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Case Report

Anaesthetic management in patient with left ventricular clot posted for non-cardiac surgery

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ABSTRACT

Cardiovascular disease remains the leading cause of mortality all over the world. Mortality from acute myocardial infarction (AMI) has decreased since the introduction of primary percutaneous coronary intervention (PCI). Left ventricular (LV) thrombus is a common complication after acute myocardial infarction (MI). One of the most feared complications is the occurrence of thromboembolic events (mostly cerebrovascular accidents) due to left ventricular (LV) thrombus formation. We present a 65 yrs old male with right traumatic foot ulcer, scheduled for right foot SSG. His ECG showed anterior wall ischemic changes. Echocardiogram showed akinetic LV apex, EF: 47%, with an organised LV apical clot of 1.5×1.4cm. Cardiologist opinion was obtained and he was put on oral nitrates, beta blocker and statins. Surgery was done under right popliteal sciatic nerve and fascisa iliaca block. Intraoperative period was uneventful. Postoperatively, serial ECG and Trop I was done on 1^{st} and 4^{th} POD and was found to be normal. He was discharged on 4^{th} POD, with advice to follow up with cardiologist.

1. Introduction

Left ventricular (LV) thrombus is a common complication after acute myocardial infarction (MI). Despite the rapid development of reperfusion and adjunctive medical therapy, complications of STEMI remain critical causes of adverse events. Among them, the formation of the left ventricular thrombus (LVT) subsequent to STEMI is not rare.[1] Anterior MI is the most critical determinant of LVT.[2] LVT in post-AMI patients indicates a worse prognosis. Anticoagulation is associated with LVT resolution and reduces embolism and mortality risk. Patients with previous history of Myocardial infarction with evidence of left ventricular clot coming for non-cardiac surgery have increased risk of developing perioperative catastrophic complications.[3] LVT is independently associated with major adverse cardiac events (MACE) at 1-year follow-up. The clot can get dislodged in the event of cardiac arrest and CPCR.[3] We are presenting a case posted for elective surgery managed without any intraoperative complications. Though it was uneventful, chance for occurance of perioperative morbidity and mortality was there.

2. Case Report

This study was done after approval of the institutional ethics committee and obtaining written informed consent from the patient. We present a 65yrs old male with right traumatic foot ulcer, scheduled for right foot SSG. He did not have any co-morbidity. He was a chronic smoker and alcoholic. Detailed history and physical examination revealed no significant clinical findings. His ECG showed anterior wall ischemic changes, Trop I, CKMB were within normal limits. Echocardiogram showed akinetic LV apex, EF: 47%, with an organised LV apical clot of 1.5×1.4cm.

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Cardiologist opinion was obtained and he was put on oral nitrates, beta blocker and statins. The preoperatrive blood sugar was within naormal range. Patient was taken up for surgery under ASA3 with high risk consent. Surgery was done under right popliteal and fascisa iliaca block.

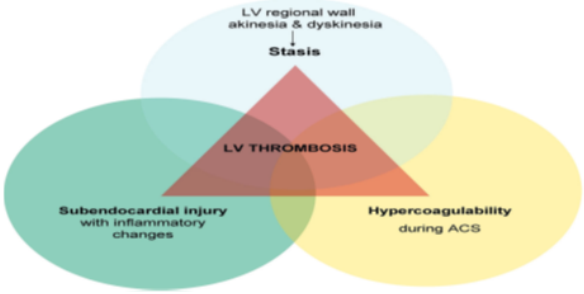

Fig. 1: The three components of the virchow's triad in left ventricular thrombus formation. ACS, acute coronary syndrome; LV, left ventricular

2.1. Anaesthetic management

After confirming adequate starvation and compliance with preoperative orders, patient shifted to the OT. IV line secured. Immediately after putting the patient on operation table, non invasive blood pressure monitoring, temperature probe, continuous ECG, and pulseoximeter were attached. He was pre-medicated with 1-2 mg IV midazolam. Patient was placed in the supine, the skin was disinfected, and transducer (linear transducer with frequency of 8-12 MHz) placed in the transverse position at the popliteal crease. Popliteal artery was identified, and then common peroneal and tibial nerves were identified. Probe was then advanced proximally till sciatic nerve was seen at separation of Tibial (TN) and Common Peroneal Nerves (CPN). Once sciatic nerve was seen, lidocaine 1% was infiltrated subcutaneously, the needle (blunt spinal needle G22) was inserted in plane 2 to 3 cm lateral to the transducer and advanced toward the sciatic nerve. Once the needle tip was adjacent to the sciatic nerve in the epi-neural sheath of the sciatic nerve between the TN and the CPN, the syringe was gently aspirated and 20 ml of bupivacaine 0.25% and 1% Lignocaine with 4 mg of dexamethasone was injected circumferentially around the sciatic nerve, causing separation of the TN and the CPN. Subsequently, the skin over the right femoral crease was disinfected and the transducer (linear transducer with frequency of 8-12 MHz) was positioned to identify the femoral artery, femoral nerve is lateral to the artery in the femoral sheath. Once visualized, lidocaine 1% was infiltrated subcutaneously 1 cm away from the lateral edge of the transducer. The needle (blunt spinal needle G22) was inserted in-plane in a lateral-to-medial orientation and advanced through the fascia iliaca towards the femoral nerve, after negative aspiration, 20 ml of bupivacaine 0.25% and 1% lignocaine with 4 mg of dexamthasone was injected around the femoral nerve. Sensation was examined by pin pricking on the distribution of sciatic nerve and femoral nerve. The surgery was allowed after confirming a complete sensory and motor block, Intraoperative period was uneventful. Postoperatively, serial ECG and Trop I was done on 1^{st} and 4^{th} POD and was found to be normal. He was discharged on 4^{th} POD, with advice to follow up with cardiologist.

