

Report of two cases of atrial premature morphology changes suggesting acute myocardial infarctionGuojie Yang^{1,2}, Chenkai Zhu², Dongbo Li¹, Guodong Li¹, Peng Qin¹, Zihan Wei¹¹Department of Geriatrics Cardiology, The First Affiliated Hospital of Zhengzhou University, Zhengzhou 450052, China²Institute of Clinical Medicine, The First Affiliated Hospital of Zhengzhou University, Zhengzhou 450052, China
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Abstract: Two Chinese patients with chest pain were admitted to the First Affiliated Hospital of Zhengzhou University with increased serum cardiac enzymes and troponins levels (cardiac troponin T, cardiac troponin I). Normal QRS complex with normal ST segment in sinus beats presented in their Electrocardiogram. Also, wide and deep QS waves and elevated ST segments were found in atrial premature beats (APBs) and a series of dynamic changes were recorded over time, just as the changes during acute myocardial infarction (AMI). As far as we know, this is the first report about the relationship between the diagnosis of AMI and the changes of QRS complex and ST segment morphology in APBs.

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Introduction

Acute myocardial infarction (AMI), also known as acute myocardial ischemic necrosis, is caused by sharply reduced blood flow or complete occlusion of the coronary artery [1]. The typical clinical and laboratory findings include severe chest pain, increased serum cardiac enzymes or troponins, arrhythmia, cardiogenic shock and heart failure (sometimes even sudden cardiac death), and typical ECG changes.

Electrocardiography is an important tool in the early assessment of patients with suspected acute myocardial infarction (AMI) [2]. The appearance of deep, wide Q waves and ST segment elevation have classically been the marker of AMI [3]. Atypical presentations are not uncommon, however, and the electrocardiogram (ECG) is normal or nearly normal in some patients with AMI, approximately 40% of AMI patients display atypical ECG changes or normal [4].

The significance of ventricular premature beats for diagnosing AMI has been previously discussed [5-7]. Changes of the QRS complex and ST segment during APBs from patients with AMI, however, have not been reported. Two patients with substernal pain were admitted to our hospital. Their ECG showed wide Q waves and elevated ST segments in APBs. Their data are presented as following.

Data**1. Clinical data****Case 1**

A 68-year-old man with a history of hypertension and 40-year smoking was admitted to the First Affiliated Hospital of Zhengzhou University because of a 4-hour precordial crushing pain, accompanied by chest tightness, shortness of breath and sweating. Physical examination: blood pressure 150/90 mmHg; few wet rales at the bottom of both lungs were heard; heart rate was 78 beats per minute (bpm); no valve murmur was heard; jugular vein was invisible.

Case 2

A 65 years old man was admitted due to crushing substernal pain for five hours, with admission due to crushing pain at the precordium for five hours, with a history of hypertension for 15 years, diabetes for 8 years, 30-year smoking and 40-year drinking. Physical examination: body temperature 37.5 °C, blood pressure 156/86 mmHg; a small amount of wet rales heard at bilateral lungs bottom; heart rate 90 beats per minute; no cardiac murmurs or jugular vein distension.

Both cases presented increased serum CK, LDH, CK-MB, and troponin T. And the cardiac enzymes and Troponin T resumed normal concentrations about 10 days later (table 1, 2).

Table 1 Changes of CK, LDH, CK-MB, and troponin T in Case 1

Case 1	CK	LDH	CK-MB	troponin T
on admission	209 U/L	848U/L	39U/L	2.33ng/ml
Two weeks later	98U/L	302U/L	12U/L	0.51ng/ml

Table 2 Changes of CK, LDH, CK-MB, and troponin T in Case 2

Case 2	CK	LDH	CK-MB	troponin T
on admission	182 U/L	300U/L	37U/L	0.80ng/ml
Two weeks later	55U/L	126U/L	20U/L	0.15ng/ml

2. ECG changes

Case 1

In ECG A, the first 2 QRS complexes were normal, and the third P wave was fused with the previous T wave. The QRS complex and ST segment of the third beat is different from those of the sinus rhythm. The QRS complex was RS type in sinus rhythm whereas appeared as QS in APBs. Meanwhile the ST segment elevated as many as 4 mm in APBs (Figures 1, 2, 3).

