


CASE REPORT

Contrast-induced early repolarization pattern and ventricular fibrillation

Ourania Kariki | Marianna Kontonika | Dimitrios Miliopoulos | George Bazoukis  |
Konstantinos Vlachos | Stylianos Dragasis | Aggeliki Gouziouta | Konstantinos P. Letsas |
Michael Efremidis | Vassilis Voudris

Department of Cardiology, Onassis
Cardiac Surgery Center, Athens, Greece

Correspondence

Konstantinos P. Letsas, Arrhythmia Unit,
Laboratory of Cardiac Electrophysiology,
Onassis Cardiac Surgery Center, 356
Syngrou Av., 17674 Athens, Greece.
Email: k.letsas@gmail.com

Abstract

Malignant arrhythmias during coronary angiography consist a complication of the procedure. Clinicians should be aware that intracoronary infusion of contrast medium can lead to physiological changes that lower the ventricular fibrillation threshold.

KEYWORDS

contrast medium, coronary angiography, J waves, ventricular fibrillation

1 | CASE PRESENTATION

A 70-year-old man was admitted to our hospital for a scheduled invasive coronary angiography due to mild exertional dyspnea followed by an equivocal treadmill stress test. His past medical history was notable for arterial hypertension, mild dyslipidemia, and smoking. He denied any family history of sudden deaths.

The patient was transferred to the cardiac catheterization laboratory asymptomatic. His resting ECG showed a slight J point elevation of 1 mm in inferior leads (II, III, aVF) (Figure 1A). The transradial coronary angiography was performed with Judkins left and Judkins right diagnostic catheters of 6F. The radiocontrast used was iopromide, a non-ionic iodine-based contrast agent of low osmolarity. During the procedure, the patient manifested ventricular fibrillation immediately after the intracoronary injections in both coronary arteries that required electrical cardioversion. Each arrhythmic episode was preceded by a further elevation of the J point in the inferior leads (II, III, aVF) during right coronary artery angiography (Figure 1B) and by J point elevation in lateral leads (I, aVL) during left main artery angiography (Figure 1C). Of note, a premature ventricular ectopic beat of

the same morphology preceded both ventricular fibrillation episodes. Angiography was then completed by additional non-selective infusions from the aortic root (Figure 1D,E,F). Coronary angiography did not reveal any sign of obstructive disease. Mechanical complications of the procedure such as stimulation of the myocardium by catheter manipulation, catheter occlusion, deep intubation into the coronaries or superselective engagement were immediately excluded by the operator. The timing as well as the mode of the initiation of the arrhythmia led us to the diagnosis of contrast media-induced ventricular fibrillation.

Even if the non-spontaneous manifestation of the arrhythmia prohibited the diagnosis of an early repolarization syndrome, the pattern in the resting ECG of the patient raised our suspicion of an underlying substrate that could increase his vulnerability to malignant arrhythmogenesis. The hospitalization of the patient was extended during which he underwent a new cardiac ultrasonography with no abnormal findings, a treadmill stress test with no signs of ischemia or arrhythmia and continuous monitoring with no arrhythmic episodes. A 'Viskin test', an isoproterenol test, and a flecainide test were conducted with negative results for long QT syndrome, premature ventricular contractions, or Brugada

This is an open access article under the terms of the Creative Commons Attribution-NonCommercial License, which permits use, distribution and reproduction in any medium, provided the original work is properly cited and is not used for commercial purposes.

© 2021 The Authors. *Clinical Case Reports* published by John Wiley & Sons Ltd.

pattern. The patient was discharged with the diagnosis of contrast-related myocardial toxicity.

2 | DISCUSSION

Malignant arrhythmogenesis is not an uncommon complication during invasive coronary angiography, even though its incidence has declined nearly by half during the last decades (1.1% sVT/VF in 1960 vs. 0.6% in 1990).¹ The majority of these events is related to catheter and wire manipulations (direct myocardial stimulation, deep coronary intubation, superselective engagement etc) and can be prevented by optimizing the equipment and the technique such as preferring smaller size catheters with sideholes, avoid prolonged injections of large amount of contrast medium, or injections into the conus branch of the right coronary artery.² Whenever the aforementioned technical issues have been excluded as the cause of the arrhythmic event, the etiology can be attributed to myocardial toxicity of the contrast agent or underlying vulnerability of the patient due to electrolyte imbalance or QT prolongation.³

Although ideally a contrast agent should be physiologically inactive, the iodine-based agents interact with the myocardial tissue when injected intracoronary leading to physiological changes, not always of clinical significance.

