



Emergency electrical cardioversion: abnormal discharge synchronization leading to ventricular fibrillation – a case report

Ratunkowa kardiowersja elektryczna: niewłaściwa synchronizacja wyładowania prowadząca do migotania komór – opis przypadku

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ABSTRACT

Ventricular tachycardia is a life-threatening arrhythmia that usually occurs secondary to coronary ischemia or as a result of structural heart disease. Abnormal synchronization can lead to the conversion of ventricular tachycardia to ventricular fibrillation and subsequent cardiac arrest, which is very rare. The study presents the case of a 56-year-old man in whom abnormal discharge synchronization led to ventricular fibrillation.

KEYWORDS

cardiac arrest, electrical cardioversion, ventricular fibrillation

STRESZCZENIE

Częstoskurcz komorowy jest zagrażającą życiu arytmia występującą najczęściej wtórnie do niedokrwienia wieńcowego lub w wyniku strukturalnej choroby serca. Nieprawidłowa synchronizacja może prowadzić do przekształcenia częstoskurczu komorowego w migotanie komór, a w konsekwencji do zatrzymania krążenia, co zdarza się bardzo rzadko. W pracy przedstawiono opis przypadku 56-letniego mężczyzny, u którego nieprawidłowa synchronizacja wyładowań doprowadziła do migotania komór.

SŁOWA KLUCZOWE

zatrzymanie krążenia, kardiowersja elektryczna, migotanie komór

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INTRODUCTION

Ventricular tachycardia (VT) is a life-threatening arrhythmia occurring most often secondary to coronary ischemia or as a result of structural heart disease. When hemodynamic instability appears, immediate electrical cardioversion (EC) is recommended, which involves the delivery of a direct current discharge synchronized with the QRS complex in the electrocardiographic (ECG) recording [1]. Improper synchronization can result in the conversion of VT to ventricular fibrillation (VF) and subsequent cardiac arrest (CA), which happens very rarely [2].

CASE REPORT

A 56-year-old obese patient (body mass index 38 kg/m²) was admitted to the Emergency Room (ER) for malaise, intermittent chest pain and a constant feeling of rapid heartbeat. More than 8 months earlier, he had experienced non-ST-segment elevation acute coronary syndrome (NSTE-ACS) with sustained peri-infarct VT. At that time, coronary angioplasty was performed with the implantation of a drug-eluting stent into the left anterior interventricular branch of the left coronary artery. Sinus rhythm was achieved. Heart failure with preserved ejection fraction (HFpEF) was diagnosed. Since the diagnosis of ACS, the patient had been taking dual antiplatelet therapy (aspirin, clopidogrel), a beta-blocker, an angiotensin-converting

enzyme inhibitor, spironolactone and a proton pump inhibitor. On the morning of the day in the ER, the patient had taken all his medication.

An ECG performed on admission showed tachycardia with wide QRS complexes at 190 bpm. The patient was conscious with full logical verbal contact. On physical examination, tachypnea (26/min) was observed, and on auscultation there were crackles in the lower lungs bilaterally. A threadlike pulse on the radial artery, moist skin and a prolonged capillary refill time of up to 4 seconds were noted. Blood pressure was 102/78 mmHg. Hemodynamically unstable monomorphic VT was diagnosed and immediate EC was decided upon. Under sedation with 14 mg etomidate (Etomidate-Lipuro, Melsungen AG, Germany), EC was performed with 300 J energy using a Lifepak 15 (Physio-Control, Inc., USA) device with manual paddles. Incorrect synchronization of the discharge resulted in the conversion of VT with pulse to VF in an R-on-T phenomenon (Figure 1).

Importantly, the myoclonus observed after EC, usually encountered after etomidate, provided momentary doubt on ECG evaluation. However, respiratory arrest and the lack of pulse on the central arteries confirmed the diagnosis of CA by VF mechanism. Defibrillation with an energy of 300 J was performed, achieving the cessation of VF, followed by temporary conversion of the rhythm to a complete third-degree atrioventricular block (Figure 2) and recurrence of VT with pulse. 150 mg of amiodarone and 100 mg of lidocaine were administered, achieving a sustained return of sinus rhythm with features of hemodynamic stability.

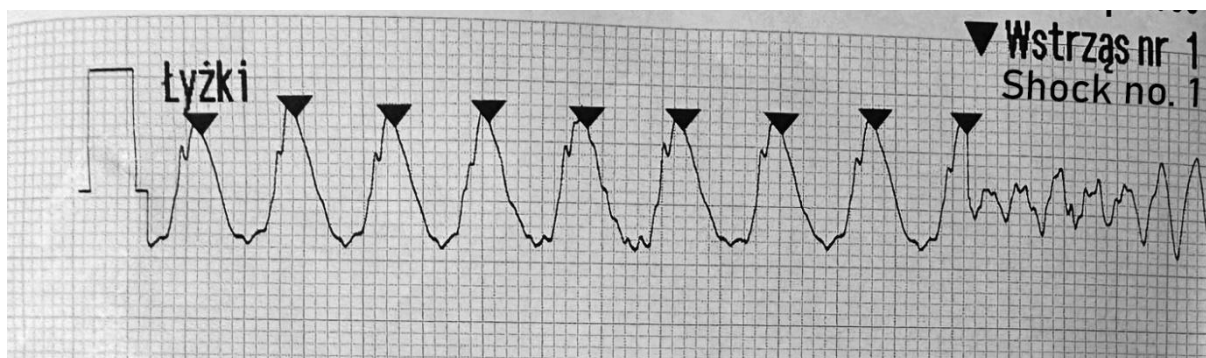


Fig. 1. Electrocardiogram. Record of course of electrical cardioversion.

