

Takotsubo Cardiomyopathy After Subarachnoid Hemorrhage: A Case Report

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Citation

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Abstract

A 36-year-old female was admitted to hospital with a cerebral hemorrhage due to ruptured aneurysm located on the posterior communicating artery. A Takotsubo syndrome was diagnosed. This rare cardiomyopathy was stressed.

INTRODUCTION

The transient left ventricular apical ballooning syndrome, also known as Takotsubo cardiomyopathy, is a recently described novel acute cardiac syndrome. The syndrome is characterized by peculiar, yet characteristic, transient regional systolic dysfunction involving the left ventricular apex and mid-ventricle with hyperkinesis of the basal left ventricular segments. Patients with Takotsubo cardiomyopathy do not have obstructive atherosclerotic coronary disease. The cause of this syndrome is unknown. We are going to present a case of subarachnoid hemorrhage complicated with a Takotsubo shaped cardiomyopathy.

CASE REPORT

A 36-year-old woman was admitted to hospital with a cerebral hemorrhage. On admission her pulse was 100 and her blood pressure was 140/90. The patient was stuporous. No seizure had been witnessed. A neurologic examination showed a "rigor nuchalis". Fine crackles were audible in the right lung base, and wheezing was audible in both lung fields. Her white blood cells count and hematocrit were increased slightly, troponin T was positive (1st hospital day). The serum level of norepinephrine and brain natriuretic peptide had increased to 3.0 ng/ml and to 967 pg/ml respectively. The ecg (fig. 1) showed a giant negative T wave in the V3-V6 leads. Radiography of the chest showed evidence of pulmonary edema. An echocardiogram revealed severe hypokinesis of the left ventricle except for the basal segment. Coronary angiography (fig 3) demonstrated normal arteries, while left ventriculography (fig 2) revealed asynergy of apical akinesis and basal hyperkinesis. A scan of the brain showed a cerebral hemorrhage and a cerebral angiography showed five cerebral aneurysms. Ruptured

aneurysm located on the posterior communicating artery was successfully treated with electrolytically detachable coils via endovascular approach. Medical treatment consisting of hydration and administration of diuretics (furosemide), angiotensin-converting enzyme inhibitor (ramipril) and beta-blocker (bisoprolol), was performed. Serum level of troponin T and norepinephrine returned progressively to the normal range. Electrocardiography obtained on pre-discharge showed improvement, however the T wave remained slightly inverted. The echocardiography obtained on day 16 showed normal wall motion in the left ventricle. The computed tomography (CT) of the brain showed that the hemorrhagic tissue had been absorbed. The patient was discharged without clinical sequelae.

Figure 1

Figure 1: Ecg (A- 1 hospital day, B- Pre discharge)

