Kardijalna astma – (ne)opravdano zaboravljeni entitet: prikaz bolesnika

Cardiac Asthma – an (Un)justly Forgotten Entity: A Case Report

Marija Tomac Stojmenović[1]*, Ana Petretić[2,3]

[1]Županijska specijalna bolnica za psihijatriju i rehabilitaciju Insula, Rab, Hrvatska
[2]Klinički bolnički centar Rijeka, Rijeka, Hrvatska
[3]Sveučilište u Rijeci, Medicinski fakultet, Rijeka, Hrvatska

[1]Insula County Hospital, Rab, Croatia
[2]University Hospital Centre Rijeka, Rijeka, Croatia
[3]University of Rijeka, Faculty of Medicine, Rijeka, Croatia

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SAŽETAK: Kardijalna astma nastaje kao posljedica zatajivanja srca, dominatno ljevostranoga. Uzrokovana je plućnom kongestijom koja je posljedica popuštanja lijeve strane srca. U ovom ćemo radu prikazati bolesnika s dilatativnom kardiomiopatijom, otvorene etiologije, reducirane sistoličke funkcije lijeve klijetke kojemu je prva manifestacija zatajivanja srca bila u obliku kardijalne astme. Ističemo važnost anamneze i kliničkoga pregleda jer svaki zvižduk na plućima ne znači astmu ili pogoršanje kronične opstruktivne plućne bolesti.

SUMMARY: Cardiac asthma is medical condition which occurs as a consequence of heart failure, predominantly left-sided. It is caused by pulmonary congestion resulting from left heart dysfunction. Herein we will describe the case of a patient with dilatative cardiomyopathy, of unknown etiology, with reduced left ventricular systolic function, in whom the first manifestation of heart failure took the form of cardiac asthma. We emphasize the importance of the clinical examination and the patient's medical history, as every wheezing sound in the lungs is not necessarily caused by asthma or exacerbation of chronic obstructive pulmonary disease.

KLJUČNE RIJEČI: zaduha, zatajivanje srca, dilatativna kardiomiopatija.

KEYWORDS: dyspnea, heart failure, dilatative cardiomyopathy.

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***ADDRESS FOR CORRESPONDENCE**: Marija Tomac Stojmenović, Županijska specijalna bolnica Insula, Kampor 224, HR-51280 Rab, Croatia. / Phone: +385-51-776-344 / E-mail: marija.tomac.mts@gmail.com

ORCID: Marija Tomac Stojmenović, https://orcid.org/0000-0003-4257-0178 • Ana Petretić, https://orcid.org/0000-0002-5767-1206

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Uvod

Zatajivanje srca (HF, prema engl. *heart failure*) klinički je sindrom karakteriziran tipičnim simptomima (nedostatak zraka, umor, otjecanje nogu) koji mogu biti praćeni kliničkim znakovima (povišen venski tlak, inspiratorne krepitacije, periferni edemi). Taj je sindrom uzrokovan strukturnim i/ili funkcionalnim abnormalnostima srca koje za posljedicu imaju smanjeni udarni volumen i/ili povišene intrakardijalne tlakove u naporu ili u mirovanju.[1]

Kardijalna (srčana) astma nije astma kao takva. Nije uzrokovana upalom i bronhokonstrikcijom. Nastaje kao posljedica HF-a, dominatno ljevostranog. Uzrokovana je plućnom kongestijom koja je posljedica popuštanja lijeve strane srca. Kardijalna je astma klinički sindrom karakteriziran paroksizmalnom dispnejom, plućnim zvižducima i kašljem.

