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## Taquicardia ventricular lenta pdf

INTRODUCTION ventricular tachycardias (TV) during the chronic phase of myocardial infarction (MI) are due in most cases, to a intramiocathetic return mechanism relating to scar1,2 infarction. Although in this context, the ischemia can act as a trigger in some cases, it does not usually plays a primordial role in its appearance, so that a rivascoruzione or an adequate treatment with antianginal drugs does not prevent the risk of recurrence. The treatment antiarrhythmic in these cases is addressed to the modification of the electrophysiological properties of the reentry circuit by means of a drug (by acting on sodium channels or potassium) or to its interruption by radiofrequency ablation or chirurgial .3. The verapamil intravenous administration is considered contraindicated in the presence of a television set, given the hemodynamic deterioration that can produce4.Preach cases of 4 patients with prior and TV who presented a response to treatment with the administration of verapamil and / or revascularisation Miocardic. CIÁ Nicos Case Ivaron 66 years came in from the side evolved IM, not treated with fibrinolitici for time criteria. After 72 h without complications, the patient was transferred to the plant, are treated with nitrates. In the coming year of income presented TV PolymorphFA syncopal that required urgent electrical carditori. In the following hours, numerous episodes of sustained tachycardia, and non-sustained (Figure 1), with an incessant behavior and occasional degenerates into ventricular fibrillation (VF), specifying multiple external shocks. These episodes are not preceded by significant bradycardia, the interval abnormally prolonged QT interval, changes in ST segment or toacco pain. The values of cardiac enzymes in the plasma have not undergone significant alterations. Treatment with intravenous lidocaine and amiodarone did not get stabilization of rhythm, which was only obtained after administration of verapamil, first intravenously and then by Oral.fig. 1. Monitor records that reveal episodes of polymorphic TV with incessant behavior in case 1. The coronarygrafy showed no significant lesions in the coronary arteries previous descendants, as well as a subtotal lesion in the proximal circumfation where angioplasty has been performed with a good result. Un'ergometria with thallium made subsequently discarded ischemia myocratica, only objectifying the presence of posterolateral necrosis. A study has revealed HOLTER only extrastolia in more scarce. The patient was discharged under oral treatment with follow-up Verapamil.tra 5, the new clinical episodes of arrhythmia have not been repeated. Recently, the patient needs a new angioplasty coronary artery anterior descending angina and progression of his disease. 2Varon 69 years with multiple cardiovascular risk factors and heart disease ischemic long evolution. Eighteen years before the present income he presented unstable angina, and surgical revascularization has been processed with a bridge of mammary artery to the previous descending. Subsequently, the patient was 2 times as im anoseptal, taking a coronary bypass where the obstruction was shown from the front and right descending coronary artery and participates © serious injury to other locations that were considered not amenable to revascularization. Similarly, the patient had severe left ventricular dysfunction, specifying the hospital income in most occasions due to heart failure. He continues usual treatment with nitrates and had a poor functional class, dyspnea and angina with minimal effort. The patient went into Hospital for a fast-supported monomorphic TV with bad clinical and hemodynamic tolerance. After carrying out an external cardiovers, the electrocardiographic monitoring highlighted an incessant behavior array that has been organized on the TV tem (Figure 2a) with unformed episodes and continuously supported. The treatment was started with intravenous lidocaine at full doses, which was not effective. The intravenous administration of 5 mg of verapamil reached the interruption of the television, to which clinical stabilization is followed in a few minutes (fig. 2b). At no time, the patient reported angina and ECG in the acute phase showed no changes to the ST or wave T-segment, even if in the following days, electrical changes of anterolateral ischemia were observed. There was no enzyme movement. A new Coronarygraph confirmed the impossibility of revascularization. The patient was discharged in treatment with amiodarone and diltiazem.fig. 2. A: AutoLimitant Episode of fast monomorphic TV (170 Lat / min) and slightly irregular, at its beginning, of the patient 2. B: simultaneous and continuous recordings (derivations I and II were observed above) when in the end Of ventricular arrhythmias is appreciated at the end of the injection of 5 mg of verapamil IV in the lower part (derivations V3 and V4) the patient's stable basic situation is appreciated after cardioversion to its entry. 