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Rare Case of Left Ventricular Thrombus Postmyocardial Infarction for Emergency Decompressive Craniectomy

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Abstract

Left ventricular (LV) thrombus formation is a notorious complication encountered in postmyocardial infarction patients. Such cases, when coming for noncardiac surgery, put the patient at greater risk of embolic events. Anesthesiologists play a pivotal role in the management of such rare and difficult cases. There is sparse evidence on management of such cases for noncardiac surgery. Hence, we would like to share our experience of a young patient with LV thrombus posted for left decompressive craniectomy.

Keywords
- craniectomy
- left ventricular thrombus
- myocardial infarction

Introduction

Left ventricular (LV) thrombus is not an uncommon complication encountered after myocardial infarction (MI). The incidence of LV thrombus is 15% with anterior wall MI (AWMI).[1] The presence of residual clot along with embolic complications pose significant challenges to an anesthesiologist in terms of managing not only the surgical procedure addressing the embolic complication but also the underlying cardiac condition that resulted in clot formation, more often MI or dilated cardiomyopathy. We present a case of post-MI LV clot with thromboembolic stroke posted for decompressive craniectomy.

Case History

A 36-year-old male presented with complaints of weakness of right upper and lower limbs, associated with opposite side facial deviation and slurring of speech for 5 days. He was diagnosed to have AWMI 4 months before presentation. Thrombolysis was done with 1.5 million units of streptokinase intravenously (IV). History revealed noncompliance to anticoagulant medication. He then developed left middle cerebral artery (MCA) infarct (►**Fig. 1**). Examination revealed right hemiplegia with 0/5 power, right upper motor neuron facial palsy, and Glasgow coma scale (GCS) score of 14/15. ECG revealed Q waves in leads 1 and aVL. Echocardiography showed severe hypokinesia of anterior, anteroseptal, and apical walls with 20% ejection fraction (EF) and large LV thrombus of size 5.3 × 4.3 cm, extending from apex to midventricular cavity. Coronary angiogram post-thrombolysis revealed recanalized left anterior descending (LAD) artery. Two days later, emergency left decompressive craniectomy was planned in view of neurological deterioration and increased midline shift despite medical management. Preinduction arterial line was secured. Opioid-based induction was done with IV fentanyl 150 mcg, 2% sevoflurane, IV rocuronium 50 mg; IV lignocaine 80 mg was given to attenuate intubation response. Airway was secured with

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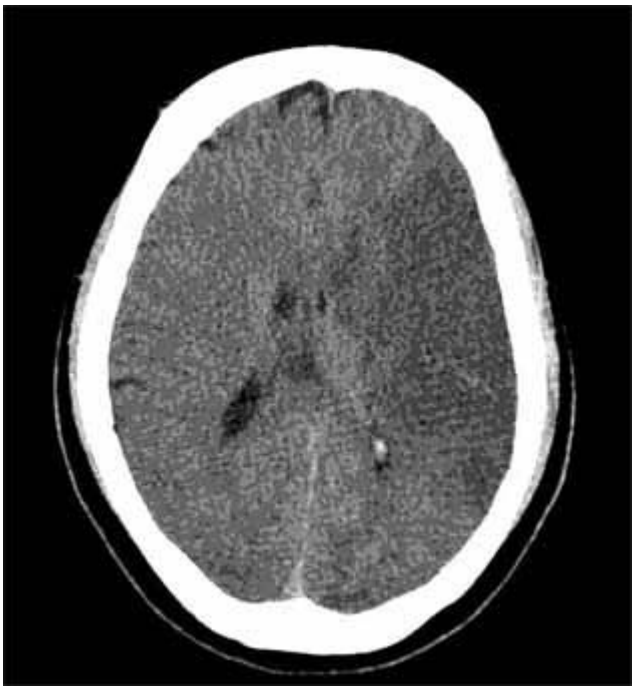

Fig. 1 Preoperative noncontrast CT brain image showing left middle cerebral artery (MCA) infarct.

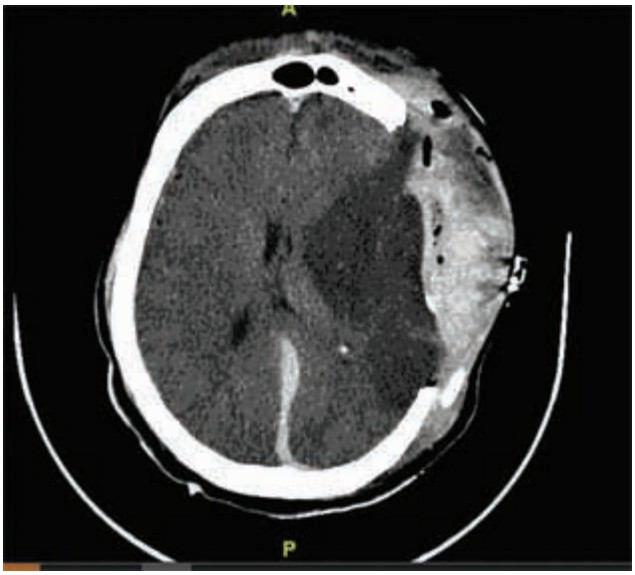

Fig. 2 Noncontrast CT brain image showing status postdecompressive craniectomy.

