

## CLINICAL CASE

Vol. 31, No. 4 October-December 2008  
pp 315-321

## Anesthetic management of a posttraumatic left ventricular pseudoaneurysm: Report of a case and literature review

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## Abbreviations:

TIMI: Thrombolysis Myocardial Infarction  
UIK: Unidades inhibitorias de caliceínas

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Received for publication: 27-11-07

Accepted for publication: 21-02-08

## SUMMARY

The following report consists of a left ventricular pseudo-aneurysm case report of a chronic complication secondary to a gun-injury traumatism. The case refers to a women who developed pain, cerebral emboli and heart palpitations after the injury. The objective of this work is to review the physiopathology, the diagnosis tools and integral anesthetic management of the studied pathology.

**Key words:** Pseudo-aneurysm, ventricular traumatism, anesthetic management.

## RESUMEN

*Reportamos el manejo anestésico de un caso de pseudoaneurisma ventricular izquierdo que se presentó como complicación crónica secundaria a un traumatismo por arma de fuego. El caso corresponde a una mujer que después del trauma presentó dolor precordial, eventos embólicos cerebrales y sensación de palpitaciones. El propósito de este trabajo es revisar la fisiopatología, métodos diagnósticos y el manejo anestésico integral de la patología.*

**Palabras clave:** Pseudoaneurisma, traumatismo ventricular, manejo anestésico.

## INTRODUCTION

The cardiac pseudoaneurysm is defined as a rupture of the myocardial wall that is contained by adhesions of the pericardial and epicardial pericardium in the rupture area. The wall of the pseudoaneurysm consists of fibrous tissue and lacks structural elements that are in a normal cardiac wall<sup>(1)</sup>. Cardiac aneurysm formation caused by cardiac trauma is a rare heart and the formation of pseudoaneurysms is considerably less frequent<sup>(2)</sup>. When present, the patient has the risk of rupture and other complications such as congestive heart failure, embolic events and cardiac arrhythmias<sup>(3)</sup>.

The purpose of this report is to present the anesthetic management used in this condition whose presentation is rare, but which involves a series of physiopathological implications to be considered for a proper anesthetic management.

## CASE REPORT

A female patient 56 years receiving in december 2002 a bullet impact in the sternal region of the chest that caused her hemopneumothorax, fracture of the sixth costal arch and type IV cardiac injury, which needed anterior and left

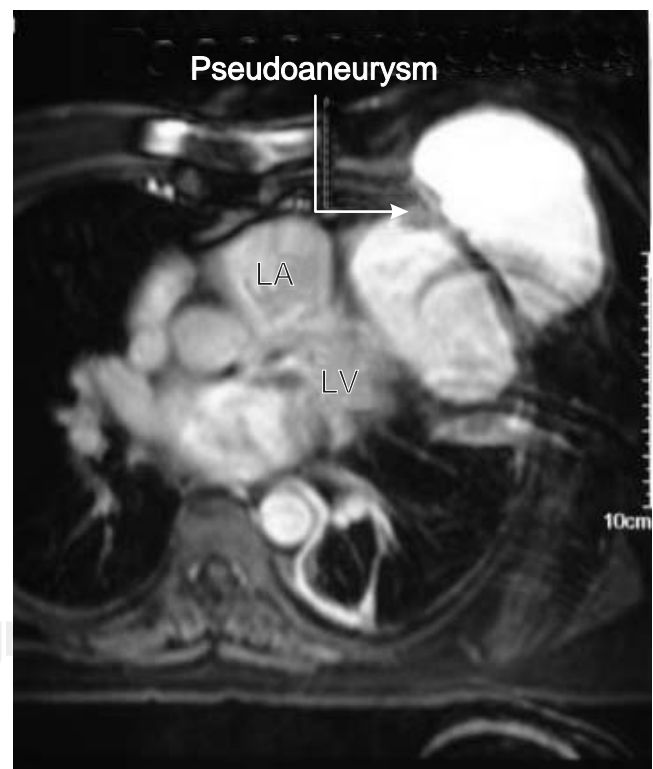
lateral exploratory thoracotomy. She remained asymptomatic throughout 2003 and then began to perceive a pulsatile mass in the left pectoral region which gradually increased in size, dyspnea at large to medium efforts, episodes of precordial pain of oppressive type and 2-3/10 in intensity. This condition progressed to dyspnea at rest, palpitations and diaphoresis, in 2005 type 2 diabetes mellitus was added and she received irregular treatment with oral hypoglycemic agents; in addition she had systemic hypertension which it irregularly treated with captopril. In January 2006, she presented a clinical picture of cardiogenic type-cerebral vascular disease of cardiogenic type resulted in a reduced visual acuity; for this reason she was studied in this institute.