Španjolski slikar Francisco Goya (1746. – 1828.) još je 1820. godine naslikao „Autoportret s dr. Arrietom". Sliku je naslikao iz zahvalnosti svojem liječniku koji ga je izliječio. Slika vjerno oslikava kliničku sliku kardijalne astme. Na slici

Introduction

Heart failure (HF) is a clinical syndrome characterized by typical symptoms (shortness of breath, fatigue, leg swelling) that may be accompanied by clinical signs (elevated venous pressure, inspiratory crackles, peripheral edema). This syndrome is caused by structural and/or functional abnormalities of the heart that result in reduced stroke volume and/or increased intracardiac pressure during exertion or at rest.[1]

Cardiac asthma is not asthma in the usual sense. It is not caused by inflammation or bronchoconstriction. Instead, it occurs as a consequence of HF, predominantly left-sided heart failure. It is caused by pulmonary congestion resulting from the failure of the left side of the heart. Cardiac asthma is a clinical syndrome characterized by paroxysmal dyspnea, as well as pulmonary wheezing and coughing.

The Spanish painter Francisco Goya (1746-1828) painted his *Self-Portrait with Dr. Arrieta* as early as 1820. He created the painting out of gratitude to his doctor, who had successfully treated him. The artwork faithfully depicts the clinical presenta-

Goya sjedi, preznojen, blijed, blago cijanotičan, otvorenih usta hvata zrak, dok ga dr. Arrieta pridržava i daje mu lijek.[2] Termin kardijalne astme prvi je objavio britanski liječnik James Hope 1832. godine.[3] Opisao je razne vrste astme, kao i simptome koji razlikuju astmu uzrokovanu plućnim bolestima od one zbog bolesti srca.[4]

Sukladno GINA (engl. *Global Initiative for Asthma*) smjernicama, astma je heterogena bolest, karakterizirana kroničnom upalom i hiperreaktivnošću dišnih puteva. Simptomi poput zviždanja, kratkoće daha, stezanja u prsima i kašlja variraju tijekom vremena, pojavljuju se u različitim vremenskim intervalima i različite jačine. Jedno je od obilježja varijabilna opstrukcija dišnih puteva, koja s vremenom može postati i trajna.[5] Osnova su liječenja inhalacijski kortikosteroidi.

Definicija kronične opstruktivne plućne bolesti (KOPB) opisuje također heterogenu bolest karakteriziranu kroničnim simptomima (zaduha, kašalj, iskašljavanje sputuma) koja nastaje zbog promjena dišnih puteva (bronhitis, bronhiolitis) i/ili alveola (emfizem) koji uzrokuju trajnu, često progresivnu opstrukciju dišnih puteva. Jedan od vodećih uzroka KOPB-a jest pušenje. Osnova su liječenja bronhodilatatori.[6]

Zatajivanje srca, KOPB i astma ubrajaju se u neke od najvažnijih zdravstvenih problema modernoga svijeta. Pridružen HF nalazi se u 20 % bolesnika sa KOPB-om, dok barem 50 % bolesnika ima sistoličku disfunkciju lijeve klijetke. Obrnuto, oko 35 % bolesnika sa HF-om ima pridružen KOPB.[7]

Aktualne smjernice Europskoga kardiološkog društva[1,8] u svojoj klasifikaciji ne spominju više kardijalnu astmu, nego HF dijele na akutno i kronično. Kronični oblik HF-a dijeli se prema ejekcijskoj frakciji (EF) lijeve klijetke na:

- kronično zatajivanje srca s očuvanom EF-om (HEpEF) – LVEF ≥50 %
- kronično zatajivanje srca s blago sniženom EF-om (HFmrEF) – LVEF 41 – 49 %
- kronično zatajivanje srca sa sniženom EF-om (HFrEF) – LVEF ≤40 %.

Američke smjernice za HF iz 2022. godine[9], osim podjele prema ejekcijskoj frakciji, opisuju 4 stadija HF-a:

- stadij A – rizik od razvoja HF-a: osoba je bez simptoma, nema strukturnu bolest srca ni povišene biomarkere (npr. osobe s arterijskom hipertenzijom, metaboličkim sindromom, obiteljskom predispozicijom za bolesti srca)
- stadij B – predzatajivanje srca: bez simptoma ili znakova HF-a uz prisutnost strukturne bolesti srca, povišenih tlakova punjenja srca ili prisutnost povišenih natriuretskih peptida (NT-proBNP, BNP)
- stadij C – simptomatski HF: strukturna bolest srca uz simptome i znakove HF-a
- stadij D – uznapredovali HF: uznapredovali simptomi koji remete svakodnevne aktivnosti i uzrokuju česte hospitalizacije.

