

Hiccups as a myocardial ischemia symptom

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Abstract: A hiccup is involuntary, paroxysmal inspiratory movements of the chest wall associated with diaphragm and accessory respiratory muscle contractions, with the synchronized closure of glottis. The mechanism underlying this common primitive reflex plays an important role in protecting airways against esophageal aspiration. The hiccup reflex mechanism is based on the afferent pathway (vagus and phrenic nerve and sympathetic fibers innervating chest organs, the abdomen, the ear, the nose and the throat stimulation, and the stimulation of hiccup center in the central nervous system, mainly reflecting psychogenic or metabolic disorders) and the efferent pathway (phrenic nerves). An incidental hiccup is a common problem, usually resolves spontaneously and does not present a clinical issue. The clinical issue arises in the case of pathologic persistent hiccups or symptomatic secondary hiccups which may lead to significant fatigue, insomnia or depression. Generally, pathologic hiccups are associated with considerable discomfort concerning both the "stigmatized" person and his or her personal surroundings in which it evokes different emotions, from amusement through impatience to uneasiness and the suggestion of a medical visit as an expression of concern for a given person. The most common causes of pathologic symptomatic hiccups are nervous system diseases, either the central nervous system (proliferative, angiogenic, inflammatory disorders), or the peripheral nervous system: the irritation of the phrenic nerve (proliferative disorders, goitre) and the vagus nerve (otolaryngologic diseases, meningitis, esophageal, stomach and duodenal diseases, hepatitis, pancreatitis, enteritis). The vagus nerve irritation with subsequent hiccups may be caused by chest disorders (injury, surgery) and heart diseases (myocardial infarction). In the present paper we describe the case of a 62-year-old male with recurrent hiccups associated with exertion as a secondary symptom of myocardial ischemia.

Key words: coronary disease, hiccup, symptom

INTRODUCTION

A hiccup is an involuntary, paroxysmal inspiratory movement of the chest wall associated with diaphragm and accessory respiratory muscles contraction, with the synchronous glottis closure [1]. An incidental hiccup is a common problem, usually resolves spontaneously and does not present a clinical issue. The clinical issue arises in the case of pathologic intractable hiccups or symptomatic secondary hiccups. The most common causes of pathologic symptomatic hiccups are nervous system diseases, either the central nervous system (proliferative, angiogenic, inflammatory disorders), or the peripheral nervous system: the irritation of the phrenic nerve (proliferative dis-

orders, goitre) and the vagus nerve (otolaryngologic diseases, meningitis, esophageal, stomach and duodenal diseases, hepatitis, pancreatitis, enteritis). The vagus nerve irritation with subsequent hiccups may cause chest disorders (injury, surgery) and heart diseases (myocardial infarction).

CASE REPORT

A 62-year-old male was admitted to the Surgical Ward of 109 Military Hospital in Szczecin for a medical checkup after hemorrhoid and rectal polyp surgery. In his medical history the patient reported recurrent hiccups associated with physical exertion (mild, e.g. while climbing to the first floor) and retrosternal pain induced by moderate exertion. Physical examination showed obesity (body mass index = 40 kg/m²). Results of laboratory tests were as follows: significant hyperlipidemia (total cholesterol 7.14 mmol/l, triglycerides – 3.77 mmol/l); the ECG showed no ischemia. During the hospitalization chest pain occurred and the patient was referred to the Internal Ward after

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the internal consultant opinion was obtained. The echocardiography revealed moderate left ventricle hypertrophy and abnormal left ventricle relaxation (E wave smaller than A wave in the mitral left ventricle inflow profile) without contractility disorders or valvular heart disease. The exercise test on a moving track was performed (according to the Bruce protocol, time: 4 min, pulse achieved 135/min, metabolic equivalents [METs]: 5.4). The test showed significant horizontal depression of the ST segment after the end of the first minute of the test (with heart rate of 100/min in II, III, aVF, V5-V6 leads). The depression of the ST segment became deeper during the test with maximum in the third minute: range -2.5 mm to -3.0 mm in II, III, aVF leads and -1.5 mm to -2.0 mm in V5-V6 leads. Clinical features observed during the test were: hiccups occurring at the end of the first minute of the test and typical anginal pain occurring in the third minute of the test. Stenocardial pain resolved in the first minute of rest and hiccups were present during 5 minutes after the exertion, resolving simultaneously with the ST segment normalization showed on the ECG. The patient was referred to the urgent coronary angiography. Advanced coronary artery abnormalities were shown: left main coronary artery – 40% distal stenosis; left anterior descending artery (LAD) – significant stenosis in proximal segment and occlusion on the level of I diagonal branch (significantly stenosed in the opening), distal LAD segments filled from its own collateral circulation and from the right coronary artery (RCA); circumflex branch of the left coronary artery – not stenosed; posterolateral branch – 50% stenoses in the mid-length; RCA – significantly obstructed in 1/3 proximal of the distal segment and critically obstructed in the end part. The patient was eligible for cardiac surgery but refused to undergo coronary artery bypass surgery. He underwent the percutaneous coronary interventions (PCI) procedure in another interventional cardiology center where the RCA angioplasty was performed. After the procedure, clinical improvement was observed. Chest pain occurred on major exertion (II class CCS) and the hiccups associated with exertion completely resolved. In the exercise test after the surgery (on the treadmill, according to the Bruce protocol, time: 10 min; pulse achieved 140/min; METs: 11.0), the ischemic feature was observed with horizontal depression of the ST segment since the ninth minute of the test in II, III, aVF, V5-V6 leads (1.0–1.5 mm), no stenocardial pain or hiccups were observed.