The physical examination found a conscious, oriented, mesomorphic body type, freely chosen attitude, full, well-formed woman. She had isocoric and normally reactive pupils, permeable nostril, oral cavity with appropriate opening, neck without jugular plethora, carotid pulses of normal intensity, and she did not have carotid bruits. Soft, 20 x 15 cm in diameter, painless, non-moving, and pulsatile mass was observed in the left lateral thoracic region. Apical impulse was palpated in the sixth left intercostal space to 2 cm from the clavicular midline. The cardiac sounds were rhythm of normal intensity, the first and second sounds were normal, and there no were cardiac bruits. There were symmetrical amplexation and amplexation movements with bibasal crepitant rales, and the abdomen had normal peristalsis and did not have visceromegalies. Lower limbs had mild ankle edema, peripheral pulses were palpated in normal intensity and capillary filling was less than 3 seconds. The electrocardiogram showed sinus rhythm, PR interval of 160 ms, QRS interval of 160 ms, QT interval of 400ms, QRSa +150°, and complete right bundle branch block of bundle of His. The echocardiogram reported post-operative status of ventricular remodeling, moderate mitral insufficiency, moderate tricuspid insufficiency, pulmonary artery systolic pressure of 61 mmHg, generalized hypokinesia, left ventricular ejection fraction of 45%. Coronariography showed anterior descending with a 90% distal lesion, and TIMI III flow. Ventriculography showed akinesia of the anterior wall and an image of two sacks compatible with a pseudoaneurysm of the anterior left ventricular wall; one of two sacks extends retrosternally and another leaves the thoracic cavity to the left breast. Left ventricular ejection fraction was 50%, and left ventricular end diastolic pressure was 35 mmHg. Computed tomography showed a large left ventricular pseudoaneurysm protruding through the fourth left intercostal space; it also showed a probable thrombus in the posteroinferior portion of the left ventricle. Postsurgical changes in the sternum and calcified aortic arch were observed. Magnetic reso-

nance imaging showed large left ventricular aneurysm which has invaginated through a hole in the chest wall, it was located in the homolateral submammary region (Figure 1), ejection fraction of left ventricle was 50%. Myocardial perfusion scintigram showed a severe perfusion defect involving the apex and the apical and middle thirds of the anterior, septal and lateral walls. There was lack of movement and thickening of the apex and the apical and middle thirds of the anterior, septal and lateral walls. The stress electrocardiogram was normal.

