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Recurrent Nocturnal Chest Pain in an Elderly Patient: A Case Report of Variant Angina

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ABSTRACT

Background: Variant angina, also referred to as Prinzmetal angina, is typified by intermittent episodes of coronary artery spasm that produce discomfort in the chest at rest and raise the ST-segment on the ECG. Diagnosing this condition may be challenging because it develops suddenly and has transient clinical signs. **Case Presentation:** A 75-year-old man arrived complaining of five weeks of night-time, largely restful, episodes of severe chest pain. A normal physical examination and a slight rise in troponin were the results of the initial testing. Following coronary angiography, a drug eluting balloon was inserted into the LAD to treat mid-LAD stenosis. Despite receiving initial treatment, the patient continued to experience chest pain; an ECG showed a transient elevation of the ST segment; angiography later revealed severe RCA stenosis and LAD restenosis. Drug-eluting stents were implanted in both arteries, and the use of GTN significantly lessened discomfort. Echocardiography revealed normal cardiac function at the time of discharge. **Conclusion:** The need of taking Variant Angina into account in patients with transient ST segment alterations and nocturnal chest discomfort is emphasized by this example. Patient quality of life can be enhanced and good results can result from appropriate diagnosis and treatment with nitrates and calcium channel blockers.

INTRODUCTION

Eminent American cardiologist Myron Prinzmetal was the first to describe a special kind of angina marked by spasms of the coronary arteries that restrict or block them[1]. The most frequent types of angina pectoris, which are usually brought on by fixed stenosis and atherosclerosis, are very different from this disorder, sometimes referred to as Variant Angina or Prinzmetal Angina[2]. At rest, sometimes in the early morning, prinzmetal angina is characterized by brief episodes of coronary artery spasm that can result in severe myocardial ischemia. When Prinzmetal Angina manifests clinically, patients may have excruciating chest pain along with related symptoms including palpitations, syncope, and, occasionally, arrhythmias[3]. Because these episodes cannot be associated with physical activity and might happen randomly, they can be very difficult to identify[4]. Because coronary artery spasms are temporary, common diagnostic procedures like stress tests or angiography might not always be able to detect the dynamic changes taking place in the coronary arteries[5, 6].

Good management and treatment of Prinzmetal Angina depend on an understanding of its pathophysiology. The underlying process is hyperactivity of the coronary arteries, which causes spasm and subsequently lowers blood flow to the myocardium. The aetiology of these spasms has been linked to endothelium dysfunction, abnormalities of the autonomic nervous system, and inflammatory processes[7-9]. There are important therapeutic ramifications for admitting Prinzmetal Angina as a separate clinical entity. Instead of responding mainly to anti-anginal drugs like nitrates and beta-blockers, Prinzmetal Angina frequently needs the usage of calcium channel blockers to successfully prevent and manage the coronary artery spasms[10]. Sometimes managing symptoms and preventing recurrence also calls for lifestyle changes and long-acting nitrates[11].

Prinzmetal Angina is still a quite uncommon and underdiagnosed illness even with the progress in our knowledge of coronary artery disease. Through the presentation of a patient with typical symptoms who

responded well to focused therapy, this case report seeks to clarify the diagnostic difficulties and clinical subtleties of Prinzmetal Angina. By looking at this instance, we want to increase understanding of this unusual and fascinating type of angina among the larger medical community.

CASE PRESENTATION

Three periods of central, non-pleuritic chest pain at repose, each lasting fifteen minutes and rousing him from sleep, brought the 75-year-old man to the Emergency Department (ED). He had experienced comparable chest pain occurrences during the previous five weeks, mostly at night. Shortness of breath, nausea, and perspiration were reported along with the severe, rating 8/10, weight of the pain. In 2023, the patient's medical history included prostate cancer with undetectable PSA values, polycythemia, chronic kidney disease stage 3 (CKD-3) and hypertension (HTN). He could walk five miles a day, lived with his family, and was self-sufficient in everyday tasks. He refuted the table 1 description of his drinking and smoking.

Table 1

Patient History and Clinical Presentation

Parameter	Details
Age	75 years
Gender	Male
Presenting Symptoms	Central non-pleuritic chest pain at rest
Duration of Pain Episodes	15 minutes
Frequency	3 episodes, woke from sleep
Intensity	8/10
Associated Symptoms	Shortness of breath, nausea, sweating
Past Medical History	Hypertension, Polycythemia, CKD-3, Prostate Carcinoma (undetectable PSA 2023)
Social History	Lives with family, independent, no smoking or alcohol use

The patient had normal findings on inspection. Urea electrolytes and a chest X-ray were normal. Troponin levels at first were 30ng/l (Table 2). A coronary angiography, considering the risk and type of his discomfort, showed mild stenosis of the right coronary artery (RCA)

and severe stenosis of the mid-left anterior descending artery (LAD). Once a drug-eluting balloon was placed in the LAD, the patient was released.

Two weeks later, the patient had another episode of chest pain at night that was similar to the ones he had before and that was cured by glyceryl trinitrate spray. Widespread ST rise in the anterior and inferior leads was seen on an ECG in the emergency department. Troponin levels were first 17 ng/L and then 18 ng/L (Table 2). His history and most recent percutaneous coronary intervention (PCI) led to another angiography that revealed significant RCA stenosis and LAD restenosis. After receiving two drug-eluting stents implanted in the RCA and LAD, the patient was brought to the CCU.

