


Chest pain: is it always what it seems to be?

Ból w klatce piersiowej – czy zawsze jest tym, czym się wydaje?

Anna Kawińska-Hamala¹ , Robert Morawiec², Jarosław Drożdż², Janusz Kawiński¹,
Zbigniew Sablik², Piotr Tyślerowicz³, Jerzy Krzysztof Wranicz¹

¹Department of Electrophysiology, Clinical and Teaching Center, Central Clinical Hospital in Lodz, Łódź, Poland

²2nd Department of Cardiology, Medical University of Lodz, Łódź, Poland

³Catheterisation Laboratory, Łódź, Poland

Abstract

A 56-year-old patient after emergency acute aortic dissection (AAD) surgery (31.03.2017), after common iliac artery (CIA) vascular surgery due to acute right lower limb ischemia (March 2017), with a 70% lesion in distal left anterior descending (LAD) in angiography computed tomography (angio-CT) detected a year ago (not yet qualified for coronary angiography/single-photon emission computed tomography), hemodynamically stable, presented to hospital due to one-week history of constant, continuous chest pain radiating to the back and left shoulder and independent of physical effort, without dyspnea. Electrocardiography (ECG) in hospital at admission (and a day ago): regular sinus rhythm, respiratory variable q III, non-specific ST-T wave abnormalities in leads I, aVL up to -0.5 mm, in leads V4–V6 up to -1 mm, no evolution. Immobilization, nitroglycerin intravenous administration – only symptoms reduction. High-sensitivity troponin T and creatine kinase-myocardial bound mass negative. Aortic angio-CT scan: in comparison to the previous study (17.10.2017) without significant changes; no leakage signs within the prosthesis; further dissection of the aortic wall from the level of the descending aorta to the bifurcation and CIA. Coronary angio-CT: significant, long stenosis in proximal LAD. Cardiac surgeon consultation: no indications for intervention. Echocardiography: left ventricular ejection fraction 65%, aortic regurgitation trace, right ventricular systolic function preserved. Diagnosis: acute coronary syndrome-unstable angina (ACS-UA). Coronarography: in LAD segm 7 isolated 80% stenosis. Aortography confirmed good prosthetic effect of the ascending aorta. Simultaneously percutaneous coronary intervention (PCI)-LAD, segm 7/IDg (bifurcation) with drug-eluting stent implantation was performed. The symptoms disappeared. Atypical chest pain in a patient with aortic aneurysm, even after successful cardiac surgery, may suggest an ACS, being an indication for coronary angiography/PCI, after exclusion aortic reasons. Delayed coronarography/coronaroplasty/antiplatelet therapy in ACS reduces the chances of successful treatment. The patient had a history of aortic diseases, negative myocardial ischemia markers, pain radiating to typical for AAD location (back pain). The first step was angio-CT to exclude another AAD.

Chest pain is not always what it seems to be. After excluding the most obvious cause, it is necessary to perform differential diagnostics, because therapeutic approach appropriate for one disease or delay in proper treatment may make it difficult/impossible to treat another one.

Key words: acute aortic syndrome, acute coronary syndrome, aortic dissection

Folia Cardiologica 2020; 15, 4: 323–326

Numerous chest pain reasons are known, the acute one require urgent medical intervention. Acute aortic syndrome (AAS) is often first symptom, which require quick diagnosis, appropriate therapy to improve extremely unfavorable prognosis [1]. Acute aortic dissection (AAD) risk is approximately 5–30/million [2–4] and is major challenge: diagnostic for cardiologists and even greater therapeutic for cardiac surgeons [2]. Completely different immediate reaction requires acute coronary syndrome (ACS).

A 56-year-old patient after emergency AAD surgery (31.03.2017, ascending aorta and arch replacement, with aortic arch arteries grafting, aortic valve repair), with well-controlled hypertension, after common iliac artery (ICA) vascular surgery due to acute right lower limb ischemia (March 2017, dissection included also abdominal aorta, ICA), with a 70% lesion in distal left anterior descending coronary artery (LAD) in angiography computed tomography (angio-CT), detected a year ago (considered as insignificant, not yet qualified for coronarography/single-photon emission computed tomography), hemodynamically stable, presented in hospital due to one-week

history of constant, continuous chest pain radiating to the back and left shoulder and independent of physical effort, without dyspnea.

