WOŁOWIEC, Łukasz, ROGOWICZ, Daniel, BUJAK, Robert, CHOMA, Anna, JAŚNIAK, Albert, OSIAK, Joanna, WOŁOWIEC, Anna, GRZEŚK, Grzegorz and ZUKOW, Walery. A clinical case of a 21-year-old patient with idiopathic acute exudative pericarditis. Journal of Education, Health and Sport. 2023;15(1):67-76. eISSN 2391-8306. DOI http://dx.doi.org/10.12775/JEHS.2023.15.01.009
https://apcz.umk.pl/JEHS/article/view/42121
https://zenodo.org/record/8117869

The journal has had 40 points in Ministry of Education and Science of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of December 21, 2021. No. 32343.
Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical Culture Sciences (Field of Medical sciences and health sciences); Health Sciences (Field of Medical Sciences and Health Sciences).
Punkty Ministerialne z 2019 - aktualny rok 40 punktów. Załącznik do komunikatu Ministra Edukacji i Nauki z dnia 21 grudnia 2021 r. Lp. 32343. Posiada Unikatowy Identyfikator Czasopisma: 201159.
Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).

The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 06.05.2023. Revised: 14.05.2023. Accepted: 25.06.2023. Published: 05.07.2023.

A clinical case of a 21-year-old patient with idiopathic acute exudative pericarditis

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Abstract

Pericarditis is a disease entity associated with the primary or secondary inflammation of its layers, which may be accompanied by the accumulation of fluid in the pericardial sac, the consequences of which depend mainly on the speed of its accumulation. The causes of pericardial diseases can be divided into infectious or non-infectious diseases, but in about 30% of cases the etiology of the disease cannot be determined. Pericarditis causes 5% of all emergency department visits related to non-ischemic chest pain.

In the study we presented a case of a 21-year-old Caucasian male with acute exudative pericarditis, which required drainage of the pericardial sac despite the use of pharmacological treatment. The patient underwent a wide range of diagnostic tests and specialist consultations, but ultimately the etiological factor of acute exudative pericarditis could not have been identified.

The therapy of acute exudative pericarditis, especially in the case of undetermined etiology, requires an individualized approach from the attending physician. Based on the observation of inflammatory parameters, imaging tests and clinical picture, the physician is to decide whether pericardiocentesis or modification of pharmacological treatment is necessary. Searching for the etiological factor is essential if it has an impact on the successive methods of diagnosis and treatment, especially in patients with recurrent idiopathic pericarditis or in the absence of a response to the therapy.

Keywords: idiopathic acute exudative pericarditis.

Background

In the pericardium, a distinction is made between serous pericardium and fibrous pericardium. The serous pericardium consists of the visceral pericardium and parietal pericardium, among which the pericardium is located. The visceral pericardium of the serous pericardium, which is epicardium, is tightly fused with the outer surface of the heart. The parietal pericardium fuses with the fibrotic pericardium so that these two parts cannot be dissected. The pericardial cavity is a space containing a small amount of serous fluid (properly about 25-50 ml) that facilitates the sliding of visceral pericardium and parietal pericardium. From the outside, the fibrous pericardium surrounds the serous pericardium and directly adheres to the chest wall only in the lower part in the medium of the pericardial

triangle. In the remaining areas from the chest wall, it is separated by the pleura and the pleural cavity as well as by the tongue of the left lung [1-3].

Pericarditis is a disease entity associated with the primary or secondary inflammation process, which may be accompanied by the accumulation of fluid in the pericardial sac, the consequences of which depend mainly on the speed of its accumulation. The criteria for acute pericarditis include pericardial chest pain, pericardial friction in physical examination, new and generalized ST-segment elevation or depression of PR-segment in electrocardiogram and the presence or increase of pre-existing fluid in the pericardial sac. The presence of at least two of the above-mentioned criteria is required for diagnosis [4-6].

Pericarditis is the most common pericardial syndrome in the world [7,8]. It causes 5% of all emergency department visits related to non-ischemic chest pain [9]. Data from the Swedish registry showed that the standardized rate of acute pericarditis was 18 out of 100,000 of the total population per year[10]. A range of data on the epidemiology of pericarditis was provided by the registry by Kytö et al [11]. The standardized rate of hospitalization for acute pericarditis was 3.32 per 100,000 people per year, and acute pericarditis was the cause of 0.20% of hospital admissions for cardiovascular diseases. Higher risk of pericarditis development was observed in the case of men aged 16-65 than in the case of women in the population of hospitalised people, and the highest risk of falling ill in the general population concerned young adults. Pericarditis more often concerned the male (65.9% of patients) than the female patients (35.1% of patients), which was attributed by the authors to the higher testosterone concentration in this group of patients. In-hospital mortality in patients with acute pericarditis was 1.10% and it increased with the age of the patient as well as in the case of coincidences of severe infections [11]. Pericardial diseases, including constrictive pericarditis, may pose a challenge in differential diagnosis in patients with exertional dyspnea of unknown etiology or symptoms of right ventricular failure [12-17].

