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CASE REPORT

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Anal-cardiac reflex leading to coronary spasm and cardiac arrest during abdominoperineal excision of the rectum (MILES): case report and review of the literature

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Abstract

Background Coronary artery spasm (CAS) has been frequently documented during regional or general anesthesia. However, severe cardiac arrest induced by CAS as a result of anal-cardiac and vasovagal reflexes is relatively uncommon.

Case presentation This report describes a patient with a history of CAS and percutaneous coronary angioplasty (PTCA) who experienced coronary artery spasm and subsequent cardiac arrest during anal anastomosis in a transabdominal perineal radical resection of rectal cancer (MILES procedure).

Conclusion This case study offers significant insights into the pathophysiology of cardiac arrest induced by surgical intervention in the anal region, which is densely innervated by the vagus nerve at the terminal portion of the rectum. This phenomenon may be associated with the anal cardiac reflex and vagus nerve reflexes. It is imperative to recognize the potential for catastrophic outcomes due to vagus nerve excitation during rectal end surgery, particularly in patients with a history of acute CAS.

Keywords MILES surgery, Anal-cardiac reflex, Cardiac arrest, Coronary artery spasm

Background

Coronary artery spasm (CAS) is characterized by which is a reversible vaso-constriction driven by a spontaneous vascular smooth muscle hypercontractility and vascular wall hypertonicity narrowing the lumen of normal or atherosclerotic coronary arteries compromising the myocardial blood flow [1]. CAS represents a pathological and physiological condition characterized by diverse clinical manifestations. These variations arise from differences in the location and severity of the spasm, as well as the presence of collateral circulation. Clinical presentations associated with CAS include typical variant angina, atypical CAS angina, acute myocardial infarction (AMI), sudden cardiac death, various arrhythmias, heart failure, and asymptomatic myocardial ischemia. Collectively, these conditions are referred to as coronary artery spasm syndrome (CASS). The vagus nerve is extensively distributed in the rectal region [2], and its stimulation is heightened during surgical procedures. Notably, manipulations such as traction or dilation of the anus can intensify the

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vagal reflex, potentially leading to significant adverse effects including bradycardia, hypotension, arrhythmias, and even cardiac arrest [3].

Case presentation

To increase awareness of this phenomenon, we present a case study involving coronary artery spasm and cardiac arrest induced by the anal cardiac reflex during general anesthesia. The subject was a 58-year-old male who underwent transabdominal perineal combined radical resection for rectal cancer (MILES) under general anesthesia. The patient has a medical history of hypertension spanning over a decade and is currently managing the condition with oral administration of amlodipine and atorvastatin tablets, resulting in well-controlled blood pressure levels. Two months prior, the patient presented to an external medical facility with symptoms of chest tightness and received a diagnosis of acute non-ST segment elevation myocardial infarction, classified as Killip grade I. Coronary angiography was conducted under local anesthesia, taking into account the possibility of coronary spasm and acute occlusion of the posterolateral artery (PLA). The patient subsequently underwent plain old balloon angioplasty (POBA), which resulted in an improvement in postoperative symptoms. Currently, the patient is on an anticoagulant regimen consisting of aspirin 100 mg once daily and ticagrelor 90 mg twice daily. This regimen has been discontinued for more than 10 days. The preoperative electrocardiogram revealed ST segment alterations, specifically a horizontal ST segment at 0.5 mm in leads II, III, aVF, V5, and V6. However, no significant abnormalities were detected on the resting electrocardiogram (Fig. 1).

Prior to the induction of anesthesia, ultrasound-guided catheterization was performed on the right internal jugular vein and the left radial artery. Anesthesia induction was achieved via target-controlled infusion (TCI) of propofol at a concentration of 3 μg/ml and remifentanil at 3–4 ng/ml. Additionally, rocuronium was administered at a dose of 0.6 mg/kg, along with lidocaine at 1 mg/kg. During the induction of general anesthesia, following the completion of tracheal intubation, 60 ml of 0.5% ropivacaine was administered for a transversus abdominis plane block. The depth of anesthesia was maintained at 0.8 minimum alveolar concentration (MAC) of desflurane. Upon reaching the stage of anal closure in the surgical procedure, the electrocardiogram exhibited notable changes(Fig. 2).

