## CASE PRESENTATION

# Acute symptomatic seizures and ventricular fibrillation - a multimodality approach to a rare complication of myocardial infarction. Case report

Miruna Florina Stefan[1], Stefania Lucia Magda[1,2]

**Abstract: Background –** 6-8% of patients with acute myocardial infarction still develop ventricular arrhythmias (VA), although their incidence has lowered due to prompt antiischemic treatment. VA might determine sometimes acute symptomatic seizures. **Case report –** A 44-year old male was admitted to our unit with acute inferior myocardial infarction. Emergency coronary angiography was performed and revealed acute occlusion of the right coronary artery. The procedure was complicated by coronary dissection, which was sealed, with good final results. After admission in the acute coronary unit, the patient developed ventricular fibrillation. He was successfully resuscitated, but developed ongoing tonic-clonic seizures, terminated after intravenous administration of several anticonvulsivant drugs. We investigated the patient for epilepsy and for other pathologies that could explain both the VA and the seizures. All investigations were within normal range. **Conclusion –** Differential diagnosis between hypoxic and epileptic seizures is difficult and important, because it further influences patient management. Cardiac arrhythmias are a rare precipitating factor for acute symptomatic seizures, due to hypotension-induced cerebral hypoxemia. Our case illustrates the value of a multimodal approach of rare complication of a myocardial infarction.
**Keywords:** myocardial infarction, ventricular fibrillation, acute seizure.

**Rezumat: Introducere –** Deși incidența aritmiilor ventriculare (VA) în faza acută a infarctului miocardic a scăzut datorită tratamentului prompt antiischemic, 6-8% dintre pacienți încă dezvoltă aceste complicații. VA pot fi urmate uneori de crize convulsive simptomatice acute, ce trebuie diferențiate de convulsiile epileptice. **Prezentare de caz –** Un bărbat de 44 de ani, a fost internat în clinica noastră pentru infarct miocardic acut inferior. S-a efectuat coronarografie de urgență, care a evidențiat ocluzia acută a arterei coronare drepte (ACD). Procedura a fost complicată de disecție la nivelul ACD, rezolvată intervențional, cu rezultat final bun. La admiterea în unitatea de Terapie Intensivă Coronariană, pacientul dezvoltă fibrilație ventriculară, resuscitată cu succes. Post-resuscitare pacientul dezvoltă crize tonico-clonice incesante, oprite prin administrarea intravenoasă de multiple anticonvulsivante. Pacientul a fost investigat pentru epilepsie și pentru alte patologii care ar putea explica atât aritmia cât și convulsiile, fără a se găsi o altă etiologie în afară de ischemia acută. **Concluzie –** Diagnosticul diferențial între crizele convulsive simptomatice acute și convulsiile epileptice este important, deoarece influențează managementul adecvat al cazului. Aritmiile cardiace pot precipita uneori convulsii simptomatice acute, prin hipoxemie cerebrală indusă de hipotensiune. Cazul nostru ilustrează valoarea abordării multimodale a unei complicatii rare a infarctului miocardic.
**Cuvinte cheie:** infarct miocardic, fibrilație ventriculară, convulsii acute.

## INTRODUCTION

Cardiovascular diseases remain the leading cause of mortality worldwide, and acute myocardial infarction (MI) is one of the most common and life-threatening cardiovascular diseases. Arrhythmias are common during the early hours of MI and are also important prognostic factors. The incidence of ventricular arrhythmias (VA) has declined over recent decades, due to reperfusion strategies and the early use of beta-blockers. However, 6–8% of patients still develop he-

[1] Department of Cardiology, Emergency University Hospital, Bucharest, Romania
[2] „Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

**Contact address:**
Stefania Lucia Magda, Department of Cardiology, Emergency University Hospital, 169 Splaiul Independentei, Bucharest, Romania.
E-mail: stefaniamagda@yahoo.com

modynamically significant ventricular tachycardia or ventricular fibrillation. Ventricular arrhythmias may sometimes be followed by a symptomatic seizure, but clear differentiation between a convulsive syncope and an epileptic seizure is essential for appropriate management. Distinguishing between the two entities may sometimes be difficult, as they share a number of common characteristics.

