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Editorial

Electrocardiographic changes in the ST segment during anesthesia: Things to consider☆

Cambios en el segmento ST del electrocardiograma durante la anestesia: en qué pensar

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Cardiac morbidity and mortality in non-cardiac surgery will depend on associated factors and the type of surgery. The incidence can be up to 30%, and the high prevalence of heart disease in the general population requires constant vigilance. Despite careful assessment and the control of triggering factors such as tachycardia, increases in catecholamine and cardiac contractility levels may lead to an imbalance in myocardial oxygen supply and consumption. Sudden electrocardiographic (EKG) changes in the ST segment may reflect a myocardium where flow and demand are up to the limit, the manifestation of acute coronary syndrome (infarction, angina), or plaque rupture in a patient with atherosclerosis. However, in a large proportion of cases, these changes are transient and do not result in irreversible damage of the cardiac muscle or a low output state. Coronary heart disease must be the first thing to rule out, which means that any change in the ST segment must prompt a clear diagnosis and appropriate management in the event coronary heart disease is confirmed.

It is important not to ignore other conditions or diseases that may surface during anesthesia. In general, changes are found to occur during intubation, and during the course of surgery because of catecholamine elevations, hypotension or bleeding.

A common characteristic is coronary, vasospasm produced by the catecholamine release of as a result of a stimulus. In most cases, catecholamine release is associated with increased catecholamine sensitivity in the coronary endothelium, as is usually the case in patients with polyneuropathies, where the loss of nerve endings increases the response to catecholamines. Since the patient report by Dote et al.,[1] a group of patients with intense coronary spasms have been recognized. This pathology, called Takotsubo cardiomyopathy[2] or transient ventricular ballooning, may present more frequently in older women under high stress. These patients have similar manifestations as those found in a coronary event: ST elevation in the EKG, contractility changes and elevation of enzymatic biomarkers (BNP being higher than troponins).[3]

In general, no associated coronary disease is found, unless the disease coexists. However, the issue is not the presence of coronary heart disease. Takotsubo cardiomyopathy is characterized by the presence of elevations in the apical leads and ballooning in the EKG due to apical, anteroapical and infraapical hypo- or dyskinesis, with sparing of the basal segments.[4,5]

Apart from these conditions, there are causes of genetic origin such as Brugada syndrome caused by a genetic mutation in the sodium channels. These patients present with

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arrhythmias, such as tachycardia and fibrillation, which may lead to sudden death. ST elevation is usually observed in leads aVR, V1, a V3, with widening of the S wave greater or equal to 0.08 s, and T wave depression or elevation. A syndrome similar to Brugada's has been observed with excess administration of propofol. Additionally, peridural anesthesia, the use of agents like oxytocin, anti-arrhythmic medications in general, nitroglycerine, psychotropic agents, or the use of cocaine, have also been documented.[6–8]

Other conditions associated with stress and increased catecholamine levels include variant angina, or Prinzmetal's angina.[9] Anaphylactic conditions and stress states may give rise to vasospasm, as is also the case with its treatment using epinephrine, as well as the treatment for anaphylaxis.

Various mechanisms have been implicated in the genesis of coronary vasospasm, which alter vasodilatation due to disturbed nitric oxide release. Vascular contraction is determined by the increase in endogenous serotonin ligands, after the inhibition of nitric oxide synthase. The interaction between the autonomic nervous system, inflammation, nitric oxide clearance, endothelial nitric oxide synthase regulation, and the activity of RhoA/ROCK in the cells of the vascular smooth muscles, and K_{ATP} channels, are some of the factors involved in coronary vasospasm.[10]

In general, in all patients with a risk of a coronary event, it is critical to examine carefully the predisposing factors and the probability of a coronary syndrome occurring. We consider the steps described by Bakker et al.,[11] together with steps to be taken during subsequent stages after the intraoperative occurrence of a coronary syndrome: (1) determine active cardiac conditions before hand; (2) determine the risk associated with the surgery; (3) determine exercise capacity; (4) identify the existing clinical factors; (5) determine the need for pre-operative testing (stratification and/or biomarker tests); (6) provide prophylactic agents (statins, beta-blockers, calcium antagonists); (7) determine the need for implantable defibrillators or ventricular assist devices; (8) intraoperative monitoring; (9) anesthetic and pain control; (10) timely diagnosis (EKG, biomarkers, echocardiogram, catheterization);[11] (11) treatment targeted to the triggering etiology (use of nitrate, oxygen, aspirin, clopidogrel, anticoagulants, angiotensin converting enzyme inhibitors and/or coronary angiography and revascularization),[12] or, in cases of normal or near-normal coronary arteries, use of calcium antagonists and/or nitrates. In refractory cases, antiarrhythmic drugs or implantable defibrillators can be used when there is a risk of sudden death. Strategies have been developed in order to avoid new episodes of coronary vasospasm: Quit cigarette smoking, use of calcium antagonists, nitrates, intravenous magnesium, RhoA/ROCK inhibitors (Fasudil), statins, and implantable cardiodefibrillators in some cases. Additional strategies include avoiding stress-inducing agents or situations, including alcohol, cigarette smoking, catecholamines, muscarinic agonists, ergot alkaloids, prostaglandins, emotional stress, and propranolol.[10]

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