In response, the anesthesiologist promptly instructed the surgical team to halt the procedure, initiated chest compressions, administered 40 μg of adrenaline intravenously in two separate doses, and commenced a continuous intravenous infusion of adrenaline at a rate of 0.1 μg/kg/min. Five milligrams of diltiazem was administered intravenously at a slow rate, resulting in the restoration of sinus rhythm approximately 4 min thereafter. Subsequently, an esophageal ultrasound was conducted, revealing no abnormalities on the echocardiogram. The

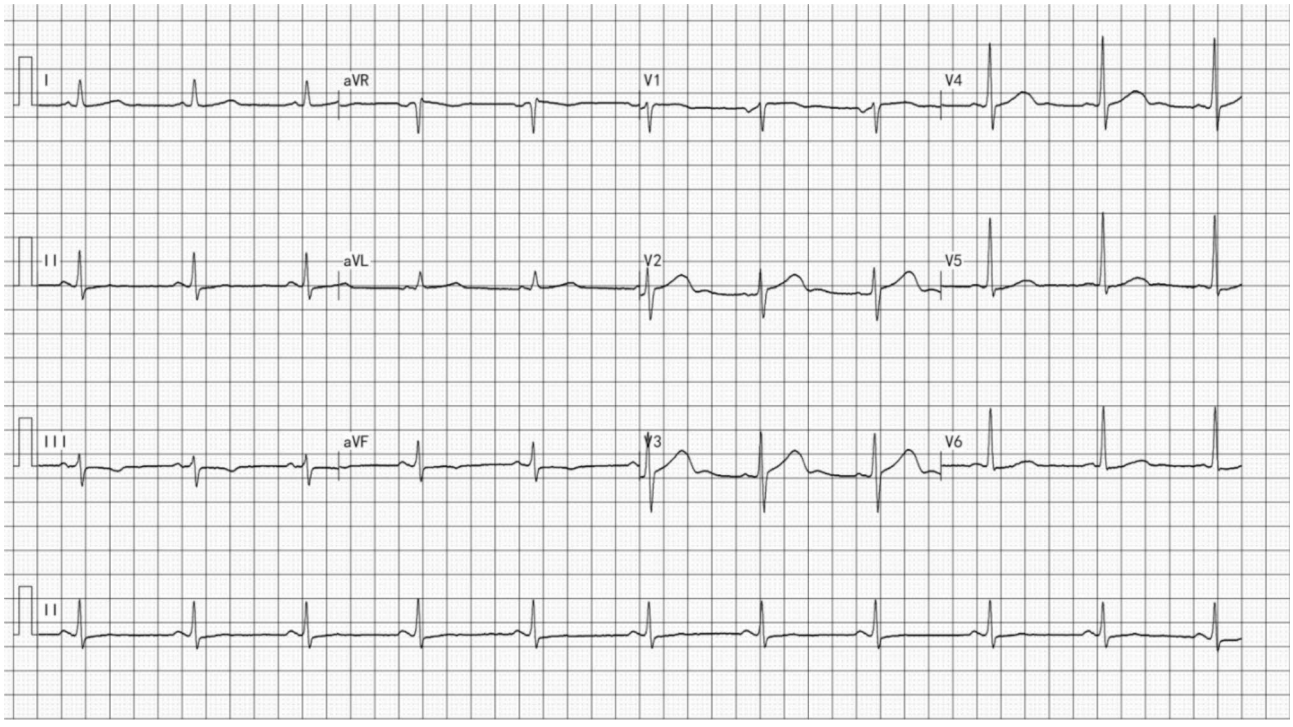

Fig. 1 Preoperative electrocardiogram

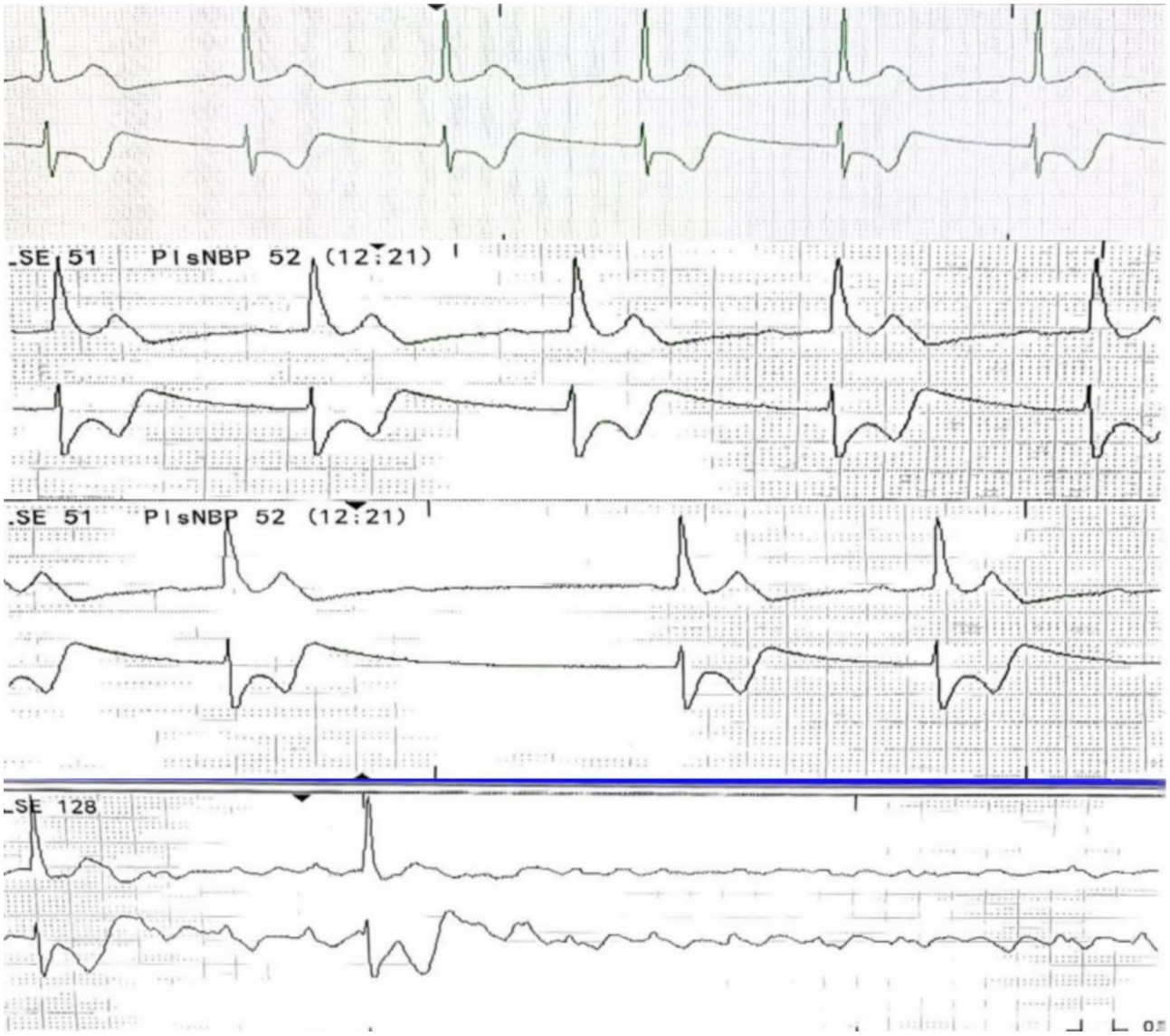

Fig. 2 ECG changes during anal suture

electrocardiogram changes are depicted in the accompanying figure (Fig. 3).