## CASE PRESENTATION

We report the case of a 44-year-old male patient who presented to our hospital's emergency room for severe chest pain and diaphoresis that started one hour prior to the presentation, after moderate physical activity. The patient had a history of untreated hyperlipidemia and was a smoker (40 pack-year). He also had significant family medical history, both his father, as well as his grandfather dying from myocardial infarction around the age of 40 and his brother having an ischemic stroke when he was 36-year old.

Upon admission, the patient was hemodynamically stable and had ongoing chest pain, with normal physical examination was normal.

The electrocardiogram (ECG) showed sinus rhythm with ST-segment elevation in the inferior leads and non-sustained ventricular tachycardia (Figure 1). There was no-ST elevation in the posterior or the right leads.

The initial high sensitivity troponin I was extremely elevated at 22827 ng/l and echocardiography at admission showed preserved left ventricle ejection fraction (LVEF) and hypokinesia of the basal segments of the interventricular septum and the inferior wall. Pulmonary radiography showed no pathological findings.

The diagnostic of acute inferior ST-elevation myocardial infarction (STEMI) was established. The patient received loading doses of aspirin and ticagrelor and underwent emergency coronary angiography, which revealed acute near-occlusion of the middle segment of the right coronary artery (RCA). The left anterior descending artery (LAD) and the circumflex artery (LCX) did not present any significant lesions (Figure 2, panel A). Right coronary artery angioplasty was performed, with the insertion of a 3.0x28 mm drug eluting stent (DES) in the second segment of the RCA. The passing of the 0.014" angioplasty guidewire into

