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A Case of Acute Myocardial Infarction with Atypical Symptoms in a Middle-Aged Male

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Abstract

Objective: To investigate the clinical characteristics and key points of emergency diagnosis and treatment of acute myocardial infarction with atypical onset symptoms, so as to reduce the rates of misdiagnosis and missed diagnosis.

Methods: A retrospective analysis was performed on the diagnosis and treatment process of a middle-aged male patient with acute ST-segment elevation myocardial infarction who presented mainly with subxiphoid pain.

Results: A 55-year-old male patient was admitted with 3 hours of subxiphoid colic and a burning sensation after alcohol consumption, without typical chest pain. Emergency electrocardiography showed Q-wave formation and ST-segment elevation in inferior wall leads (II, III, aVF), consistent with acute inferior ST-segment elevation myocardial infarction. Coronary angiography revealed acute complete occlusion of the proximal right coronary artery and diffuse stenosis in the proximal and middle segments of the left anterior descending artery (maximum approximately 80%). Emergency percutaneous coronary intervention was performed, with one stent implanted in the right coronary artery. After the operation, the patient received standardized treatment, including lipid regulation, antiplatelet therapy, anticoagulation, blood glucose control, and gastric protection. The patient was discharged with an improved condition.

Conclusion: Acute myocardial infarction with abdominal pain as the main manifestation is highly prone to misdiagnosis. Routine electrocardiography and dynamic monitoring of myocardial necrosis markers in the emergency department are crucial for early diagnosis and can significantly improve patient prognosis.

Keywords

Acute Myocardial Infarction, Middle-Aged Male, Atypical Symptoms, Abdominal Pain, Emergency Diagnosis and Treatment

Introduction

Acute myocardial infarction is a common critical clinical emergency characterized by rapid onset, rapid progression, and high mortality, which seriously

threatens public health and life safety [1]. Typical acute myocardial infarction is dominated by retrosternal crushing chest pain and is relatively easy to diagnose based on medical history, electrocardiography, and myocardial necrosis marker tests. However, about 20%–25% of patients in clinical practice present with atypical chest pain, manifesting as abdominal pain, toothache, neck and shoulder pain, dyspnea, and other extracardiac symptoms as the initial or main manifestation, which is defined as acute myocardial infarction with atypical symptoms (including painless myocardial infarction) [2].

Such patients are mostly middle-aged and elderly, and the incidence in young and middle-aged individuals has increased significantly in recent years. They often have underlying diseases such as hypertension, type 2 diabetes mellitus, and hyperlipidemia, or risk factors including smoking, excessive alcohol consumption, a high-fat and high-sugar diet, an irregular daily routine, and high mental stress. Atypical symptoms easily lead to delayed diagnosis and treatment, misdiagnosis, and missed diagnosis, resulting in a significantly higher mortality rate than those with typical chest pain [3].

This paper reports the diagnosis and treatment of a middle-aged male patient with atypical acute myocardial infarction initially presenting with abdominal pain, summarizes clinical experience, and provides a reference for emergency diagnosis and treatment.

Clinical Data

A 55-year-old male patient was admitted to the emergency department of our hospital in the early morning of November 4, 2024, due to abdominal pain for 3 hours. Three hours before admission, the patient developed abdominal pain without an obvious inducement after drinking alcohol, mainly presenting as subxiphoid colic with paroxysmal attacks and heartburn. There was no nausea, vomiting, acid regurgitation, belching, chest tightness, chest pain, dyspnea, diarrhea, frequent urination, urgency, or dysuria.

The patient was previously healthy, with no history of hypertension or diabetes mellitus, but had a long history of smoking and drinking, as well as a habitual high-fat and high-sugar diet.

Physical examination on admission: body temperature 36.6°C, pulse 77 beats/min, respiration 20 breaths/min, blood pressure 114/80 mmHg, and blood oxygen saturation 98%. The patient was conscious and responsive. Breath sounds were clear in both lungs, without dry or moist rales. The heart border was not enlarged; the heart rate was 77 beats/min with a regular rhythm, and no pathological murmurs were heard in any valve auscultation area. The abdomen was flat and soft, with no tenderness, rebound tenderness, or muscular rigidity. Muscle strength of the extremities was grade V, and no edema was observed in both lower extremities.