3. Discussion

Cardiovascular disease remains the leading cause of mortality all over the world. Mortality from acute myocardial infarction (AMI) has decreased since the introduction of primary percutaneous coronary intervention (PCI) which helps in decreasing mortality rates and clinical adverse events. Still, postinfarct complications are not rare. One of the most feared complications is the occurrence of thromboembolic events (mostly cerebrovascular accidents) due to left ventricular (LV) thrombus formation.[1]

The incidence of STEMI-related LVT could be as higher as 31%-56% in the earlier time when thrombolysis was the mainstream of reperfusion. The risk lowers in the era of the primary PCI, but LVT can still be detected in around 15% patients.[4,5] The embolic complications in the prethrombolytic era were reported in to be around 10%,while in the thrombolytic era its 2% to 3%.[6] The modern era of rapid reperfusion, effective neurohumoral blockade and DAPT plus vitamin K antagonist has lowered the incidence.

Left ventricular thrombus (LVT) can occur both in ischemic and nonischemic cardiomyopathies and can lead to thromboembolic complications such as stroke.LV thrombus appears earlier in the course of the disease when initial ejection fraction <40%, in the presence of multivessel coronary artery disease, or a high peak creatine kinase value.8.w10 Thrombus formation reflects the presence of factors that represent the Virchow's triad in the ventricle – reduced wall motion, local myocardial injury and hypercoagulability/stasis of blood flow.[7,8] LVT formation is a major complication after acute ST-segment–elevation myocardial infarction (STEMI) more so in Anterior wall MI(apical hypokinesia).It may lead to devastating complications, such as reinfarction, congestive cardiac failture, embolic stroke and death.[1]

Overall, current evidence suggests that the most important means of reducing LVT formation is prompt revascularization and preservation of LV myocardial function. However, the two studies with an incidence rate higher than 20% in the current era makes us to look for in high risk cases, diagnose and treat LVT. Once diagnosed, rapid percutaneous coronary intervention (PCI), concomitant use of potent triple drug (DAPT plus vitamin

K antagonist- triple antithrombotic) along with ventricular reverse remodeling drugs are used in the management of LVT.[9] There was no significant association between anticoagulation and bleeding risk. The risk of LV thrombus formation is highest during the first 3 months following acute myocardial infarction, but the potential for cerebral emboli persists in the large population of patients with chronic LV dysfunction.

Usually TEE/contrast TEE is used in clinical practise, Contrast-enhanced CMR has a significantly better diagnostic accuracy than echocardiography, and thus it is considered as the reference standard imaging modality for LV thrombus detection. Its useful to perform contrast CMR in all patients with reduced apical wall motion on noncontrast echo. This is 100% sensitive for detection of LVT after MI.[10]

While the ACCF/AHA guidelines do not prescribe a defined duration, the ACCP guidelines suggest 3 months of anticoagulation The ESC guidelines define 6 months of anti coagulation. Target INR for warfarin therapy is a range between 2–3. Though low molecular weight heparin has been studied, the use of long term subcutaneous injections and cost make it a less favoured treatment option.[11–14]

LVT occurred in 93.2% of patients with the occlusion of left artery descending (LAD). More than 2/3 of LVT was reported within the first two weeks of STEMI, late thrombus can be found in three months or even later. The existence of LVT is clearly related to increased risk of embolic events and death. STEMI patients with LVT demonstrated a 3.97 times higher risk of embolic events than those without LVT. In a recent study, the rate of 5-year embolism in STEMI patients with LVT was up to 16.9% if without effective therapy, significantly higher than the rate of 2.9% in patients without LVT and 3% in patients with LVT but undergoing ideal therapy.

PNBs provide more stable intraoperative hemodynamics than general anesthesia or a central neuraxial block.[15–17] Ultrasound-guided combined femoral and sciatic nerve blocks are mainly chosen for lower limb surgery. USG guided regional nerve blocks became popular and frequently used for anaesthesia and postoperative pain control of such cases, having the advantages of providing a good intraoperative anaesthesia as well as a prolonged postoperative analgesia that may last for up to 20 hours with addition of dexamethasone.[18,19]

4. Conclusion

LV clot formation is the most common complication following MI. Especially the risk of LV clot formation is higher in first 3months following acute MI. Though in this case intraoperative and postoperative period may be uneventful, the risk of thromboembolic events following LV clot formation is higher in most of the patients. The existence of LVT is clearly related to increased risk of embolic events and death. STEMI patients with LVT demonstrated a 3.97 times higher risk of embolic events than those without LVT.

5. Source of Funding

None.

6. Conflict of Interest

None.

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