Acute Myocardial Infarction Induced by Alternating Exposure to Heat in a Sauna and Rapid Cooling in Cold Water

Yukiko Imai
Sachihiko Nobuoka
Junzo Nagashima
Toru Awaya
Jiro Aono
Fumihiko Miyake
Masahiro Murayma

Second Department of Internal Medicine,
St. Marianna University School of Medicine, Kawasaki City, Japan

Key Words
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Abstract
We describe a patient with acute myocardial infarction, which was thought to result from plaque rupture or thrombosis because of coronary artery spasm. The vasospasm was most likely induced by stimulation of the α-adrenergic receptors during alternating heat exposure during sauna bathing and rapid cooling during cold water bathing. This report emphasizes the dangers of rapid cooling after sauna bathing in patients with coronary risk factors.

Introduction
Several authors [1–3] have described the effects of sauna bathing on the cardiovascular system and its associated hazards. However, coronary artery spasm caused by sauna bathing has not been reported. We report a case of acute myocardial infarction induced by alternating exposure to heat during sauna bathing and rapid cooling during cold water bathing.

Case Report
A 37-year-old Japanese man tried to lose weight by sauna bathing. His height was 172 cm and his weight was 60 kg. He had no history of hypertension and his family history was unremarkable. He had smoked one and a half packs of cigarettes daily for 20 years.

On the evening of December 14, 1996, he alternated between a hot sauna bath and a cold water bath repeatedly for about 2 h. During this time, chest pain and chest discomfort occurred. The chest pain resolved after 2 h, but the chest discomfort persisted all night long. On December 16, he visited a local community hospital. His electrocardiogram showed poor R-wave progression in leads V1–V3, abnormal Q waves in lead aVL, as QS pattern in lead V4, ST-segment elevation in leads V2–V4 and negative T waves in leads I, aVL, and V2–V6 (fig. 1A). Elevated serum cardiac enzyme activities confirmed the diagnosis of acute myocardial infarction.

On December 21, the patient was admitted to our hospital. Physical examination on admission revealed a temperature of 36.3 °C, a regular pulse rate of 72/min and a blood pressure of 110/64 mm Hg. The heart sounds were normal and there were no murmurs. The lungs were clear to auscultation. Laboratory findings revealed high creatine kinase activity (206 IU/l) and increased lactate dehydrogenase activity (504 IU/l). However, glutamatic oxaloacetic transaminase and glutamic pyruvic transaminase were normal.

A repeat electrocardiogram showed a normal sinus rhythm at a rate of 60, with poor R-wave progression in leads V1–V4 (fig. 1B). A chest radiograph showed a normal cardiac silhouette without evidence of pulmonary congestion. These findings were consistent with a diagnosis of acute anteroseptal myocardial infarction.

Cardiac catheterization was performed on December 25. Coronary angiography revealed no significant stenosis of the coronary arteries, although mild stenosis was noted in the proximal portion of the left anterior descending artery (fig. 2). Left ventriculography demonstrated hypokinesis of the anteroseptal segment of the left ventricular wall.
Discussion

In the present patient, coronary angiography revealed no significant stenosis of the coronary arteries. However, mild stenosis was present in the infarct-related artery, suggesting the presence of a plaque. Several investigators [4–6] have reported that in patients with no significant coronary stenosis, coronary artery spasm plays an important role in the pathogenesis of acute myocardial infarction. In addition, coronary artery spasm most likely causes plaque rupture.

With respect to the effects of sauna bathing on the cardiovascular system, increased cardiac output because of an increased heart rate and decreased peripheral resistance because of dilation of the peripheral vascular bed have been reported [1, 2]. In addition, dehydration,
hemoconcentration, and hypotension have been reported in association with sauna bathing [2, 3]. However, coronary spasm induced by sauna bathing or exposure to heat has not been described.

It is of interest that the patient in the present case alternated between sauna bathing and rapid cooling for about 2 h. Rapid cooling following sauna bathing increases both systolic and diastolic blood pressure through sympathetic stimulation. Increases in the systolic blood pressure of more than 300 mm Hg and in the diastolic pressure of more than 200 mm Hg have been described in this setting [2]. Sudden stimulation of the sympathetic system enhances activation of the α-adrenergic receptors and induces coronary artery spasm [7]. In fact, Raizner et al. [8] reported that cold pressor testing, a sympathetic reflex stimulus, provokes coronary artery spasm, resulting in focal coronary artery spasm in patients with an atheromatous plaque at the site of spasm. In addition, Vincent et al. [9] described the occurrence of coronary spasm causing thrombosis and myocardial infarction.

We believe that the myocardial infarction was not caused by sauna bathing or exposure to heat. Rather, myocardial infarction most likely resulted from plaque rupture or thrombosis due to coronary artery spasm induced by α-adrenergic stimulation during alternating between sauna bathing and cold water bathing.

It has been reported that there were 6,175 sudden and unexpected deaths during 1 year in Finland. Of these, 102 occurred during or within 24 h of sauna bathing. In 67 of these 102 deaths in connection with sauna bathing, sudden death was caused by myocardial infarction, primarily in people with existing ischemic heart disease [2]. Romo [10] reported that there were 1,631 acute myocardial infarctions during a 16-month period in Helsinki, of which 21 occurred during or within 3 h of sauna bathing. These reports suggest that the risk of sudden cardiac death or acute myocardial infarction associated with sauna bathing is small. However, the present report demonstrates the risks of rapid cooling in cold water after sauna bathing.

References