2 months of follow-up income is according to another center, in which a new TV episode has been described in the anterior ischemia context unanswered to the lidocaine, which was treated with amiodarone and procainamide.caso 3 a male of 46 Years, smoker and without other pathological background, placed by a lower, rear and side IM, treated with fibrinolytics with doubtful reperfusion criteria. The patient has evolved without complications and a coronary it was carried out, which revealed an obstruction in the middle segment of the right coronary artery, with the distal heterocoronary flow, and stored on the left ventricular global systolic function. It was not acted on that lesion and an ergometry was previously performed at the top which was clinically negative for angina and highly positive late. Later, in a routine review, slowly supported asyptom tica TV (fig. 3). The treatment was started with beta-blockers and a Holter studio was carried out, which highlighted an incessant behavior of arrhythmia, alternating TV and sinus rhythm. A new ergometry was clinically and electrically negative, presenting to the end of a television similar to the previously documented one. The study with TALLIO demonstrated lower and inferolateral ischemia with partial and total reperfusion, consequently, consequently, the angioplasty was carried out on prejudice in the right coronary. It is interesting to note that, at the beginning of the procedure, the ECG has been appreciated a television that disappeared during dilatation.fig. 3. 12-resulting electrocardiogram and the rhythm strip of slow monomorphic TV (110 lat / min) in the patient 3. Annex dissociation is clearly visible. 11 months of non-documented recurrence follow-up. An ergometry made 6 months after angioplasty has been negative for angina or ischemia, and only frequent ventricular extras appeared in the maximum effort.cases 4varon of 59 years with cardiovascular risk factors and im prosthainferior with primary FV, 8 years earlier of income. In subsequent years it detects various income and percutaneous interventions (angioplasty in the previous descendant and the second diagonal, double stents in the previous descendant) clinical angina. The last of them took place in another center of 6 months before the current one. In the was performed an ergometric test For angina, but electrically positive for ischemia, which appears in a sustained monomorphic TV postpack, reversed with lidocaine. The coronarygraph revealed the permeability of the two previous stent and significant stenosis in the descending branches and above average (fine caliber vessels) on which has not been done. The patient has been discharged on treatment with diltiazem and nitrates. Episodes of self-limiting emphasized dizziness and palpitations, documenting a sustained monomorphic tv sustained behavior slowly incessant (Figures 4A and B), which has not responded to the administration of lidocaine. He did not present clinical angina or electrocardiographic signs of ischemia. He was presented to our hospital for study, in which treatment with beta-blockers was started, although the episodes arrometric continued. A coronary artery has been performed, which revealed a restenosis stent in the anterior descending level has been satisfactorily resolved. Subsequently, the patient does not present recurrence. A subsequent effort had negative test for angina, ischemia or tachycardia and an electrophysiological study was conducted in which ventricular arrhythmias were not induced.fig. 4. A: electrocardiogram during TV monomorphic slow (110 lat / min) In the case 4. The presence of visible retrograde wave is appreciated in the final port of the QRS complexes as a demonstration of ventriculoatrial guide 1. 1. B: It is observed a patient's system register 4 in sinus rhythm. 10 months of follow-up the patient has not presented new recurrences. Nest "Nestos 4 cases of tv sustained in patients with IM scarboard have different clinical presentations, sharing of myocardial ischemia discovery AA Adida the scar of the myocardium and the incessant behavior. Two cases (3-04) presented a TV "slow "(about 120 lat / min), with a tendency to incessant behavior, which resembles idioventricular accelerated rhythms that are often accompanied by reperfusion RDICA myocal, in both cases, revascularization by percutaneous angioplasty myocatic terminated the arrhythmia. In the other two cases (1 and 2) the clinical course was stormy, with tachycardia tolerated very fast and soon, even from different cardioversions if 1. 