Table 2

Laboratory and Diagnostic Findings

Test/Procedure	Result
Initial Troponin	30 ng/L
Subsequent Troponin (First Episode)	17 ng/L, 18 ng/L
Troponin (CCU Episode)	16 ng/L
Chest X-ray	Normal
Urea and Electrolytes	Normal
ECG (First Presentation)	Widespread ST elevation in anterior and inferior leads
ECG (CCU Episode)	ST elevation in anterior leads
Echocardiogram	Normal study, EF > 55%, no valvular abnormalities, no wall motion abnormalities

He had another episode of chest pain while in the CCU; the ECG showed ST elevation in the anterior leads, which was corrected with GTN and thereafter resolved. Troponin levels were only 16ng/L once more. As table 3 summarizes, an echocardiography performed at discharge revealed a normal study with left ventricular function (ejection fraction >55%), no valve abnormalities, and no wall motion abnormalities.

Table 3

Interventions and Outcomes

Intervention	Details
Coronary Angiogram (First Episode)	Moderate RCA stenosis, severe mid LAD stenosis, drug eluting balloon in LAD
PCI (Second Episode)	Restenosis of LAD, severe RCA stenosis, 2 drug-eluting stents in LAD and RCA
Medication	GTN spray
Outcome	Relief of chest pain, resolution of ST elevation

DISCUSSION

This patient exhibits the classic symptoms of Variant Angina, also known as Prinzmetal Angina: short elevation of the ST-segment on the ECG, no rise in troponin levels, and intermittent chest pain during rest. The symptoms and clinical results of the 75-year-old man, who had a complex medical history including polycythemia, stage 3 chronic renal diseases, hypertension, and prostate cancer, show how challenging it may be to diagnose and treat variant angina.

Variant angina, a kind of angina pectoris, is caused by transient myocardial ischemia resulting from coronary artery spasms. In contrast to typical angina, which is typically triggered by physical exertion and linked to atherosclerotic plaque, variant angina occurs during rest and typically worsens at night[12]. Chest pain and ST-segment elevation are brought on by the spasms' momentary reduction in blood flow, which passes after the spasm is over. In this case, the patient experienced waking episodes of chest pain that occurred primarily at night, as is common with Variant Angina. This diagnosis is further supported by the associated symptoms of nausea, sweating, and dyspnea in addition to the severe pain. Diagnosis of variant angina might be challenging due to its transient symptoms and potentially variable clinical outcomes[13]. In this case, there was a slight elevation in troponin (30 ng/L) during the patient's initial episode, but no other noteworthy physical abnormalities were

observed. Following a coronary angiography that revealed significant stenosis in the mid-LAD and mild stenosis in the RCA, the first intervention was performed using a drug-eluting balloon in the LAD. However, additional investigation was necessary due to the persistence of the symptoms and the episodes that followed[14].

During episodes, the ECG displayed widespread ST elevation in the anterior and inferior leads; GTN therapy brought this back to normal. This transient ST elevation along with little troponin increase (17 ng/L and 18 ng/L) matches the typical presentation of Variant Angina, in which the myocardial ischemia is brief and not extensive enough to produce severe cardiac damage, as would be seen in a myocardial infarction (MI). Preventing further episodes and reducing coronary artery spasms are the goals of treating variant angina. This patient's ST segment elevation and chest pain were relieved by GTN spray[15]. This rapid response to nitrates distinguishes Variant Angina from other forms of angina and MI, in which ST segment abnormalities persist even after nitrate delivery[16, 17].

Following a second coronary angiography that revealed severe RCA stenosis and LAD restenosis, two drug-eluting stents were placed. The decision to proceed with further angiography and stenting was supported by the angiographic results and the recurrent nature of the symptoms, even after the initial intervention[18-20]. This highlights how crucial a thorough and dynamic approach to treating patients with recurrent chest discomfort is, particularly in the context of Variant Angina. The patient's discharge echocardiogram showed normal left ventricular function and no valve anomalies, indicating that the repeated ischemia episodes had not resulted in any long-term myocardial damage. This positive outcome further demonstrates the efficacy of the therapy procedures, which included PCI with drug-eluting stents and GTN.

Limitations and Future Directions

This case report's limitations include the single patient viewpoint and the absence of long-term follow-up data, despite the fact that it offers insightful information on the diagnosis and treatment of Variant Angina. Subsequent research endeavors may concentrate on the enduring consequences of individuals suffering with Variant Angina, specifically those with intricate medical backgrounds and recurring symptoms. Furthermore, additional investigation into the pathophysiological mechanisms causing coronary artery spasms may result in more focused and efficient therapies.

CONCLUSION

This case emphasizes the need of taking Variant Angina into account when patients have intermittent ECG abnormalities and persistent chest discomfort at night. The unique characteristics of this disease are highlighted by the patient's episodes of chest discomfort, which are controlled by nitrates and marked by little or no troponin increase. Stable cardiac function and symptom alleviation with reverting ECG changes were obtained with GTN spray and drug-eluting stents. Patient outcomes can be improved and repeated symptoms of variant angina can be avoided by identifying and treating it properly.

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