## ANESTHETIC MANAGEMENT

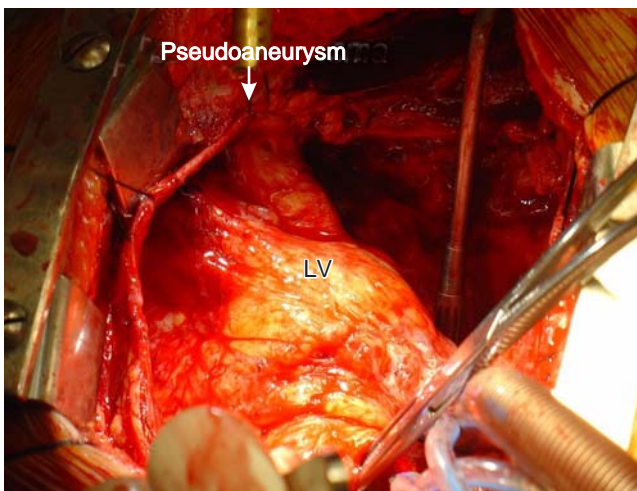
Resection of the pseudoaneurysm and remodeling of the left ventricle under balanced general anesthesia was performed. Monitoring included continuous electrocardiogram (leads DII and V5), invasive blood pressure in the left radial artery, pulse oximetry, central venous pressure, pulmonary flotation catheter, urine output, esophageal and rectal temperature. Peripheral via is channeled using a 14G catheter. The induction of intravenous anesthesia with fentanyl, etomidate and pancuronium was performed, an intubation was executed using an 8.0 mm ID cannula, and she



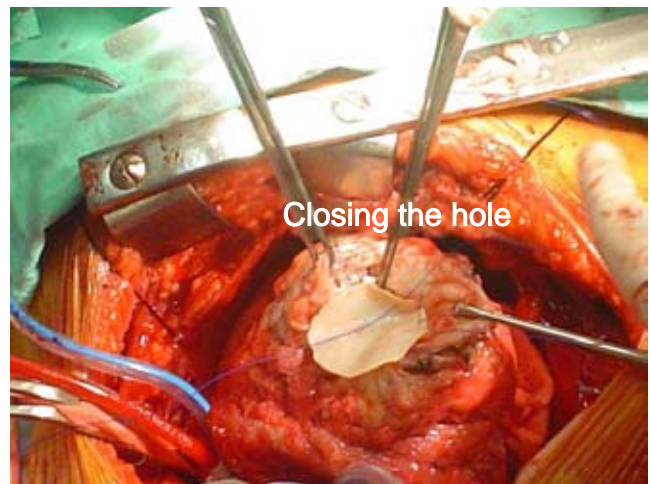
**Figure 1.** Magnetic resonance imaging of ventricular pseudoaneurysm. Left atrium (LA), left ventricle (LV).

was administrated 100% oxygen, sevoflurane, and fentanyl infusion. A dose of 2 million units aprotinin was administered and support with dobutamine was started by a hemodynamic profile that showed evidence of low output syndrome. At the prebypass stage, after administration of heparin and after the surgical dissection, bleeding presented in layers that committed the patient's hemodynamics, required fluids were administered and extracorporeal circulation for circulatory assistance was started to complete the surgical dissection (Figure 2). Aortic cross-clamping and cardiac arrest with hyperkalemic cold blood cardioplegia solution were performed to accomplish the resection of ventricular pseudoaneurysm (Figure 3) and ventricular re-

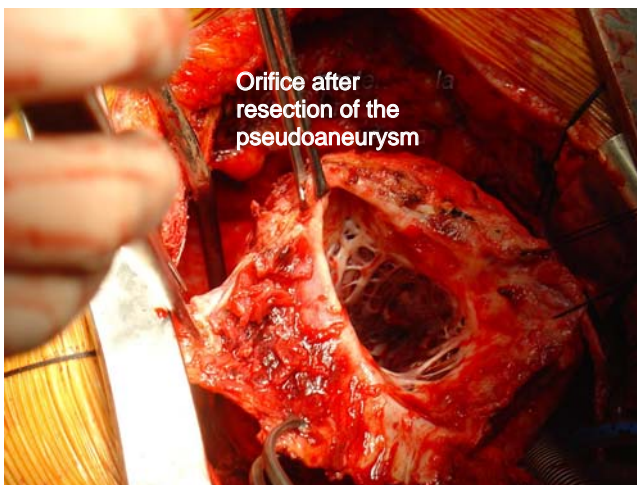
modeling (Figures 4 and 5). After a clamping of 71 minutes, the aorta is declamped with normothermia, ventricular fibrillation occurred which became ventricular tachycardia without a pulse, which lidocaine and magnesium and a direct electric shock of 30J were administered, and then sinus rhythm was observed. Ventricular function was assisted with dobutamine, milrinone, and norepinephrine obtaining a response sufficient to separate the patient from cardiopulmonary bypass. Heparin was reversed with protamine, the addition the dose of 2 million units aprotinin was completed in transoperative and after the extracorporeal circulation. However, hemodynamic parameters were in low normal range, considerable bleeding occurred which



