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ACUTE HEART FAILURE (AHF) - HOW TO EVALUATE THE HEMODYNAMIC PROFILE AND WHEN TO HOSPITALIZE

INSUFICIÊNCIA CARDÍACA AGUDA (ICA) - COMO AVALIAR O PERFIL HEMODINÂMICO E QUANDO INTERNAR

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ABSTRACT

Heart failure is a clinical syndrome that has been increasing over the last few years and is currently one of the main causes of hospitalization in Brazil and in the world. Acute heart failure has two forms of presentation - chronic acute decompensated heart failure and new heart failure, both conditions being associated with high morbidity and mortality. The first approach to these patients should be conducted systematically in order to establish the diagnosis, the hemodynamic classification, and the risk stratification in order to adequately guide the initial conducts. An immediate therapeutic decision is essential to optimize the intra- and extra-hospital management of these patients for better prognostic evolution.

Keywords: Heart Failure; Diagnosis; Treatment.

RESUMO

A Insuficiência cardíaca é uma síndrome clínica cuja prevalência vem aumentado nos últimos anos, sendo uma das principais causas de internação no Brasil e no mundo. A apresentação da insuficiência cardíaca aguda compreende dois espectros, quais sejam, insuficiência cardíaca crônica agudizada e insuficiência cardíaca nova e ambas estão associadas a alta morbidade e mortalidade. A primeira abordagem desses pacientes deve ser realizada de maneira sistemática para estabelecer o diagnóstico, classificação hemodinâmica e estratificação de risco para orientar adequadamente as condutas iniciais. A decisão terapêutica imediata é essencial para otimizar o manejo desses pacientes, visando a melhor evolução prognóstica intra e extra-hospitalar.

Descritores: *Insuficiência Cardíaca; Diagnóstico; Tratamento.*

INTRODUCTION

Congestive heart failure (HF) is a clinical syndrome characterized by signs and symptoms resulting from the reduction in cardiac output and/or increase in cardiac filling pressure. There are two presentations of this clinical syndrome: chronic HF, a condition that manifests as persistent and progressive characteristics of the disease; and acute HF, represented by the sudden onset (new-onset acute HF) or intensification of a chronic (acute chronic HF) condition. Both conditions require immediate treatment. This article discusses acute HF (Figure 1).[1,2]

EPIDEMIOLOGY

The increase in life expectancy of the population and advances in the clinical treatment of chronic diseases has led to an increase in the number of patients with HF. Acute HF is currently one of the main causes of hospitalization worldwide. Approximately 190,000 patients per year are admitted in Brazil, with an in-hospital mortality rate of 13%. In accordance with international records, the mortality rate among patients hospitalized for acute HF is 11–17% per year, with rehospitalization rates of 44–66%. These numbers may be even higher in Brazil, creating a serious public health problem.[3,4]

Epidemiological data of hospitalized patients with acute HF in Brazil were published in the BREATHE registry, which included 1263 patients from 51 centers in Brazil. When the etiology of HF was evaluated, most patients had ischemic (30%) and hypertensive (20%) cardiomyopathy, while the rest of the causes being dilated, valve, and Chagas cardiomyopathy as well as other causes (Figure 2).[4]

The initial evaluation of patients with suspected acute HF should be performed objectively to exclude conditions that confer a high risk, such as acute myocardial infarction (AMI), acute dissection of the aorta, pulmonary thromboembolism

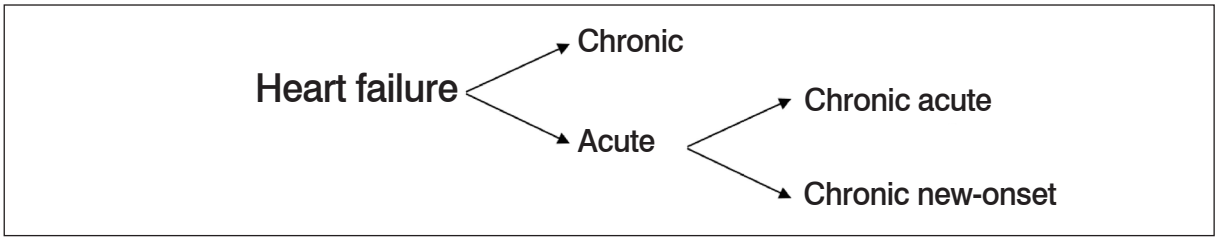

Figure 1. Classification of heart failure.

