Journal of Cardiology Cases 16 (2017) 26–29

Contents lists available at ScienceDirect

Journal of Cardiology Cases

journal homepage: www.elsevier.com/locate/jccase

Case Report

A case of ventricular fibrillation as a consequence of capecitabine-induced secondary QT prolongation: A case report

Kazuto Hayasaka (MD)[a], Masateru Takigawa (MD)[a,*], Atsushi Takahashi (MD)[a], Taishi Kuwahara (MD)[a], Kenji Okubo (MD)[a], Yasuaki Tanaka (MD)[a], Toru Misawa (MD)[a], Masafumi Mizusawa (MD)[a], Yosuke Yamakami (MD)[a], Keisuke Kojima (MD)[a], Yuichiro Sagawa (MD)[a], Keiichi Hishikari (MD)[a], Kazuya Yamao (MD)[a], Emiko Nakashima (MD)[a], Jun Nakajima (MD)[a], Shigeki Kimura (MD)[a], Katsumasa Takagi (MD)[a], Hiroyuki Hikita (MD)[a], Mitsuaki Isobe (MD, FJCC)[b]

[a] Department of Cardiovascular Center, Yokosuka Kyosai Hospital, Yokosuka, Japan
[b] Department of Cardiovascular Medicine, Tokyo Medical and Dental University, Tokyo, Japan

ARTICLE INFO

Article history:
Received 17 November 2016
Received in revised form 7 March 2017
Accepted 24 March 2017

Keywords:
Capecitabine
Secondary long QT syndrome
Ventricular fibrillation

ABSTRACT

Capecitabine is an oral fluoropyrimidine which can prolong QT interval. However, there have been no reports that capecitabine induced ventricular fibrillation (VF) due to secondary QT prolongation in patients with no structural heart disease. A 39-year-old woman developed VF during the chemotherapy of capecitabine for colon cancer. At the administration, corrected QT interval (QTc) was prolonged to 559 ms despite no evidence of organic heart disease. Discontinuation of capecitabline normalized the QTc (414 ms). During the follow-up of eight years, neither the QTc prolongation nor the recurrent VF has been detected. We report the rare case of capecitabine-related VF without any organic heart disease.
<**Learning objective:** Capecitabine is an oral fluoropyrimidine carbamate commonly used to treat colorectal and breast cancer. Capecitabine has been reported to be associated with VF due to vasospasm. However, capecitabine is also associated with QT elongation. This is the first report to describe VF due to capecitabine-related secondary long QT syndrome in a patient with no cardiac heart disease. Physicians must carefully follow up patients during capecitabine chemotherapy with serial electrocardiograms.>
© 2017 Japanese College of Cardiology. Published by Elsevier Ltd. All rights reserved.

Introduction

Capecitabine is an oral fluoropyrimidine carbamate that is converted to 5-fluorouracil (5-FU) and selectively delivered to tumors. It is commonly used to treat colorectal and breast cancer. Although QT prolongation due to capecitabine was described [1], ventricular fibrillation (VF) associated with QT prolongation due to capecitabine has not been reported. One case report described the association between capecitabine and VF due to vasospasm [2]. Furthermore, 5-FU, which is converted from capecitabine by thymidine phosphorylase in tumor sites, is experimentally proved to have an effect on vascular smooth muscle cells, inducing concentration-dependent coronary vasospasm [3]. Here, we report the case of VF not due to vasospasm but due to capeciatabine-related secondary long QT syndrome in a young woman in the course of chemotherapy for colorectal cancer.

