Rev Esp Cardiol. 2022;75(1):9–11

Editorial

Acute heart failure. The quintessential cardiac emergency

Insuficiencia cardiaca aguda. La urgencia cardiológica por excelencia

Carlos Peña-Gil,[a,b,*] Violeta González-Salvado,[a,b] and Jaume Figueras-Bellot[c]

[a] Servicio de Cardiología y Unidad Coronaria, Hospital Clínico Universitario de Santiago de Compostela, SERGAS, IDIS, Santiago de Compostela, A Coruña, Spain
[b] Centro de Investigación Biomédica en Red de Enfermedades Cardiovasculares (CIBERCV), Spain
[c] Servicio de Cardiología, Hospital Universitari Vall d'Hebron, Barcelona, Spain

Article history:
Available online 3 September 2021

Acute heart failure (AHF) is a common cause of emergency care and hospitalization, and up to 80% of affected patients are hospitalized. In-patient mortality and early rehospitalization rates are also high.[1]

This clinical syndrome can arise when a specific underlying cardiac cause is combined with 1 or more triggers, and the spectrum of presentation can include resting dyspnea, acute respiratory failure in the form of acute pulmonary edema, and cardiogenic shock.[1,2] The condition can be the first form of presentation of heart disease (de novo AHF), or it can be the stage to which various chronic cardiologic processes can progress, including decompensated chronic heart failure.[2] Appropriate diagnosis of AHF requires a step-by-step process to identify the clinical syndrome and the type of AHF and to recognize its underlying causes and triggers.[1,2] Each patient is a challenge for emergency departments. An adequate workup should cover the patient's history, accompanying symptoms, physical signs (including heart murmurs), and the results of additional tests, namely, electrocardiogram, chest x-ray, laboratory tests, and cardiac and pulmonary ultrasound. These diagnostic tests should be performed as soon as possible, preferably when the patient arrives at the emergency department. Combining clinical information and additional tests may be complex. For instance, the presence of a structural heart abnormality does not necessarily mean that it is the cause of the current clinical picture.[2]

Successful management of patients with AHF depends on early, accurate diagnosis of the underlying cause and appropriate treatment of reversible triggers.[2] It is essential to avoid diagnostic delays in serious conditions, such as acute myocardial infarction or pulmonary thromboembolism, as well as improper medical decisions with counterproductive results, such as excessive fluid therapy in acute pulmonary edema or beta-blocker use in AHF with severely impaired systolic function.[3] Multiple studies have highlighted the importance of shortening the time from initial care to administration of the appropriate treatment, including early start of diuretics in the prehospital setting.[1,3,4] Reversible triggers should be treated as soon as possible to help stabilize the patient promptly and to prevent organ damage and progression to severe respiratory failure, acute kidney failure, refractory shock, or death.

Most patients who present to emergency departments with AHF are older and have comorbidities, and treatment is often conservative.[2] However, some patients have AHF triggers requiring specific treatments and need to be transferred to a referral hospital.[5] It is vitally important to maintain high alert when first evaluating this heterogeneous group of patients and monitoring their progress during the first few hours in the emergency department.

Systolic blood pressure is a prognostic marker in AHF, and better prognoses are seen at higher blood pressures. The hypertensive response associated with acute pulmonary edema may result from adrenergic activation, rather than a trigger of edema.[6] Consequently, acute pulmonary edema may be accompanied by blood pressure levels $\geq$ 170 mmHg in the same proportion, whether or not ischemic heart disease is present.[6] In fact, nowadays, hypertensive emergencies rarely lead to acute pulmonary edema.[6] A similar situation can arise with rapid atrial fibrillation: although it can trigger AHF in some underlying cardiologic conditions, it is often precipitated by AHF and by the activation mechanisms triggered.

Two key additional tests for the etiological diagnosis of AHF are electrocardiography and cardiac and pulmonary ultrasound.[2,5] Electrocardiography can provide information on possible triggers, such as tachyarrhythmia (new-onset atrial fibrillation), myocardial ischemia (ST-segment changes), or bradyarrhythmia (advanced atrioventricular block), as well as on possible underlying issues (left ventricular hypertrophy, prior myocardial necrosis, and atrial dilatation).[2,5]

Advances in ultrasound equipment quality and portability, coupled with widespread training, have improved the learning curve and allowed the technique to be performed by noncardiologists who gain adequate technical training in emergency departments.[7] Moreover, telemedicine tools and communication apps have made it easier to share the findings of these techniques with consultants in referral cardiology departments in real time.

