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CASE REPORT

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Episode of ventricular fibrillation in patient with coronary artery ectasia during coronary angiography

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Key Clinical Message

There was an 83-year-old man having coronary artery disease associated with coronary artery ectasia who occurred ventricular fibrillation suddenly during coronary artery angiography. As Kawasaki disease was suspected to the most likely reason which led to coronary artery lesion.

KEYWORDS

coronary artery angiography, coronary artery ectasia, Kawasaki disease, ventricular fibrillation

1 | INTRODUCTION

Coronary artery angiography (CAG) has been regarded as an effective method to diagnose coronary artery disease (CAD) and has become increasingly popular and mature, but it also has a potential risk leading to some serious complications, such as death (<0.2%), myocardial infarction (<0.5%), stroke (<0.7%), malignant ventricular arrhythmia (<0.5%), etc.[1] However, the probability of these complication occurring is low, as is usually the case in patient/s with severe left main disease, multi-vessel disease, dissection or rupture of plaque/s, and hemodynamic instability. The development of VF in CAD associated with coronary artery stenosis during CAG is infrequent worldwide and related reports are rare. Only one similar case has previously been reported on the Chinese island of Taiwan. An 84-year-old female with diffuse, dilated multi-vessel coronary artery ectasia experienced a similar accident during CAG. She experienced a sudden onset of VF during CAG, and then regained consciousness after defibrillation.[1] Coronary artery ectasia is strongly associated with many adverse cardiovascular events. Coronary artery ectasia, which accompanies these complications, tends to have a poor prognosis. In this case, an 83-year-old male with massive coronary artery ectasia who developed sudden ventricular fibrillation (VF) during CAG came to

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our attention. Through analyzing angiogram and searching literature, we speculated that the cause of patient with paroxysmal VF might closely related to the huge coronary artery ectasia in the left circumflex (LCX)'s that was considered relating to Kawasaki disease (KD). Deep lessons and inspiration were obtained.

2 | CASE DATA

An 83-year-old man was admitted to the hospital with the chief complaint of recurrent chest pains and tightness in his chest. He had a history of coronary heart disease, hypercholesterolemia, diabetes mellitus, and hypertension, and a history of smoking or drinking alcohol. The patient has had a total of five stents implanted since 2016. The patient's cardiopulmonary examination after admission revealed no obvious abnormalities. But the electrocardiogram (ECG) indicated left anterior hemiblock (LAH) and changes in T-wave (Figure 1). Echocardiography showed that the integrated systolic function of left ventricle was normal and the ejection fraction (EF) was 53.5%. CAG re-examination showed a tumor-like artery ectasia in the middle of LCX (Figure 2). In the process of left anterior oblique (LAO) projection in fluoroscopy, the patient suddenly developed VF, 150 joules (J) biphasic defibrillation was given immediately, and then the heart was defibrillated successfully.

3 | DISCUSSION

Myocardial ischemia (MI) is the most common cause of VF in CAG. MI can be subdivided into three types, including primary MI, secondary MI, and Iatrogenic MI. Primary MI is often referred to as coronary atherosclerotic heart disease. Secondary MI can occur due to various reasons, including moderate to severe anemia, rapid atrial fibrillation (AF), hyperthyroidism, hypertrophic cardiomyopathy, etc. These primary cardiomyopathies can lead to myocardial insufficiency, or over oxygen consumption of the myocardium, resulting in secondary MI. Iatrogenic MI refers to MI due to occlusion of the coronary arteries caused by medical mal-practise during PTA or PTCA. But along with