Case report

A 21-year-old, previously health Caucasian, has been admitted for suspicion of pericarditis. The patient reported that for 2 days prior to the admission to the hospital, he felt a stabbing retrosternal chest pain, which awakened the patient from sleep, intensified with deep breath, decreased after NSAIDs and after adopting a sitting posture with the trunk leaning

forward. The pain was accompanied by periodic dyspnea, a 38 ° C fever and a papular, itchy, erythematous rash near the right shoulder. About 4 weeks prior to the admission, the patient had an upper respiratory tract infection treated with cefuroxime in outpatient settings. The patient denied smoking, drinking alcohol, taking drugs or other dangerous substances. In physical examination on the admission to hospital, the patient was cardiorespiratory stable, the body temperature – 38 ° C, the skin of the right shoulder area was found to have a maculopapular rash, arterial blood pressure – 112/66 mmHg, heart rate – 110 / min. ECG recorded: sinus tachycardia – 110 / min, normal axis, depression of PR-segment and concave ST-segment elevation up to 2 mm in leads II, III, aVF, V4-V6, ST-segment depression in aVR and V1.

In laboratory tests, the features of active inflammation were observed (white blood cells 16 G / l, OB 81 mm / h, CRP 321 mg / l, negative procalcitonin), whereas renal function, ionogram, thyroid hormones, markers of myocardial damage (NT-proBNP, highly specific troponin T, CK-MB mass) were normal. Echocardiography revealed: correct size of heart cavities and wall thickness; normal left ventricular ejection fraction with EF 58%, no segmental contractility disorders and diastolic dysfunction; construction of valves in the standard; dialted and decreased respiratory motility of the inferior vena cava; separation of pericardial layers (free fluid with a small amount of fibrin) in the parasternal long-axis view: 5mm behind the posterior wall of the left ventricle, in four-chamber view in front of the right ventricle apically up to 12mm, behind the lateral wall of the left ventricle − 6mm and around the right atrium − maximum 3mm, in subcostal view up to 15mm in front of the right ventricle.

The following drugs were included: colchicine 1x0.5mg, ibuprofen 3x600mg, proton pump inhibitor 1x20mg, piperacillin with tazobactam 3 times a day at a dose of 4g + 0.5g. On the second day of hospitalization in the follow-up echocardiogram there were observed separations of pericardial layers in the parasternal long-axis view: 11mm in front of the right ventricle and 11mm behind the posterior wall of the left ventricle, in the four-chamber view in front of the right ventricle apically up to 20mm, behind the lateral of the left ventricle − 6mm and around the right atrium − maximum 5mm, in the subcostal view up to 20mm in front of the right ventricle; flattening of the free wall of the right ventricle and features of right atrial compression, respiratory variability of E-wave of the mitral inflow − 28%. Due to deterioration of the hemodynamic function of the heart, pericardiocentesis was performed by evacuating 200 ml of fluid with exudate features. The results of all microbiological

examinations of pericardial fluid (enteroviruses, tuberculosis, fungi, bacteria) were negative, and in the pathomorphological examination inflammatory cells with the predominance of neutrocytes were described. During the treatment, a two-fold increase in inflammatory parameters, relapse of fever and chest pain with a temporary increase in the amount of pericardial fluid were observed. For this reason, the dose of NSAIDs and colchicine was increased, the antifungal drug and probiotic were added, and piperacillin with tazobactam was replaced with meropenem 3x1g, afterwards meropenem was replaced with linezolid 2x600mg.