Electrocardiography (ECG) in hospital at admission (and a day ago): regular sinus rhythm 75/min., respiratory variable q III, correct progression r in leads V1–V6, non-specific ST-T wave abnormalities in leads I, aVL up to –0.5 mm, in leads V4–V6 up to –1 mm, no evolution. Physical examination: aortic regurgitation (AR), low mitral regurgitation murmur, without gallop; proper peripheral pulse in typical sites symmetrical. Immobilization, nitroglycerin intravenous (NTG i.v.) administration – only symptoms reduction. Hemoglobin (Hgb) 15.1 g/dL, high-sensitivity troponin T 11 ng/L, creatine kinase-myocardial bound mass 1.36 ng/mL.

Angio-CT scan was performed from the level of the ascending aorta to the CIA – in comparison to the previous study (17.10.2017) without significant changes; no leakage signs within the prosthesis; arteries protruding from the aortic arch without dissection (Figure 1); further dissection of the aortic wall from the level of the descending aorta to the bifurcation and CIA (Figures 2, 3). Coronary angio-CT: significant, long stenosis in proximal

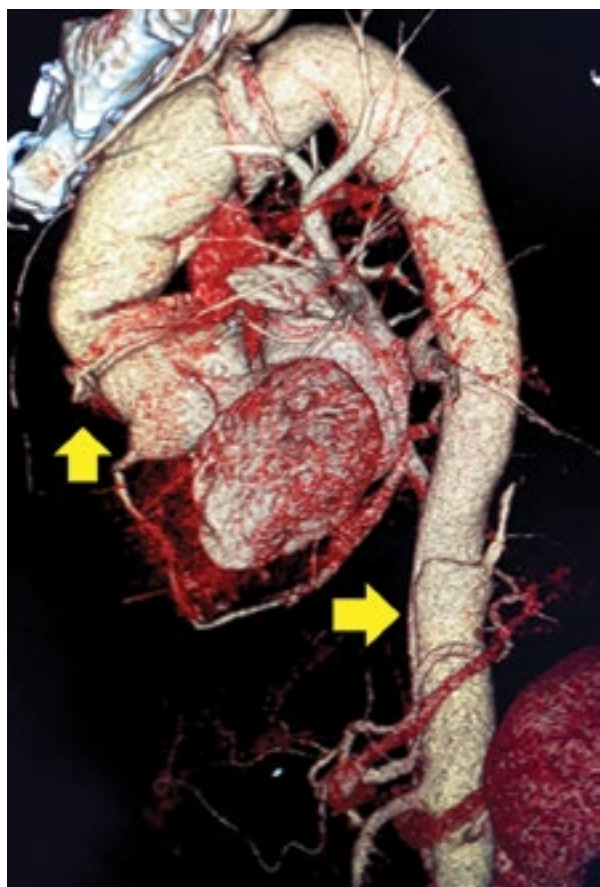


Figure 1. Rendered three-dimensional computed tomography (3D CT) scan: no leakage signs within the prosthesis

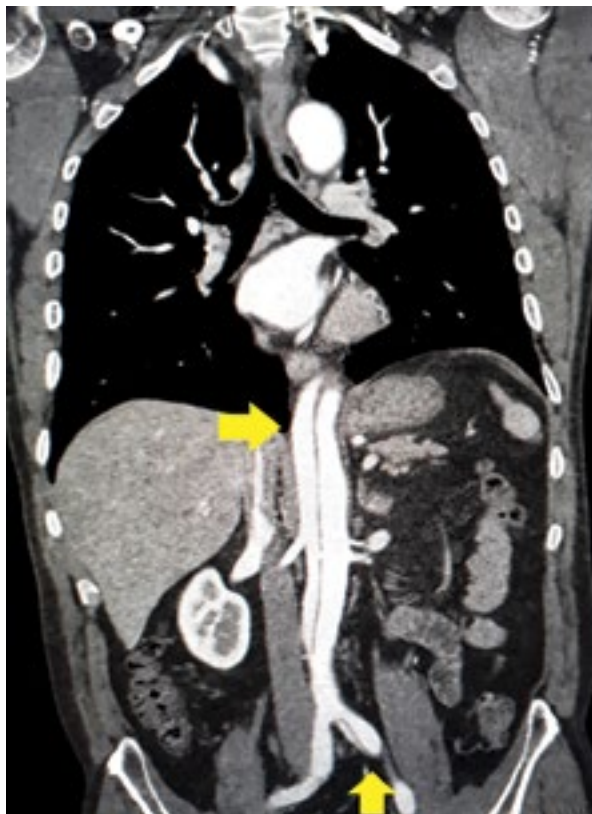


Figure 2. Computed tomography (CT) scan: dissection of the aortic wall from the level of the descending aorta to the bifurcation and common iliac artery (CIA)



Figure 3. Computed tomography (CT) scan: dissection of the aortic wall from the level of the descending aorta to the bifurcation and common iliac artery (CIA)