endotracheal intubation. Right subclavian central venous catheter was secured. To prevent hemodynamic response to surgical incision, scalp block was given with 20 mL of 0.25% bupivacaine. Anesthesia was maintained with isoflurane in air-oxygen mixture. Hypertonic saline and furosemide were administered as antiedema measures. Surgery lasted for 2 hours and the patient was not extubated in view of diffuse brain bulge and was electively ventilated (►**Fig. 2**). The patient was restarted on cardiac medications and enoxaparin 60 mg OD from postoperative day 1 (POD-1) and was extubated with full GCS score on POD-2. Enoxaparin dosage was stepped up to BD dosage on POD-3 and later switched over to clopidogrel on POD-5.

Discussion

LV thrombus formation occurs due to presence of Virchow's triad; reduced contractility and increased LV internal diameter leads to stasis, local myocardial injury (endothelial injury), and presence of proinflammatory or procoagulable state. Determining factors for clot formation include degree of myocardial injury, ST-segment elevation myocardial infarction (STEMI) more than non-ST-segment elevation myocardial infarction (NSTEMI), apicoanterior wall lesions more than others, and EF less than 40%.[2] The risk of embolization is uncertain, but it can be up to 10 to 20% in the first 3 months after MI and thereafter risk decreases as the thrombus becomes organized.[1] Mobile pedunculated clots are associated with increased risk of embolization.[3] LV clot accounts for 10% cases of cardioembolic stroke.[4] Our patient had risk factors associated with LV thrombus formation like previous AWMI, low EF, and noncompliance to systemic anticoagulation. Although he had an organized clot, and the inciting MI event had occurred more than 3 months ago, he still developed cardioembolic stroke due to refraining from anticoagulation.

Intraoperative plan was to maintain preload, avoid tachycardia, maintain systemic vascular resistance (SVR), avoid hypotension, and maintain a normal sinus rhythm. Sevoflurane has little effect on cardiac output and SVR in graduated doses compared with IV induction agents.[5] Hence, a combination of opioid-based induction with sevoflurane was used for induction. An IV bolus of lignocaine effectively attenuated the pressor response of tracheal intubation. Surgeons were advised to avoid adrenaline infiltration to prevent tachycardia. Surgical response was well blunted by scalp block, thereby allowing us to use lesser anesthetic concentrations at 0.5 minimum alveolar concentration (MAC), which was beneficial both for negating the cardiodepressant action on the poorly contracting heart and avoiding increase in cerebral blood flow by inhalational anesthetics. The general principle to avoid clot embolization revolved around avoiding a hyperdynamic heart and avoiding tachycardia, arrhythmias and increased contractility, and maintaining perfusion without the use of inotropes. Inotropes, by increasing contractility, can theoretically cause embolization. If the dislodged embolus size is big enough to cause LV outflow tract obstruction, then on-table cardiac arrest is inevitable, and cardiopulmonary resuscitation (CPR) and defibrillation increases the risk of further embolization. So, the case was operated under cardiopulmonary bypass (CPB) backup in case of any untoward events as a resuscitative measure, and measures to minimize the chances of clot dislodgement were taken. Intraoperatively, dynamic fluid responsiveness parameters were used to administer fluids and blood. Since the space inside LV cavity other than thrombus is small, despite dilatation of the ventricles, patient cannot tolerate hypovolemia or hypervolemia. Hence, maintaining adequate volume is of paramount importance. To reduce intracranial pressure,

furosemide and hypertonic saline were used. Mannitol was avoided, as it can precipitate cardiac failure in the setting of poor cardiac function. Postoperatively, enoxaparin 60 mg subcutaneously (SC) was started on POD-1 as once daily dose and stepped up to 60 mg SC twice daily dosage to prevent recurrent systemic embolization. Anticoagulation was increased gradually to avoid the risk of intracranial bleed from recent surgery and to allow adequate time for surveillance for bleeding. Although the possibility of concomitant surgical thrombectomy and decompressive craniectomy could have been ideal, it was not practically possible as LV thrombectomy would have required anticoagulation and risked intracranial bleeding. Moreover, LV thrombectomy is usually allocated to patients with high risk of embolization like prior embolic episodes, floating thrombus, and unsuccessful anticoagulation therapy. This procedure usually requires LV incision which, in turn, can lead to further LV dysfunction, arrhythmias, and aneurysm formation. Hence, we did not combine LV thrombectomy and decompressive craniectomy in the same sitting.

Such cases coming for noncardiac surgery in an emergency setting are rare and difficult to manage due to complexity in decision-making and also due to their complex cardioneurological hemodynamic interplays. Although speculations that LV thrombus plays a positive role in the acutely damaged myocardium by offering mechanical support to the infarcted areas by reducing myocardial bulge and preventing rupture resulting in effective myocardial contraction have been proposed,[6] LV clot presents as "sword of the Damocles," producing an imminent everlasting risk of systemic embolism.

Conflict of Interest

None declared.

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