STUDIUM PRZYPADKU / CLINICAL VIGNETTE

Coronary spasm secondary to hypocalcaemia and hypomagnesaemia

Skurcz naczyń wieńcowych w przebiegu hipokalcemii i hipomagnezemii

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A 43-year-old man was admitted to the Endocrinology Department because of hypocalcaemia and hypomagnesaemia developed after surgical treatment of hyperparathyroidism. There was no history of coronary heart disease and hypercholesterolemia before admission, only moderate hypertension. At about 2 pm the patient experienced sudden chest pain radiating to the jaw and upper limbs. Electrocardiogram revealed temporary horizontal ST-segment elevation in II, III and aVF leads (Fig. 1). The patient was referred to the Cardiology Department and coroangiography was performed. There were neither atherosclerotic changes nor contraction of coronary arteries during angiography (Fig. 2A–C). Laboratory test made shortly after the onset of pain revealed severe ionised hypocalcaemia - 0.69 mmol/L (1.13-1.29 mmol/L) and hypomagnesaemia — 0.52 mmol/L (0.7–1.0 mmol/L). Troponin I level was within the normal range — 0.039 ng/mL (0.0-0.056 ng/mL) but a slight elevation of creatine kinase-MB mass was present — 4.6 ng/mL (0.0-3.6 ng/mL). The chest pain ceased following intravenous administration of calcium and magnesium. Two-dimensional transthoracic echocardiography showed normal left ventricular size and function with ejection fraction of 57%, mild left ventricular hypertrophy and mild mitral and tricuspid regurgitation (Fig. 3). Although coronary spasm secondary to hypocalcaemia is a very rare facet of angina, failing to consider it in differential diagnoses in all cases of variant angina might pose a grave threat to the patient's life.



Electrocardiogram during chest pain



Figure 3. Two-dimensional transthoracic echocardiography showed left ventricular size and function with ejection fraction of 57%



anterior descending arteries

Figure 2C. Right coronary artery

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and left circumflex coronary arteries