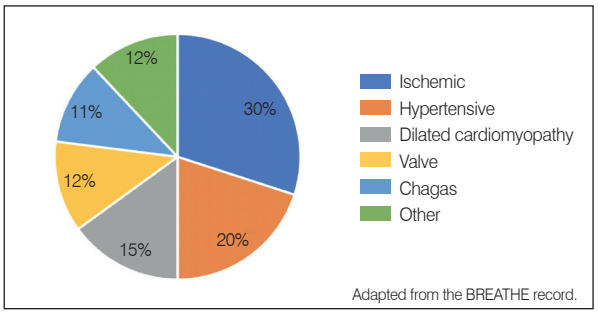

Figure 2. Etiologies of acute heart failure.

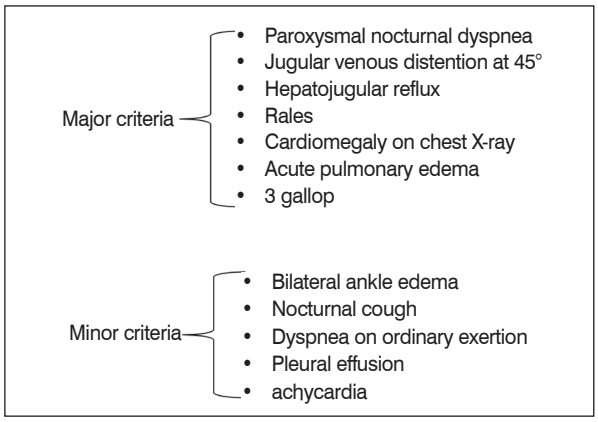

Figure 3. Framingham criteria.

(PTE), stroke, potentially fatal arrhythmias, and metabolic/infectious disorders. These patients should receive a diagnosis and initial treatment (Table 1) within 30 minutes after admission.[5,6]

DIAGNOSIS

Excluding the conditions that cause greater risk, the focus will be on the diagnostic definition for the implementation of targeted treatment. The evaluation should be performed systematically for up to 120 minutes[7] following the judgement of rational diagnostic construction to evaluate the following points:

Diagnosis of acute HF;

- Model of presentation of HF (new-onset acute or chronic acute);
- HF with preserved ejection fraction or HF with reduced ejection fraction;
- Assessment of decompensation factors;
- Decompensated non-cardiovascular comorbidities; and
- Clinical-hemodynamic model.

Diagnosis of acute HF

The diagnosis of HF is established with the assessment of the clinical history and the evaluation of signs and symptoms that are usually present in this syndrome targeted through the Framingham criteria (Figure 3), which requires the presence of two major and one minor criteria for the diagnosis or one major and two minor criteria.

There are two scenarios of acute HF presentation. The most frequent, occurring in about 60–75% of cases, is called acute chronic HF, a process in which the patient already had a stable presentation of prior HF and that for some reason destabilizes with an HF decompensation. The other less common presentation occurs in approximately 25–40% of cases in patients without a prior history of HF or structural heart disease who present with HF, called acute new-onset HF.[8]

The distinction between the two presentations (Table 2) is essential to increasing our understanding of the pathophysiological mechanisms involved in cardiac decompensation and direct the strategy of therapeutic intervention.[9]

HF can be classified according to the ventricular function estimated by ejection fraction (Table 3) and is of extreme importance in terms of the definition for both the implementation of the immediate therapeutic strategy and stabilization after hospital discharge.[10]

Assessment of decompensation factors

At least half of patients with acute HF have associated clinical factors that precipitate the decompensation. The study of these factors is essential for case management

Table 1. Conditions with a high risk of in-hospital mortality.