Case report

A 39-year-old woman with an ascending colon cancer, received an adjuvant chemotherapy with capecitabine (3600 mg/day) subsequent to a right hemicolectomy and lymph node dissection. Following a few periods of faintness while walking, she collapsed for the first time in her life without any chest pain, during the course of third chemotherapy, resulting in a cardiopulmonary arrest (CPA). She was given bystander cardiopulmonary resuscitation soon and recovered with automated external defibrillator

* Corresponding author at: Department of Cardiovascular Center, Yokosuka Kyosai Hospital, 1-16 Yonegahamadori, Yokosuka-shi, Kanagawa-ken 238-8558, Japan. Fax: +81 46 825 2103.
E-mail address: teru.takigawa@gmail.com (M. Takigawa).

http://dx.doi.org/10.1016/j.jccase.2017.03.009
1878-5409/© 2017 Japanese College of Cardiology. Published by Elsevier Ltd. All rights reserved.

8 min after CPA (Fig. 1). When she arrived at hospital, 12-lead electrocardiogram demonstrated prolonged QT interval at 482 ms (QTc 559 ms) without ST change (Fig. 2a). Electrolytes (K: 3.9 mEq/l, Ca: 8.8 mg/dl, Alb: 4.4 g/dl, Mg: 2.0 mg/dl) and atrial blood gas were within normal range as well as the other biochemical tests. Her chest X-ray was normal. Cardiac ultrasonography, left- and right-ventriculography, and coronary angiography revealed no evidence of organic heart disease (left ventricular ejection fraction 72.7%). Magnetic resonance imaging showed no evidence of delayed enhancement. Acetylcholine provocation test was also negative (Fig. 3). Pilsicainide provocation test was negative. Holter electrocardiogram demonstrated no evidence of premature ventricular contraction (PVC), and signal averaged electrocardiogram showed no late potentials. Although she did not have a family history of long QT syndrome or sudden cardiac death, genetic mutation examination was performed to rule out congenital long QT syndrome associated with KCNQ1, KCNH2, SCN5A, and SLMAP; the result was found to be negative two months later. After the discontinuation of capecitabine, fusion of TU wave appeared (Fig. 2b) followed by gradual QT interval shortening. Finally, four days after the discontinuation of capecitabine, QT returned to 442 ms (QTc 414 ms) (Fig. 2c). The VF was suspected to be

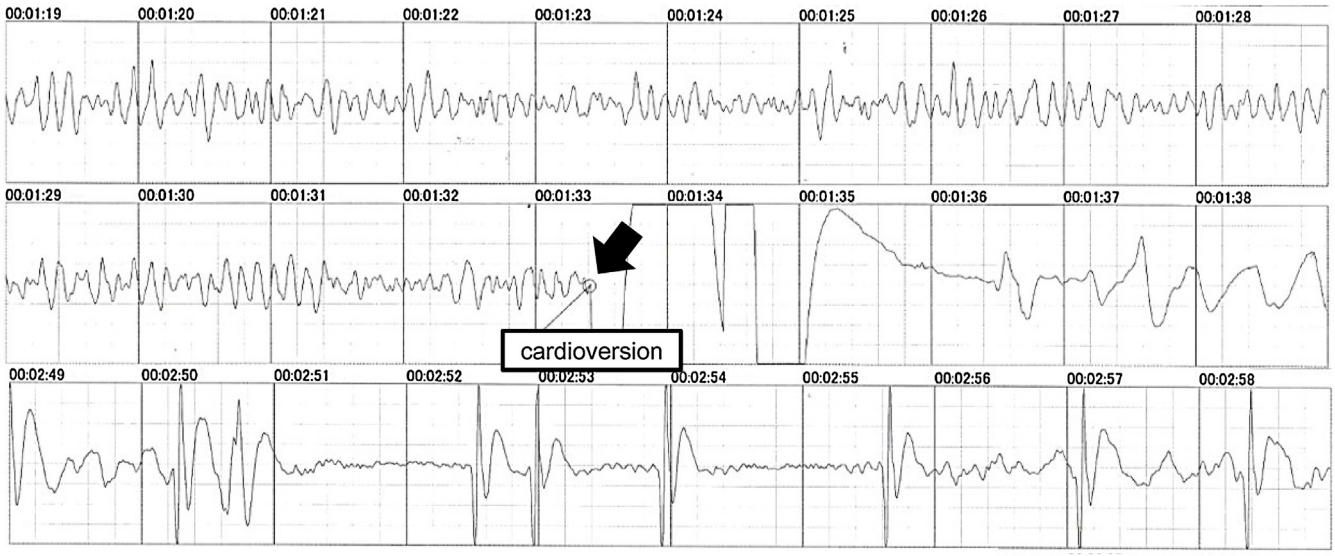

Fig. 1. Intracardiac electrocardiogram in automated external defibrillator (AED) during the event. Cardioversion from the AED terminated the ventricular fibrillation (black arrow).