The first emergency electrocardiogram (ECG) at 01:38 on November 4, 2024, showed sinus rhythm, frequent atrial premature beats, Q-wave formation, and ST-segment elevation with upward convexity in inferior wall leads (II, III, aVF), suggesting acute inferior ST-segment elevation myocardial infarction. See **Fig-1**.

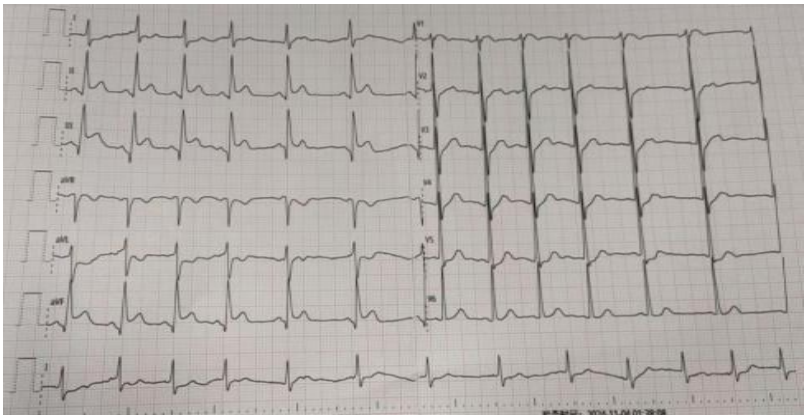

Fig-1: Electrocardiogram on Admission

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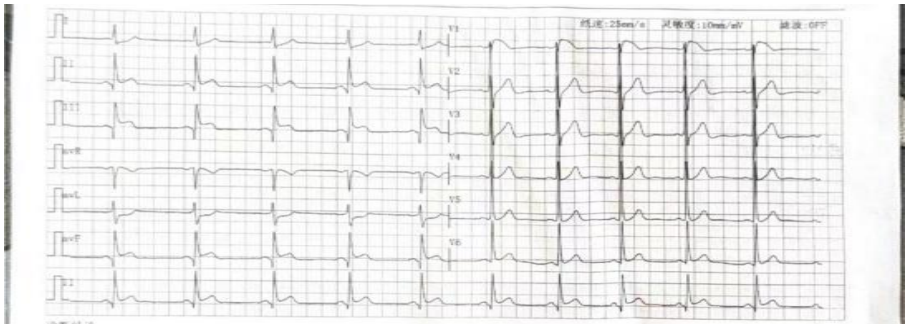

Fig-2: Electrocardiogram before Operation

Consultation with the Department of Cardiology was arranged immediately, and the patient was transferred to the resuscitation room. Loading doses of aspirin 300 mg, clopidogrel 300 mg, and atorvastatin 20 mg were administered as chewable tablets immediately, followed by an intravenous bolus injection of low-molecular-weight heparin sodium 3000 IU. Bedside and laboratory myocardial marker tests were performed simultaneously, and repeated ECG findings continued to support the diagnosis of acute inferior myocardial infarction. See **Fig-2**.

After full communication with the patient and his family regarding the condition, the patient was transferred directly to the catheterization laboratory for emergency percutaneous coronary intervention (PCI), bypassing the coronary care unit (CCU).

Coronary Angiography Results:

No obvious abnormality in the left main trunk;

Diffuse atherosclerotic lesions in the proximal and middle segments of the left anterior descending artery, with maximum stenosis of approximately 80%;

No significant stenosis in the left circumflex artery;

Acute complete occlusion in the proximal segment of the right coronary artery, with distal antegrade flow of TIMI grade 0.

The patient was diagnosed with coronary atherosclerotic heart disease and acute occlusion of the right coronary artery. One Boston Scientific stent (3.0 mm × 32 mm) was implanted at the occlusive lesion of the right coronary artery. See **Fig-3** to **Fig-7**.