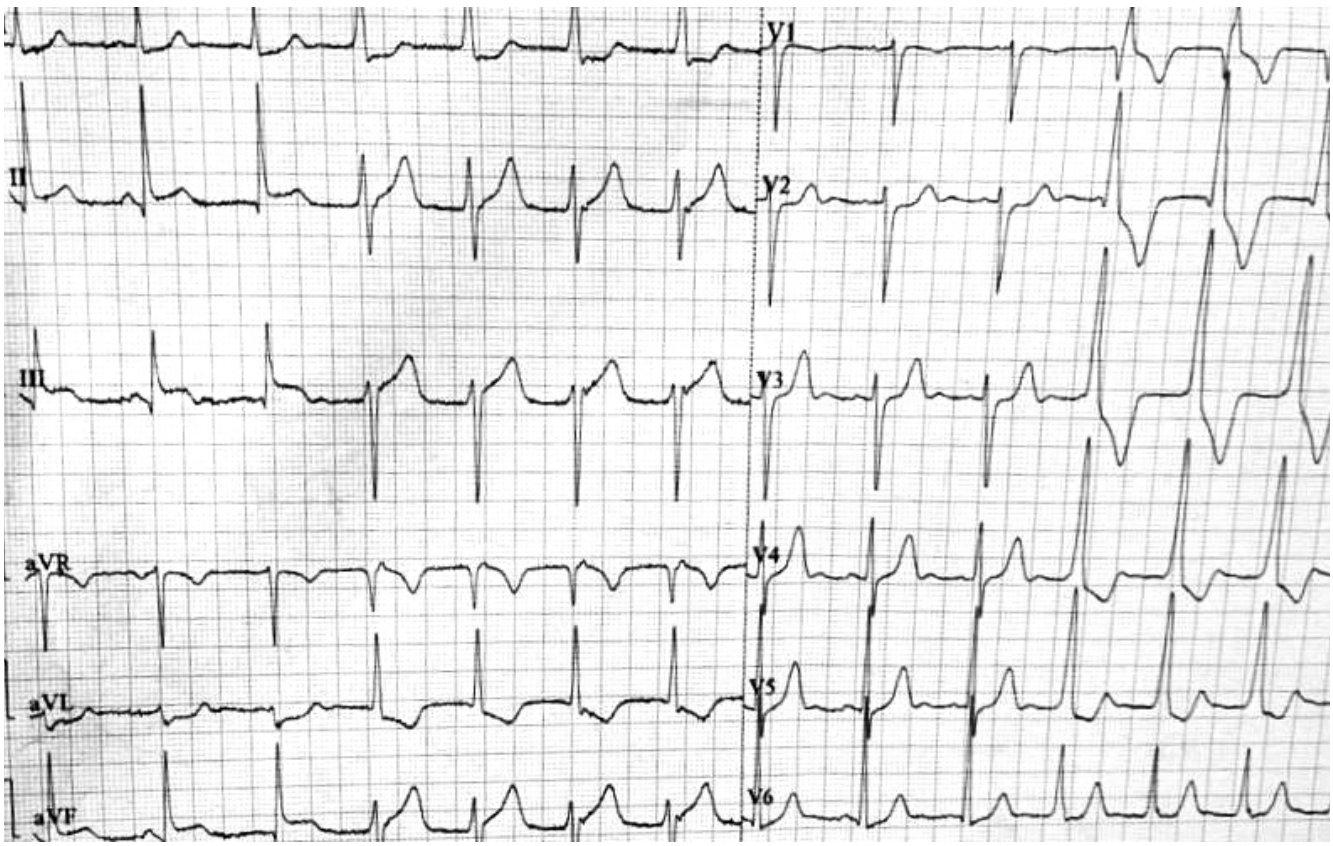


**Figure 1.** 12-leads electrocardiogram (ECG) at hospital admission showing sinus rhythm with ST-segment elevation in the inferior leads and non-sustained ventricular tachycardia.

the distal RCA was complicated by dissection at the site of the culprit lesion. Another 0.014" guidewire was placed in the false lumen and was maintained there in order to seal the dissection (Figure 2, panels B and C). Two iv boluses of eptifibatide glycoprotein IIb/IIIa inhibitor were administered during the procedure. The final result was good, without any residual stenoses and TIMI3 distal flux in all the coronary arteries (Figure 2, panel D).

Upon admission in our coronary intensive care unit the patient was angina-free, but he developed atrial fibrillation, with a heart rate of 148 beats per minute and good hemodynamic tolerance. He received intravenous amiodarone for chemical conversion.

