INTRODUCTION
Penetrating aortic ulcer (PAU) is ulceration of an aortic atherosclerotic plaque penetrating through the internal lamina into the media. PAU is a rare condition and occurs in 2% – 7% of acute aortic syndromes (AAS); however, the actual incidence is unknown because of asymptomatic patients. One may treat it conservatively as well as surgically. We present a case of a 54-year-old man, who was admitted to hospital due to the exaggeration of exertional chest pain and persistent headaches. During coronary angiography, the suspicion of PAU was raised. Contrast-enhanced computed tomography confirmed the diagnosis. Transesophageal echocardiography showed bicuspid aortic valve with minimal calcification, the dilated ascending aorta, large atherosclerotic plaques in the aortic arch with ulceration (thickness: 5.0 – 5.5 mm, diameter: 5 – 6 mm, depth: 3 – 4 mm), without intramural hematoma. Conservative treatment was chosen with uneventful 2-year follow-up. Although surgical management is advocated for patients with PAU type A, we demonstrated that type A PAU can be successfully treated conservatively as well.

CASE REPORT
A 56-year-old male patient with a history of arterial hypertension and tobacco-smoking was admitted to our department due to the intensification of typical exertional chest pain for a couple of weeks. On admission, he was stable and complained about persistent headaches.

Physical examination revealed elevated blood pressure (BP, 170/100 mmHg), and a systolic heart murmur in the right second intercostal space radiating towards carotid arteries. Three-months earlier, transthoracic echocardiography (TTE) showed left ventricle ejection fraction (LVEF) of 65%, left ventricular diastolic diameter (LVDd) of 55 mm, left atrium (LAD) of 47 mm. Also, the suspicion of the bicuspid aortic valve (BAV) was raised. Additionally, aortic dilatation was observed with the aortic bulb (AoB) of 44 mm, the aortic arch of 40 mm, the middle part of the aorta of 32 mm, and the dilated descending aorta (AoD).

The patient was being diagnosed in the direction of arterial hypertension and ischemic heart disease. At discharge, a patient received atorvastatin, acetylsalicylic acid, ramipril, and amlodipine. Planned coronary angiography took place three months later and showed non-obstructive coronary arteries (Fig. 1 A – B). During the procedure, the operator raised the suspicion of “something” in the aorta. Further computed tomography (CT) showed ulceration near the brachiocephalic trunk within atherosclerotic plaque (Fig. 2 A – B) consistent with diagnosis of type A PAU. It also showed the following: AoB 46 mm, AoA 45 mm, AoD 28 mm, the aortic arch 37 mm and BAV was confirmed. After treatment modification, the patient had satisfactory...
BP control and remained chest pain-free. At the Heart Team the decision about conservative treatment was made, and bisoprolol was added to the regimen.

Two months later, transesophageal echocardiography (TEE) showed BAV with minimal calcification, the dilated ascending aorta, large atherosclerotic plaques in the aortic arch with ulceration (thickness: 5.0 – 5.5mm, diameter: 5 – 6 mm, depth: 3 – 4 mm), without intramural hematoma or penetration (Fig. 2 C). Conservative treatment was continued.

After one year, TTE showed LVEF 65%, LVDd 48 mm, AoA 46 mm, BAV, AoB 45 mm, the aortic arch of 36 mm, proximal segment of AoD 29 mm. In comparison with previous examinations valves function improved. Moreover, TEE revealed: AoA as in previous examination, large plaques in AoD, in aortic arch a smaller atherosclerotic plaque with ulceration (diameter: 3 mm, depth: 2.5 mm). The CT results were similar to the previous study.

After two years, TTE results were comparable with previous examination, and CT findings showed new thrombi around previous ulceration, AoB 46 mm, AoA 43 mm, aortic arch 37 mm, and AoD 28 mm. Again, the decision about conservative treatment was made.

**DISCUSSION**

Our patient was initially diagnosed with arterial hypertension. It is difficult to say whether the pain at admission was caused by the exacerbation of the comorbid disease or PAU. Differentiation of PAU from other AAS and other cardiovascular diseases, based only on clinical symptoms, may be difficult due to non-specific symptoms; therefore, radiological diagnostics is very useful and reveals a characteristic picture. In making the diagnosis of PAU contrast-enhanced CT remains the method of choice [3]. During CT scans, it is essential to use contrast because otherwise, PAU may be mistaken for intramural hematoma (IMH) [3].

Our patient had only arterial hypertension; however, patients with PAU are usually older (>70 years). Other common comorbidities, not present in our case, are ischemic heart disease, chronic obstructive pulmonary disease, renal insufficiency, and coexisting abdominal aneurysm [3, 4].

The natural course of PAU is a matter of debate. The ulcer may remain stable or may lead to the development of saccular or fusiform aneurysms, aortic enlargement, transmural rupture, and formation of medial hematoma [3, 4]. The main goal of PAU treatment is to prevent aortic rupture and progression to acute aortic dissection. The risk of aortic rupture for symptomatic PAU may be up to 40%, even without the enlarged aortic diameter [1, 5].

European Society of Cardiology (ESC) guidelines from 2014 recommend conservative treatment with analgesic therapy and BP control is recommended in all patients with PAU [3]. The most commonly used antihypertensives are beta-blockers and calcium channel blockers. Pain control is also vital to limit catecholamine burst and subsequent tachycardia and BP increase [1]. Target values for BP and heart rate are 100-120 mmHg and 60-80 bpm, respectively, and are the same as in other AAS [6].
The further strategy depends on the type of lesion. In type A symptomatic cases, due to more frequent complications, patients should undergo urgent surgery [1, 3]. In type B cases, it is recommended to introduce conservative treatment under careful observation. Among patients with uncomplicated type B PAU, imaging surveillance is suggested. In complicated cases, patients should have interventional therapy considered. There are two therapeutic options in this case: thoracic endovascular aortic repair (TEVAR) and open surgery. However, because of elderly age and comorbidities open surgery is often not recommended. TEVAR seems to be a better option for these patients and is more preferred than surgery by ESC (class of recommendation IIa vs. IIb, respectively) [3]. TEVAR is mainly used to treat the descending aorta. Endovascular treatment of ulcers localized in the aortic arch is more difficult and complicated. However there are more and more techniques available for treating ulcers in the aortic arch, where endografts with branches and fenestrations are used [7].

Our patient had no predictors of the disease progression like refractory and recurrent pain, increase in pleural effusion and PAU diameter > 20 mm and PAU neck > 10 mm. These risk factors are useful to determine the need for interventional treatment [2, 3, 6]. Not all studies confirmed the validity of the PAU dimension criterium [1].

In our case, pharmacology treatment introduced at the beginning was not focused at PAU. We managed to achieve proper BP values and relieve pain. After treatment modification, the patient's response was satisfactory. Moreover, our patient had no negative prognostic factors, which would indicate an urgent need for surgery. By the used treatment, it was possible not only to stop the development of the disease but also to reduce the size of the ulcer by almost 50% after two years.

CONCLUSIONS
In making a diagnosis of PAU contrast-enhanced CT remains the method of choice. However, in presented case, coronary angiography with aortography appeared to be crucial to raise the suspicion of PAU. In type A PAU, patients are usually treated surgically unless intractable comorbidities are present. Although surgical management is advocated for patients with PAU type A, we demonstrated that type A PAU can be successfully treated conservatively as well.

REFERENCES

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Conflict of interest
Authors declare no conflict of interest

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