Ozbiljnost simptoma HF-a i funkcionalni kapacitet bolesnika klasificiraju se sukladno NYHA (engl. *New York Heart Association*) klasifikaciji koja težinu simptoma dijeli u četiri stupnja. Iako ima svoje slabosti, klasifikacija NYHA neovisni je prediktor smrtnosti bolesnika.

- Klasa 1. – bez ograničenja u tjelesnoj aktivnosti. Uobičajena tjelesna aktivnost ne izaziva zaduhu, umor ili palpitacije.

tion of cardiac asthma. In the painting, Goya is seated, sweating, pale, slightly cyanotic, and gasping for air with his mouth open, while Dr. Arrieta supports him and administers medication.[2] The term cardiac asthma was first employed by British physician James Hope in 1832.[3] He described various types of asthma as well as the symptoms that differentiate asthma caused by lung diseases from asthma due to heart disease.[4]

According to GINA (Global Initiative for Asthma) guidelines, asthma is a heterogeneous disease characterized by chronic inflammation and airway hyperreactivity. Symptoms such as wheezing, shortness of breath, chest tightness, and coughing vary over time, occurring at different intervals and with varying intensity. One of its key characteristics is variable airway obstruction, which may become persistent over time.[5] Treatment is based on inhaled corticosteroids.

The definition of chronic obstructive pulmonary disease (COPD) also describes a heterogeneous disease characterized by chronic symptoms (shortness of breath, cough, sputum production) resulting from changes in the airways (bronchitis, bronchiolitis) and/or alveoli (emphysema), which cause persistent, often progressive airway obstruction. One of the leading causes of COPD is smoking. Bronchodilators represent the basis for the treatment of the disease.[6]

Heart failure, COPD, and asthma are among the most significant health issues in the modern world. HF as comorbidity is present in 20% of patients with COPD, while at least 50% of them have systolic dysfunction of the left ventricle. Conversely, approximately 35% of patients with HF have COPD as comorbidity.[7]

The current guidelines of the European Society of Cardiology[1,8] no longer mention cardiac asthma in their classification but instead categorize HF as acute or chronic. Chronic HF is further classified into the following types based on left ventricular ejection fraction (EF):

- Chronic heart failure with preserved ejection fraction (HFpEF) – LVEF ≥50%
- Chronic heart failure with mildly reduced ejection fraction (HFmrEF) – LVEF 41–49%
- Chronic heart failure with reduced ejection fraction (HFrEF) – LVEF ≤40%

In addition to classification by ejection fraction, the 2022 American guidelines for HF[9] also describe four stages of HF:

- Stage A – At risk for HF: The individual has no symptoms, no structural heart disease, and no elevated biomarkers (e.g., individuals with hypertension, metabolic syndrome, or a family history of heart disease).
- Stage B – Pre-heart failure: No symptoms or signs of HF, but with structural heart disease, elevated cardiac filling pressures, or increased levels of natriuretic peptides (NT-proBNP, BNP).
- Stage C – Symptomatic HF: Structural heart disease with symptoms and signs of HF.
- Stage D – Advanced HF: Severe symptoms that interfere with daily activities and lead to frequent hospitalizations.

The severity of HF symptoms and the functional capacity of the patient are classified according to the NYHA (New York Heart Association) classification, which ranks symptom severity into four grades. Despite its limitations, the NYHA classification remains an independent predictor of patient mortality.

- Klasa 2. – blaga ograničenja u tjelesnoj aktivnosti. Osoba je bez simptoma u mirovanju, ali uobičajena aktivnost izaziva simptome nedostatka zraka, umora ili palpitacija.
- Klasa 3. – znatna ograničenja u tjelesnoj aktivnosti. Osoba je bez simptoma u mirovanju, ali i manja tjelesna aktivnost uzrokuje simptome.
- Klasa 4. – osoba nije sposobna ni za kakvu tjelesnu aktivnost, a simptomi mogu biti prisutni i u mirovanju.