However, at 21 min postrestoration of sinus rhythm, the patient experienced another cardiac arrest. Cardiopulmonary resuscitation was initiated with continuous chest compressions, intravenous administration of adrenaline, and placement of a temporary pacemaker. The subsequent electrocardiogram changes are illustrated in Fig. 4, which depicts the ECG alterations following the second cardiac arrest and successful resuscitation:

Following a 20-minute interval, the spontaneous heart rate was reestablished, and an intravenous administration of esmolol was employed to regulate both the heart rate and blood pressure. To facilitate diuresis and correct acidosis, 125 ml of mannitol, 125 ml of sodium bicarbonate, and furosemide were utilized. Bispectral index (BIS) monitoring indicated that the cognitive index exceeded 90. Upon stabilization of the circulatory system, the surgical team proceeded with the operation without further incidents of cardiac arrest. The surgery lasted of 246 min, after which the patient was transferred to the intensive care unit (ICU) for postoperative care.

Two hours following admission to the intensive care unit, the bedside electrocardiogram revealed an elevated ST segment in leads II, III, aVF, and V3R-V5R, with the posterior arch measuring ≤ 0.8 mm, as illustrated in the accompanying figure (Fig. 5):

Following a detailed discussion with the patient's family regarding the advantages and potential risks associated with coronary angiography, the decision was made to proceed with the examination. The results indicated the absence of any coronary stenosis or obstruction (Fig. 6).