After the operation, the patient was transferred to the CCU for continuous monitoring and treatment. Repeated electrocardiography showed persistent pathological Q waves in leads II, III, and aVF, with significant regression of ST-segment elevation compared with preoperation, accompanied by dynamic evolution of T waves. See **Fig-8**.

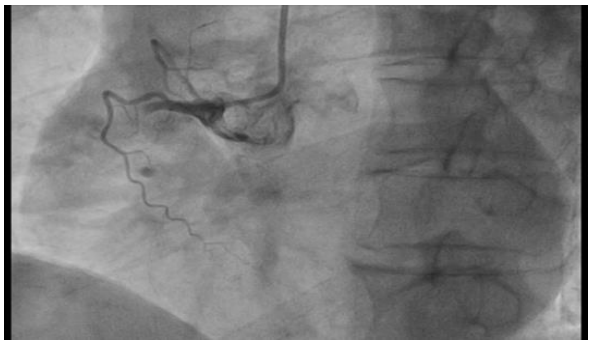

Fig-3: Coronary Angiography before Intervention

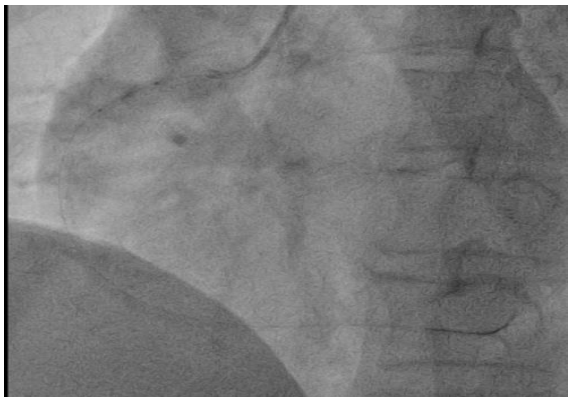

Fig-4: Guidewire Passing through the Coronary Occlusive Lesion

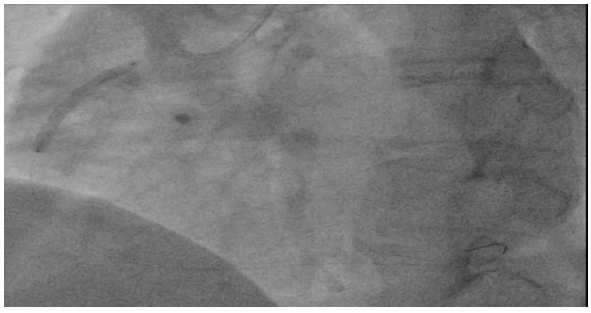

Fig-5: Balloon Angioplasty and Stent Implantation

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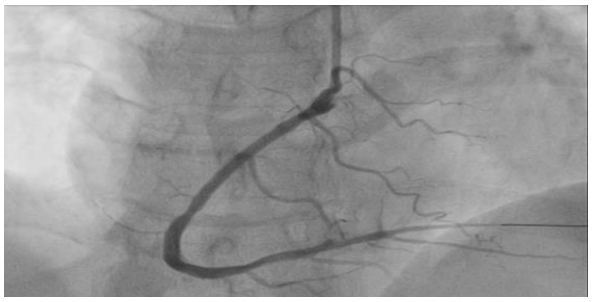