Stupovi u medikamentnom liječenju HF-a jesu lijekovi: inhibitori renin-angiotenzin-aldosteronskog sustava (ACE – inhibitori i blokatori angiotenzinskih receptora – ARB s inhibitorom angiotenzinskih receptora – neprilizinom – ARNI – ili bez njega), beta-blokatori, inhibitori kotransportera natrija i glukoze 2 (SGLT 2 inhibitori), antagonisti mineralokortikoidnih receptora (MRA) te diuretici, najčešće Henleove petlje, za olakšavanje simptoma. Također u liječenju HF-a važnu ulogu imaju i drugi lijekovi te liječenje ostalih komorbiditeta. U naprednom liječenju HF-a uporabljuju se i implantabilni uređaji kao kardioverterski defibrilatori, uređaji za srčanu resinkronizaciju te uređaji za trajnu cirkulacijsku potporu i, na kraju, transplantacija srca.[1]

Prikaz bolesnika

U ovom radu prikazujemo bolesnika s dilatativnom kardiomiopatijom otvorene etiologije, reducirane sistoličke funkcije lijeve klijetke u kojeg je prva manifestacija HF-a bila u obliku kardijalne astme, NYHA klasa 4.

Šezdesetčetverogodišnji dugogodišnji pušač s otprije preboljelim moždanim udarom inicijalno je liječen u Županijskoj bolnici zbog pogoršanja psihičkoga stanja u sklopu posttraumatskoga stresnog poremećaja. Tijekom hospitalizacije počeo se žaliti na otežano disanje u naporu. U terapiji je otprije imao brzodjelujući beta-agonist salbutamol, kojeg mu je propisao obiteljski liječnik, ali mu taj lijek više nije pomagao. Nikada prije nije bio na pulmološkom pregledu te nije imao jasnu dijagnozu astme ili KOPB-a. Pri prvom internističkom pregledu bolesnik je bio tahidispnoičan u mirovanju te se koristio pomoćnom respiratornom muskulaturom. U fizikalnom je nalazu obostrano nad plućima registriran produljen ekspirij uz zvižduke. Nije bilo znakova periferne kongestije. Radiološki nalaz prsnih organa opisao je pojačanu transparenciju obaju plućnih krila bez znakova svježe infiltracije, dok su na periferiji srednjih plućnih polja opisani areali diskretnoga patološkog intersticija. Nije bilo pleuralnog izljeva, širina sjene medijastinuma bila je uredna, a hilusi bez patoloških promjena. Srce je opisano kao uredne veličine i zaobljene konture lijeve klijetke. U inicijalnom 12-kanalnom elektrokardiografskom zapisu opisani su: srednja električna os, atrijska tahikardija frekvencije klijetki od 131/min uz pojedinačne ventrikularne ekstrasistole. Inicijalna radna dijagnoza na odjelu bila je pogoršanje KOPB-a ili astme pa je stoga liječenje započeto metilprednizolonom i. v. te inhalacijama kratkodjelujućih bronhodilatatora. Na navedenu terapiju nije uočeno znatnije kliničko poboljšanje bolesnikova stanja. U laboratorijskim se nalazima uz akutnu bubrežnu ozljedu, alterirane jetrene enzime i blago povišen C-reaktivni protein isticao nalaz NT-proBNP koji je bio povišen iznad granica mjernog intervala laboratorija. Nakon toga uvodi se terapija za HF, započelo se s liječenjem diuretikom, uz empirijski antibiotik širokoga spektra ceftriakson, nebivolol i dapagliflozin. Usto su i dalje

- Class 1 – No limitation of physical activity. Ordinary physical activity does not cause shortness of breath, fatigue, or palpitations.
- Class 2 – Mild limitation of physical activity. No symptoms at rest, but ordinary activity causes shortness of breath, fatigue, or palpitations.
- Class 3 – Significant limitation of physical activity. No symptoms at rest, but even minimal physical activity triggers symptoms.
- Class 4 – Unable to perform any physical activity; symptoms may be present even at rest.