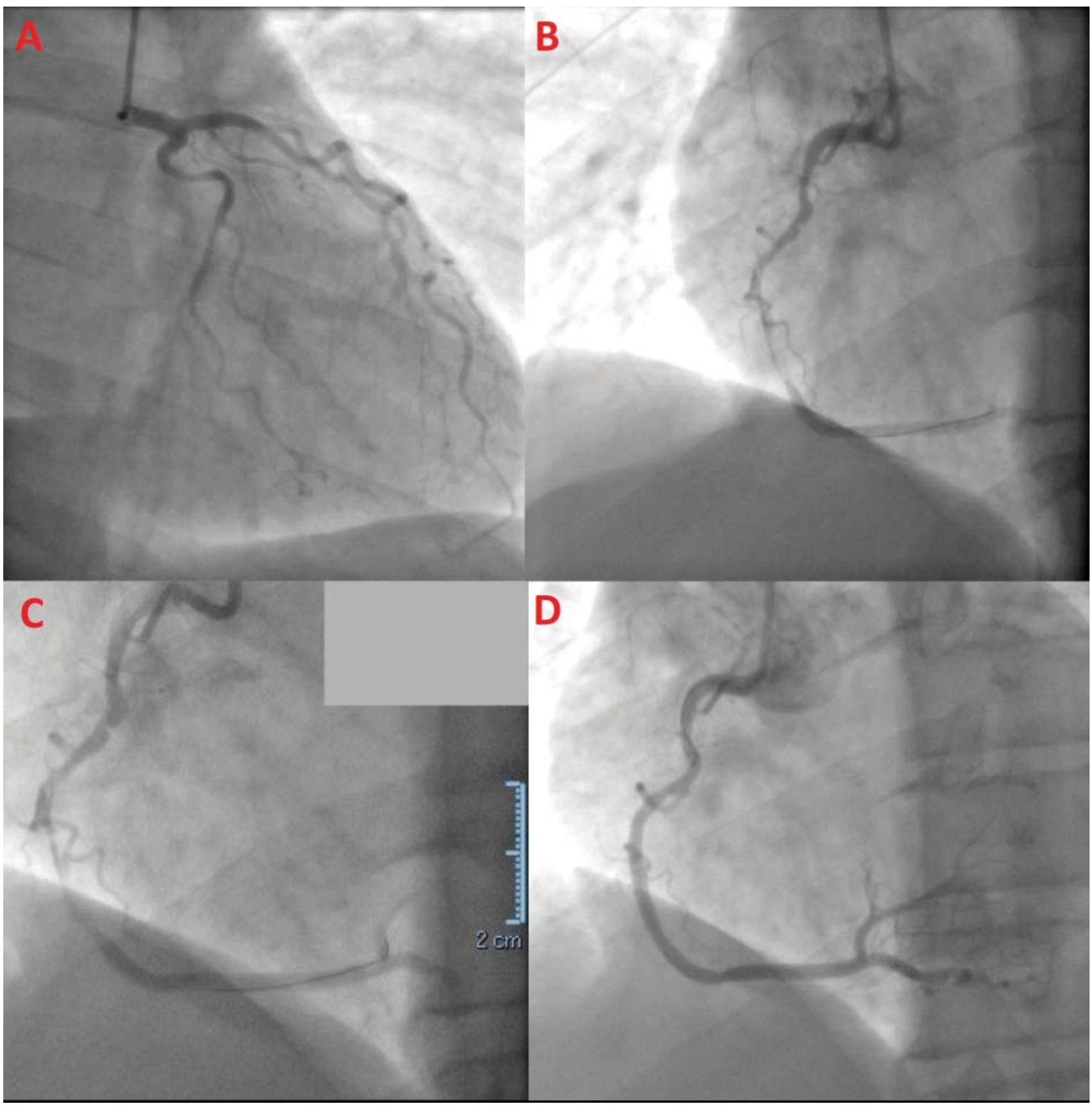


**Figure 2.** Coronary angiography. Panel A- The left anterior descending artery (LAD) and the circumflex artery (LCX) with no significant lesions; Panel B and C- showing acute near-occlusion of the middle segment of the right coronary artery (RCA) and intraprocedural dissection at the site of the culprit lesion; Panel D- Drug eluting stent in the second segment of the RCA, without any residual stenoses and TIMI3 distal flux.

After a short time, the patient developed recurrent ventricular fibrillation and he needed resuscitation by five 200 J electric shocks in approximately 5 minutes. We concomitantly administered intravenous adrenaline, as well as lidocaine. After resuscitation the blood pressure was 120/90 mm Hg, the heart rate 150 beats per minute (sinus tachycardia on the monitor), and the oxygen saturation 95% in ambient air. Persistent tonic-clonic seizures appeared, with alteration of the consciousness. The patient was sedated with diazepam and propofol and intubated.

The cardiac echography performed after the resuscitation showed moderate left ventricle systolic disfunction (LVEF 40%), diffuse kinetics disorder, no