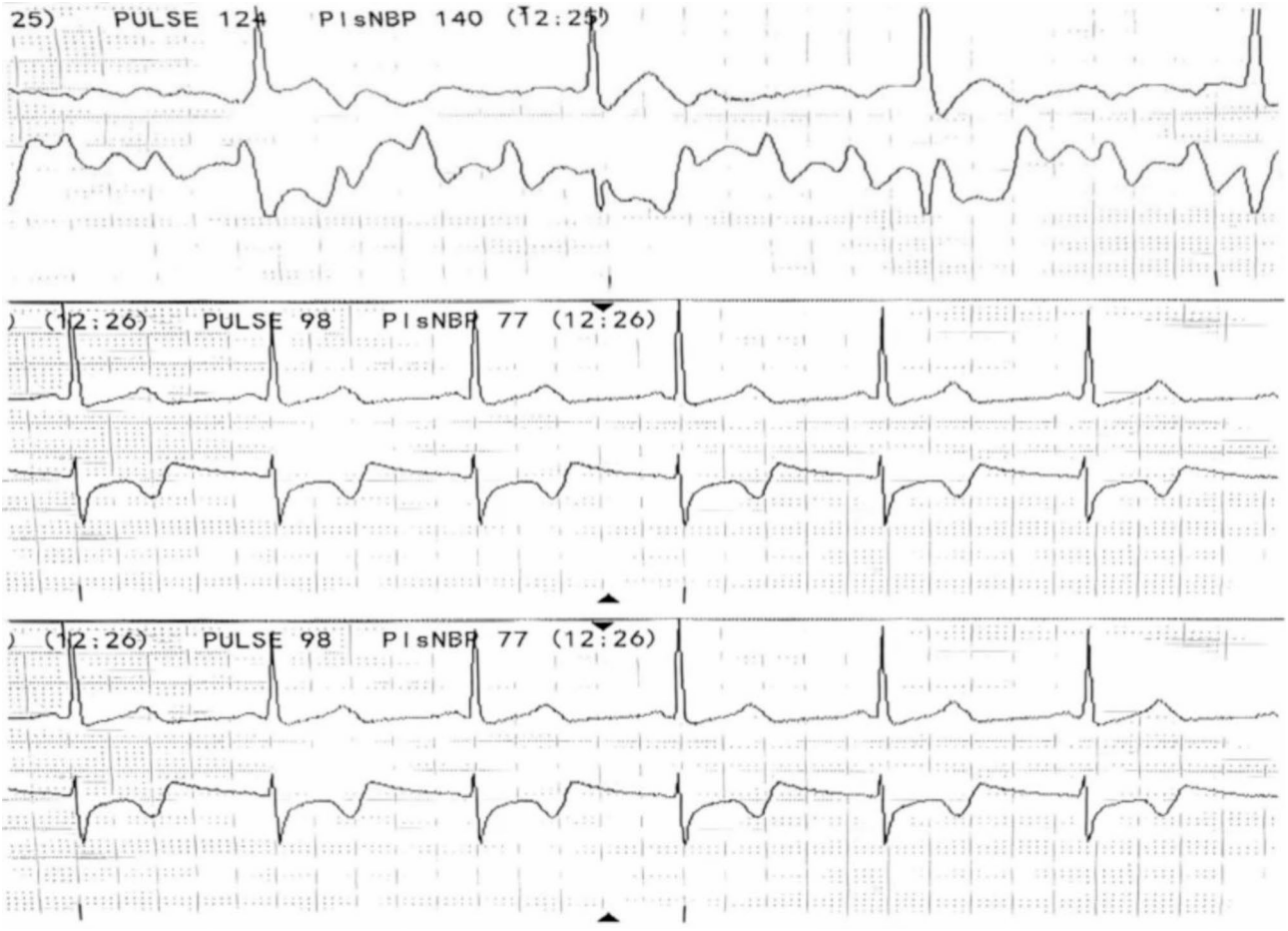

Fig. 3 ECG after recovery from the first cardiac arrest

A bedside echocardiographic examination revealed segmental wall motion abnormalities of the left ventricle: reduced systolic activity in the left ventricular septum, anterior wall, and inferior wall, while the remaining segments show acceptable systolic activity. Following the cessation of sedation, the patient gradually regained consciousness by the subsequent afternoon, at which point the tracheal tube was successfully removed. Four days thereafter, the patient was transferred back to the general ward. Five days posttransfer, 24-hour electrocardiographic monitoring revealed a sinus rhythm accompanied by a shortened P-R interval, whereas QRS and ST-T segment changes remained within normal limits.

The following year, the patient regularly visited our hospital for stoma care and cardiology outpatient treatment, and there were no related sequelae following cardiac arrest.

Discussion and conclusions

Cardiac arrest in surgical patients is frequently a critical event, with documented mortality rates exceeding 50%. The overall incidence of perioperative cardiac arrest is estimated to range from 4.3 to 34.6 per 10,000 procedures, increasing to 54.4 per 10,000 procedures in the elderly population [4]. Factors contributing to intraoperative cardiac arrest include anaphylaxis, pulmonary embolism, hypertension, trauma-related arrest, malignant hyperthermia, tension pneumothorax, severe hyperkalemia, and local anesthetic systemic toxicity [5]. In this patient, however, these potential causes were systematically excluded, and the vital signs remained stable and free of abnormalities until the anal suture was conducted. This observation led us to consider the previously overlooked anal cardiac reflex, also known as the vagus-vagus reflex, which is associated with the dense network of vagus nerve fibers in the anal region. When these fibers are stimulated or compressed, they can exert an inhibitory effect on the heart via the vagus nerve, potentially resulting in cardiac arrest. Inducing factors include excessively deep or shallow anesthesia, intense surgical stimulation, patient-specific sensitivity and specificity to stimulation, instability in autonomic nervous system function, and preexisting conditions such as heart

