

## Troubleshooting for Epileptiform Activity during Percutaneous Transvenous Mitral Commisurotomy

### Abstract

Percutaneous transvenous mitral commisurotomy (PTMC) is a frequently used minimally invasive procedure for patients with symptomatic mitral stenosis. However, it is not without complications. Few complications which are distinctive to the procedure are thromboembolism, left-to-right shunts, mitral regurgitation, cardiac tamponade and complete heart block. We present the case of a 32-year-old female patient scheduled for a PTMC, who had multiple complications during the procedure. She developed cardiac tamponade for which pericardiocentesis and autotransfusion was done. Subsequently she exhibited epileptiform activity for which there was a diagnostic dilemma due to the presence of multiple confounding factors. However, she had a complete recovery without any residual sequelae at the time of discharge.

**Keywords:** Cardioemboli, epileptiform activity, fentanyl, local anesthetic systemic toxicity, percutaneous transvenous mitral commisurotomy

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### Introduction

Rheumatic heart disease (RHD) contributes largely to the burden of valvular heart diseases in the Indian subcontinent with prevalence rates varying from 4.54 to 6/1000 population.<sup>[1]</sup> Mitral stenosis (MS) is probably one of the most common valvular pathologies to occur as a sequelae of RHD.<sup>[2]</sup> Inoue *et al.* described percutaneous transmitral commissurotomy (PTMC) as a modality of treatment for mitral stenosis. PTMC can be associated with complications like thromboembolism, left-to-right shunts, mitral regurgitation, cardiac tamponade and complete heart block.<sup>[3,4]</sup>

We present the combined occurrence of cardiac tamponade and epileptiform activity during PTMC in a 32-year-old female patient.

The cause for the seizures proved to be a diagnostic dilemma.

### Case Report

Our patient was a 32-year-old female, 149 cm in height, weighing 36 kg with severe symptomatic MS. She did not have any comorbidities and her past history was insignificant. The transthoracic

echocardiography (TTE) examination prior to the procedure revealed critical MS (mitral valve orifice area of 0.6 cm<sup>2</sup>) with thickened leaflets, submitral fusion, mitral valve gradient of 26/13, pulmonary artery systolic pressure of 59 mmHg and a suspected clot in the roof of the left atrium (LA). In order to confirm the findings, a transesophageal echocardiography (TEE) examination was performed. This confirmed the diagnosis of critical MS, but ruled out the presence of a clot in the LA. Instead, the TEE exam documented grade 3 spontaneous echo contrast in the LA.

In view of the above mentioned findings, she was scheduled to undergo a high risk PTMC. Her preoperative blood investigations were unremarkable except for a hemoglobin level of 10.5 g%.

Her baseline vitals were as follows - blood pressure (BP) - 122/76 mmHg, Pulse Rate (PR) - 101 beats per minute (bpm), room air saturation (SpO<sub>2</sub>) - 98%. She had an 18G peripheral intravenous (IV) catheter *in situ* in the left upper limb.

Right femoral venous and arterial access were secured using Seldinger's technique under local anesthesia - 10 ml 1% lignocaine was used for infiltration. 8F sheaths were inserted through both the

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vascular punctures. Mullin's sheath and Brockenbrough needle were used for transseptal puncture during which the patient developed hypotension (74/40 mmHg) and bradycardia (38 bpm). Diminished movement of the left cardiac border was visualized on fluoroscopy simultaneously and hence a diagnosis of cardiac tamponade was made. Injection atropine 0.6 mg was administered IV and second large bore peripheral IV cannula was inserted. Oxygen was supplemented via facemask. A 7F pigtail catheter was immediately inserted using echo and fluoroscopy guidance into the pericardial sac and autotransfusion was started. Poststabilization, her vitals were BP – 140/80 mmHg, PR– 110 bpm, SpO<sub>2</sub> –100%. A blood gas sample revealed all parameters to be within normal limits except for the hemoglobin which was 8.5 g%. The right-sided sheaths were kept *in situ* and the procedure was resumed by inserting a second Mullin's sheath through a left femoral venous access after infiltrating 10 ml 1% plain lignocaine.