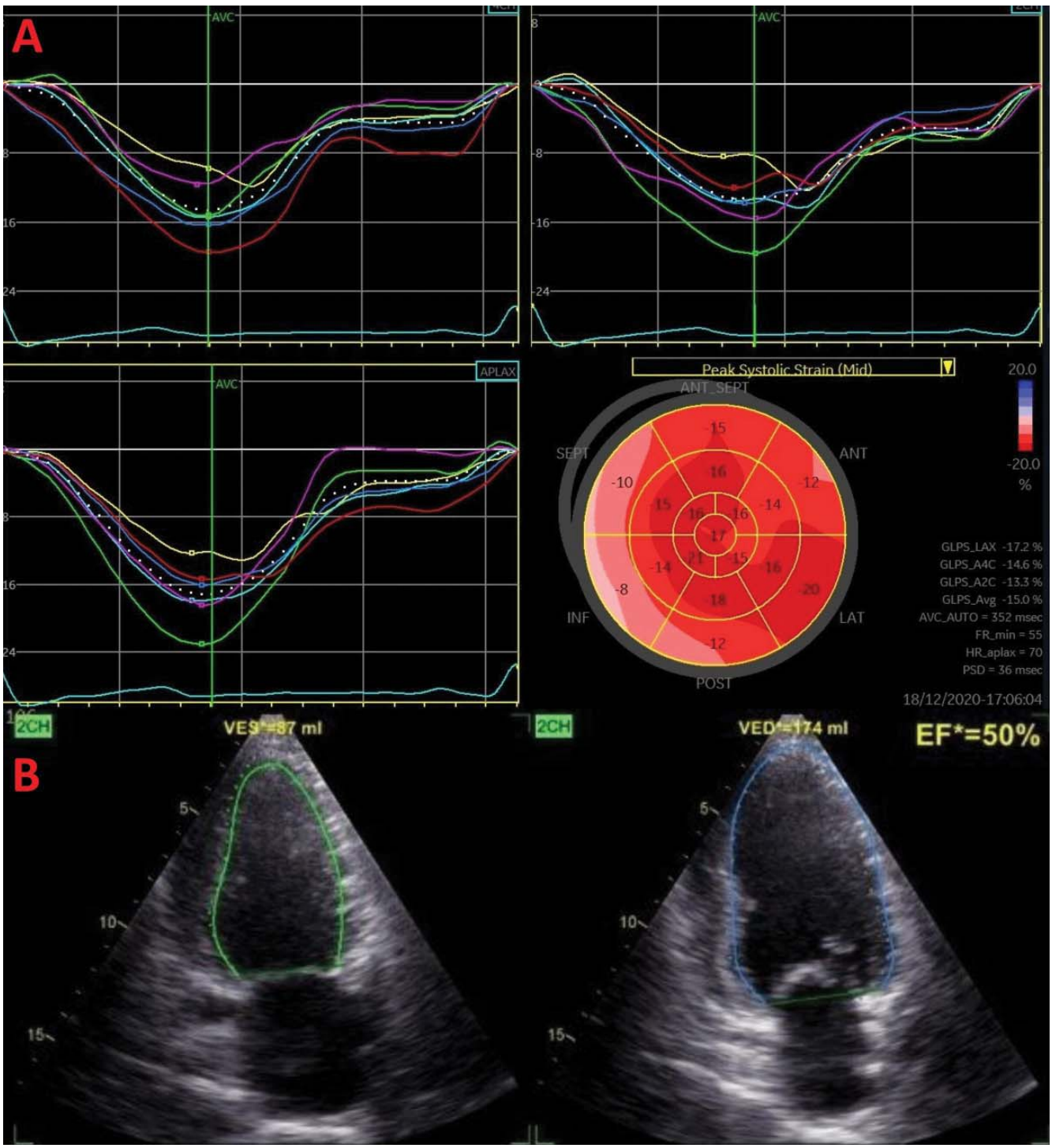


**Figure 3.** Transthoracic echocardiography. Panel A- strain pattern showing reduced strain in the inferior and posterior walls, Panel B – apical 2 Chamber view showing the calculation of left ventricle ejection fraction.

mechanical complications and no pericardial effusion. The patient had a good evolution and was retransferred to our clinic the next day.

The follow-up echocardiography showed mild systolic dysfunction (LVEF 50%), inferior and posterior wall hypokinesia, type I diastolic dysfunction, no significant valvular disease, no pericardial effusion. The strain pattern was compatible with inferior myocardial infarction, but didn't show features suggestive of other myocardial pathologies (Figure 3).

Biologically, there were to pathologic findings. The criteria for familial hypercholesterolemia were not met.

Taking into account the tonic-clonic seizure, a neurological examination was performed, without pathological findings. Moreover, both a cerebral CT scan, as