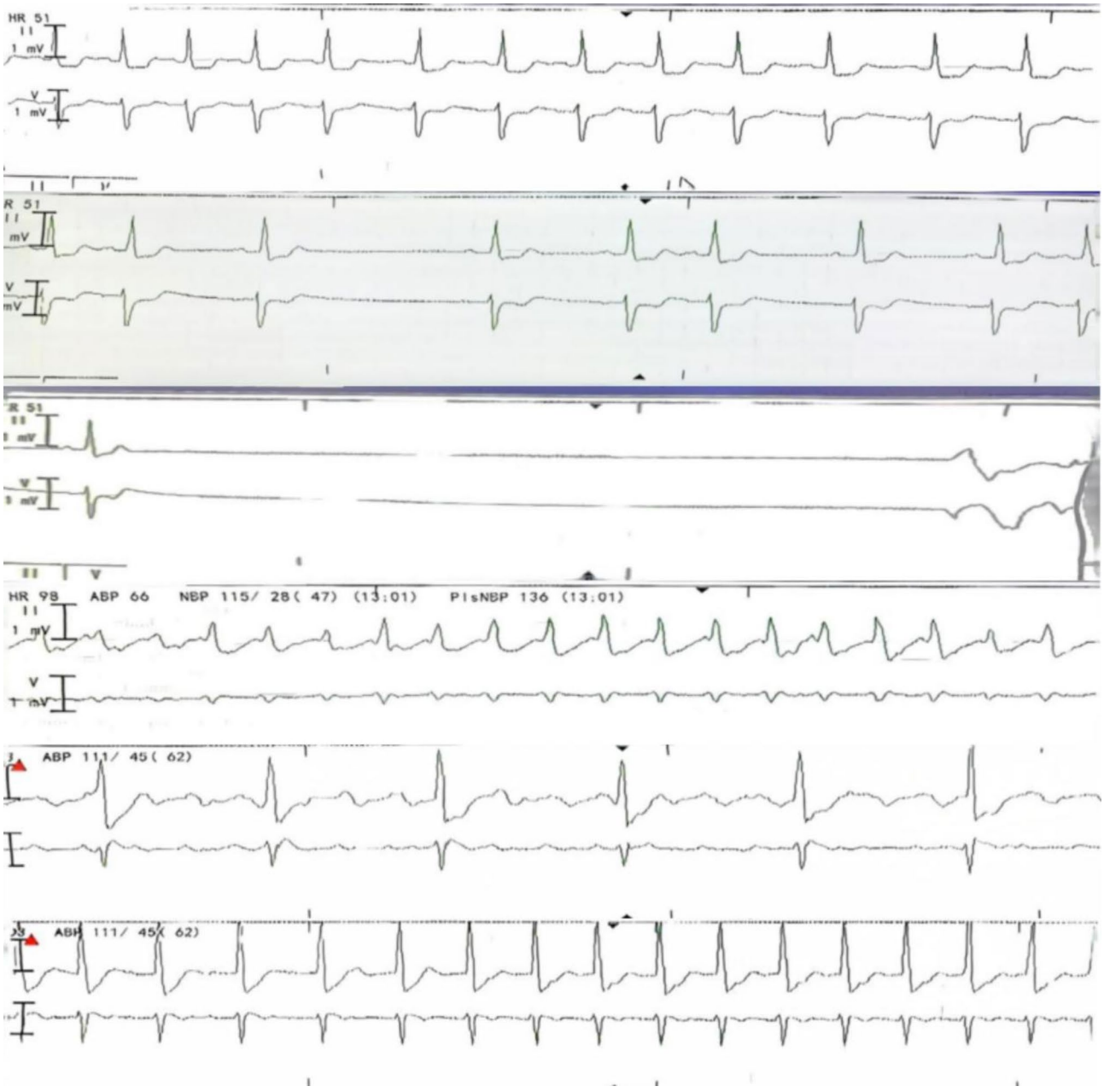

Fig. 4 ECG changes after the second cardiac arrest and successful resuscitation

disease, which may increase the risk of developing a vagal reflex [6].

The patient's history of CAS and PTCA was the most likely precipitating factor for CAS in this case. The impairment of endothelium-dependent vasodilation associated with atherosclerosis and hypercholesterolemia also contributes to this condition. However, the precise mechanism underlying the occurrence of CAS remains unclear [7]. The vascular endothelium is the primary target of cardiovascular risk factors, which alter endothelial function and result in endothelial dysfunction, manifesting as vasospasm, thrombosis, and atherosclerosis [8]. Balloon injury after angioplasty triggers a similar pathological cell proliferation process, manifested as restenosis in a relatively short period of time. In this case, the patient had a history of CAS and PTCA within 2 months, which increased the likelihood of recurrent coronary stenosis after PTCA. The exaggerated reparative response to arterial injury induced by balloon dilation results in intimal hyperplasia, which constitutes the principal mechanism underlying restenosis [9]. The restenosis process is most pronounced within the initial three to four months following balloon dilation. In a cohort of patients subjected to serial vascular imaging studies, the