The pharmacological effects of the iodine-based agents depend on their osmolarity, their sodium concentration, and their calcium-binding properties. In greater detail, iodine-based contrast agents are categorized according to their iodine ratio forms. The iodine ratio is calculated by dividing the number of iodine atoms in the molecule to the number of osmotically active particles that the molecule produces in solution. Agents with a ratio of 1.5 belong to the high-osmolar category (osmolarity 2000 mOsm/kg water), agents with a ratio of 3.0 are considered as having low osmolarity (600–900 mOsm/kg water), and agents of 6.0 ratio represent the iso-osmolar category. High-osmolar contrast agents are by definition ionic and can be further divided as calcium or noncalcium binding. On the contrary, low-osmolar agents can be either ionic or non-ionic.⁴

Intracoronary injection of iodine contrast agents induces negative inotropic and chronotropic myocardial effects which are far more prominent when agents of 1.5 ratio with calcium-binding properties are used since they tend to extract intracellular calcium. Regarding cardiac conductivity, contrast infusion decreases sinoatrial automaticity and atrioventricular conduction leading to transient bradycardic events. Moreover, it is well documented that the use of contrast agents increases the vulnerability of the patient to ventricular fibrillation or sustained ventricular tachycardia.⁵

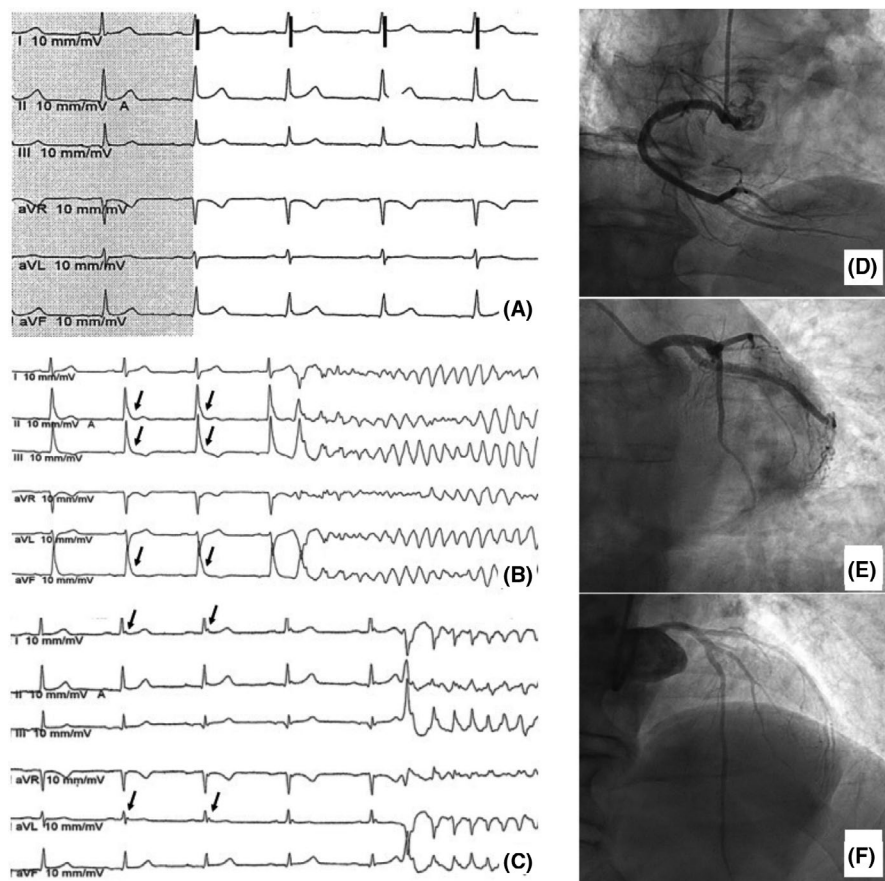


FIGURE 1 (A) Baseline ECG of the limb leads showing a slight J point elevation of 1 mm in inferior leads (II, III, aVF); (B) Significant J point elevation in inferior leads (II, III, aVF) with a slurred morphology preceded ventricular fibrillation during right coronary artery angiography; (C) J point elevation in lateral leads (I, aVL) preceded ventricular fibrillation following left main coronary artery angiography; (D) Selective right coronary artery angiography; (E) Selective left main coronary artery angiography; (F) Non-selective left main coronary artery angiography

Murdock et al. in 1984 explored the mechanisms of contrast-induced ventricular fibrillation in an experimental model. The iodine-based agent used was the most popular by the time in the USA, Renografin 76, an agent of high osmolality. The study revealed that the intracoronary injection of the agent led to a marked QT prolongation of 116 ± 18 msec during the first 6–12 s of the infusion exclusively in the myocardial territory of the LAD, which resolved back to normal after 30–45 s. During this time, QT interval was unchanged in the LCX territory betraying a marked temporal diversity of repolarization between these myocardial regions. When a premature ventricular beat was introduced spontaneously or by pacing during this time of inhomogeneous repolarization, intracardiac bipolar electrograms recorded marked conduction delay and fractionated electrical activity that ended to ventricular fibrillation in 23/25 cases.⁶ In a following study using iohexol—a low osmolar non-ionic agent—a similar mechanism of VF induction was proposed. Antiarrhythmic drugs of all four classes of Vaughan William's classification failed to prevent any contrast medium induced arrhythmias.⁷ On the contrary, the addition of 30 mmol/L of sodium reduced the incidence of ventricular fibrillation during this study, a controversial finding according to the rest of the literature. Although sodium concentration is recognized as critical for the prevention of malignant arrhythmogenesis caused by ionic agents, its ideal concentration differs among studies.⁸