The medication treatment for HF is based on the following medications: renin-angiotensin-aldosterone system inhibitors (ACE inhibitors and angiotensin receptor blockers (ARBs), with or without angiotensin receptor-neprilysin inhibitors – ARNI), beta-blockers, sodium-glucose co-transporter 2 (SGLT2) inhibitors, mineralocorticoid receptor antagonists (MRA) and diuretics, most commonly loop diuretics, used to relieve symptoms. Other medications and the management of other comorbidities also play a crucial role in HF treatment. In advanced HF management, implantable devices such as cardioverter defibrillators, cardiac resynchronization therapy devices and long-term mechanical circulatory support devices may be used, with heart transplantation as the final treatment option.[1]

Case report

Herein we present the case of a male patient with dilated cardiomyopathy of unknown etiology and reduced systolic function of the left ventricle, whose first manifestation of heart failure (HF) was in the form of cardiac asthma (NYHA Class 4).

A 64-year-old long-term smoker with a history of previous stroke was initially treated at the County Hospital for the exacerbation of a psychiatric condition related to post-traumatic stress disorder. During hospitalization, the patient began complaining of exertional dyspnea. His prior treatment included a short-acting beta-agonist (salbutamol) prescribed by his primary care physician, but it was no longer effective. The patient had never been evaluated by a pulmonologist and did not have a clear diagnosis of asthma or COPD.

At the first examination by an internal medicine specialist, the patient was tachypneic at rest and using accessory respiratory muscles. Physical examination revealed prolonged expiration with wheezing bilaterally, but with no signs of peripheral congestion. The chest X-ray showed increased transparency of both lung fields without fresh infiltrates, with discreet pathological interstitial changes in the mid-lung periphery. There was no pleural effusion, mediastinal shadow width was normal, and with no hilar pathological changes. Heart size was normal, with rounded contours of the left ventricle. The initial 12-lead ECG recorded a normal electrical axis and atrial tachycardia with a ventricular rate of 131 bpm, with occasional ventricular extrasystoles. The initial working diagnosis established at the ward was COPD or asthma exacerbation, and treatment was started with IV methylprednisolone and short-acting bronchodilator inhalations. No significant clinical improvement occurred on this therapy. Laboratory findings showed acute kidney injury, elevated liver enzymes, and mildly elevated C-reactive protein (CRP), with markedly elevated NT-proBNP, exceeding the measurement range in the laboratory. HF treatment was initiated based on these findings,

nastavljeni kortikosteroidi i bronhodilatatori. Inicijalno nije uveden ACEi/ARB zbog nižih vrijednosti arterijskoga tlaka. Nakon pet dana liječenja prati se znatno poboljšanje kliničkoga stanja uz značajan pad vrijednosti NTproBNP-a koji je sada iznosio 5386 pg/mL, no uz daljnje pogoršanje bubrežne funkcije. U međuvremenu je provedena i ostala obrada. Nalaz spirometrije potvrdio je KOPB. Ultrazvukom srca opisane su uvećane sve četiri srčane šupljine (LVIDd 60 mm, LA 52 mm, RV 35 mm, RA 52 x 34 mm), globalna akinezija s hipokinezijom u području lateralne stijenke te septuma koji funkcionalno pripada desnoj klijetki, dok su inferiorna i inferolateralna stijenka stanjene uz aneurizmu bazalne trećine inferiorne stijenke. Teško je smanjena ejekcijska funkcija lijeve klijetke (EF 15 %). Dopplerom je prikazana sekundarna teška mitralna i trikuspidalna regurgitacija uz indirektne pokazatelje povišenoga tlaka u plućnoj cirkulaciji te dijastolička disfunkcija III. stupnja. S obzirom na nalaz, u terapiju je uključen i antagonist mineralokortikoidnih receptora eplerenon. ARNI nije uključen jer bolesnik zbog simptomatske hipotenzije nije tolerirao povećanje doze valsartana na više od 40 mg. Dvadesetčetverosatnim holterom EKG-a zabilježene su asimptomatske nepostojane ventrikularne tahikardije.