2 patients had an immediate response to Verapamil intravenous therapy, when the intravenous amiodarone administration had failed in case 1 and lidocaine in 2 cases some features of clinical presentation allow a mechanism other than intramiocardic of falling TV-related myocardial scar : the tendency to incessant behavior, in the absence of antiarrhythmic drugs (type I or type III), the lack of efficacy of conventional treatment and, ultimately, the response to intravenous verapamil.The patient 2 presented a monomorphic fast television, infrequent variant in the context of acute ischemia. Peters et al described a case monomorphic VT antiarrhmic resistant to drugs that, however, responded to anti-sequicity therapy (beta blockers, calcium channel blockers and nitrates) 5. Three of our patients (cases 1, 2 and 4) were receiving nitrates before the appearance of arrhythmia, despite having developed tachycardia. In another treatment (case 3) was started with beta-blockers in which the TV has been detected, but this has not prevented the arrhythmia persistence. Verapamil could suppress arrhythmia with a esquedic trigger myocatic improving perfusion and reducing consumption6 oxygen. However, resistance to certain drug treatments and anti-seated anti-Artmic and yet the therapeal response to verapamil may suggest that calcium channels are involved in Of these tachycardia and soccer antagonists can have a purely anti-SEQUILÉ independent therapy effect. The calcium channels can intervene in the Genesis arrhythmias through different mechanisms. In the ischemic areas or heart attack, the rest potential of the cells can be altered, with partial or total inactivation of sodium channels. In this situation, calcium channels can be activated that would lead to abnormal automatic arrhythmias. The use of calcium antagonists would be hypothely useful for treatment in these cases, at least in experimental models7.8. Patients 3 and 4 presented, as already mentioned, a slow TV similar to arrhythmias that are observed in the context of myocatic reperfusion. These arrhythmias are probably an increase in automation, even if they were also linked to postpotentials9.10. Some studies have demonstrated protection for the development of their soccer -10.11 antagonists. In the triggered activity, it can also play a role, both calcium channels and itI2 intracellular overload, having been described an occasional favorable effect by FÁIrmacos antagonists of Calcium13. 14. Postpotentials could also be important in the polymorphs TV Gevesis in the Patients with Qt Long14.15. Polymorphs televisions are generally associated with the presence of long QT, congenital or acquired interval, or a marked dispersion of repolarization15. This unequal repolarization could occur in some clinical situations, such as ischemia, even with a normal QT interval in ECG16. Some authors have hypothesized the possible role of M cells in the GÁf © Nesis of different arrhythmias. They constitute a subpopulation of cells located in the average ventricular myocardial rule, with electrophysiological characteristics that make them prone to the extension of the action potential and the development of Postpenzials before multiple stimuli, including AGONISTS15,17 calcium channels. They could be involved mainly on the Classically arrhythmias attributed to the unleashed activity, such as reperfusion or arrhythmias ischemia. Furthermore, it could also be the substrate for a resentment mechanism by creating dispersion of refractority and blocks of conduction15. The patient 1 of our series has developed a recurrent polymorphic TV with a normal QT interval. Wolfe et described the execrable of polymorpha postinfacto TV related to ischemia, in the absence of electrolytic alterations, of the QT, pauses or bradycardia19 intervals. As in our case, the use of the type the antiarrhythmic drug was not effective, although unlike our patient, the amiodarone was useful in some of them. In this series, the revascularization also seems to prevent recurrence. The absence of recurrence after the revascularization in patients 1, 3 and 4 confirms an important pathogenous card of ischemia, when acting on an adequate anatomical substrate. It is important to emphasize that none of them presented clinical or electrocardiographic data from ischemia before the frightening of symptoms, even if a patient subsequently presented them. It should also be emphasized that the only patient who could not carry out an adequate myocating revascularization presented a recurrence. For this reason, in these cases, the main objective should be to restore adequate coronary perfusion, since even if calcium-antagonists can be useful for acute stabilization, its long-term benefits are very much doubt. The patient 2 presented recurrence despite the treatment with Diltiazem and the patient 4 took it when the arrhythmia developed, myocratic ischemia can play an important role in Ventricular monoomorfin or incessant polymorphic behavior in patients with heart attack, even in the absence of angina or histle changes in ECG. The traditional antiarrhythmic treatment can be ineffective, sometimes stabilizing in the acute phase with intravenous administration of Vepamilo. Therefore, this therapy option should be considered in the event of failure of the usual drugs. The coronary rivascularization should be the main objective and can constitute a definitive therapy. The alterations of the automatism and secondary trigger activity at myocardial incision could be involved in the mechanism of these arrhythmias. Corrubesporence: Dr. F. Arribas. Cardiology service. Arrhythmic unity. University Hospital 12 October. Avda de CA'rdoba, S / N. 28041 Madrid. Electronic mail: Farbas@secardiologia.es Farbas@secardiologia.es

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