Newly developed J waves or augmentation of preexisting ones during invasive coronary angiography of the right coronary artery have also been reported in a case series of patients with vasospastic angina.⁹ Since vasospasm was not provoked during the infusions, the authors hypothesized that the J waves were caused by transient ischemia from the contrast medium with subsequent conduction delay in the myocardial territory of the infused coronary artery. Right coronary artery perfuses mainly the inferior wall, the latest myocardial wall to be activated during normal conduction. A further delay in this area could be visible as elevation of the J point on the surface ECG. On the contrary, possible local conduction delays in the rest of the myocardial walls from left coronary artery infusion occur during systole and can be concealed in the QRS complex.⁹

In our case, every intracoronary injection of contrast agent led to intensification of the previously noticed pattern of early repolarization with further elevation of the J point and manifestation of ventricular fibrillation within 10 s of the infusion, initiated by the same premature ventricular ectopic beat. These observations led to the diagnosis of myocardial toxicity induced by the contrast agent. However, it is not clear whether the underlying pattern of early repolarization in the resting ECG played an additional role in the event.

ACKNOWLEDGMENT

Published with written consent of the patient.

CONFLICT OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

OK contributed to management of the patient, major revision, and approval of the final manuscript. MK contributed to management of the patient, major revision, and approval of the final manuscript. DM contributed to management of the patient, major revision, and approval of the final manuscript. GB contributed to major revision and approval of the final manuscript. KV contributed to management of the patient, major revision, and approval of the final manuscript. SD contributed to management of the patient, major revision, and approval of the final manuscript. AG contributed to management of the patient, major revision, and approval of the final manuscript. KPL wrote the first draft, contributed to management of the patient, major revision, and approval of the final manuscript. ME contributed to management of the patient, major revision, and approval of the final manuscript. VV contributed to management of the patient, major revision, and approval of the final manuscript.

ETHICAL APPROVAL

Data contained were obtained through routine clinical care and for a diagnosis and treatment purposes. No ethical approval was recommended.

DATA AVAILABILITY STATEMENT

Data available on request from the authors.

ORCID

George Bazoukis  <https://orcid.org/0000-0003-1009-9772>

REFERENCES

1. Shaik FA, Slotwiner DJ, Gustafson GM, Dai X. Intra-procedural arrhythmia during cardiac catheterization: a systematic review of literature. *World J Cardiol.* 2020;12(6):269-284.
2. Chen J, Gao L, Yao M, Chen J. Ventricular arrhythmia onset during diagnostic coronary angiography with a 5F or 4F universal catheter. *Rev Esp Cardiol.* 2008;61(10):1092-1095.
3. Chen J, Gao LJ, Chen JL, Song HJ. Contemporary analysis of predictors and etiology of ventricular fibrillation during diagnostic coronary angiography. *Clin Cardiol.* 2009;32(5):283-287.
4. Brinker J. What every cardiologist should know about intravascular contrast [published correction appears in *Rev Cardiovasc Med.* 2004 Spring;5(2):following 138]. *Rev Cardiovasc Med.* 2003;4(Suppl 5):S19-S27.
5. Hirshfeld JW Jr. Cardiovascular effects of iodinated contrast agents. *Am J Cardiol.* 1990;66(14):9F-17F.
6. Murdock DK, Euler DE, Becker DM, Murdock JD, Scanlon PJ, Gunnar RM. Ventricular fibrillation during coronary angiography: an analysis of mechanisms. *Am Heart J.* 1985;109(2):265-273.
7. Pedersen HK, Jacobsen EA, Mortensen E, Refsum H. Contrast-medium-induced ventricular fibrillation: arrhythmogenic mechanisms and the role of antiarrhythmic drugs in dogs. *Acad Radiol.* 1995;2(12):1082-1088.

8. Idée JM, Matalon C, Koeltz B, Bonnemain B, Lefevre T. Contrast-medium-induced ventricular fibrillation: arrhythmogenic mechanisms and the role of antiarrhythmic drugs in dogs. *Acad Radiol.* 1996;3(9):781-785.
9. Kitazawa H, Sato A, Okabe M, Aizawa Y. J waves induced during coronary angiography in patients with vasospastic angina and its implication. *J Electrocardiol.* 2021;64:99-101.

How to cite this article: Kariki O, Kontonika M, Miliopoulos D, et al. Contrast-induced early repolarization pattern and ventricular fibrillation. *Clin Case Rep.* 2021;9:e04630. <https://doi.org/10.1002/ccr3.4630>