A 58 year old woman was referred to our department for evaluation of chest pain. She did not smoke, have diabetes or obesity, and there was no family history of early sudden death of cardiac origin.

At the present admission, cardiac auscultation revealed no cardiac murmurs, and no signs of heart failure. Electrocardiography performed on admission [Figure 1] showed normal sinus rhythm with a P-pulmonale wave pattern, minimal QTc interval prolongation (471 msec), and 1 mm ST segment elevation in leads V3-V6. Troponin-I level was elevated (4.020 μg/L), but decreased the next day (1.920 μg/L). Total creatinine phosphokinase level was 439 IU/L (CPK-MB accounted for 16.4%) and dropped to 97 IU/L two days later.

Echocardiography, performed on day 2 of hospitalization, showed a normal-sized left ventricle, mild decrease in global contractility (despite a preserved ejection fraction of 45%), segmental apical akinesis, and ballooning [Figure 2, arrows]. Given the presence of chest pain accompanied by electrocardiographic changes and elevated troponin level, the initial tentative diagnosis was myocardial infarction.

However, the coronary cardiac topography angiogram demonstrated normal coronary arteries, and the calcium score was zero (indicating a high negative predictive value for a significant obstructive coronary artery disease in asymptomatic, but not in symptomatic patients) [1]. Furthermore, cardiac magnetic resonance imaging excluded myocarditis and older myocardial scars, but demonstrated left ventricular apical ballooning syndrome [Figure 3, arrows]. No other anatomic abnormalities or major pericardial abnormalities were observed.

On the basis of the classic findings of segmental wall motion dyskinesis and normal coronary arteries on MRI, a diagnosis of apical ballooning syndrome (Takotsubo cardiomyopathy) was made. Following supportive treatment, the patient was released on day 9 in good general condition with preserved myocardial function.

In postmenopausal woman, Takotsubo cardiomyopathy is a common cause of chest pain and ECG changes with elevated cardiac enzyme levels usually (but not always) following psychological stress.
or physiologic stress. Pathophysiology remains controversial. Unlike myocardial infarction, the prognosis is usually benign with full restoration of contractile functions [2, 3].

Corresponding author:
Dr. Y. Shoenfeld
Center for Autoimmune Diseases, Sheba Medical Center, Tel Hashomer 52621, Israel
Phone: (972-3) 530-2652
Fax: (972-3) 535-2855
email: shoenfel@post.tau.ac.il

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References

Figure 3. Steady-state free precession ("white blood") acquired in vertical long axis (two-chamber view) in end systole. Apical ballooning is demonstrated (white arrows), as compared to normal systole when the ventricle is contracted. LA = left atrium, LV = left ventricle.

Capsule

Influenza from 1918 to 2011

Analyses of historical data from the devastating influenza pandemic of 1918 have facilitated our understanding and preparations for controlling contemporary outbreaks. Fraser and colleagues accessed previously unpublished data from the fall of 1918, gathered during a household survey of over 7000 Maryland households conducted by Wade Hampton Frost, who led the U.S. Public Health Service’s investigations into the Spanish flu. Frost made a mathematical model of disease transmission, which Fraser et al. have expanded on. The most important parameter in epidemic control is transmissibility, and the new analysis shows that rates of transmission within households were actually quite low and very variable. It appears that not only were roughly a fifth of the population immune before the fall wave of infection, but also that there appeared to have been very few asymptomatic infections. These revelations show that influenza is consistently only moderately transmissible and thus always potentially controllable, provided that the measures and tools available to us now remain available.

Eitan Israeli

Capsule

How individual heart muscle cells respond to stretching

When the heart fills with blood, the muscle cells relax and stretch. To find out how individual heart muscle cells, or cardiomyocytes, respond to stretching, Prosser et al. suspended single rodent cardiomyocytes between glass rods, stretched them just a little, and monitored the release of tiny amounts of calcium through channels known as ryanodine receptors. Loading the cells with fluorescent dyes sensitive to the binding of calcium or reactive oxygen species (ROS) revealed that activation of the enzyme nicotinamide adenine dinucleotide phosphate oxidase 2 triggered ROS generation, which then led to opening of the ryanodine receptor pore. Cells from animals with muscular dystrophy showed excessive calcium release, which may contribute to impaired muscle function.

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Eitan Israeli