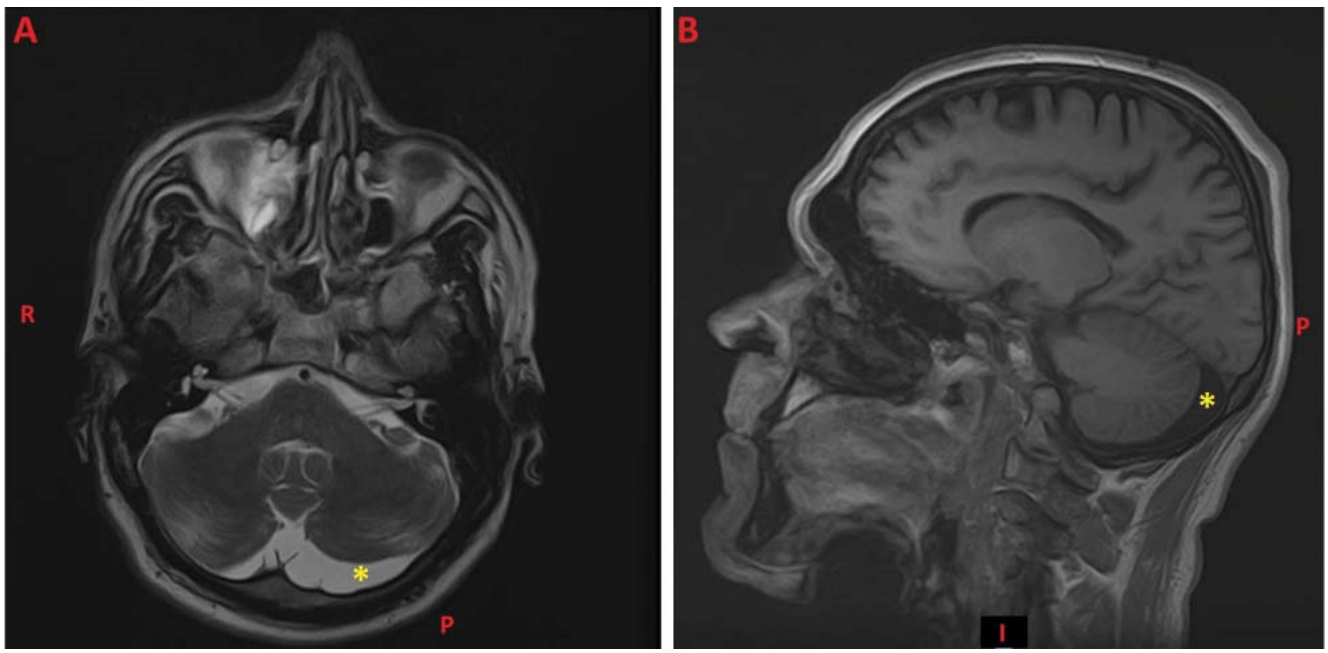


**Figure 4.** Cerebral magnetic resonance imaging (MRI); Panel A- transversal section, T2 sequence; Panel B – sagittal section, T1 sequence; *- posterior fossa arachnoid cyst of 47/11/46 mm; R- right, P-posterior, I-inferior.

