Open a senescent’s heart and look into his brain to find the Takotsubo trigger!

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J Geriatr Cardiol 2016; 13: 97–98. doi:10.11909/j.issn.1671-5411.2016.01.011

Keywords: ECG; Mental stress; Takotsubo cardiomyopathy

To the editor

With interest we read the case report of Budnik, et al.[1] about the oldest patient with Takotsubo cardiomyopathy (TTC) so far reported. We have the following comments and questions: TTC is frequently triggered by emotional or physical stress.[2] Were there any emotional or physical triggers for TTC in the presented patient? It is indicated that the patient felt chronic stress associated with her memories of World War II. Since the World War II ended more than 70 years ago, it would be interesting if any recent event exacerbated her memories.

TTC is frequently associated with neurological diseases, in particular central nervous system diseases such as subarachnoid bleeding, epilepsy, or stroke.[3] Central nervous system diseases more rarely associated with TTC include intra-cerebral bleeding, posterior reversible encephalopathy syndrome, or migraine. Thus it would be of interest to know if the patient had any of these disorders in her recent history. Since aneurysm formation, frequency of epilepsy and stroke rate increase with age, these neurological disorders need to be excluded as stressor for TTC. Subarachnoid bleeding may go along with minimal symptoms, as well as stroke or seizures. Patients with TTC but without an obvious acute stressful event in their recent history need to be actively investigated for acute central nervous system disease since it may be occasionally overlooked, especially if cardiac manifestations dominate the symptomatology. Additionally, neuromuscular disorders such as amyotrophic lateral sclerosis or deteriorating metabolic myopathy need to be excluded as stressors of TTC.

It is reported that the patient received catecholamines because of hypotension. This therapy is problematic in TTC since increased endogenous catecholamine levels might be responsible for TTC.[4] Furthermore, several case reports suggest that the administration of catecholamines may lead to further hemodynamic deterioration in patients with TTC by aggravating an intraventricular pressure gradient or by a paradoxical negative inotropic effect.[5,6]

It would be of interest to know the pharmacotherapy of the patient before occurrence of TTC, especially if she received beta-blocking agents. Furthermore, we are interested in her creatinine clearance at admission and at discharge and on her liver function parameters. Which imaging studies were carried out in the acute phase and during follow-up?[7]

Which pharmacotherapy did she receive during the time in the hospital? The occurrence of bradycardia with multiple pauses requiring implantation of a pacemaker is rarely reported in TTC.[8] Did follow-up ECGs during the hospital observation show a continuous decrease in her heart rate or PQ-time or did the bradycardia develop suddenly? Did she suffer from atrioventricular block or from other causes of bradycardia? Did follow-up investigations after pacemaker-implantation show that her cardiac rhythm was pacemaker-dependent or did she develop intrinsic activity?

Overall, this interesting case merits further investigations for central nervous system disease or NMD which could have triggered TTC and may have manifested with only mild or no symptoms. Patients without an obvious trigger for TTC need to be actively investigated for the most frequent physical triggers but also for acute psychological stress. Particularly senescent patients may not always open their inner life and often do not talk frankly about their emotional disturbances.

References

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Authors’ reply

We would like to thank for the very interesting comment on our case report, and fully agree that in the vast majority of patients with TTC can find the trigger. Our patient did not indicate that pain was preceded by mental stress or physical exertion. However, as she was 98 years old, her memories of the World War II returned, even several times a day. In our registry, about 10% of cases were not able to identify a specific triggering factor. In another registry it was possible only in 57% patients.[1]

If the neurological diseases are concerned, the patient did not have any problems in the anamnesis. She did not have any symptoms at admission and during hospitalization, which indicating subarachnoid bleeding, epilepsy or stroke. Additionally, there were no abnormalities in the neurological physical examination.

Because of hypotension, the patient received an infusion of dopamine in the regional hospital before transporting to our hospital. Dopamine was discontinued during coronary angiography. We would like to emphasize that intraventricular pressure gradient as well as left ventricular outflow tract obstruction was absent in this patient; and the first echocardiography exam was performed after 15 min from the admission to the cardiac intensive care unit. Fortunately, despite the risk of hemodynamic deterioration,[2] the patient was stable in further observation. The patient had not received beta-adrenolytic before occurrence of TTC because beta adrenolytic administration did not protect against TTC in predisposed patients in our observation.[3]

The patient received dopamine during transport to our clinic as we mentioned above, aspirin, clopidogrel and unfractioned heparin, were discontinued after coronary angiography. She also received diuretic, statins and low molecular weight heparin until disappearance of wall motion abnormalities. Because of the trends to bradycardia she did not receive β-adrenaline. At discharge the patient received β-adrenaline, diuretic and statins.

In the comment, there are the questions about creatinine clearance at admission and at discharge and on the liver function parameters of the patient. As we mentioned, the patient suffered from third stage of chronic kidney disease. The creatinine clearance at admission was 31 mL/min per 1.73 m² and at discharge 30 mL/min per 1.73 m². If the liver parameters are concerned aspartate aminotransferase was 51 U/L, alanine transaminase 34 U/L. The coagulation parameters were normal.

The reason to implant dual chamber pacemaker was symptomatic bradycardia with pauses because of sinus node disease. The patient did not suffer from atrioventricular block. In our opinion, bradycardia was not the complication of TTC.

References

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