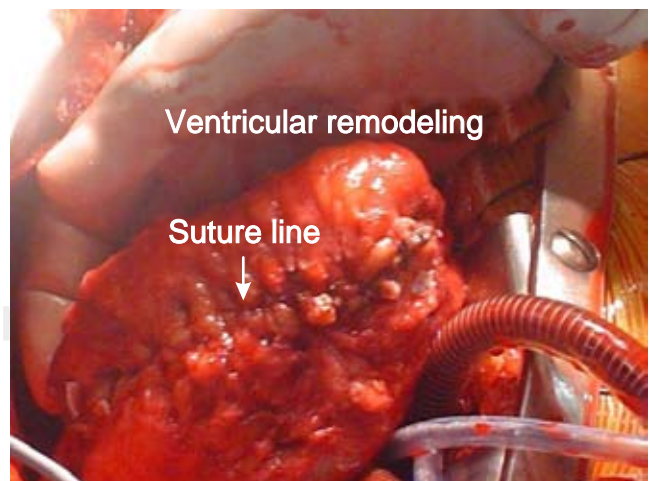
**Figure 2.** Dissection of ventricular pseudoaneurysm. Left ventricle (LV).



**Figure 4.** Closing the hole in the ventricular pseudoaneurysm with bovine pericardium.



**Figure 3.** Resection of ventricular pseudoaneurysm.



**Figure 5.** Ventricular remodeling.



required polytransfusion with 250 mL albumin at a concentration of 20%, 10 units erythrocyte concentrate, 6 units fresh frozen plasma, 5 platelet apheresis and 2 pool cryoprecipitates. In the intensive care unit made there was 2,180 mL hemorrhage which required mediastinal re-examination and packaging. Unpacking and sternal closure were performed 48 hours later. Inotropic support was instituted and prone to low output syndrome, prolonged intubation, and mechanical ventilation were observed. This situation necessitated tracheostomy. Twenty days after she had *Morganella* and *E. coli* pneumonia, as well as *Pseudomonas aeruginosa* and *Candida albicans* in blood cultures. Also secretion occurred from the sternal wound caused by *Aspergillus*. The latter was treated with triple antimicrobial schemes. An antibiogram showed that these antibiotics had antibiotic sensitivity. Control computed tomography reported data suggestive of osteomyelitis and therefore total sternotomy and pectoral rotation were performed.

The patient, in spite of using imidazoles, amphotericin B and triple antimicrobial scheme, evolved to septic shock, renal failure by renal tubular acidosis was added, this situation required hemodialysis. Systemic hypotension and non-pulse electrical activity were presented during the hemodialysis. Advanced cardiopulmonary resuscitation maneuvers were done but without answer.

## DISCUSSION

The reports described in the international field are intended for diagnosis and management, primarily medical diagnosis and surgical management of posttraumatic left ventricular pseudoaneurysm, and they made no mention or emphasis of the anesthetic management of these patients.

Although presentation of these cases is very rare, there are reports of them since 1969 when Killen<sup>(4)</sup> et al reviewed and reported the first cases of post-traumatic aneurysms secondary to external violence and contusion and/or pervasive trauma.

Killen mentions that the interval between the chest trauma and diagnosis was between 5 months and up to 24 years after trauma; two of these patients underwent extensive resection of the ventricular pseudoaneurysm and observed complications were severe, complex and difficult to management, such as ventricular pseudoaneurysm rupture, cardiac failure, blood embolism, and severe and fatal cardiac arrhythmias.