Suspicion	Diagnosis	Treatment
Acute myocardial index	Electrocardiography	Primary angioplasty/thrombolytic therapy
Pulmonary thromboembolism	Angiotomography of thorax	Thrombolytic therapy
Acute aortic dissection	Angiotomography of thorax	Surgery
Acute respiratory failure	Physical examination	Tracheal Intubation
Cardiogenic shock	Physical examination	Inotropes/intra-aortic balloon/ mechanical circulatory support
Hypertensive emergencies	Physical examination	Sodium nitroprusside/nitroglycerin
Mechanical complication or valve heart disease	Physical examination/echocardiography	Surgical or percutaneous intervention
Potentially fatal arrhythmias	Electrocardiography	Electrical cardioversion/temporary pacemaker
Stroke	Physical examination	Stroke protocol

since most of the time only with the resolution of the clinical presentation is it possible to stabilize the HF presentation. The main associated factors that must always be assessed include infections, metabolic changes (thyroid disease or diabetes), intake of alcohol or drugs, arrhythmia, valvular heart disease, anemia, poor adherence to medication, diet (increased salt intake), and others.[11,12]

Uncompensated non-cardiovascular comorbidities

Complementary questioning with the assessment of other comorbidities is essential to case management. Approximately 75% of patients have at least one associated comorbidity, with diabetes, chronic obstructive pulmonary disease, hypothyroidism, depression, and chronic kidney disease being the most prevalent. Identification and therapeutic control are part of the treatment of acute HF since they influence the therapeutic response and patient prognosis.[13]

Hemodynamic and clinical profile

Two parameters must be identified for the establishment of the clinical hemodynamic profile: congestion and perfusion (Figure 4). Congestion is present in approximately 90% of patients and low cardiac output in 10%, but the accuracy of clinical examination in detecting these variables is low, requiring complementation with laboratory and imaging exams for the real identification of the hemodynamic profile.[14]

The assessment of signs and symptoms of congestion and perfusion allows one to classify a patient's hemodynamic profile. Regarding congestion, the patient may be considered "wet" if congested or "dry" if not congested. Regarding perfusion, the patient can be considered "cold" if poorly perfused or "warm" if well perfused.[15]

With the evaluation of the parameters of congestion and perfusion, four hemodynamic phenotypes can be revealed: profile A, when the patient is considered "warm" and "dry"; profile B, a more common presentation when the patient is "warm" and "wet"; profile C, corresponding to about 20% of cases when the patient is "cold" and "wet"; and finally profile L, when the patient is "cold" and "dry" (Figure 5).[16]

Table 2. Distinction between acute chronic HF and acute new-onset HF.

	Acute chronic HF	Acute new-onset HF
Pathophysiology	Systolic dysfunction/ hydrosaline retention	Diastolic dysfunction
Congestion	Global	Pulmonary
Symptom	Fatigue/dyspnea	Dyspnea
Start of symptoms	Progressive (days)	Fast (hours)
Blood pressure	Reduced	High or normal
Ejection fraction	Reduced	Normal or reduced
Peripheral edema	Frequent	Infrequent
Increased weight	Yes	No

Table 3. Classification of HF according to ejection fraction.

Ejection fraction	Classification
Menor 40%	Reduced ejection fraction HF
40-49%	Intermediary ejection fraction HF
Maior ou igual 50%	Preserved ejection fraction heart rate

COMPLEMENTARY EXAMS

In emergency care, some exams are essential for the diagnosis of acute HF and should be performed as early as possible so that appropriate treatment is implemented quickly.

Laboratory exams

Laboratory and imaging exams should be requested at admission to complement the clinical assessment in the diagnosis of acute heart failure (AHF), define the causal factors and differential diagnosis, and assist in the establishment of the risk profile on admission. Among the laboratory tests, natriuretic peptides (NP) are of high diagnostic value and should be used whenever possible in the emergency room, as the presence of high levels of B-type natriuretic peptide (BNP) > 500 pg/mL and NT-proBNP > 900 pg/mL

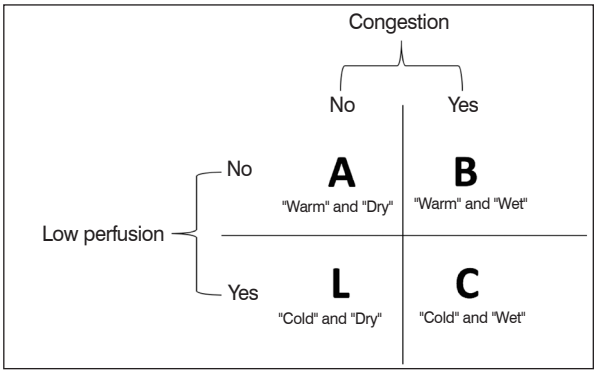

Figure 5. Hemodynamic profile.

