left atrium thrombus. We considered the acute extensive anterior myocardial infarction had nothing to do with coronary atherosclerosis, so we did not use anti-platelet and hypolipidemic drugs, only gave anticoagulant therapy with warfarin when leaving hospital. The echocardiography 3 months later showed that the thrombosis in left atrium was disappeared. The patient was performed replacement of mitral valve successfully in cardiac surgery in December 2011. Now she felt well and keep taking warfarin all the time.

Methods The most of myocardial infarctions are caused by broken of unstable plaque in coronary artery and some are related with the serious spasm of coronary artery. The rest are caused by some rare reasons such as coronary embolism, injury, deformity or inflammation.

Results The patient had not yet entered menopause and had on traditional risk factors of coronary heart disease such as high blood-fat, blood-pressure, diabetes, smoking, fatness, hyperhomocysteinemia and premature cardiovascular disease family history. In the whole process of the disease there were typical ischaemic chest pain and the dynamic evolution of ECG and myocardial enzyme which were in accord with the change of the myocardial infarction.

Conclusions The patient was diagnosed as acute extensive anterior myocardial infarction finally. Considered the coronary angiography of the patient was normal, we summarised that the myocardial infarction was likely caused by break off of the thrombus which brought embolism of left anterior descending arterial and disappeared after intravenous thrombolysis.

GW23-e2180 **ONE CASE REPORT OF MYOCARDIAL INFARCTION INDUCED BY LEFT ATRIAL MURAL THROMBUS SHEDDING IN RHEUMATIC HEART DISEASE**

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Objectives I. Clinical data

The patient was a 49-year-old female who presented with complaints of recurrent chest discomfort and serious jaw pain for 7 days. The ECG of the patient made in local hospital showed that the ST-segments were camponotus-like elevation in V2-V6 leads. Then, she was diagnosed as acute anterior wall myocardial infarction and given intravenous thrombolytic therapy with urokinase. The discomfort of chest pain and chest tightness was disappeared 1 h later. The ECG and myocardial enzyme were consistent with the dynamic changes of acute myocardial infarction. The patient had no history of hypertension, coronary heart disease, hyperlipidaemia, diabetes and no family history of premature cardiovascular disease. In recent 3 years, the sick often felt dyspnoea when she was doing hard work. Physical examination showed BP was 126/72 mm Hg and the heart rate was 66/min with a fairly regular rhythm. ECG showed poor R wave progression and abnormal ST-T in lead V3-V6. The echocardiography showed that the sick were rheumatic heart disease with moderate mitral valve stenosis, left atrium thrombus, and weak anterior wall movement. Coronary artery angiography was normal. According to the medical history, we gave the following diagnosis: (1) Acute extensive anterior myocardial infarction (the possible reason was that the falling off of thrombus in left atrium caused anterior descending artery embolism and it became clear after a intravenous thrombolysis); (2) Rheumatic heart disease with moderate mitral valve stenosis and

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