The ventricular pseudoaneurysm may occur after chest trauma injuring the heart and thus cause a myocardial infarction. In these cases, Alain<sup>(5)</sup> et al recommend that the determination of serum troponin I values to help assess the extent of existing myocardial injury. It can also occur after a heart surgery such as the correction of con-

genital cardiac anomalies and mitral and aortic valve replacement; in these latter it is caused by manipulation of the surgical areas or it occurs in sites of the various incisions<sup>(1)</sup>. Other less common causes of ventricular pseudoaneurysm include infective endocarditis, syphilis, traumatic myocarditis, disseminated tuberculosis, rheumatic carditis, and lymphoma<sup>(1,6)</sup>.

Yeo<sup>(1)</sup> et al revised 52 patients with cardiac pseudoaneurysms and found that 48% of these pseudoaneurysms were discovered incidentally in asymptomatic patients, 8% of them were found when the patient had a picture of acute myocardial infarction and cardiac tamponade; another form of clinical presentation was congestive heart failure in 15% of patients, chest pain in 13%, syncope or arrhythmia in 10%, and systemic embolism in 6%. The ventricular pseudoaneurysm occurred after cardiac surgery in 58% of patients and after myocardial infarction in 42%. The pseudoaneurysms were located in the posterolateral and inferior wall of the left ventricle in 82% after acute myocardial infarction. Were they also found in the outflow tract of right ventricle in 87% of patients after congenital heart surgery, and in the wall of posterior subannular region of the mitral valve in 100% of the cases after mitral valve replacement and in the subaortic region in 100% of cases after aortic valve replacement. The post-traumatic pseudoaneurysms are typically found in the anterior wall of the left ventricle.

Symptoms include recurrent chest pain that may be associated with symptoms of hypotension, decreased heart sounds, pericardial rub, elevated right and left filling pressures, sinus bradycardia or junctional rhythm. When the pseudoaneurysm is large as in this case can produce an apical impulse<sup>(3)</sup>.

The signs and symptoms rarely indicate the diagnosis of pseudoaneurysm. These symptoms may be chest pain or congestive heart failure and arrhythmias when they are secondary to acute myocardial infarction; but non-cardiac symptoms such as data on syncope and systemic embolism may be observed<sup>(1)</sup>.

A substantial number of patients with left ventricular pseudoaneurysm are asymptomatic, approximately 10%<sup>(6)</sup>. Although surgical repair is the treatment of choice, the conservative management in selected patients with high surgical risk it seems reasonable as dies for cause of future disruption do not occur<sup>(1)</sup>.

Gill<sup>(7)</sup> et al reported a case of a woman of 69 years with a history of palpitations from 11 years of age, condition which was assessed using an electrophysiological study, and after this study, the patient developed a small submitral left ventricular pseudoaneurysm. The patient remained asymptomatic and the lesion was detected in a routine echocardiographic procedure, which resolved spontaneously and no injury was observed by echocardiography months later.

There are several imaging methods that can be used for diagnosis, including two-dimensional echocardiography<sup>(8)</sup>, computed tomography, magnetic resonance imaging<sup>(9)</sup>, and ventricular angiography<sup>(10)</sup>. Moen<sup>(11)</sup> et al reported a rare case of a patient of 80 years of age with an aneurysm of right ventricular outflow tract and right ventricular pseudoaneurysm caused by a vehicular accident in which the diagnosis was made properly by echocardiography .

The large pseudoaneurysms, as in our case, indicate that after a severe thoracic trauma it may develop slowly and do not break because it is contained within the chest wall and this delays their breakdown<sup>(11)</sup>. Although acute pseudoaneurysms often cause death by tamponade, the chronic pseudoaneurysm may be relatively less threatening, because the clinical course is the chronic adhesion created by a hematoma.