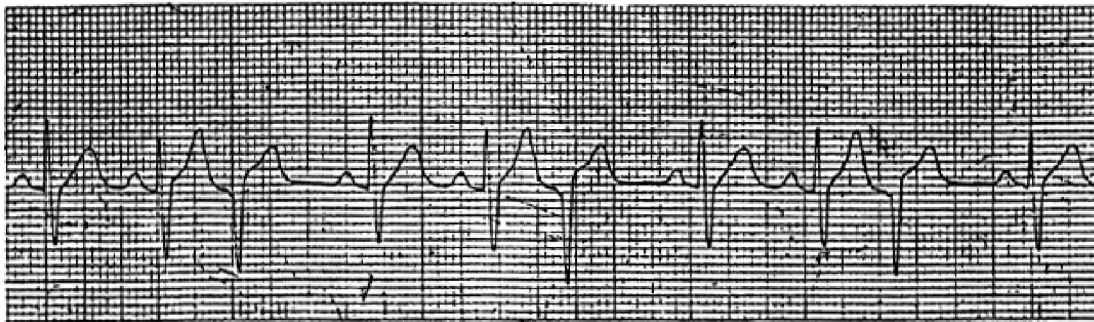


Figure 1. Electrocardiogram (ECG) on admission. The third and sixth QRS complexes were atrial premature beats (APBs). P waves were fused with the previous T waves. QRS complex of APBs were QS type, with approximately 4 mm of ST-segment elevation.



Figure 2. ECG at 2 weeks after admission. QRS complex of APBs remained the QS-type. ST segment has almost returned to baseline (the third QRS).

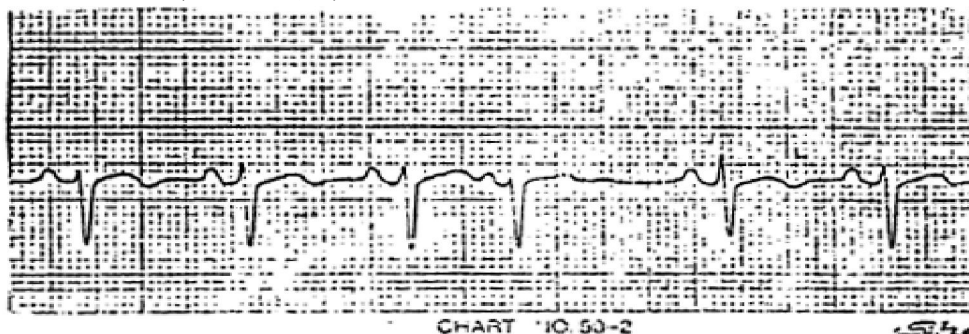


Figure 3. ECG 3 weeks later. The rS-type of QRS was recorded during APBs. The initial QRS vector was the same as sinus excitation. These changes were same to the typical QRS complex and ST segment dynamic changes during AMI. They were also consistent with the clinical symptoms and fluctuation of the cardiac enzymes.

The elevated ST segment in APBs returned to baseline 2 weeks later.

Case 2

There were normal QRS-T complexes in sinus rhythm in leads I, II and QS-type QRS complexes in leads II, III, aVF and V1-V6. The ST segment elevated up to 0.3mV in leads V3-V6. A R-type QRS complex and a 0.1mV depression of the segment in leads I and aVL were presented, which were typical changes in AMI (Figure 4).

