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The oldest patient with takotsubo cardiomyopathy

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Takotsubo cardiomyopathy (TTC) is a rare condition that affects mainly aging women. According to a retrospective review, patients with TTC accounted for approximately 2% of all the patients with suspected acute coronary syndrome (ACS). A few reports indicated that the average age of TTC patients was 68 years, although children or young adults may also be affected. In US and Europe, a number of contemporary TTC studies report that 90% of patients with TTC are women aged 65–70 years. Meta analysis showed that the age ranged from 10 to 89 years.[1] There was also one case study of a 90-year-old patient with TTC — the oldest patient known so far. In that case, the patient died during the course of treatment from severe multi-organ failure.[2] In the present case report, we present a 98-years old woman with TTC admitted to our clinic.

A 98-years old patient was admitted to our clinic because of significant chest pain and general weakness accompanied by hypotension that required catecholamine administration with ST-segment elevation in the anterolateral leads in ECG. The patient suffered from hypertension and third stage of chronic kidney disease. Urgent cardiac catheterization and ventriculography confirmed the absence of any critical coronary disease, but also the presence of a typical apical ballooning and midventricular hypokinesis. Troponin I (TnI) at admission was 5.555 ng/mL and creatine kinase soenzyme MB (CK-MB) mass was 14.5 ng/mL. Inflammatory parameters were not elevated, whereas N-terminal pro brain natriuretic peptide (NT-proBNP) concentration was markedly elevated, at 18,623 pg/mL. NT-proBNP/TnI ratio was 3352.48 on the first day and even higher after 24 h, at 7113.36. This markers profile is characteristic of TTC. There is a relatively small increase in creatine kinase and troponin concentrations in relation to the extent of wall motion abnormalities. BNP is always elevated in patients with TTC and is higher than in patients with ST-segment elevation myocardial infarction.

Some researchers suggest that TTC can be distinguished from ACS on the basis of the characteristic profile of cardiac markers consisting of a sudden increase in the concentration of NT-proBNP in the first few days when there is only a small increase in markers of myocardial necrosis (the ratio of NT-pro BNP/troponin).[3]

The ECG performed at admission showed ST segment elevation in leads I, aVL, V2-V6. There was no reciprocal ST-segment depression in inferior leads that could facilitate the distinction of TTC from anterior acute myocardium infarction,[4] nor were there any abnormal Q waves in anterior leads that is also more common in TTC than in anterior myocardial infarction (Figure 1).

The patient's admission transthoracic echocardiography showed a depressed left ventricular ejection fraction of 25% with akinesis and dilation of the mid- and apical left ventricle segments, as well as a diastolic dysfunction of left ventricle. There was intraventricular septal hypertrophy (1.6 cm), but without left ventricular outflow tract obstruction. We also found a severe functional mitral regurgitation and a moderate tricuspid regurgitation.

During hospital observation, we found typical to TTC changes in the ECG. After two days, there was an initial T-wave inversion, following which we observed transient improvement in T-wave inversion. After the next few days, we found a second deeper T-wave inversion, which persisted until the patient's discharge. T-wave inversion usually occurs within the first 24–72 h from the onset symptoms and is present in many leads, but most often in V2-V6 and I, II, aVL.[5] In our patient, we observed T-wave inversion after 48 h in I, aVL, V2-V6 (Figure 1). After three days, there was contractility improvement and ejection fraction (EF) was 43%. After 7 days, EF was 46%. In Holter-ECG

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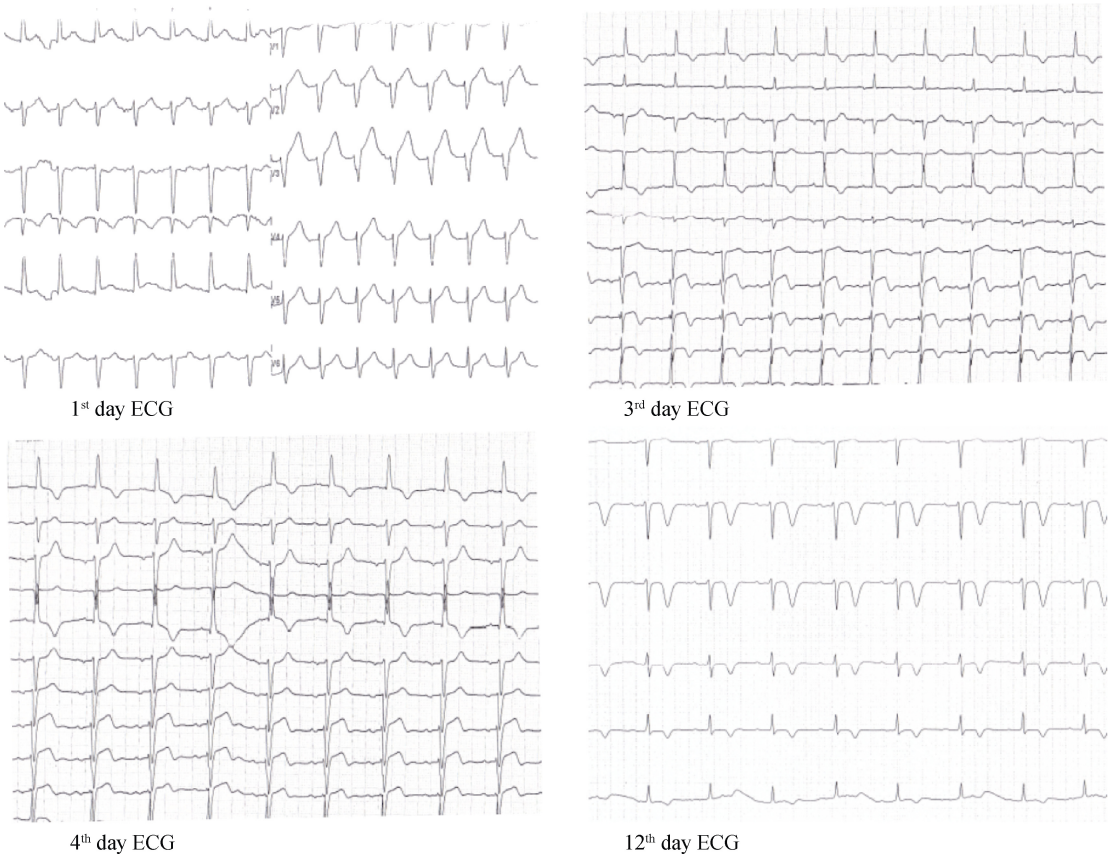

Figure 1. Changes in the ECG during hospitalization.

performed on the 7th day, we noticed numerous pauses, the longest being 6.08 s. For this reason, on the 9th day of her hospitalization, the patient had a dual chamber pacemaker implanted. A transthoracic echocardiogram performed 10 days after admission showed full recovery of the patient's left ventricular function, consistent with a diagnosis of TTC. Moreover, there was also a reduction in the degree of mitral regurgitation. The patient recovered completely under the supportive conservative and cardiological therapy.

Available literature allows us to believe that our patient is the oldest patient with TTC to date. Interestingly, the patient did not have acute stress just before the onset of symptoms. She did, however, feel chronic stress associated with her memories of World War II.

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