Direct Cardiac rupture is the cause of death in the 7-10% in patients with an acute myocardial infarction; contained rupture of the pseudoaneurysm is a rare complication of acute myocardial infarction<sup>(12)</sup>. The left ventricular pseudoaneurysms are prone to rupture in 30 to 45% of cases, so it is recommended to be repaired surgically as in asymptomatic patients, as there is a high risk for congestive heart failure due to is a non-contractile cavity and the high possibility of embolic events<sup>(3)</sup>. As is the case of a man 47 years of age reported by Makaryus<sup>(13)</sup> et al, who underwent a surgery for a penetrating chest wound by a knife that occurred 25 years ago and subsequently developed a rare sequel of the contained rupture of the left ventricle, forming a pseudoaneurysm. After angina the repair and immediate reconstruction of injury were needed because there was a high probability of rupture and death.

## ANESTHETIC CONSIDERATIONS

### Preoperative assessment

The preoperative cardiovascular evaluation is indispensable in this type of procedure because it allows to know the anatomy of the pseudoaneurysm, ventricular function, and general conditions such as hypertension that forced to do an anesthetic induction without tenesmus, cough, or hypertension to prevent ventricular rupture. Know then the characteristics of the ventricular defect and patient will increase safety of surgical anesthesia, plan immediate intraoperative and peroperative approach, in order to reduce complications and outcomes.

The preanesthetic evaluation should include the basic aspect from the clinical history, and identify information on the risk of bleeding, impaired renal function, development of arrhythmia, previous infections, low output syndrome, lung diseases, and nutritional disorders and development or

exacerbation of neurological deficit secondary to cerebrovascular disease, and previous medication.

The evaluation should also include the physical examination and highlight the presence of fever due to infections and the presence of hidden systemic arterial hypertension, which shall be treated in order to avoid difficulties in the postoperative management, because it was observed that the association of systemic arterial hypertension with pseudoaneurysm causes frequently rhythm disturbances and electrocardiographic changes, increasing cardiac morbidity<sup>(14)</sup>.

In the evaluation of laboratory analysis should have a hematocrit greater than 35% due to the risk of bleeding and transfusion requirements with these patients, in addition to a platelet count greater than 100,000/mm<sup>3</sup>. The increase in the plasma creatinine is a strong predictor of postoperative renal failure, because it is necessary to optimize renal function by taking adequate measures<sup>(15)</sup>.

Changes in the bilirubin values and liver enzymes should be evaluated to rule out some degree of hepatic dysfunction, which significantly increases the perioperative morbidity.

The evaluation must complemented with studies such as an electrocardiogram, which will identify abnormalities in heart rhythm, must completed with, old or recent myocardial infarction and left and right ventricular hypertrophy: a hemodynamic study to identify the patients with coronary heart disease adjacent to the pseudoaneurysm, additionally a chest X-ray, magnetic resonance, computed tomography, and echocardiography to establish an final anatomic diagnosis of the pseudoaneurysm.

The existence of recoverable myocardial regions should established using additional studies justifying the perioperative risk such as a Thallium-201 myocardial perfusion scintigraphy, technetium 99 radioisotope ventriculogram, echocardiogram under stimulus inotropic drugs, and proton emission tomography in patients with a ventricular pseudoaneurysm and significant deterioration of cardiac function

### Intraoperative monitoring

The patients with a ventricular pseudoaneurysm undergoing heart surgery have fast hemodynamic changes, so it is necessary to perform an appropriate invasive monitoring at all times for obtaining a correct interpretation and implementing appropriate therapeutic measures.

Pseudoaneurysm monitoring includes: Electrocardiogram (leads DII and V5), invasive blood pressure, central catheter for evaluation of central venous pressure, pulmonary artery catheter, urinary output, esophageal and rectal temperature, capnography, pulse oximetry, measurement of cardiac output, periodic analysis of arterial blood gases, transesoph-

ageal echocardiography and measurement of O<sub>2</sub> transport to tissues. Additionally, it is necessary an appropriate size of venous access for the potential administration of fluids and blood.