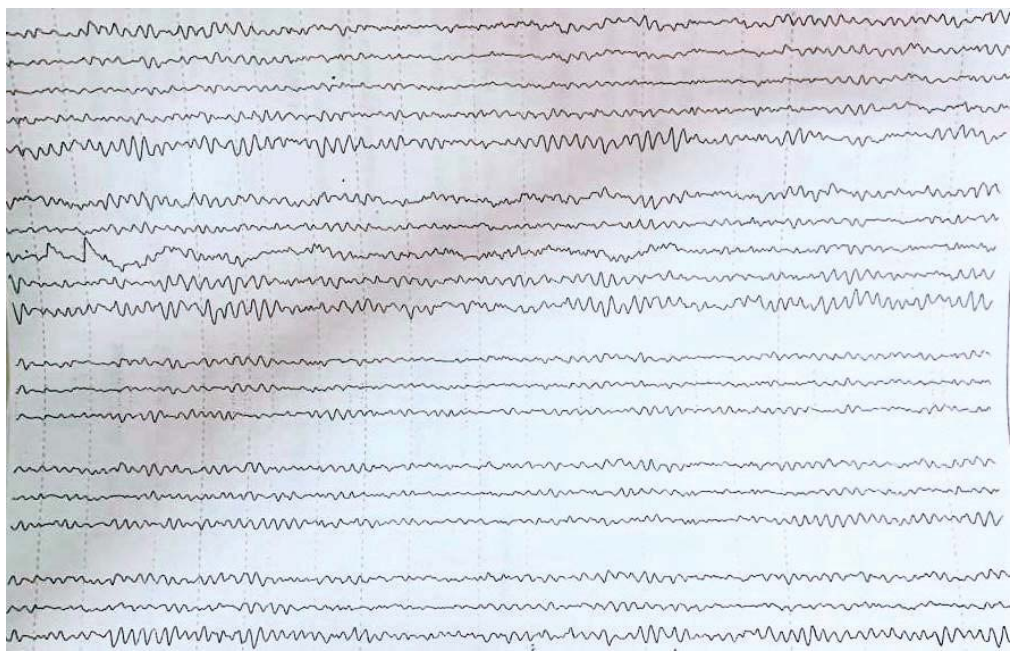

**Figure 5.** Electroencephalogram showing normal alpha pattern.

well as an MRI were made. No recent vascular changes were identified. A posterior fossa arachnoid cyst of 47/11/46 mm was identified, but without clinical implications (Figure 4). Doppler ultrasound of cervical vessels identified bilateral atheroma plaques in the internal carotid arteries without hemodynamic significance. The electroencephalogram (EEG) was normal (Figure 5).

Suspicion of Fabry disease was raised, but refuted by normal serum alpha galactosidase levels. Direct and indirect Coombs tests were also normal and so were thyroid hormones, blood homocysteine and blood cobalamin.

24-hour ECG Holter monitoring revealed background sinus rhythm, with rare monomorphic atrial and ventricular extrasystoles, without systematization.

The patient was discharged with double antiplatelet therapy (acetylsalicylic acid and ticagrelor), beta blocker, angiotensin converting enzyme inhibitor, statin at maximum dose and proton pump inhibitor. No neurological treatment was recommended so far.

Therefore, the tonic-clonic seizures were interpreted in the context of cerebral hypoxemia secondary to recurrent arrhythmic episodes. The patient will repeat the cerebral imagistic investigations and the EEG at one year after the event.

At 2-month follow-up the patient was neurological and cardiovascular asymptomatic.

## DISCUSSION

Our case describes a patient with an acute symptomatic seizure occurring in the setting of acute myocardial infarction complicated by intraprocedural right coronary artery dissection and followed by ventricular fibrillation. The seizure occurred promptly following a systemic stimulus, differentiating it from an epileptic seizure. Coronary artery dissection implies a tear in the wall of the artery, with the compression of the lumen, which leads to a reduction in blood flow and additional ischemia[1].

Acute seizures represent 1% of all visits to emergency departments in the United States. One-third of acute seizures are not due to epilepsy, but provoked by underlying potentially life-threatening acute conditions[2]. The two entities have many similar clinical features. Both involve transient loss of consciousness (LOC). Syncope is the LOC and loss of muscle tone due to reversible cerebral hypoperfusion. It is characterized by sudden onset, spontaneous and complete recovery. Epileptic seizure is the result of an abnormal, excessive and hypersynchronous neuronal activity in the brain[3,4].

Convulsive syncope (or symptomatic seizure) are terms previously used to describe generalized seizure-like activity attributed to transient cerebral hypoperfusion secondary to cardiac arrhythmia[5].

Appropriate differentiation between the two pathologies is essential, so that unnecessary medications or procedures may be avoided[6,7]. Moreover, anticonvulsant medications have been reported to have cardiotoxicity. Pregabalin has been related to heart failure[8], oxcarbazepine was reported to induce resistant VF[9], and carbamazepine can cause atrioventricular (AV) block[10,11], hypotension[12] or hypertension[13]. All those adverse effects, might, in fact, worsen the initial cause of the generalized seizure, if the right diagnosis has not been made.

Cardiac arrhythmias are a rare precipitant factor for acute symptomatic seizures, due to hypotension-induced cerebral hypoxemia. A case published by Cunnington et al. described a patient who developed seizure in the setting of cardiac arrest in Takotsubo cardiomyopathy with ventricular fibrillation[14]. Sabu et al. described a patient with ventricular tachycardia presenting with acute symptomatic seizure[15]. Yin et al. reported a similar case of acute seizure following polymorphic ventricular tachycardia[16].

