中文題目:心電圖表現為 ST 波段上升心肌梗塞的顱內出血

英文題目:Intracranial hemorrhage presented as ST elevation myocardial infarction in EKG

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Background: ST elevation in EKG usually implies myocardial infarction. Reperfusion therapy should be arranged immediately, such as primary percutaneous intervention, with the door to balloon time less than 90 minutes; or thrombolytic therapy with r-TPA in 30 minutes from door to drug. However, the differential diagnosis of ST elevation in EKG should include coronary spasm, myocarditis or pericarditis, and direct contusion injury of heart. Occasionally, some patients with intracranial hemorrhage may have similar presentation in EKG, just mimic ST elevation myocardial infarction. We would like to present a case with intracranial hemorrhage presented as ST elevation myocardial infarction in EKG

Case report: This is a 74-year-old man with history of ESRD, hypertension, and DM was admitted due to traumatic brain injury, presented as the chief complaint of headache and dizziness after falling down since four days ago. He was brought to ER with GCS of E4M5V6, BP: 128/91 mmHg, HR: 100/min RR: 18/min T:37C, warm extremities without cyanosis or hemiplegia. Otherwise, brain CT scan during admission showed negative of intracranial hemorrhage. Under the impression of traumatic brain injury, he was admitted to general ward for further evaluation and management.

After admission, regular hemodialysis course was arranged as usual and sudden onset chest tightness and dizziness with low blood pressure was noted during hemodialysis process, complete EKG was done and showed ST elevation over lead II, III, aVF, without inverted T waves or Q waves; and hyperacute T waves over lead V3 to V5. Therefore, emergent coronary angiography was done. Surprisingly, there was no evidence of coronary occlusion in all coronary arteries. The coronary blood flow was TIMI 3 and no significant lesion was found. The cardiac enzyme was only 0.04 to 0.05 IU/L, and echocardiography showed preserved LV contractility with diastolic dysfunction and moderate to severe tricuspid regurgitation. Because of dizziness was still complained of after blood pressure became stable, we rechecked brain CT scan, which revealed bilateral frontal hemorrhage and occipital subdural hemorrhage. After conservative treatment, dizziness improved and the ST segment change also went back to baseline. He was discharged on the 20th hospital day.

Discussion: Prevalence and characterization of ECG abnormalities after intracerebral hemorrhage had been discussed in previous study. About eighty percents of patient with intracranial hemorrhage

had one or more ECG abnormalities. The most frequently observed ECG abnormality was QTc prolongation (30-40%), followed by ST-T morphologic changes (20-30%), sinus bradycardia (10-20%), and inverted T wave (10-20%). The incidence of being misdiagnosed for having myocardial ischemia was very low in published papers, because the EKG was usually ignored or tolerated. In our patient, initial brain CT scan showed no intracranial hemorrhage and new EKG change was noted during hemodialysis with typical chest pain, series of ST elevation over inferior leads was noted and coronary angiorgraphy was done, though negative for coronary occlusion was reported. Recheck brain CT showed bilateral frontal hemorrhage and occipital subdural hemorrhage, which is the real cause of ST elevation..

Conclusion: Via this case, we should learn two lessons: No ICH initially do not mean free of ICH later. And the second, coronary angiography without heparinization will be a safe method if STEMI cannot be fully excluded.