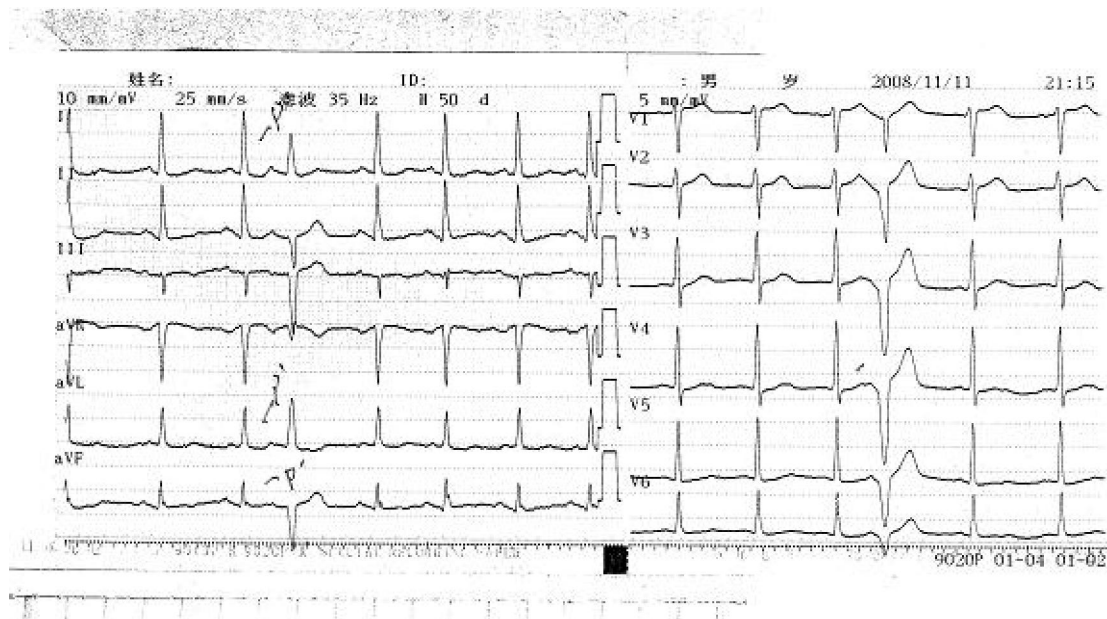


Figure 4. ECG of the 2nd patient. The Fourth QRS complex in APBs. The P wave was fused with the former T wave. QS-type QRS complexes were recorded on leads II, III, aVF, and V1–V6. The ST segment was elevated up to 0.3mV on leads V3–V6. An R-type QRS complex and a 0.1mV depression of the ST segment were found on leads I and aVL.

Discussion

Although the QRS complex in sinus rhythm was normal in both patients, we were still able to diagnose AMI on the basis of clinical findings and the elevated levels of cardiac enzymes and troponin T. The changes of the QRS and ST segment in atrial premature beats demonstrated a series of typical changes just as there in AMI. This appears to be the first report in the literature.

The underlying mechanisms of this phenomenon are unknown. As early as in 1961, after conducting animal experiments and clinical studies on infarction and ventricular premature beats, Bisteni et al.^[8] noted that the diagnosis of AMI should be considered if the RS-type QRS complex along with any abnormal conduction was found on the leads displaying ventricular premature changes, regardless of their supraventricular or ventricular origin. They thought that the infarction could be diagnosed from the ectopic beats—and not from the sinus beats—because of the asynchronous ventricular activation and the vector change. Our two cases were different from those in Bisteni's report in that our patients displayed no left or right bundle branch blocks (LBBB or RBBB).

We supposed that the morphological changes in APBs might be related to the ventricular volume or the myocardial blood supply. According to the Brody effect^[9], the amplitude of the R wave was

reduced if the left ventricular end-diastolic volume (LVEDV) decreased and was strengthened if the LVEDV increased.

Daniels^[10] measured the LV volume (EDV and ESV) and ejection fraction of patients with supraventricular tachycardia induced by rapid atrial pacing. They discovered that both EDV and ESV were reduced with an increased heart rate. The R amplitude on a surface ECG was also reduced. During APBs the diastolic filling was less, further reducing the coronary blood supply, thereby aggravating myocardial ischemia and injury, leading to impaired depolarizing function. The integrated depolarizing vector pointed to the infarct area, depicting a pathological Q wave on the surface ECG. Therefore, the QRS variation in APBs was correlated with the undermined diastolic filling. These results indicate that variation of the R-R interval is associated with the shape of the QRS complex, and this variation might be more remarkable in the presence of myocardial ischemia.

Conclusion

The wide, deep Q wave and elevated ST segment during the atrial premature beat may be sensitive indicators of acute myocardial infarction, even though the sinus beats are normal. The mechanism, however, is not fully understood.

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