Tijekom daljnjeg liječenja optimizirana je doza lijekova, uz oporavak bubrežne funkcije, te je bolesnik otpušten s terapijom: nebivolol 10 mg, empagliflozin 10 mg, eplerenon 50 mg, valsartan 40 mg, furosemid 80 mg, acetilsalicilatna kiselina 100 mg, atorvastatin 40 mg te umeklinidijev bromid / vilanterol 55/22 mcg 1 inhalacija na dan.

Pri otpustu je preporučena kontrola kod pulmologa te kardiologa/aritmologa radi daljnjega naprednog liječenja. Da bi se definirala etiologija dilatativne kardiomiopatije, u bolesnika je potrebno učiniti još koronarografiju, dalje, ovisno o nalazu, dolaze u obzir magnetna rezonancija srca i genetsko testiranje. S obzirom na to da to nije bilo moguće učiniti tijekom hospitalizacije, bolesnik je upućen na daljnju obradu u mjesto boravka.

Rasprava

Razlikovanje HF-a od astme i KOPB-a većinom je jednostavno, osim u bolesnika u kojih su prisutne obje bolesti. Tada je za sigurno razlikovanje potrebno učiniti EKG, kompletne testove plućne funkcije, ehokardiografski pregled i natriuretski peptid. No u praksi dijagnoza se često postavlja na osnovi simptoma. U mladih osoba pojava kašlja i zviždanja ima visoku senzitivnost i specifičnost za astmu, no u starijih osoba postoji veći rizik od pogrešnog postavljanja dijagnoze.[10] Pogrešna dijagnoza donosi velik rizik za bolesnika, ne samo da se bolesnici onda pogrešno liječe i time im je uskraćena adekvatna skrb nego imaju rizik i od razvoja nuspojava na kardiovaskularni sustav. Lijekovi koji se uporabljuju u liječenju astme (kortikosteroidi, bronhodilatatori) nisu učinkoviti u liječenju kardijalne astme. Dapače, neki od tih lijekova mogu dodatno pogoršati HF. Primjerice, teofilin, bronhodilatator koji se rabi u drugoj liniji, kontraindiciran je u bolesnika s HF-om.[11] Kortikosteroidi, kada se primjenjuju peroralno, uzrokuju zadržavanje natrija te time i dodatne tekućine, čime uzrokuju pogoršanje HF-a.[12] Beta-2-agonisti mogu utjecati na beta-1-receptore u srcu te na taj način uzrokovati tahikardiju koja dovodi do porasta ishemijskih događaja. Efekt beta-2-agonista izaziva se neovisno o načinu primjene (inhalacijski i peroralni). Ipak postoje dokazi da beta-2-agonist vilanterol, sam ili u kombi-

comprising treatment with diuretics along with an empirical broad-spectrum antibiotic (ceftriaxone), nebivolol, and dapagliflozin. In addition to the above, treatment with corticosteroids and bronchodilators was continued as well. ACEi/ARB were not initially introduced due to low arterial blood pressure. After five days of treatment, the patient showed significant clinical improvement and a significant decrease in NT-proBNP levels (now 5386 pg/mL), but with further worsening of kidney function. Additional diagnostics were performed as well. Spirometry findings confirmed COPD. Echocardiography showed dilation of all four cardiac chambers (LVIDd 60 mm, LA 52 mm, RV 35 mm, RA 52×34 mm), global akinesia with hypokinesis of the lateral wall and septum (functionally belonging to the right ventricle), along with inferior and inferolateral wall thinning with an aneurysm of the basal third of the inferior wall. Left ventricular ejection fraction was severely reduced (EF 15%). Doppler findings indicated severe secondary mitral and tricuspid regurgitation with indirect markers of pulmonary hypertension and grade III diastolic dysfunction. Based on these findings, eplerenone, a mineralocorticoid receptor antagonist, was added to the therapy. An angiotensin receptor-neprilysin inhibitor was not introduced due to symptomatic hypotension preventing valsartan titration above 40 mg. A 24-hour Holter ECG recorded asymptomatic, non-sustained ventricular tachycardias.