Echocardiography is the technique of choice to confirm an anatomic diagnosis and identify the causes, estimate hemodynamic status, and monitor treatment response.[7] A full study will report on whether global left ventricular systolic function is reduced or preserved, as well as on the presence of regional contractility defects indicating acute myocardial ischemia or prior

SEE RELATED CONTENT:
https://doi.org/10.1016/j.rec.2020.11.022

* Corresponding author: Servicio de Cardiología, Hospital Clínico Universitario de Santiago de Compostela, Travesía Choupana s/n, 15706 Santiago de Compostela, A Coruña, Spain.
E-mail address: Carlos.pena.gil@sergas.es (C. Peña-Gil).

https://doi.org/10.1016/j.rec.2021.06.017

myocardial necrosis. The presence of significant left valve disease (mitral or aortic) should also be evaluated, and it is particularly important to detect degenerative aortic stenosis in elderly patients. Doppler echocardiography can be used to accurately estimate the patient's hemodynamic parameters, filling and pulmonary pressures, and intravascular volume status. These findings will help define more precise treatment. In patients with shock, echocardiography can identify the underlying cause, diagnose cardiac tamponade, and establish the suspicion of hypovolemic or distributive shock and acute pulmonary thromboembolism.[7]

When performed in the acute phase of AHF, echocardiography can reveal transient systolic dysfunctions or dynamic mitral regurgitation that might not be detected in echocardiograms carried out after patient stabilization.[8] Significant mitral regurgitation caused by a functional mechanism is present in a high percentage of patients with acute pulmonary edema and is often silent.[8] The degree and severity of mitral regurgitation may be influenced by the conditions under which it is studied: acute phase, subacute phase, or after the start of diuretic or vasodilator treatment.[8,9]

In addition to emergency echocardiography, pulmonary ultrasound can identify and quantify Kerley B lines in various areas of the lungs to provide useful information for both diagnostic confirmation and prognosis.[7] Repeating these studies and recording the trends on electronic media allow monitoring of the response to the various treatments.[7]

In a recent article published in *Revista Española de Cardiología,* Miró et al.[10] examined short-term results to analyze the impact of structural and organizational components of emergency departments and hospitals managing AHF patients. The analysis was based on a retrospective multicenter cohort from 40 Spanish emergency departments of the Epidemiology of Acute Heart Failure in Emergency Departments (EAHFE) registry. The registry provides useful information for daily practice on the approach to AHF in Spanish emergency departments and offers demographic, clinical, and prognostic data on these patients. The population was older, with a mean age of 82 years, and 30-day mortality was 10.3%. One of the main findings of the study was that AHF patients treated in referral hospitals had lower in-patient mortality (odds ratio = 0.78; 95% confidence interval, 0.65-0.94) and fewer lengthy stays. The existence of heart failure units in hospitals was also related to better results after hospital discharge.[10]

The authors state that ejection fraction and natriuretic peptide levels were not available for 48% and 52% of patients, respectively. Acute coronary syndrome was detected as a trigger in 14.8% of patients and was treated by noninvasive ventilation in 6.7%.[10] These data may suggest that improved equity is needed, compared with referral centers, both in access to crucial additional tests for diagnosis, such as echocardiography in emergency departments, and access to first-line treatments (eg, noninvasive ventilation) shown to improve the prognosis of patients with acute pulmonary edema.[11]

An accurate AHF diagnosis will allow appropriate treatment to be tailored to the patient's hemodynamic needs. In patients with acute pulmonary edema, adjustments can be made to the initial therapeutic approach, which is usually based on diuretic treatment, vasodilators, and oxygen therapy. It is important to identify each patient's profile and characteristics early and to establish individual therapeutic objectives for blood pressure control, preload and postload (vasodilators), reduced congestion (diuretics), heart rate and rhythm (antiarrhythmic agents and negative chronotropes), improved cardiac output (inotropics), and correction of reversible triggers (coronary revascularization, cardiac surgery, advanced therapies).[2,5]

Patients with AHF accompanied by acute coronary syndrome may benefit from early coronary angiography and from coronary revascularization.[12] In our setting, ischemic heart disease is the most common underlying cause and trigger of AHF episodes, although it can sometimes go unnoticed due to the absence of angina or due to the presence of a conduction disorder on electrocardiography, masking ischemia.[13] In many cases, coronary angiography is not performed because of the patient's age and, therefore, the role of ischemia as an underlying cause is further underestimated in these patients.[13]

Vasodilator treatments or excessive diuretics must be used with caution in patients with severe aortic stenosis or hypertrophic obstructive cardiomyopathy, as they can trigger arterial hypotension. Patients with serious systolic dysfunction and rapid atrial fibrillation can deteriorate when heart rate control is attempted with beta-blockers.[2,5] Urgent implantation of a ventricular pacemaker may be indicated in some severe bradyarrhythmias with AHF, and some isolated cases of rapid atrial fibrillation may require synchronized electrical cardioversion. Acute aortic or mitral valve regurgitation, particularly in the context of acute endocarditis or type A aortic dissection, cannot be treated by medical therapies alone, and will require emergency surgical repair. Some patients with advanced or exacerbated kidney failure may require treatments for prompt extrarenal purification to stabilize congestive symptoms. Patients with preserved global systolic function do not have an indication for inotropic treatment in the case of hypotension.[2,5] Certain patients in cardiogenic shock may benefit from advanced ventricular assist treatments.

Therefore, the diagnostic and therapeutic needs of patients presenting to emergency departments for symptoms of AHF are high, and must be addressed immediately.[2,5] Accurate diagnosis and early treatment of the triggers and aggravating factors, such as neurohormonal response or ischemia, as well as the early detection of patients at risk for complications (respiratory fatigue, hypotension, or kidney failure) will improve the prognosis of these patients.[2–5] Healthcare inertia and frequent visits by patients with decompensated chronic heart failure is a risk for the safety of patients with de novo AHF who could experience delays in appropriate diagnosis and treatment.