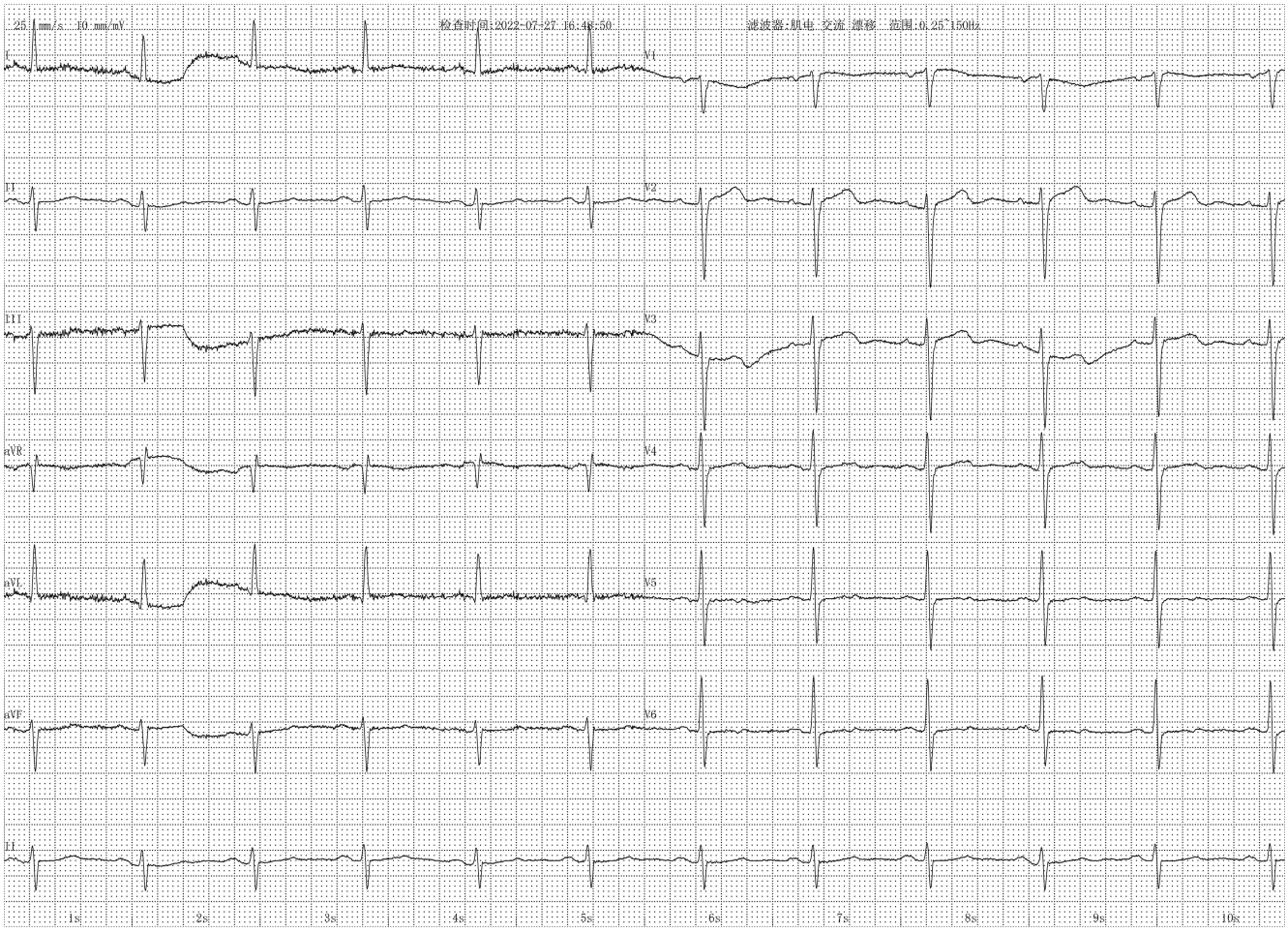

FIGURE 1 ECG suggests left anterior branch hemiblock, T wave changes in I, avL, V4, V5, V6 lead.