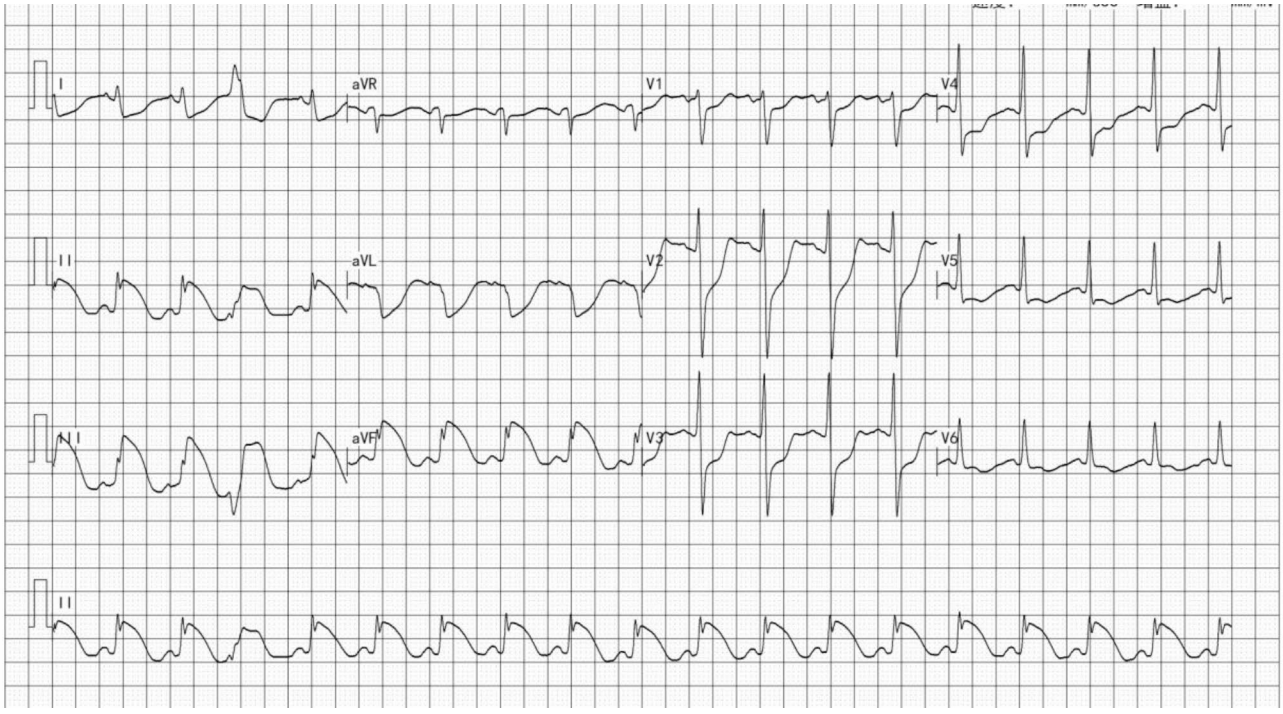

Fig. 5 ECG taken 2 h postoperation

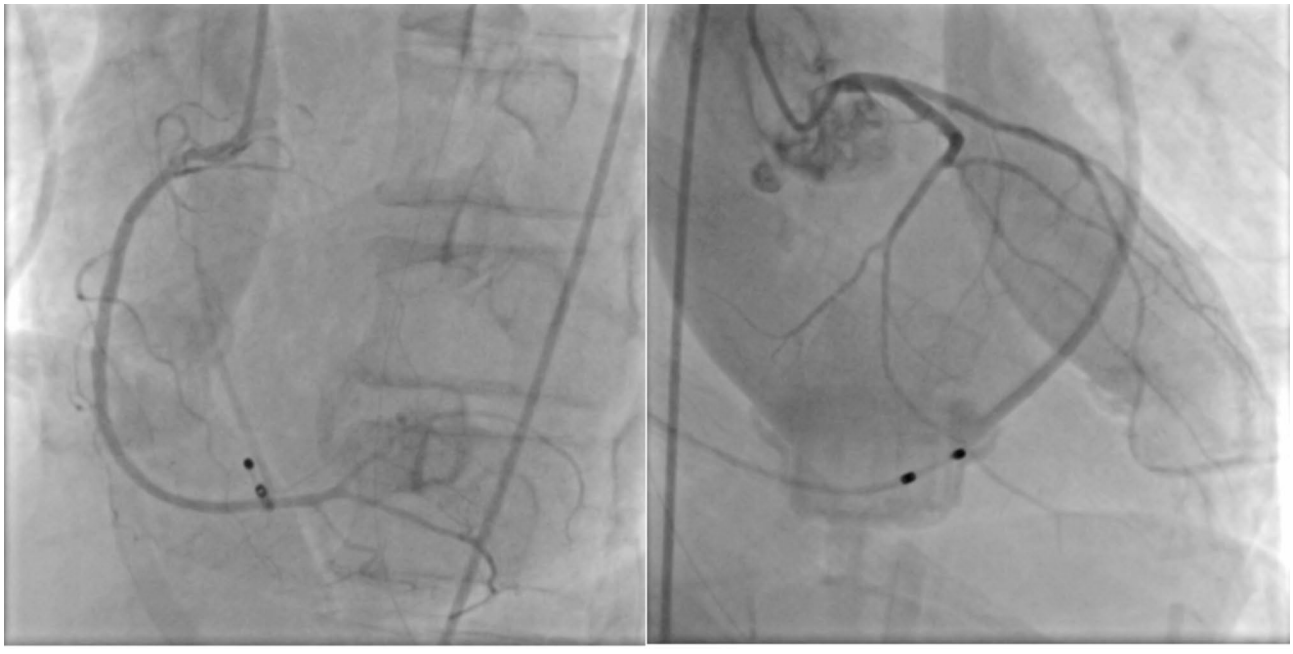

Fig. 6 Postoperative coronary angiography image

incidence of restenosis at 1, 3, 6, and 12 months was 12%, 43%, 49%, and 52%, respectively [10]. The probability of experiencing adverse cardiac events during the perioperative phase of non-cardiac surgery following PTCA is double that observed in the normal control group [11]. In determining the optimal timing for elective noncardiac surgery post-PTCA and conducting preoperative risk assessments, it is recommended that such surgeries be deferred until at least 14 days following balloon angioplasty [12]. The patient underwent a moderate-risk surgical procedure, with a major adverse cardiovascular event (MACE) score ranging from 1 to 5%. The patient's preoperative cardiac risk was assessed as moderate, with an activity tolerance of ≥4 METs and a preoperative

hemoglobin concentration of 12.8 g/dL. Consequently, no additional invasive cardiac evaluations were deemed necessary. The etiology of CAS during the perioperative period is multifaceted. Potential general mechanisms contributing to the occurrence of CAS in these patients include the redistribution of blood flow, alterations in humoral factors, heightened catecholamine responses secondary to anesthesia, and an imbalance between vasoconstriction and vasodilation. Various specific factors are thought to contribute to the occurrence of spasms during the perioperative period. These include elevated blood pH, heightened alpha-adrenergic activity, stimulation of the parasympathetic nervous system, physical manipulation of coronary arteries, and the release of vasoconstrictors by platelets [13].