The study by Miró et al. is relevant because it considers aspects that can be improved to enhance quality in AHF patient outcomes and prognosis in Spain.[10] Widespread availability of echocardiography is needed in all hospital emergency departments. The healthcare process of AHF patients should promote the coordination of hospital emergency departments with cardiology departments to make urgent diagnostic and therapeutic resources available to patients.

Creating multidisciplinary teams coordinated in a network with referral hospitals can offer advanced treatments to more patients, as seen in the treatment of cardiogenic shock.[14] In AHF, as in acute coronary syndrome or stroke, time is important for preventing patient complications and decline. AHF is the quintessential cardiac emergency, and has a poorer prognosis in the acute phase than most acute coronary syndromes. Similar to the need for implementation of a shock code in Spain to centralize multidisciplinary teams with experience in mechanical circulatory support,[15] the scope of these proposals should be broadened to include urgent referral of other types of AHF requiring advanced diagnostic and therapeutic procedures, with considerable potential clinical impact achieved despite small investments in resources. This will help provide equitable health care, in which AHF patients' health outcomes are not overly dependent on the emergency department where they are attended.

In conclusion, AHF is a common cardiac emergency that necessitates comprehensive care to ensure appropriate diagnoses and therapeutic resources for all patients. Early diagnosis requires that emergency departments have echocardiography units available, and that they are able to start early personalized treatment

based on the underlying cardiologic cause and on the AHF triggers. Proper care of these patients calls for an adequate organizational response, using the available technological resources, as needed in other cardiovascular emergencies, such as acute coronary syndromes and stroke.

FUNDING

None.

CONFLICTS OF INTEREST

None declared.

REFERENCES

1. Ponikowski P, Voors AA, Anker SD, et al. 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC). Developed with the special contribution of the Heart Failure Association (HFA) of the ESC. *Eur J Heart Fail.* 2016;18:891–975.
2. Gheorghiade M, Pang PS. Acute heart failure syndromes. *J Am Coll Cardiol.* 2009;53:557–573.
3. Mebazaa A, Yilmaz MB, Levyet P, et al. Recommendations on pre-hospital and early hospital management of acute heart failure: a consensus paper from the Heart Failure Association of the European Society of Cardiology, the European Society of Emergency Medicine and the Society of Academic Emergency Medicine — short version. *Eur Heart J.* 2015;36:1958–1966.
4. Matsue Y, Damman K, Voors AA, et al. Time-to-furosemide treatment and mortality in patients hospitalized with acute heart failure. *J Am Coll Cardiol.* 2017;69:3042–3051.
5. Pang PS, Collins SP, Gheorghiade M, et al. Acute dyspnea and decompensated heart failure. *Cardiol Clin.* 2018;36:63–72.
6. Figueras J, Bañeras J, Peña-Gil C, et al. Acute arterial hypertension in acute pulmonary edema: mostly a trigger or an associated phenomenon? *Can J Cardiol.* 2016;32:1214–1220.
7. Price S, Platz E, Cullen L, et al. Expert consensus document: Echocardiography and lung ultrasonography for the assessment and management of acute heart failure. *Nat Rev Cardiol.* 2017;14:427–440.
8. Stone GW, Griffin B, Shah PK, et al. Prevalence of unsuspected mitral regurgitation and left ventricular diastolic dysfunction in patients with coronary artery disease and acute pulmonary edema associated with normal or depressed left ventricular systolic function. *Am J Cardiol.* 1991;67:37–41.
9. Pierard LA, Lancellotti P. The role of ischemic mitral regurgitation in the pathogenesis of acute pulmonary edema. *N Engl J Med.* 2004;351:1627–1634.
10. Miró Óaue, López-Díez MP, Cardozo C, et al. Impacto de las características estructurales y organizativas hospitalarias y de urgencias en el resultado evolutivo de la insuficiencia cardiaca aguda. *Rev Esp Cardiol.* 2022;75:39–49.
11. Masip J, Peacock WF, Price S, et al. Indications and practical approach to non-invasive ventilation in acute heart failure. *Eur Heart J.* 2018;39:17–25.
12. Collet JP, Thiele H, Barbato E, et al. ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. *Eur Heart J.* 2021;42:1289–1367.
13. Pena-Gil C, Figueras J, Soler-Soler J. Acute cardiogenic pulmonary edema: Relevance of multivessel disease, conduction abnormalities and silent ischemia. *Int J Cardiol.* 2005;103:59–66.
14. Hernández-Pérez FJ, Álvarez-Avelló JM, Forteza A, et al. Initial outcomes of a multidisciplinary network for the care of patients with cardiogenic shock. *Rev Esp Cardiol.* 2021;74:33–43.
15. Díaz Molina B, González Costello J, Barge-Caballero E. The shock code in Spain, The next quality leap in cardiological care is here. *Rev Esp Cardiol.* 2021;74:5–7.