Fig-6: Coronary Angiography after Stent Implantation

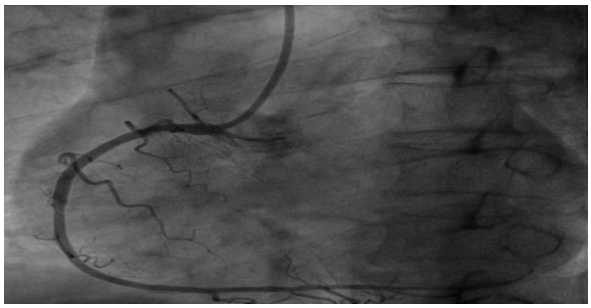

Fig-7: Coronary Angiography after Operation

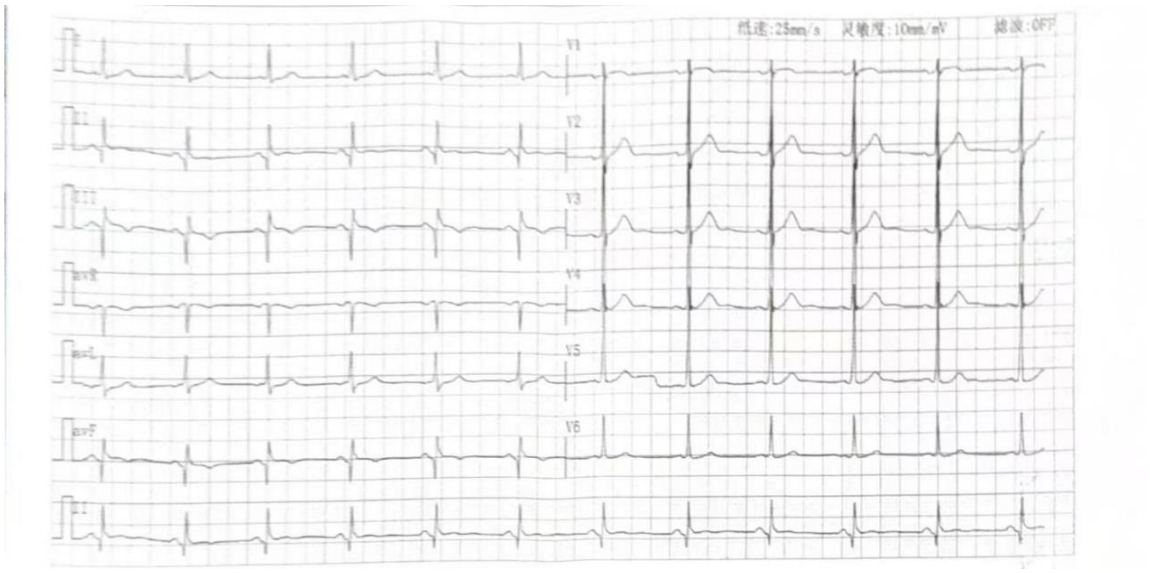

Fig-8: Electrocardiogram on the Day of Operation

Postoperative Laboratory Examinations:

Initial bedside emergency myocardial markers were negative. Subsequent reexamination revealed creatine kinase isoenzyme MB 19.90 ng/mL, myoglobin 417.00 ng/mL, N-terminal pro-B-type natriuretic peptide 45 pg/mL, and cardiac troponin T 129.0 ng/L. Emergency fasting blood glucose was 19.71 mmol/L, and triglyceride was 6.6 mmol/L.

Bedside Echocardiography on November 5, 2024:

All cardiac chambers were normal in size. The interventricular septum was slightly thickened, myocardial motion amplitude of the inferior left ventricular wall was decreased, and motion of other ventricular walls was coordinated. No obvious abnormalities were found in valvular morphology and structure, and no pericardial effusion was detected. Left ventricular ejection fraction (LVEF) was 57%. Other related examinations showed no obvious abnormalities in electrolytes, thyroid function, liver and renal function, and routine stool test. Low-density lipoprotein cholesterol was 2.11 mmol/L, and procalcitonin was 0.18 ng/mL. Hepatitis B serological markers indicated recovery from a previous infection. Glycated hemoglobin was 10.5%. Postoperative peak myocardial enzymes increased significantly: creatine kinase isoenzyme MB >300.00 ng/mL, and cardiac troponin T >10000.0 ng/L.

Endocrinology consultation was performed. Combined with blood glucose and glycated hemoglobin results, type 2 diabetes mellitus was confirmed. The patient received daily subcutaneous injection of insulin glargine for hypoglycemic treatment. During hospitalization, standardized secondary prevention treatment for coronary heart disease was administered continuously, including statin lipid regulation and plaque stabilization, dual antiplatelet therapy, anticoagulation, myocardial nutrition, strict blood glucose control, and gastric protection.