### **Management in the pre-cardiopulmonary bypass**

The induction of anesthesia must be deep, based on high doses of opiates, hypnosis with benzodiazepines or other hypnotic induction agent which does not cause cardiovascular depression. Muscle relaxation should select non-vagolytic drug, though it will depend on the cardiovascular condition that the patient has at this time.

Often there is a high risk of bleeding in these patients, therefore, fresh frozen plasma, vitamin K, aminocaproic acid or aprotinin should be considered to minimize bleeding.

Transesophageal echocardiography is used before cardiopulmonary bypass to quantify the end-diastolic volume of left ventricle, end diastolic diameter, stroke volume, and the diameter of the ventricular chamber.

### **Management in the cardiopulmonary bypass period**

When the cardiopulmonary bypass period is carried out and after the cardiac incision of ventricular pseudoaneurysm has been closed, and once adequate anastomosis has been established, residual air must be carefully removed from the heart through left ventricle using echocardiographic guidance and by allowing a gradual ejection.

When, before the separation of the cardiopulmonary bypass, a pulmonary flotation catheter is not placed; then a left atrial line is placed and added to the hemodynamic monitoring to measure the left preload. Care must be taken not to overfill the heart because it presents a poor compliance and it can easily Care must be taken not to, and severe dilation leads to acute heart failure. The overall ST-segment elevation is typically seen with the restoration of a heartbeat, and it is probably due to the combination of the inevitable entry of air into the coronary arteries and myocardial edema. These electrocardiographic changes tend to persist even after a gradual improvement in cardiac function has been observed, and they returns to baseline after two hours of separation from cardiopulmonary bypass.

### **Management in the post-cardiopulmonary bypass**

After separation from cardiopulmonary bypass, mean arterial pressure should be maintained between 60 and

70 mmHg because the cardiac suture line is big, as there is a substantial potential of catastrophic bleeding at the stage of hypertension. Vasodilators are frequently used (eg, nitroglycerin, nitroprusside), and in some cases these patients may tend to vasodilation, thus requiring vasopressor support with norepinephrine. This discrepancy may be partly because these patients are under chronic treatment with large doses of inhibitors of angiotensin-converting enzyme. if it is necessary inotropic support, the potential effects of hypercontractility on the suture line are again significant. In the post-cardiopulmonary bypass period, transesophageal echocardiography is used for monitoring the progress of the restoration of ventricular function. It has been observed that the force of contraction gradually recovers for 20-30 minutes after removal from cardiopulmonary bypass. The presence of ventricular air is associated with transient abnormalities of wall motion. Ventilatory management is facilitated by the use of pressure-control ventilation in the intensive care unit, post, since these patients have some degree of edema pulmonar<sup>(16)</sup>.

## **CONCLUSIONS**

- The cardiac pseudoaneurysm is a rupture of the myocardial wall that is contained by adhesions of the pericardial and epicardial pericardium in the rupture area.
- The cardiac aneurysm formation as sequelae of cardiac trauma is a rare entity and the formation of pseudoaneurysms is considerably less frequent.
- The preoperative cardiovascular evaluation is indispensable in this type of procedures because it increases the safety of surgery and improves immediate intraoperative and postoperative management by reducing complications and thus improving outcomes.
- The patients with a ventricular pseudoaneurysm undergoing heart surgery have fast and large hemodynamic changes, so it is necessary to perform an appropriate invasive monitoring at all times for obtaining a correct interpretation and implementing appropriate therapeutic measures.
- The anesthetic management is specific to each period of cardiopulmonary bypass and focuses primarily on monitoring, hemodynamic stability and maintenance of the balance between oxygen supply and consumption, primarily by avoiding systemic hypertension, tachycardia, hypercontractility and the presence of heart arrhythmias.

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