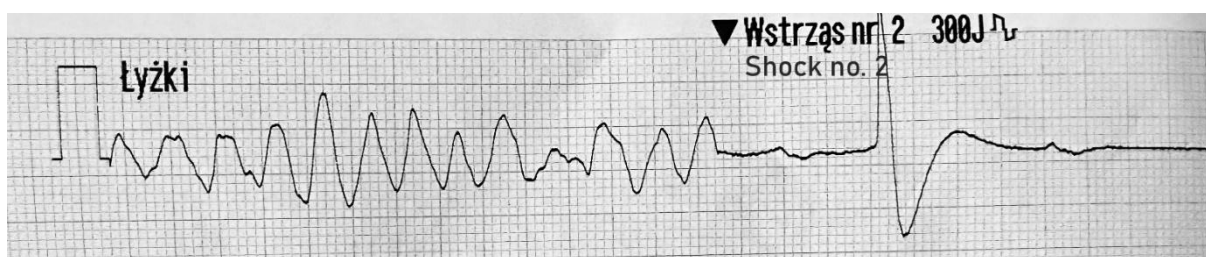


Fig. 2. Electrocardiogram. Defibrillation record. Conversion of ventricular fibrillation to complete third-degree atrioventricular block.



The return of spontaneous breathing was observed. Passive oxygen therapy through a face mask was continued. After a few minutes, the patient regained consciousness and was graded 10 points on the modified Aldrete score. The results of the basic laboratory tests performed, including markers of myocardial damage and electrolyte levels, revealed no significant abnormalities. A resting 12-ECG showed no features of fresh myocardial ischemia. Urgent angiography of the coronary arteries showed no significant changes. The patient in stable condition was transferred for further treatment in the cardiology department, where he underwent the implantation of a cardioverter-defibrillator on the following day. On day 3, the patient left the hospital in good condition (Cerebral Performance Category 1).

DISCUSSION

This case shows that incorrect discharge synchronisation during EC can lead to CA with the need to implement advanced life support (ALS). Electrical cardioversion is a life-saving procedure for tachyarrhythmias presenting with hemodynamic instability. Nonetheless, like any procedure, it is fraught with the risk of complications. They result from the need for anesthetics under extremely adverse hemodynamic conditions, and less frequently, from the direct effect of an electrical impulse delivered at the wrong moment in the heart's electrical cycle. In the phase associated with T-wave formation, there are areas of the myocardium where repolarization and the return of excitability have already occurred. The delivery of an impulse in the predisposed zone of the T-wave can lead to the initiation of the R-on-T phenomenon, followed by VF [3]. The therapeutic procedure is to perform immediate defibrillation.

Danon et al. [2] identified 7 case reports of VF after EC. In all the cases, the cause of VF was improper synchronization, and one case ended in death. The European Resuscitation Council (ERC) guidelines recommend using energy of 120–150 J for the first discharge [4]. Nevertheless, there are reports suggesting that using the highest energy for the first cardioversion increases the chance of success of the first cardioversion attempt by avoiding multiple discharges of increasing energy with repeated exposure of the heart to further damage. In the described case, due to the significant haemodynamic instability, it was decided to use a higher initial energy strategy than recommended by the ERC. Gallagher et al. [5] hypothesized that the use of low discharge energy may increase the risk of VF. This observation was not confirmed in our case as a 300 J pulse was delivered.

Our case is important in the context of everyday clinical practice. In each case of EC, it is essential to confirm correct synchronisation before performing a discharge. In some cases, concomitant hyperkalaemia may result in the presence of high T-waves that will be incorrectly identified by the device as QRS complexes, although in our case the potassium level was normal. If there is any doubt about the correct synchrony, consideration may be given to changing the lead, amplifying the signal or changing the position of the electrodes. However, this does not eliminate the risk that, despite correct synchronisation, a discharge is delivered at the incorrect time. Finally, when proper synchronisation cannot be achieved, non-synchronised defibrillation is possible in cases of haemodynamic instability. Nonetheless, one should be aware of the consequences of this strategy.

In conclusion, one must keep in mind that improper synchronization of the electrical impulse during EC can lead to VF. For this reason, the procedure should be carried out by personnel trained in ALS.

Author's contribution

Study design – M.P. Pluta

Manuscript preparation – M.P. Pluta, Ł.J. Krzych

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