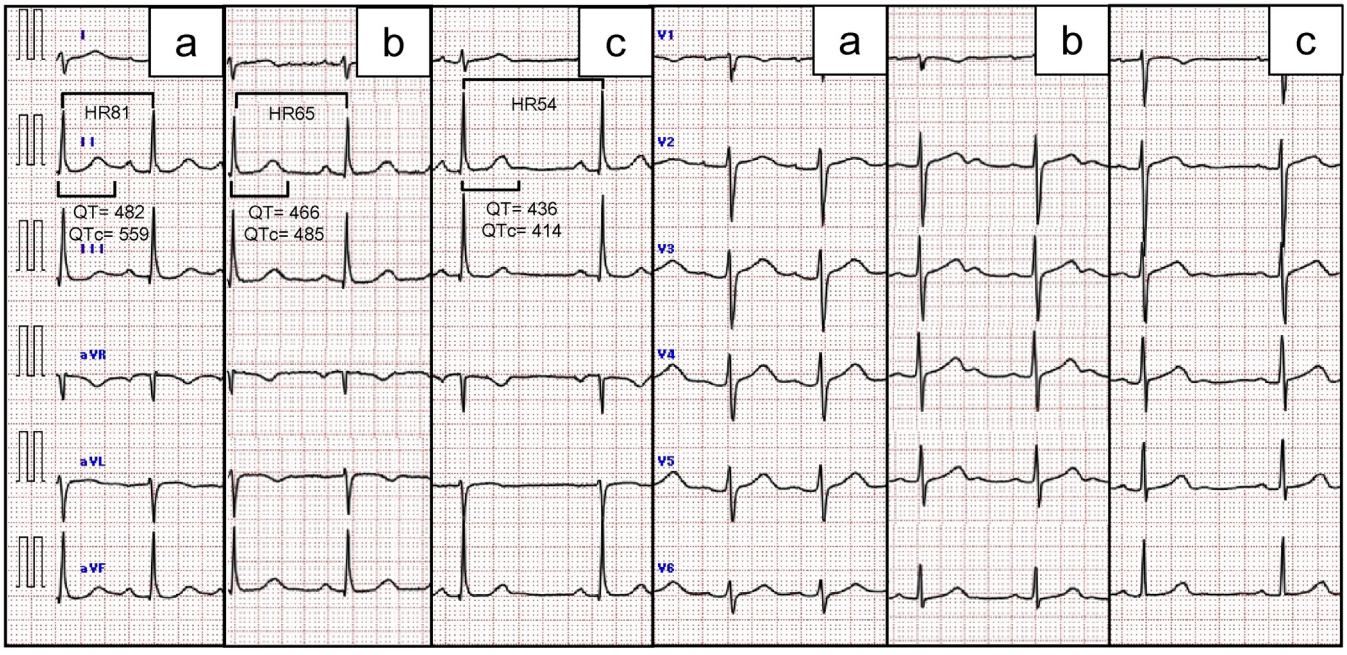

Fig. 2. 12-lead electrocardiogram at the admission (a), on the day after admission (b), and four days later (c). A corrected QT interval (QTc) shortened from 559 ms to 414 ms after the discontinuation of capecitabine (c). A QTc interval in lead II was calculated based on Bazett's formula as follows: QTc = QT interval/square root of the RR interval (in s) [13].

Download English Version:

https://daneshyari.com/en/article/5614809

Download Persian Version:

https://daneshyari.com/article/5614809

Daneshyari.com