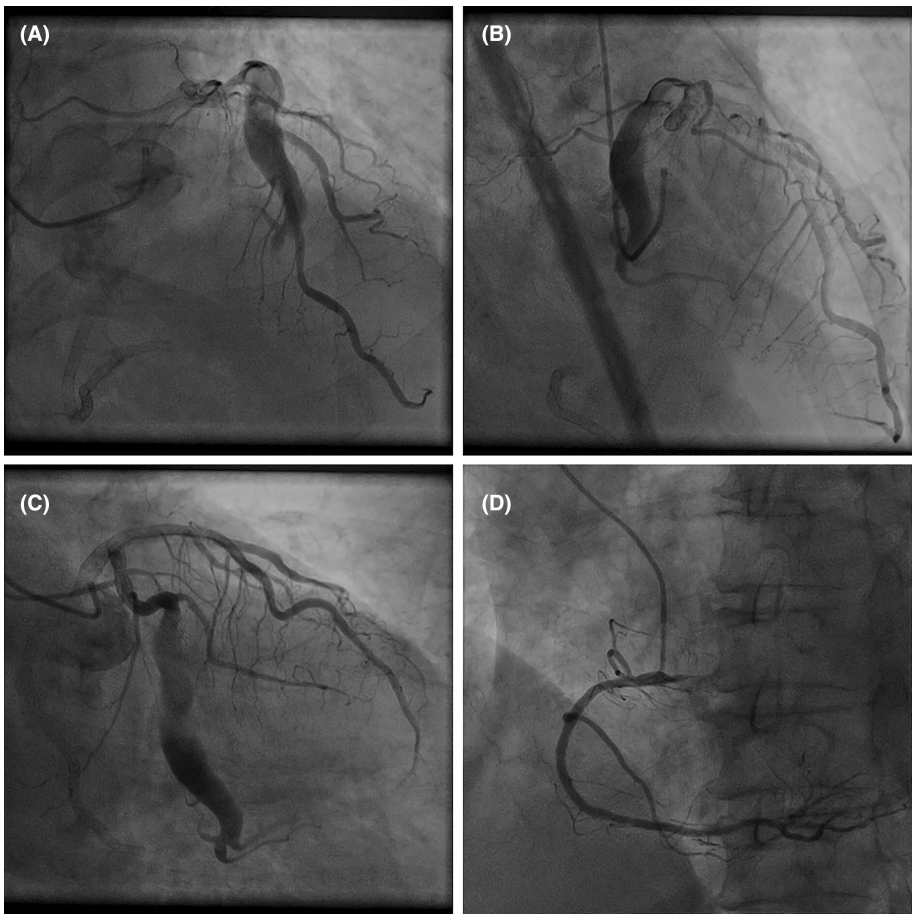

FIGURE 2 Picture A, B, C showed LCX's ectasia and the inner space is full of blood perfusion. Picture D showed there was no obvious stenosis or ectasia in RCA after PTCA.

advances in medical care and imaging techniques, the incidence of VF during CAG is extremely low.

No significant narrowing of the coronary arteries was seen during this patient's CAG, including the previously treated RCA, and no stenosis was found with the stent. However, slow blood flow was observed. Therefore, the possibility of VF occurring during CAG may not be related to MI caused by vascular stenosis. We hypothesize that the cause of the occurrence of VF in this patient may be related to the slow blood flow in the LCX caused by the presence of giant coronary ectasia. We speculate that the occurrence of the VF may be due to the MI caused by the short time interval between the second and third contrasts. The third contrast was performed when the contrast medium of the second contrast was not completely emptied out from the vessel, resulting in MI.

We found that the speed of iodine-based contrast medium emptied very slowly from the dilated LCX, consistent with the pathophysiology of slow blood flow due to vortex formation in the dilated vessel. Therefore, we hypothesize that the coronary artery ectasia may be the trigger for VF. Besides that, process of iodine-based contrast medium's injection may induced myocardial stretch, lead to certain pathophysiological changes and cardiac arrhythmia on the basis of coronary aneurysm, and then triggered the onset of VF.[2] Next, thrombosis can easily occur in the dilated coronary arteries due to abnormal blood flow and impaired vascular endothelium. But we did not find any significant clots during the CAG process, and this probability can be ruled out.

The pathophysiological changes that triggered VF due to coronary artery ectasia can be described in the following aspect: the coronary artery ectasia in the middle of LCX lead to rearrangement in coronary vascular network system, accompanied with the presence of triple vessel disease (TVD), make the normal coronary arteries lose their function in transporting blood. In order to reduce turbulence of blood flow, the coronary circulatory system developed compensatory collateral blood circulation, which lead to further rearrangement in the vascular network, form a vicious cycle that gradually results in MI.[3,4] MI with coronary artery ectasia is prone to electrophysiological instabilities due to concentration gradients produced by various metabolites between the perfusion and nonperfusion regions. This abnormal electrophysiological

activity will distort the shape of the action potential (AP) and accelerate myocardial diastolic depolarization. As a result, there is a significant reduction in myocardial AP conduction, which interferes with the recovery of myocardial excitability and the onset of arrhythmia symptoms. These arrhythmias always precede the VF and contribute to its development. Above activity progression is possibly a fundamental mechanism of VF in patients with CAD and coronary artery ectasia.[5,6]

The slow emptying of the contrast agent in the LCX might be responsible for MI. However, due to the sudden onset of VF, the operator needed to perform non-synchronized cardioversion and did not record the intraoperative ECG in a timely manner. After the CAG, the patient refused to have blood drawn again and the myocardial enzymes were not rechecked. Therefore, we lacked evidence of MI and could only speculate that the patient may have had local MI.