During manipulation of the second Mullin's sheath, the patient complained of severe pain over the left lower limb catheterisation site, for which, IV fentanyl 40 mcg was administered. Following this, she developed altered sensorium associated with tonic posturing and jaw clenching. As there was no response to IV midazolam 2 mg, IV propofol 30 mg was administered which terminated the episode. Simultaneously her respiration was assisted with bag and mask. Her vitals remained stable except for tachycardia (147 bpm) and post ictal confusion.

PTMC was resumed and the procedure was completed without any further complications. Postprocedure, she was shifted to the recovery room where she recovered completely within half an hour. Rest of the hospital stay was uneventful and the patient did not have any residual neurological deficit at the time of discharge.

## Discussion

The peak incidence of rheumatic MS has been known to occur between 30 and 39 years of age with nearly two-thirds (65.9%) of the patients being females.<sup>[1]</sup> PTMC is recommended for symptomatic patients with severe MS and favorable valve morphology as determined by Wilkin's score.<sup>[5]</sup> It was first used by Inoue in 1982 to provide immediate symptomatic and hemodynamic improvement.<sup>[6]</sup>

PTMC, like any other procedure, is not bereft of complications. Bleeding, thromboembolic events, seizures, development of mitral regurgitation, residual atrial septal defect, perforation of cardiac chambers, cardiac tamponade<sup>[3,4,6]</sup> are few of the reported complications.

During the course of the procedure, our patient developed cardiac tamponade. The incidence of tamponade during PTMC varies from 0% to 9%.<sup>[7]</sup> The tamponade was immediately diagnosed and treated. Although our patient did not require any inotropic support or surgical intervention, there have been individual and case series

reports where patients have required the same, contributing to increased morbidity and mortality.<sup>[3,7]</sup>

An additional dose of local anesthetic was infiltrated to secure the second femoral access. Fentanyl was also administered as the patient complained of pain following which she exhibited epileptiform activity. The differential diagnoses considered for the etiology of the epileptiform activity were cardiogenic embolic phenomena, local anesthetic neurotoxicity and fentanyl induced seizures.

Although cerebral angiography was considered on table to rule out cardioemboli, it was not done since the presenting feature was predominant epileptiform activity without any associated focal neurological deficits. Arboix and Alió in their review on cardioembolic strokes<sup>[8]</sup> state that although there is no gold standard to provide a diagnosis, 79.7% of such embolic events are associated with sudden onset of focal neurological deficits. They also state that emboli arising from the cardiac chambers are often large and hence more likely to cause severe stroke, disability and death with a low frequency of symptom free hospital discharge.

The second possible differential diagnosis considered was local anesthetic systemic toxicity (LAST), as a result of either intravascular injection or higher cumulative dose. Direct intravascular injection leading to LAST may not have any premonitory symptoms and the patient can directly develop seizure activity that may progress to cardiac excitation (tachycardia, ventricular arrhythmias).<sup>[9]</sup> After exhibiting epileptiform activity, the patient developed tachycardia (147 bpm). This pattern correlated with the described pattern of initial neurotoxicity followed by cardiotoxicity in LAST following intravascular injection.

Epileptiform activity has also been described after the administration of opioids including fentanyl.<sup>[10,11]</sup> Since the occurrence of seizures concurred with the administration of fentanyl, opioid-induced seizure was a possibility. Postulates proposed to explain the neuronal excitation are dose dependent selective stimulation of  $\kappa$  and  $\mu$  opioid receptors, decreased GABA mediated interneuronal inhibition of pyramidal neurons and inhibition of hyperpolarization activated potassium currents.<sup>[11]</sup> There also have been case reports describing the potentiation of epileptiform activity due to lignocaine by fentanyl.<sup>[12]</sup> Since the administration of fentanyl and lignocaine concurred in our patient, the potentiation could have led to development of seizures.

## Conclusion

Seizures during PTMC is not an uncommon occurrence. Both anesthetic and procedural factors must be considered during development of complications. Caution should be exercised while using local anesthetic, even if it is for surface infiltration, especially when used in conjunction with fentanyl.

### Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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### Conflicts of interest

There are no conflicts of interest.

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