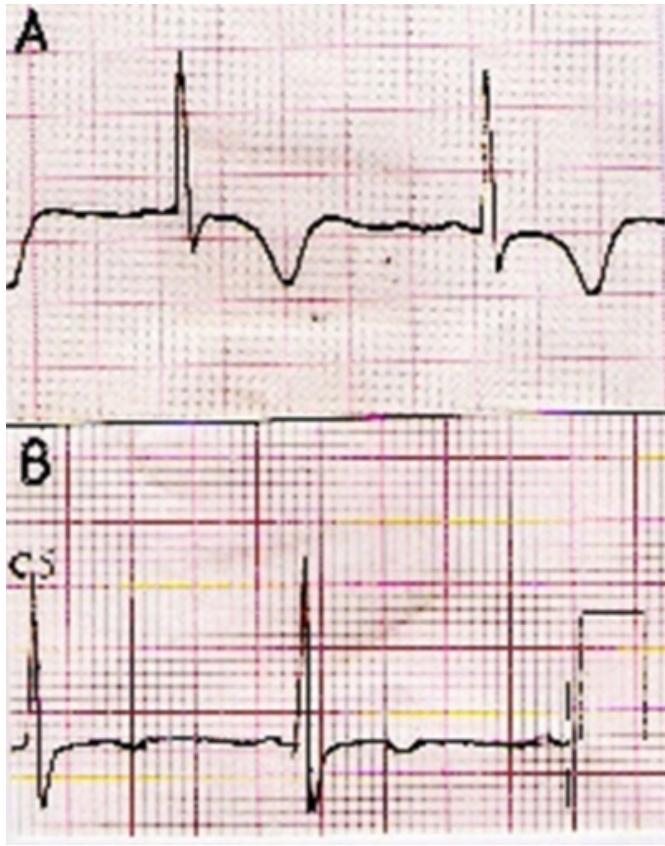


Figure 2

Figure 2: Left ventriculography

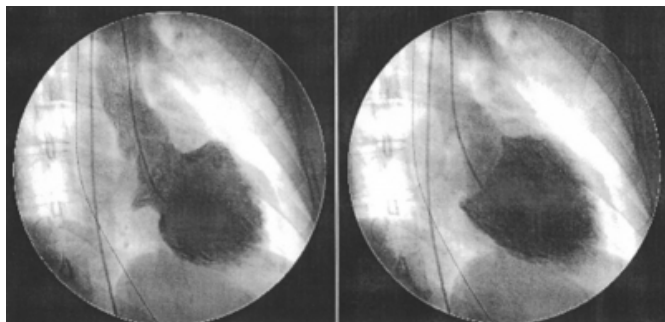
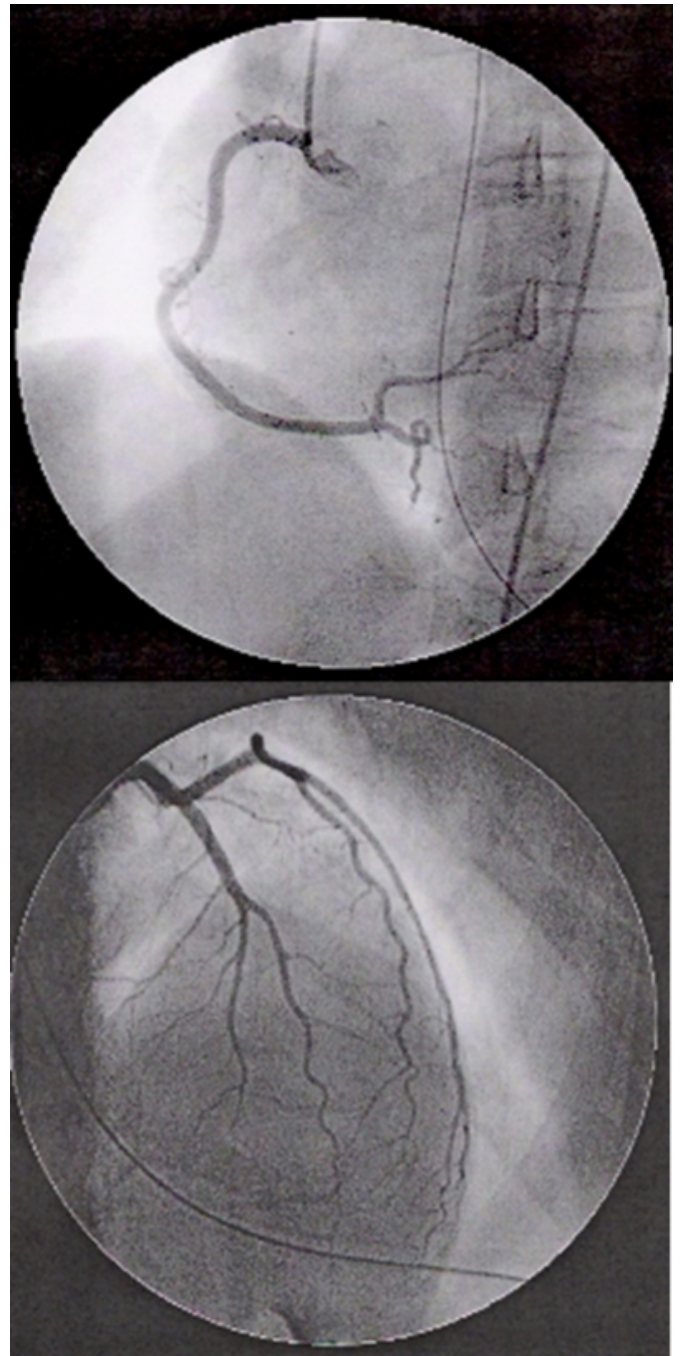


Figure 3

Figure 3: Coronary angiography



DISCUSSION

Takotsubo- shaped cardiomyopathy is a unique heart syndrome characterized by reversible left ventricular apical wall motion abnormalities mimicking acute myocardial infarction without coronary stenosis. The clinical characteristics of the syndrome include the acute onset of ischemic-like chest pain or dyspnea, transient apical and mid-ventricular regional wall motion abnormalities, minor elevation of cardiac enzyme and biomarker levels, and

electrocardiographic st-segment changes with QT interval prolongation and evolutionary T-wave inversion. The depressed left ventricular systolic function and characteristic left ventricular apical and mid-ventricular regional wall-motion abnormalities are transient features and generally resolve within days to weeks after they appearance. The overall prognosis seems to be favourable. The most common reported clinical complication is left heart failure. Isolated cases of death have been reported. Although the exact mechanism of the transient left ventricular dysfunction is unclear, myocardial damage may be mediated by norepinephrine-induced coronary vasoconstriction or by the direct toxic effect of norepinephrine. It may also represent a catecholamine-mediated myocardial stunning that result from a combination of myocardial ischemia related to diffuse microvascular dysfunction and metabolic injury or multivessel transient epicardial spasm. He should be noted that the wall-motion abnormalities in the syndrome are not typical of those generally found with subarachnoid hemorrhage in which the apex is generally spared and the basal left ventricular segments are affected. Effectively, like in our case, only isolated reports described a Takotsubo syndrome associated with subarachnoid hemorrhage. The

syndrome should be suspected in patients with characteristic left ventricular wall-motion abnormalities in the absence of obvious coronary artery disease. An appropriate approach to the syndrome seems to involve medical management with beta-blockers, angiotensin-converting enzyme inhibitors, aspirin (if there are not any contra-indications), diuretics.

CONCLUSION

Several reports of Takotsubo syndrome or transient left ventricular apical ballooning have been described, especially in Japan. We treated a case with the typical features of the syndrome after acute subarachnoid hemorrhage due to cerebral aneurysm rupture.

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