Although the patient in this report and the above mentioned Chinese patient in Taiwan all had occurred VF during CAG, the character of their coronary dilation was different. Coronary dilation in this patient was not diffuse and multiple, restricted only to LCX, though the extent of the expansion is striking.

As the patient in this case is an elderly individual with an incomplete description of his previous medical history and family history, we could not confirm whether he had been previously diagnosed with KD. However, we consider on the basis of GAG that the limited but large coronary artery ectasia in this patient could be caused by a prior KD history rather than coronary atherosclerosis. Initially, the extent of atherosclerosis and stenosis in the LCX of this patient was not severe, and was out of proportion to the extent of dilatation. In contrast, severe atherosclerotic stenosis is seen in the distal of right coronary artery (RCA) and almost blocked, but no obvious vasodilation was found in the proximal and middle segment of RCA. Second, coronary ectasia caused by atherosclerosis often occurs in RCA (40%–61%), followed by the left anterior descending (LAD) artery (15%–32%) and LCX (15%–23%),[7] whereas coronary lesions involved by KD are prominent seen in left coronary artery.[8] This might explain the fact that the patient's coronary artery ectasia is confined to the LCX. In third, atherosclerotic coronary ectasia is diffuse and often involves multiple vessels,[9] which is inconsistent to the patient's CAG result. Therefore, we speculate that the coronary artery ectasia may be involved in KD.

It is regrettable that we were not able to provide direct evidence of MI, such as ECG, myocardial enzyme test results, or myocardial nuclei scans. We can only speculate that the localized MI in the patient may have been caused by delayed contrast emulsion due to slow blood flow in the coronary artery ectasia.

In addition, according to Rittger H's research, complication rates increased significantly with age in CAG.[10] There is a possibility that the increased risk of complications during CAG is due to the advanced age of the patient. And the incidence of VF also increases with age. According to Tseng WC's research, VF incidence rates were low in patients younger than 10 years and increased steadily after adolescence out of hospital.[11] Similarly, the probability of VF occurring in-hospital patients increases with aging.[12] Paradoxically, in Jabbari R et al.'s data, they drew a conclusion, there was a higher risk of developing VF among those aged <60 years. But they supposed that this association could be due to higher resuscitation and survival rates for out-of-hospital VF in younger persons.[13]

We found the reasons of VF during CAG includes multi-vessel disease (especially more likely to occur in the proximal or opening area of RCA on multi-vessel disease), operating means, types of contrast agent, and some unidentified causes by searching for the related reference.[14] However, the analysis of the correlation between VF during CAG and age seems to have not been discussed separately, the age dependence of the pattern of VF might indicate different underlying external or endogenous triggers,[15] and further research is needed to confirm.

4 | CONCLUSION

Although the patient was defibrillated and regained consciousness shortly after the onset of VF during CAG, the accident was still unexpected and profound. Hitherto, for those who are suspects of having both coronary artery ectasia and coronary stenosis, we should be on the alert and ready for immediate rescue. If the operator suspect that the coronary artery has ectasia and slow blood flow before or during the CAG, they can avoid the risk of VF by (1) eliminating any potential causes of VF before CAG, such as MI, electrolyte disorders, and electrical storms, etc. (2) properly extend the time for each shot of contrast agent injection. (3) dilute the contrast agent appropriately with physiological saline to facilitate the emptying of the contrast agent. If VF occurs unfortunately during CAG, non-synchronized cardioversion can quickly restore the patient's sinus rhythm.

It is well known that VF can occur during CAG, athough with a lower probability, each medical member still needs to be cautious and careful during each procedure, thus being able to handle most unexpected events calmly. At the same time, a differential diagnosis should be performed to exclude those with coronary artery ectasia caused by KD. The correct diagnosis and treatment of patients with coronary artery ectasia due to KD can reduce or even avoid the occurrence of adverse cardiovascular

events such as bleeding, intracavity thrombosis, myocardial infarction, heart failure, and sudden cardiac death.

AUTHOR CONTRIBUTIONS

Shujiao Zheng: Conceptualization; writing – original draft. **Zuheng Liu:** Data curation; funding acquisition; supervision. **Shaofeng Zhang:** Resources; validation. **Qiang Xie:** Resources; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from The First Affiliated Hospital of Xiamen University. Data are available from the authors with the permission of The First Affiliated Hospital of Xiamen University.

CONSENT

I confirm that written patient consent has been signed and collected in accordance with the journal's patient consent policy, and that I have added a patient consent statement asserting this at the bottom of the manuscript's title page. I will retain the original written consent form and provide it to the Publisher if requested.

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