Repeated Coronary Angiography on November 8, 2024:

20% stenosis at the distal left main trunk; 50% stenosis in the middle segment of the left anterior descending artery, with a myocardial bridge in the middle and distal segments presenting 50% systolic compression. No abnormalities in the left circumflex artery. Blood flow in the right coronary artery stent was unobstructed, without residual stenosis. Stenosis of the left anterior descending artery was alleviated compared with the acute phase, complicated with coronary spasm. No interventional treatment was required temporarily, and conservative medical treatment was continued.

The patient's clinical symptoms were completely relieved, without chest tightness, chest pain, or abdominal discomfort, and vital signs remained stable. He was discharged on November 11, 2024. The patient took medications regularly after discharge and followed up regularly in the cardiology outpatient department, with no recurrence of chest tightness, chest pain, abdominal pain, or other discomforts.

Discussion

A 55-year-old middle-aged male presented solely with subxiphoid colic and a burning sensation after drinking alcohol, without typical myocardial infarction symptoms such as chest distress, chest pain, profuse sweating, or dyspnea. The condition was highly susceptible to misdiagnosis as digestive system diseases, including acute gastritis, gastroesophageal reflux disease, and alcoholic gastric mucosal injury [1,4]. If clinicians made a diagnosis based only on abdominal symptoms without routine electrocardiography, missed diagnosis and misdiagnosis could easily occur, delaying reperfusion therapy and inducing severe adverse cardiovascular events such as malignant arrhythmia, cardiogenic shock, and sudden cardiac death.

Combined with domestic and international literature, the clinical characteristics, misdiagnosis mechanisms, and emergency diagnosis and treatment strategies of atypical abdominal pain-type myocardial infarction are analyzed as follows.

Clinical Characteristics and Misdiagnosis Risk Factors of Abdominal Pain Myocardial Infarction:

Acute myocardial infarction presenting with abdominal symptoms as the initial manifestation is the most common atypical myocardial infarction, mostly inferior wall myocardial infarction [2,5]. The inferior ventricular wall is closely adjacent to the diaphragm, and lesion stimulation easily causes referred abdominal pain with vague localization, which is easily confused with acute abdomen. Clinical data show that the initial emergency misdiagnosis rate of such myocardial infarction reaches up to 57.18%, often misdiagnosed as acute gastritis, cholecystitis, gallstones, acute pancreatitis, and other digestive diseases, and short-term mortality increases significantly after misdiagnosis [6].

Misdiagnosis results from three aspects: patients, disease characteristics, and clinicians.

- **Patient factors:** Middle-aged patients had no previous hypertension or diabetes and considered themselves healthy. Abdominal discomfort induced by alcohol was subjectively judged as gastrointestinal disease. They refused cardiovascular examinations and had poor treatment compliance, interfering with clinical judgment.
- **Disease characteristics:** There was no typical chest pain throughout the course, only upper abdominal pain, acid regurgitation, and heartburn. Vital signs were stable, and abdominal physical examination showed no definite positive signs, lacking specific diagnostic clues.
- **Clinical diagnosis and treatment factors:** Emergency physicians had limited inertial thinking, prioritized common digestive diseases, and lacked vigilance regarding myocardial infarction risk in post-alcohol upper abdominal pain. They had insufficient ability to identify abnormal electrocardiographic changes such as atypical ST-segment changes and atrial premature beats, and failed to implement routine ECG screening for middle-aged patients with acute abdomen.

Pathophysiological Mechanism of Abdominal Pain in Inferior Wall Myocardial Infarction:

Abdominal pain in abdominal pain-type acute myocardial infarction is caused by multiple pathophysiological pathways rather than a single mechanism. Cardiac sympathetic afferent fibers and

visceral afferent fibers of upper abdominal organs converge on the same spinal neurons at T7-T9 spinal segments and transmit signals to the central nervous system through the same ascending pathway. After myocardial ischemia and necrosis, the central nervous system cannot accurately distinguish the source of pain signals, misidentifying cardiac ischemia as abdominal pain, and typical referred pain occurs [7].