Medication dosages were optimized during further treatment, and recovery of kidney function was observed; consequently, the patient was discharged with the following therapy: nebivolol 10 mg, empagliflozin 10 mg, eplerenone 50 mg, valsartan 40 mg, furosemide 80 mg, acetylsalicylic acid 100 mg, atorvastatin 40 mg, umeclidinium bromide/vilanterol 55/22 mcg (1 inhalation daily).

At discharge, the patient was advised to follow up with a pulmonologist and a cardiologist/electrophysiologist for further advanced treatment. A coronary angiography would be required in order to determine the etiology of dilated cardiomyopathy, with cardiac MRI and genetic testing to be considered depending on the findings. Since these tests could not be performed during hospitalization, the patient was referred for further evaluation in his place of residence.

Discussion

Differentiating heart failure (HF) from asthma and COPD is usually straightforward, except in cases where both conditions are present. In such cases, a definitive diagnosis requires an ECG, complete pulmonary function tests, echocardiography, and determining natriuretic peptide levels. However, in clinical practice, the diagnosis is often established based on symptoms alone. In younger patients, the presence of coughing and wheezing has high sensitivity and specificity for asthma, but the risk of misdiagnosis increases in older adults.[10] A misdiagnosis poses a significant risk to patients, as they not only receive incorrect treatment, thus being denied appropriate care, but they also face a higher risk of developing cardiovascular side effects. Medications used to treat asthma (corticosteroids, bronchodilators) are not effective in managing cardiac asthma. In fact, some of these medications can worsen HF. For example, theophylline, a second-line bronchodilator, is contraindicated in patients with HF.[11] When administered orally, corticosteroids cause sodium retention and thus also fluid retention, resulting in HF exacerbation.[12] Beta-2 agonists can affect beta-1 receptors in the heart, thus causing tachycardia that leads to an in-

naciji s inhalacijskim kortikosteroidom flutikazonom, ne povećava rizik od smrtnosti u kardiovaskularnih bolesnika.[13] Također se inhalaciiski antikolinergici smatraju sigurnom alternativom za bolesnike s HF-om i pridruženim KOPB-om.[14]

U starijih osoba u kojih se prvi put očituju simptomi paroksizmalne dispneje i kašlja uvijek je potrebno razmotriti mogućnost za postojanje HF-a. U tome nam mogu pomoći elektrokardiogram, anamnestički podatci o uzimanju diuretika, o postojanju arterijske hipertenzije, ishemijske bolesti srca i slično. Dijagnoza kardijalne astme i nije toliko jednostavna, jer se pod njom vole sakriti različiti klinički entiteti, pa njezina točna dijagnoza zahtijeva veliku kliničku oštroumnost.

Habaruiga *i sur.*[15] opisali su slučaj 52-godišnjeg bolesnika koji je dvije godine pogrešno i neuspješno liječen s dijagnozom astme sve dok nije postavljena točna dijagnoza HFrEF-a. Pod kardijalnom se astmom mogu kriti i drugi klinički sindromi. Na primjer, kao kardijalna astma se mogu očitovati ishemijska bolest srca, akutni infarkt miokarda, nestabilna angina, miokarditis, postpartalna kardiomiopatija, bolesti mitralne valvule itd.[16] Veeraraghavan *i sur.* opisali su slučaj gigantskog miksoma lijevog atrija u 32-godišnje bolesnice koja se godinu dana liječila zbog rekurentnih napadaja astme.[17]

