CASE REPORT

MitraClip implantation under sedation

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Abstract The percutaneous MitraClip system is a catheter-based device designed to perform edge-to-edge mitral valve (MV) leaflet repair at the site of regurgitation. MitraClip implantation is an alternative procedure in patients at high surgical risk with symptomatic severe mitral regurgitation (MR) who are not candidates for MV repair/replacement due to their degree of comorbidity and associated high mortality risk. The procedure is guided by 3-dimensional (3D) transesophageal echocardiography (TEE) and fluoroscopy. A clip is positioned between the anterior and posterior leaflet to reduce valve regurgitation. Quantitatively, the reduction in MR is less than with surgical repair, but it significantly improves patients’ quality of life and functional capacity. Advantages are avoidance of sternotomy and cardiopulmonary bypass, beating-heart repair of the MV and reduction in post-operative duration of mechanical ventilation, intensive care unit (ICU) stay and need for blood transfusion.

General anesthesia (GA) with orotracheal intubation is the most common approach in the literature because of the TEE probe and the need for the patient to be immobilized during the procedure. Since May 2014, of the 39 patients who have had MitraClip implantation in our hospital, only two were under deep sedation.

We describe here the case of a MitraClip implantation performed under deep sedation with ketamine and propofol infusion in a patient unsuitable for surgical repair because of her comorbidities.

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Background

Mitral regurgitation (MR) is one of the most common valve disease. Valve repair is often recommended in severe cases. In presence of low surgical risk and high probability of durable repair, early surgery may represent the preferred approach. However, it might not be feasible because of patients’ co-morbidities. This paper reports case of a Percutaneous MitraClip (MC) procedure performed under deep sedation with ketamine and propofol perfusion in a patient refused for surgical repair because of her co-morbidities.

Case report

A 73-year-old female with severe MR diagnosed 2-3 years previously, New York Heart Association Class III heart failure (with normal left ventricular ejection fraction), Global Initiative for Chronic Obstructive Lung Disease stage 4 chronic obstructive pulmonary disease, Roizen’s Classification of Dyspnea Grade 4, with air trapping, a negative bronchodilatation test and nocturnal oxygen therapy (2 L/min 12 h), ruled out for surgery. American Society of Anesthesiologists (ASA) physical status 4.

Three weeks before the procedure, the patient had a multidisciplinary consultation (with the anesthesiologist, cardiologist assistant, interventional cardiologist and imaging cardiologist) to stratify risk, explain the entire procedure to the patient and companion, answer questions and read, understand and sign the informed consent form.

She was admitted the day before the procedure.

Before being brought to the hemodynamic intervention room, cefazolin 2 g intravenous (IV) was administered for antibiotic prophylaxis and fasting confirmed.

ASA standard monitoring was carried out with pulse oximetry, 5-lead electrocardiogram, and non-invasive blood pressure 5-5 minutes. End tidal carbon dioxide and capnography (via nasal cannula) and bispectral index were also monitored, not for the absolute value but rather for tendency and variation. Once the interventional cardiologists catheterized the femoral artery, we had invasive blood pressure monitoring visible to the whole team on-screen, so we chose not to catheterize a peripheral artery, as most anesthetic plans describe, due to lack of added clinical benefit and an increased risk of complications.

Another IV line was inserted in the opposite limb. A standard bite block was introduced with the cooperation of the patient. Propofol 1% (1-2 mg/kg/h) and ketamine (0.5 mg/kg/h) infusions were started in different IV sites until deep sedation was achieved. The TEE probe was gently introduced after applying topical lidocaine to the oropharynx and cardiac assessment was repeated before the intervention.