This case offers significant insights into the pathophysiology of cardiac arrest induced by surgical procedures in the region densely innervated by the anal vagus nerve at the terminal part of the rectum [14], This phenomenon may be associated with the anal cardiac and vagus nerve reflexes. Almquist et al. [15] reported two instances of cardiac arrest attributed to intraoperative neuromonitoring (IONM) stimulation of the vagus nerve, which occurred in patients undergoing thyroid and parathyroid surgeries. The case of sudden cardiac arrest in one of these patients warrants particular attention. It is imperative to consider the potential catastrophic outcomes of vagal nerve excitation during surgeries at specific anatomical sites, especially in patients with a history of acute coronary syndrome.

Abbreviations

AMI	Acute myocardial infarction
CAS	Coronary artery spasm
CASS	Coronary artery spasm syndrome
ICU	Intensive care unit
IOMN	Intraoperative neuromonitoring
MACE	Major adverse cardiovascular event
PLA	Posterolateral artery
PTCA	Percutaneous coronary angioplasty

Author contributions

YML contributed to writing and revising the manuscript. JMW and JLL contributed to performing of anesthesia. JYW contributed to collection of data. CL contributed to revising the manuscript. All authors read and approved the final manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Written informed consent was obtained from the patients for publication of these Case reports and any accompanying images. A copy of the written consents is available for review by the Editor of this journal.

Competing interests

The authors declare no competing interests.

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