Considering the important family history of the patient, we screened him for epilepsy, as well as for the most frequent systemic diseases that could predispose to both cardiac arrhythmias or epileptic seizures. Blood biomarkers, as well as the EEG and the transthoracic echocardiography (including strain imaging) showed no pathological findings compatible with the aforementioned afflictions.

## CONCLUSION

This case emphasizes the importance of the correct differentiation between acute symptomatic seizure (or convulsive syncope) and an epileptic seizure in a patient presenting with seizure-like activity. We illustrate the value of thorough history taking, and multimodal imagistic, electrocardiographic and electroencephalographic screening in identifying the underlying cause of the seizure-like activity, in order to correctly treat it and prevent its recurrence. Moreover, our case highlights the importance of modern imaging techniques in managing rare complications of myocardial infarction.

**Conflict of interest:** none declared.

## References:

1. Dash D. Complications of coronary intervention: abrupt closure, dissection, perforation. Heart Asia. 2013; 5: 61-5.
2. Van Dijk JG, Thijs RD, Benditt DG, Wieling W. A guide to disorders causing transient loss of consciousness: focus on syncope. Nat Rev Neurol. 2009 ;5: 438–48.
3. Pallin DJ, Goldstein JN, Moussally JS, et al. Seizure visits in US emergency departments: epidemiology and potential disparities in care. Int J Emerg Med 2008; 1: 97–105.
4. Fisher RS, van Emde Boas W, Blume W, Elger C, Genton P, Lee P, Engel J Jr. Epileptic seizures and epilepsy: definitions proposed by the International League Against Epilepsy (ILAE) and the International Bureau for Epilepsy. Epilepsia. 2005; 46: 470–72.
5. Akhtar MJ. All seizures are not epilepsy: many have a cardiovascular cause. J Pak Med Assoc. 2002; 52: 116–20.
6. Linzer M, Grubb BP, Ho S, Ramakrishnan L, Bromfield E, Estes NA 3rd. Cardiovascular causes of loss of consciousness in patients with presumed epilepsy: a cause of the increased sudden death rate in people with epilepsy? Am J Med.1994; 96:146–54.
7. Erdogan G, Ceyhan D, Gulec S. Possible heart failure associated with pregabalin use: case report. Agri. 2011; 23: 80–83.
8. El-Menyar A, Khan M, Al Suwaidi J, Eljerjawy E, Asaad N. Oxcarbazepine-induced resistant ventricular fibrillation in an apparently healthy young man. Am J Emerg Med. 2011; 29: 693. e691-693.
9. Ide A, Kamijo Y. Intermittent complete atrioventricular block after long term low-dose carbamazepine therapy with a serum concentration less than the therapeutic level. Intern Med. 2007; 46: 627–29.
10. Takayanagi K, Hisauchi I, Watanabe J, Maekawa Y, Fujito T, Sakai Y, Hoshi K. et al. Carbamazepine-induced sinus node dysfunction and atrioventricular block in elderly women. Jpn Heart J. 1998; 39: 469–79.
11. Tibballs J. Acute toxic reaction to carbamazepine: clinical effects and serum concentrations. J Pediatr. 1992; 121: 295–99.
12. Transient neurologic deficits associated with carbamazepine-induced hypertension. Clin Neuropharmacol. 2003; 26:174–76.
13. Sheldon R, Rose S, Ritchie D, Connolly SJ, Koshman ML, Lee MA, Frenneaux M. et al. Historical criteria that distinguish syncope from seizures. J Am Coll Cardiol. 2002; 40: 142–48.
14. Cunnington C, Garg S, Balachandran KP. Seizure-associated takotsubo cardiomyopathy presenting with unheralded ventricular fibrillation. Int J Cardiol 2012; 162: e21
15. Sabu J, Regeti K, Mallappallil M, Kassotis J, Islam H, Zafar S, Khan R, Ibrahim H, Kanta R, Sen S, Yousif A, Nai Q. Convulsive Syncope Induced by Ventricular Arrhythmia Masquerading as Epileptic Seizures: Case Report and Literature Review. J Clin Med Res. 2016; 8: 610-5.
16. Yin HC, MN Wu, Chen CH, Huang P. Ventricular tachycardia manifested as tonic seizure. Epilepsy Behav 2012; 24: 146–7.