The posterior inferior ventricular wall is rich in vagal afferent receptors. Myocardial ischemia and hypoxia directly activate the vagus nerve, inducing gastrointestinal circulation disturbance and smooth muscle spasm via the vagovagal reflex, which is the core mechanism of abdominal symptoms in inferior wall myocardial infarction. Meanwhile, the inferior wall is adjacent to the diaphragm. Severe ischemia and hypoxia cause inflammatory stimulation and diaphragmatic reflex spasm, presenting as abdominal muscle tension, traction pain, or hiccups similar to digestive symptoms.

Impaired cardiac pump function and reduced cardiac output lead to systemic hypoperfusion, hepatic congestion, increased hepatic capsule tension, and gastrointestinal congestion, aggravating gastrointestinal smooth muscle spasm and severe abdominal pain similar to acute abdomen. In some patients, hepatic congestion progresses rapidly while pulmonary congestion develops slowly, so heart failure symptoms are concealed by severe abdominal pain. Severe mesenteric hypoperfusion can induce intestinal spasm and pseudo-peritonitis signs, further complicating diagnosis. Severe stress may also cause stress ulcers and gastrointestinal bleeding, leading to early misdiagnosis as primary digestive diseases [8].

Standardized Emergency Screening and Treatment Strategies:

Combined with treatment experience and domestic and international guidelines, an integrated emergency process of screening, identification, and treatment should be established for atypical abdominal pain-type myocardial infarction [9]. Routine ECG screening should be performed for all patients ≥40 years old with unexplained upper abdominal pain or subxiphoid discomfort, regardless of chest pain or digestive incentives, to reduce missed diagnosis.

High-risk stratification should be carried out simultaneously. Young and middle-aged males with long-term smoking, excessive drinking, a high-fat diet, and irregular schedules are high-risk groups. Atypical myocardial infarction should be prioritized for differential diagnosis in those with two or more risk factors.

Dynamic multi-index monitoring is required for highly suspected patients. A normal single ECG and myocardial markers cannot exclude hyperacute myocardial infarction. ECG and myocardial enzymes should be rechecked every 30-60 minutes to capture dynamic ischemic changes. Clinicians should pay attention to the mismatch between severe symptoms and mild abdominal signs. When symptomatic treatment for digestive diseases is ineffective and accompanied by palpitations, fatigue, or blood pressure fluctuation, cardiogenic abdominal pain should be highly suspected.

Once STEMI is confirmed, the emergency reperfusion green channel should be activated. Patients should be transferred directly to the catheterization laboratory, bypassing the CCU to minimize door-to-balloon time, reopen culprit vessels early, and improve long-term prognosis [10].

Clinical Implications of Young and Middle-Aged Myocardial Infarction:

The incidence of acute myocardial infarction in young and middle-aged people has continued to rise in recent years. Individuals without traditional hypertension and diabetes are no longer considered low cardiovascular risk populations [11]. Long-term smoking, excessive drinking, a high-fat diet, irregular lifestyle, and chronic stress are major risk factors. Such patients often present with atypical symptoms and have a higher risk of sudden death.

Clinicians should abandon the misconception that myocardial infarction occurs only in the elderly. Emergency physicians should strictly implement routine ECG screening for acute abdomen and avoid interference from patients' self-diagnosis. Public cardiovascular health education should be promoted to achieve early screening and intervention, reducing

mortality and disability caused by atypical myocardial infarction [12-14].

Conclusion

In conclusion, atypical inferior wall myocardial infarction with isolated abdominal pain has strong concealment and an extremely high risk of misdiagnosis. Emergency departments should strengthen screening of high-risk populations, implement routine ECG examination, dynamically combine ECG and myocardial marker changes, achieve early identification and rapid revascularization, and significantly improve patient prognosis.

Conflict of Interest

The authors have read and approved the final version of the manuscript. The authors have no conflicts of interest to declare.

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