Jedan od razloga zašto je spomenute dijagnoze teško razlučiti krije se i u maskiranju testova plućne funkcije. Epizoda pogoršanja HF-a utječe na nalaz testova plućne funkcije ne samo tijekom pogoršanja nego čak do 3 mjeseca poslije. Opstruktivne plućne bolesti uzrokuju hiperinflaciju pluća i opstruktivni obrazac (Tiffeneauov indeks /FEV1/FVC/ <0,7 i pad u forsiranome ekspiratornom volumenu u prvoj sekundi /FEV1/), dok HF uzrokuje restriktivni obrazac (proporcionalno smanjenje FEV1 i forsiranoga vitalnog kapaciteta /FVC/). U bolesnika s kroničnim HF-om nalaz povećanoga rezidualnog volumena (RV) i funkcionalnoga rezidualnog kapaciteta (FRC) brzo će nas usmjeriti prema KOPB-u. No u bolesnika s KOPB-om koji ima i HF situacija je teža. Kongestija, niski srčani *output* i slabija plućna popustljivost uzrokuju restrikciju. Restrikcija će, ako je dovoljno teška, sakriti hiperinflaciju pluća i dovesti do lažne „normalizacije" nalaza testova plućne funkcije.[18,19]

Zaključak

Umjesto zaključka istaknuli bismo važnost anamneze, dobroga kliničkog pregleda te kliničkog razmišljanja. Bitno je ne brzati sa zaključcima jer nije svaki zvižduk astma ili pogoršanje KOPB-a. Pod zvižducima se može kriti i akutizacija HF-a ili takozvana kardijalna astma. Termin se prije više upotrebljavao, no i danas ga se možemo pokatkad sjetiti da nas brzanje sa zaključcima ne bi odvelo u pogrešnom smjeru.

creased risk of ischemic events. This effect of beta-2 agonists is present regardless of whether they are administered orally or via inhalation. However, some evidence suggests that the beta-2 agonist vilanterol, either alone or in combination with inhaled corticosteroid fluticasone, does not increase cardiovascular mortality risk.[13] Additionally, inhaled anticholinergics are considered a safe alternative for patients with HF and coexisting COPD.[14]

In older individuals presenting with paroxysmal dyspnea and cough for the first time, it is always necessary to consider the possibility of HF. Diagnostic clues that can facilitate this process include electrocardiography, medical history of diuretic use, the presence of arterial hypertension and ischemic heart disease, etc. Diagnosing cardiac asthma is not straightforward and simple, as shares symptoms with various other clinical entities, requiring sharp clinical judgment to establish an accurate diagnosis.

Habaruiga *et al.*[15] described the case of a 52-year-old patient who was misdiagnosed and unsuccessfully treated for asthma for two years before finally being accurately diagnosed with HFrEF. Other clinical syndromes can also present as cardiac asthma. For example, ischemic heart disease, acute myocardial infarction, unstable angina, myocarditis, postpartum cardiomyopathy, mitral valve diseases, and other diseases can present as cardiac asthma.[16] Veeraraghavan *et al.* described a case of a giant left atrial myxoma in a 32-year-old woman who was misdiagnosed and treated for recurrent asthma attacks for a year.[17]

One of the reasons why these diagnoses are difficult to differentiate lies in the masking effect on pulmonary function tests. Episodes of heart failure exacerbation can affect pulmonary function test results not only during the exacerbation itself but for up to three months afterwards. Obstructive lung diseases cause lung hyperinflation and an obstructive pattern (Tiffeneau index FEV1/FVC <0.7 and a decrease in forced expiratory volume in the first second (FEV1)), whereas HF causes a restrictive pattern (proportional reduction in FEV1 and forced vital capacity (FVC)). In patients with chronic HF, findings of increased residual volume (RV) and functional residual capacity (FRC) can quickly guide us toward a COPD diagnosis. However, in patients who have COPD along with coexisting HF, the situation is more complex. Congestion, low cardiac output, and reduced pulmonary compliance contribute to restriction. Restriction, if severe enough, will mask lung hyperinflation and lead to a false "normalization" of pulmonary function test results.[18,19]

Conclusion

Instead of a formal conclusion, we would like to emphasize the importance of taking the patient's medical history, performing a thorough clinical examination, and careful clinical reasoning. It is crucial not to jump to conclusions, as not every wheeze is caused by asthma or COPD exacerbation. Wheezing can also be caused by HF exacerbation or so-called cardiac asthma. Although the term cardiac asthma is less frequently used today, it is still worth bearing in mind in order to avoid hasty conclusions that could lead us astray in diagnosis and treatment.

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