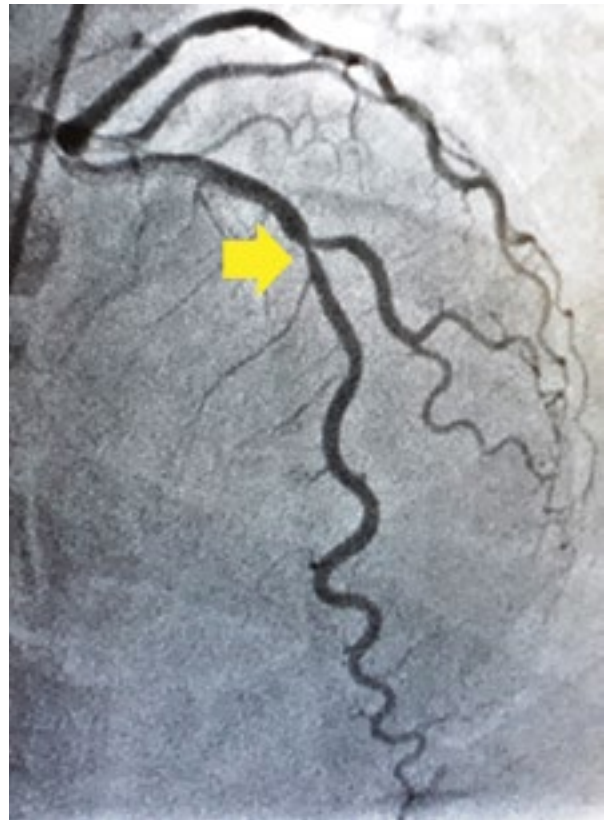


Figure 4. Coronarography: isolated 80% stenosis in descending coronary artery (LAD) segm 7

LAD. Cardiac surgeon consultation: no indications for intervention.

Echocardiography: good systolic function with left ventricular ejection fraction (LVEF) 65%, AR trace (good distant valve plastics effect, V_{max} 1.06 m/s), right ventricular systolic function preserved. Diagnosis: ACS-unstable angina (UA). Coronarography: isolated 80% stenosis in LAD, just after IDg (Figure 4). Aortography confirmed good prosthetic effect of the ascending aorta. Simultaneously percutaneous coronary intervention PCI-LAD, segm 7/IDg (bifurcation) with stent implantation (sirolimus) 3.5×26 mm (16 atm, 25 s) was performed, with the result: PCI: 80–0%, flow TIMI (Thrombolysis in Myocardial Infarction) 3. The symptoms disappeared.

Numerous patient cases with initial ACS suspicion finally verified as AAS, e.g. AAD, were described [2, 3]. Nineteen out of 820 people with suspected ACS–ST-elevation myocardial infarction (STEMI) had different diagnosis, including 3 AAD [5]. The distinction between these two diseases is crucial because anticoagulation necessary for ACS is contraindicated because it exacerbates

course of AAD [5]. No papers emphasize the importance of differential diagnostics in the opposite direction, although delayed coronarography/coronaroplasty/antiplatelet therapy in ACS reduces the chances of successful treatment. The patient had a history of aortic diseases, negative myocardial ischemia markers, pain radiating to typical for AAD location (back pain occurs in 40% in type A, 70% in type B AD [1]). The first step was CTA to exclude another AAD.

Chest pain is not always what it seems to be. After excluding the most obvious cause, it is necessary to perform differential diagnostics, because therapeutic approach appropriate for one disease or delay in proper treatment may make it difficult/impossible to treat another one. Following DeBakey: “no doctor will recognize a disease he will not think about” [4].

Conflict of interest

The authors declare no conflict of interest.