Unfractionated heparin (70-100 IU/kg) was given after trans-septal puncture, which is a critical point of the procedure. Once the MitraClip system had been aligned, the clip was advanced into the left ventricle with arms opened and, under TEE guidance, the arms grasped the leaflets creating a double orifice. Two clips were needed to attain an acute reduction from severe to mild-to-moderate MR (2+/4+) with improved cardiac output, confirmed by an increase in the pressure profile. The TEE showed maximum and medium
gradients of 14 mmHg and 8 mmHg respectively. The infusions were stopped at the end of the procedure, which took about two hours. The patient was wakened and transferred to coronary ICU without complications.

While in the hospital, the patient had no exacerbation of her respiratory or cardiovascular disease or hemodynamic or electrical instability. She was discharged the day after the procedure.

Discussion

MitraClip implantation is a successful alternative in high-risk patients with symptomatic severe MR. The desired outcome of the procedure is MR reduction <2+, which is usually obtained with one clip. However, more clips may be needed, as in our case. The main complications are mechanical (clip entrapment in the chordae within the left ventricle (LV), partial clip detachment, pericardial tamponade, atrial septal rupture) and functional (MR >2+ after implantation of 2/3 clips, acute LV dysfunction due to abrupt increase in LV afterload following reduction of the MR). The role of 3D TEE in guiding the procedure is crucial in terms of improving the grasping phase and verifying the final outcome. Perioperative management requires a team approach and the same precautions as in the surgical setting, including postoperative monitoring in the ICU. Consequently, if we are moving toward less invasive therapeutic interventions, why not allow anesthetic management to evolve, and interfere less with a patient’s baseline homeostasis, reducing the risks associated with GA in patients with severe comorbidities. A few articles have already been published describing deep sedation as an alternative to maintain the patient’s baseline physiological state.

As we know, the most common intravenous anesthetics used for sedation induce a reduction in peripheral vascular resistance and heart rate (HR) leading to decreased cardiac output (CO), which will underestimate MR. We therefore aimed to use a drug that would achieve deep sedation, but maintain hemodynamic stability, remaining as close as possible to the patient’s baseline and with the severity of the patient’s respiratory condition in mind, maintain spontaneous ventilation.

Ketamine is useful for short procedures. Functionally it "dissociates" the thalamus from the limbic cortex so the patient is unable to process or respond to sensory input. It has a wide spectrum of pharmacological effects including sedation, catalepsy and somatic analgesia, allowing the patient to tolerate TEE, bronchodilation and maintenance of spontaneous ventilation, and sympathetic nervous system stimulation (in contrast to other anesthetics) it increases arterial blood pressure, HR and CO. However we cannot forget that large bolus injections of ketamine can increase pulmonary artery pressure and myocardial workload, so we have to be selective about using it in patients with impaired ventricular function or coronary disease. The increase in salivation can be troublesome, requiring extra vigilance from anesthesiologists, with frequent suction of oropharyngeal secretions or premedication with an anticholinergic agent.

Of all the induction agents, propofol has the most profound cardiovascular depressant effects. It inhibits sympathetic vasoconstrictor activity, reducing systemic vascular resistance, preload and cardiac contractility, and lowering arterial blood pressure and CO, which can be severe in very young or very old patients, patients receiving β adrenergic blockers or those with impaired ventricular function. Debate surrounds whether propofol has a direct depressant effect on the myocardium or not. Adequate intravascular volume, as well as slow and titrated dosing, can minimize cardiovascular effects. It is also a respiratory depressant and it inhibits normal response to hypercapnia even at sedative doses. However, rapid onset of action, short distribution half-life and high clearance rate contribute to relatively quick recovery following continuous infusion. Propofol also potentiates immobility.

Conclusion

Using both agents we were able to achieve a balance between their beneficial effects and minimizing the less desirable ones and, as in this case, the residual mitral regurgitation during the procedure was more faithfully reproduced, which may also contribute to the success of the intervention. Obviously, sedation requires that anesthesiologists and the rest of the team take extra care, but evolution does not necessarily mean less work, effort or commitment.

Conflicts of interest

The authors have no conflicts of interest to declare.

References