are use Aspirin at a lower dose of 75 mg, use Clopidogrel instead of more powerful other antiplatelet agents (like Prasugrel or Ticagrelor) and maintain PT INR at around 2 (instead of between 2 to 3) or if a DOAC is used, use one step lower dose (15 mg Rivaroxaban instead of 20 mg and 2.5 mg bid Apixaban instead of 5 mg bid).
- Once 3 days to 6 weeks time is over, de-escalate the therapy to SAPT + VKA till the clot dissolves: Clopidogrel is preferred over Aspirin, Prasugrel or Ticagrelor
- Once clot is dissolved/ after 6-12 months, stop VKA/DOAC and switch to DAPT or SAPT as per the demand of the situation.

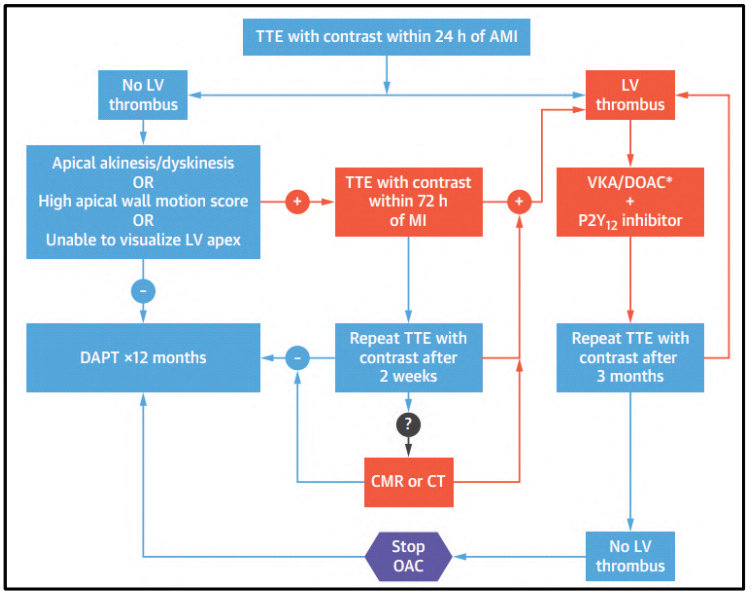
7. Treatment duration for VKA/DOAC in post MI LV Clot:

- Generally anticoagulation is continued for 3 months.
- Repeat LV imaging with Echocardiography or CMR to be carried out after 3 months of therapy.
- If the LV thrombus has resolved, we discontinue VKA and continue with DAPT as per management of AMI.
- If the LV thrombus is persistent, we continue VKA for further 3 months and we repeat imaging every 3 months.
- Once anticoagulation has been discontinued, repeat imaging 3 months later is advised to check absence of reappearance of LV clot.

8. Should we use VKA/DOAC for prevention of an LV Clot in post-MI patients?:

- No prospective trial has examined the role of anticoagulation plus contemporary antiplatelet therapy in the modern percutaneous coronary intervention era.
- Such a preventive strategy has never gained Class I or IIa recommendation in any international guidelines in past.
- So as a general recommendation, no prophylactic anticoagulation should be used. Rather we need to pay proper attention to the use of better DAPT regimen and guideline directed heart failure therapy.
- Yet it may be considered for selected patients with STEMI and anterior apical akinesis or dyskinesis balancing ischemic vs bleeding risks.
- If a decision to use VKA is made, guidelines recommend use of a vitamin K antagonist (VKA) with a lower INR target of 2.0-2.5.

9. Post MI LV Clot: Algorithm of Diagnosis and Management



10. Post MI LV Clot: Take Home Messages

Incidence of LV thrombus following AMI has markedly declined, advancements in reperfusion and antithrombotic therapies have played a major role.

LV thrombus development depends on Virchow's triad and that's why we need to address all the 3 aspects of clotting mechanisms: Stasis, Hypercoagubility and Tissue injury

Most LV thrombi develop within the first 2 weeks post–AMI, diagnosis is made by a TTE (-/+ enhancing agents) and cardiac MR.

Embolic events are most feared complications and that is why a systemic anticoagulation is needed. VKA is the mainstay of therapy, as the efficacy of DOACs is less well established.

There is not much data to support the routine use of prophylactic anticoagulation, even in high-risk patients.

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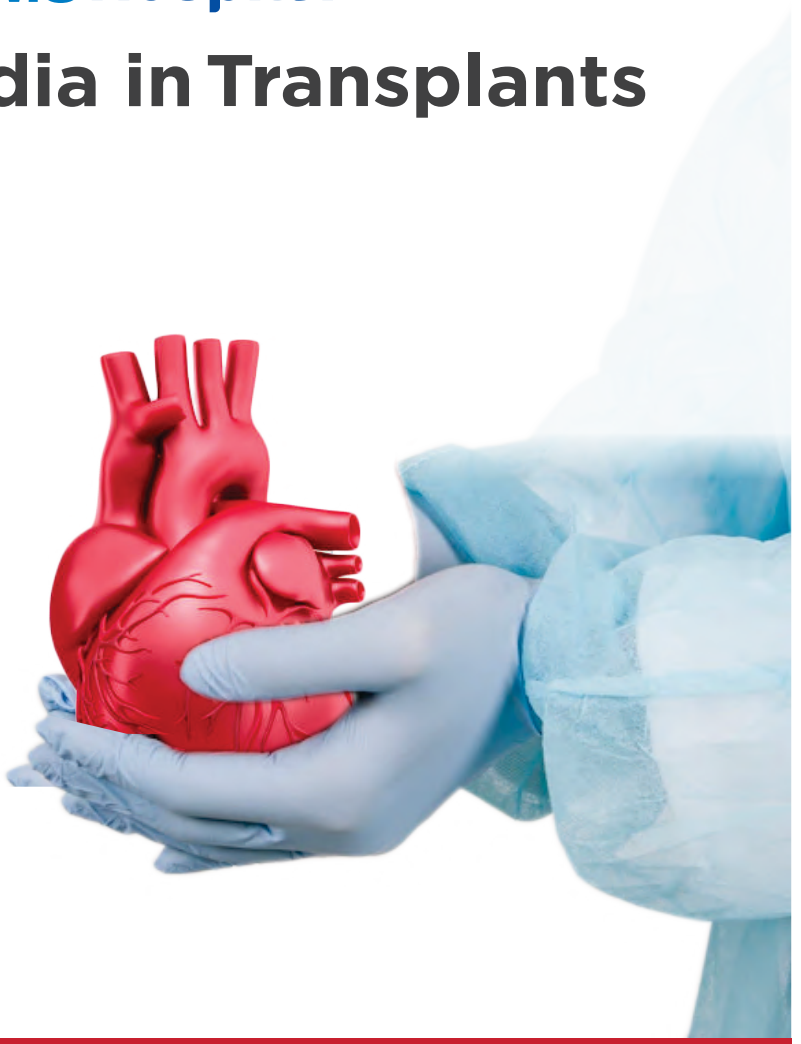

To the heroes who gave the ultimate gift: your generosity echoes in the beating hearts you donated.

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