Streszczenie

Pacjent w wieku 56 lat, po pilnej operacji tętniaka rozwarstwiającego aorty (AAD) (31.03.2017 r.), po udrożnieniu tętnicy biodrowej wspólnej (CIA) z powodu ostrego niedokrwienia kończyny dolnej (marzec, 2017 r.), z 70-procentowym dystalnym zwężeniem gałęzi przedniej zstępującej (LAD) (w angiografii tomografii komputerowej [angio-CT], od roku, dotąd niekwalifikowanym do koronarografii/tomografii emisyjnej pojedynczego fotonu), wydolny krążeniowo-oddechowo, przyjęty z tygodniowym wywiadem stałego bólu w klatce piersiowej, promieniującego do okolicy międzyłopatkowej, lewego barku, niezależnego od wysiłku fizycznego, bez duszności. Kolejne elektrokardiogramy: rytm zatokowy miarowy, zmienny oddechowo q III, nieswoiste zmiany odcinka ST-T w odprowadzeniach I, aVL do $-0,5$ mm, w odprowadzeniach V4–V6 do -1 mm, bez ewolucji. Wartości troponiny T oznaczanej metodą wysokoczułą i frakcji sercowej kinazy kreatynowej w normie. Nitrogliceryna podawana dożylnie spowodowała tylko zmniejszenie dolegliwości. Angio-CT aorty bez zmian w porównaniu z wcześniejszym badaniem (z 17.10.2017 r.): proteza bez przecieku, stare rozwarstwienie od aorty zstępującej do rozwidlenia, obejmujące CIA. Angio-CT tętnic wieńcowych: istotne długie zwężenie LAD. Bez wskazań do interwencji kardiologicznej. Echokardiografia serca: frakcja wyrzutowa lewej komory 65%, dobry odległy efekt operacji naprawczej AV. Rozpoznano ostry zespół wieńcowo–dławicę piersiową (ACS-UA). Koronarografia: LAD segm. 7 długie zwężenie 80%, izolowane. Aortografia: dobry efekt protezowania aorty wstępującej. Jednocześnie przezskórna interwencja wieńcowa (PCI)-LAD: segm. 7/IDg (bifurkacja) z implantacją stentu uwalniającego lek. Dolegliwości ustąpiły. Nietypowe bóle w klatce piersiowej u chorego z tętniakiem aorty, nawet po skutecznej operacji, mogą odpowiadać ACS, stanowiąc wskazanie do koronarografii/PCI, po wykluczeniu przyczyn ze strony aorty. Opóźniona koronarografia/PCI/leczenie przeciwplatek w ACS zmniejsza szanse powodzenia leczenia. U opisanego pacjenta stwierdzono dodatni wywiad w kierunku chorób aorty, ujemne markery niedokrwienia mięśnia sercowego, ból promieniujący do typowej dla AAD lokalizacji (do pleców). Pierwszym krokiem była angio-CT w celu wykluczenia kolejnego AAD.

Ból w klatce piersiowej nie zawsze jest tym, czym się wydaje. Po wykluczeniu najbardziej oczywistej przyczyny konieczne jest przeprowadzenie diagnostyki różnicowej, ponieważ leczenie terapeutyczne odpowiednie dla jednej choroby lub opóźnienie właściwego leczenia może utrudnić/uniemożliwić leczenie innej.

Słowa kluczowe: ostry zespół aortalny, ostry zespół wieńcowy, rozwarstwienie aorty

Folia Cardiologica 2020; 15, 4: 323–326

References

1. Erbel R, Aboyans V, Boileau C, et al. [2014 ESC Guidelines on the diagnosis and treatment of aortic diseases] [Article in Polish]. *Kardiol Pol.* 2014; 72(12): 1169–1252, doi: [10.5603/KP.2014.0225](https://doi.org/10.5603/KP.2014.0225), indexed in Pubmed: [25524604](https://pubmed.ncbi.nlm.nih.gov/25524604/).
2. Kubiak G, Glanowska G, Kubiak L, et al. [Acute aortic syndrome mimicking anterior wall ST-elevation myocardial infarction] [Article in Polish]. *Kardiol Pol.* 2013; 71(8): 852–854, doi: [10.5603/KP.2013.0199](https://doi.org/10.5603/KP.2013.0199), indexed in Pubmed: [24049027](https://pubmed.ncbi.nlm.nih.gov/24049027/).
3. Hawatmeh A, Abu Arqoub A, Isbitan A, et al. A case of ascending aortic dissection mimicking acute myocardial infarction and complicated with pericardial tamponade. *Cardiovasc Diagn Ther.* 2016; 6(2): 166–171, doi: [10.21037/cdt.2015.11.06](https://doi.org/10.21037/cdt.2015.11.06), indexed in Pubmed: [27054106](https://pubmed.ncbi.nlm.nih.gov/27054106/).
4. Dixon M. Aortic dissection or MI? It could be both. *Emergency Med.* 2014; 4(4), doi: [10.4172/2165-7548.1000199](https://doi.org/10.4172/2165-7548.1000199).
5. Gu YL, Svilaas T, van der Horst ICC, et al. Conditions mimicking acute ST-segment elevation myocardial infarction in patients referred for primary percutaneous coronary intervention. *Neth Heart J.* 2008; 16(10): 325–331, doi: [10.1007/BF03086173](https://doi.org/10.1007/BF03086173), indexed in Pubmed: [18958255](https://pubmed.ncbi.nlm.nih.gov/18958255/).