DISCUSSION

Hiccups are the pathologic effect of reflex arc activation. The center of hiccups is localized in C3–C5 of the spinal cord. The center is activated by an impulse conducted in afferent fibers of the phrenic nerve, vagus nerve and sympathetic trunk. From the heart lead two potential afferent pathways of the hiccup reflex arc: vagus nerve branches and sympathetic branches. Parasympathetic and sensory innervation of the heart is mediated by the vagus nerve: cervical segment

branches (cardiac cervical upper branches and cardiac cervical lower branches from the laryngeal recurrent nerve) and thoracic segment branches (cardiac thoracic branches with, among others, pericardial branches). Sympathetic innervation of the heart is mediated by celiac branches leading from the cervical segment of the sympathetic trunk, that is, most often, the three cardiac carotid nerves: upper, middle and lower. Cardiac carotid nerves form the cardiac plexus and constitute the main sympathetic innervation of the heart [2,3]. Pathological process of the heart or pericardium may therefore stop the reflex arc and release hiccups. The phenomenon was already observed during myocardial infarction [4–11] and as the only symptom of infarction [12]. Hiccups were also observed in the postmyocardial infarction syndrome (Dressler syndrome) [13].

In the case presented here the hiccups were the symptom of myocardial ischemia, outstripping typical stenocardial pain. It occurred together with ischemic features revealed on ECG during the exercise test and resolved together with the ischemic features withdrawal. The resolving of hiccups after interventional coronary disease PCI treatment also suggest the myocardial ischemia to be the releasing factor for the hiccup reflex arch. Interestingly, hiccups resolved after the successful PCI on the RCA despite significant obstructions in other coronary vessels, including two localizations in the proximal segment of LAD. It may be explained by the fact that the phrenic nerve, which leads motor fibers to the phrenic muscle, also leads sensory branches (30% of fibers) to the pleura, pericardium and peritoneum [14]. Moreover, it forms anastomoses through branches leading from the subclavicular loop of cervical part of the sympathetic trunk and the celiac plexus, with the major part of the fibers reaching the pericardium from the right side [2]. This point of view is confirmed by available evidence indicating that hiccups occurred much more frequently in patients with inferior myocardial infarction (and the right ventricle infarction) compared with those with infarctions of other localizations [15,16].

REFERENCES

1. Jakimowicz W. *Neurologia kliniczna w zarysie*. Warszawa, PZWL, 1981.
2. Bochenek A, Reicher M. *Anatomia człowieka*. Warszawa, PZWL, 1989.
3. Budrewicz S, Góral M, Podemski R. *Patofizjologia i leczenie czkawki*. *Przegl Lek*. 2002; 59: 924–926.
4. Reiss M, Reiss G. Singultus: indication of an atypical myocardial infarct? *MMW Fortschr Med*. 2006; 148: 55–56.
5. Cento D, Saporito F, Arrigo F. Hiccups during acute myocardial infarction. *Recenti Prog Med*. 1987; 78: 241–242.
6. Kounis NG. Persistent hiccuping in acute myocardial infarction – report of case. *Ir Med J*. 1974; 67: 644–645.
7. Douthwaite AH. Intractable hiccuping in acute myocardial infarction. *Br Med J*. 1971; 2: 709.
8. Ikram H, Orchard RT, Read SE. Intractable hiccuping in acute myocardial infarction. *Br Med J*. 1971; 2: 504.
9. Akaoglu C, Berkmen R, Yilmaz S. A rare initial symptom of myocardial infarct: hiccup. *Türk Tip Cemiy Mecm*. 1961; 27: 542–546.
10. Swan HR, Simson LH. Hiccups complicating myocardial infarction. *N Engl J Med*. 1952; 247: 726–728.
11. Abaza A, Lepage M. Prognostic and diagnostic significance of hiccup during myocardial infarction; therapeutic effect of procaine amide. *Clinique (Paris)*. 1952; 47: 105–113.

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12. Gilbert NS. Hiccups as sole presenting symptom of myocardial infarction. *J Fla Med Assoc.* 1958; 44: 960-963.
13. Jayasekara HT, Fernando HJ. The post-myocardial infarction syndrome: (Dressler's Syndrome). *Ceylon Med J.* 1963; 18: 92-98.
14. Gołąb B. Anatomia czynnościowa obwodowego układu nerwowego. Lublin, Wydawnictwo Czelej, 1998.
15. Culić V, Mirić D, Eterović D. Correlation between symptomatology and site of acute myocardial infarction. *Int J Cardiol.* 2001; 77: 163-168.
16. Sinha N, Ahuja RC, Saran RK, Jasin GC. Clinical correlates of acute right ventricular infarction in acute inferior myocardial infarction. *Int J Cardiol.* 1989; 24: 55-61.