Blood and urine cultures were taken, which were negative, prior to including the antibiotic and prior to each change of it. Cerebrospinal fluid culture was negative, and the normal flora of the upper respiratory tract was found in the pharyngeal swab. In imaging tests (chest X-ray, CT of the head and paranasal sinuses without contrast, CT of the chest and abdominal cavity with contrast) no changes were found that could be the cause of acute exudative pericarditis in the patient. Hematology, neurology, rheumatology, laryngology consultations were carried out and a maxillofacial surgeon was consulted as well (2 teeth were removed due to caries). Diagnostics for tuberculosis (QuantiFeron-TB Gold test, sputum test in GeneXpert system), syphilis, borreliosis, toxoplasmosis, viral infection (HCV, HBV, HIV, CMV, EBV, adenoviruses, enteroviruses - Poliviruses, Coxsackie A and B groups, Echoviruses, Enteroviruses types 68-72), immune disorders (manual peripheral blood smear, proteinogram, concentration of IgA, IgM and IgG 1-4), systemic connective tissue disease (dsDNA antibodies, Ro, La, Rib-P, ANCA (MPO, PR3), ACA, ANA 3, antiCCP, RF IgM, complement components C3 and C4) and tumor process (imaging tests, beta 2 microglobulin, LDH, AFP, CEA) were negative.

After the treatment, clinical improvement, normalization of inflammatory parameters and reduction of pericardial fluid were obtained. Despite the extended diagnosis, the cause of acute exudative pericarditis could not be determined. After the end of the treatment observation of the patient was carried out for 3 months by telephone, during which no recurrences were observed.

Discussion

It is difficult to determine the etiology of acute pericarditis. In most cases, the etiological factor remains unknown, despite the use of invasive methods when collecting

material. Idiopathic pericarditis accounts for 78% to 86% of pericarditis cases, the majority of which are responsible for viral infections [18,19].

The causes of pericardial diseases can be divided into infectious diseases (2/3 cases: viruses and bacteria) or non-infectious diseases (1/3 of cases: systemic inflammatory diseases, cancer, and postcardiac injury syndromes). The viruses most often responsible for acute pericarditis are enteroviruses (coxsackieviruses, echoviruses), herpes viruses (Epstein-Barr virus, cytomegalovirus), adenovirus or parvovirus B19. Bacteria that cause pericarditis include mainly Mycobacterium tuberculosis (common, especially in developing countries), Coxiella burnetii, Borrelia burgdorferi, gram-positive and gram-negative cocci, as well as Chlamydia spp., Mycoplasma spp., Listeria spp., Legionella spp., Leptospira spp., Providencia stuartii [20].

Hammer et al. showed that the incidence of acute idiopathic pericarditis is seasonal. This rate was twice as high in the colder half of the year (October - March) compared to the warmer one (April - September, $p = 0.001$). The average CRP concentration in the first quarter of the year was 11.5mg / dl (mean 9), while in the remaining quarters of the year 14.1mg / dl (mean 20.3), so they were definitely lower than those observed in the presented patient [21].

The study by Levy et al. which included 204 patients with exudative pericarditis, aimed to find the etiological factor for the largest percentage of subjects by using the diagnostic algorithm developed by the authors. Despite a number of diagnostic methods, idiopathic exudative pericarditis, Levy et al. eventually determined 97 cases (47.5%). However, it should be emphasized that the patient we presented had to undergo additional imaging diagnostic tests and lumbar puncture which, if applied to the algorithm by Levy`s et al., would reduce the outlined aboved percentage with high probability [22].

In other studies conducted on large groups of patients with exudative pericarditis, the percentage of inflammations of unknown cause was in the range of 7 to 28%, which is a much smaller percentage than in the case of the absence of more fluid in the pericardial sac [23-26].

Conclusion

The therapy of acute exudative pericarditis, requires an individualized approach from the attending physician, especially in the case of undetermined etiology. The guidelines by the European Society of Cardiology from 2015 do not precisely determine the optimal duration of

anti-inflammatory therapy and doses of drugs used in the therapy of this disease. Based on the observation of inflammatory paramaters, imaging tests and clinical picture, the physician is to decide whether pericardiocentesis or modification of pharmacological treatment is necessary [27]. Persistent search for the etiological factor is important if it has an impact on the successive methods of diagnosis and treatment, especially in patients with recurrent idiopathic pericarditis or in the absence of a response to the therapy.

Author Contributions

Conceptualization, Ł.W., D.R.; methodology: Ł.W., D.R., R.B.; formal analysis Ł.W., D.R., R.B.; data curation Ł.W., D.R., R.B., A.C., A.J., J.O., A.W., G.G.; draft writing Ł.W., D.R., R.B., A.C., A.J., J.O., A.W., G.G.; writing Ł.W., D.R., R.B., A.C., A.J., J.O., A.W., G.G.; supervision Ł.W., D.R., R.B., W.Z. All authors have read and agreed to the published version of the manuscript.

Funding

This research received no external funding.

Institutional Review Board Statement

Not applicable.

Informed Consent Statement

Not applicable.

Data Availability Statement

Not applicable.

Conflicts of Interest

The authors declare no conflict of interest.

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