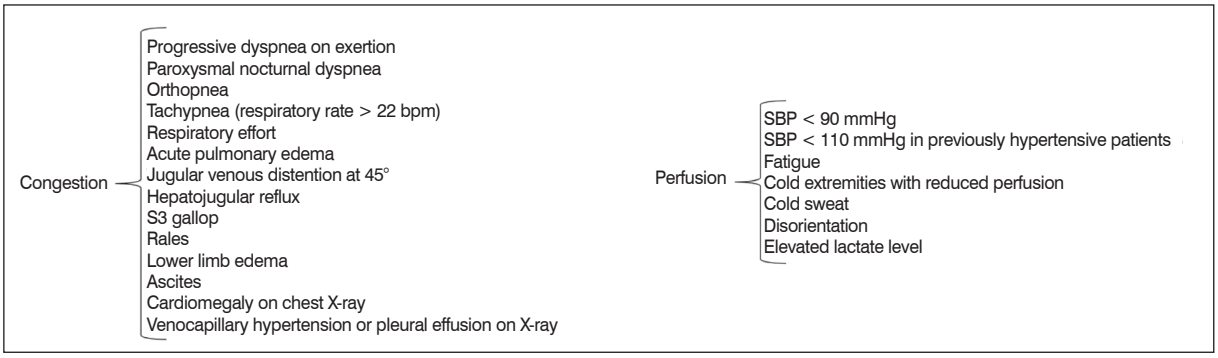

Figure 4. Signs and symptoms of congestion and low perfusion. SBP, systolic blood pressure.

strongly indicates a diagnosis of AHF.[17,18] NP levels present high sensitivity; the presence of serum BNP < 100 pg/mL and NT-proBNP < 300 pg/mL almost always excludes the diagnosis of AHF,[19-22] while intermediate serum levels of BNP require correlation to confirm the diagnosis.

One should pay attention to patients treated with sacubitril-valsartan since the inhibition of neprilysin by sacubitril promotes an elevation in serum BNP levels but not NT-proBNP, interfering with the diagnostic accuracy of BNP levels.[23]

Other important laboratory exams include troponin levels (suggestive of ACS, myocarditis, or Takotsubo syndrome), renal function, hemogram, electrolytes, thyroid function, liver enzymes, glycemia, venous blood gases, and venous lactate levels.[24]

Electrocardiography

Among the imaging exams, findings of 12-lead ECG help one narrow down the etiology of HF and the cause of decompensation, such as ACS, tachyarrhythmia, or bradyarrhythmia, which demand immediate therapeutic intervention.[25]

Chest X-rays should be performed since it allows the evaluation of the cardiac area and pulmonary congestion and helps in the differential diagnosis of thoracic and pulmonary causes of dyspnea.[26]

Echocardiography should be performed in all patients within the first 48 hours after admission, especially in those patients with cardiogenic shock and/or new-onset acute HF since it allows for definitions of etiological factors and degrees of ventricular dysfunction, assessments of pulmonary and systemic congestion, and the identification of mechanical factors to enable early targeting of therapy on admission.

Chest ultrasonography is complementary to the bedside clinical assessment and can be performed by a non-specialist with high accuracy for the detection of pulmonary and systemic congestion due to its portability, which allows for repeated evaluations and provides clinical and hemodynamic monitoring in response to therapy.[27-30]

Coronary angiography should be implemented upon admission to evaluate a suspected diagnosis of ACS or Takotsubo syndrome.[31,32]

Cardiac magnetic resonance imaging has significant accuracy for functional and morphological evaluations in patients with suspected acute myocarditis or Takotsubo syndrome; however, this method is used when the patient is clinically stable.[5,7,10]

RISK STRATIFICATION

The patient should immediately be stratified based on the risk on admission to determine the need of hospitalization according to the patient's severity. According to the hemodynamic clinical profile, the most appropriate therapy should be implemented, and the patient should be directed to the appropriate inpatient unit.

The estimate of the patient's admission risk profile of in-hospital mortality should be established based on the clinical risk profile, which evaluates the clinical presentation of acute HF (Figure 6) and the risk score.[11,33,34] The most validated scoring system is the ADHERE risk scale, which uses the variables systolic blood pressure, urea, and serum creatinine levels. (Table 4)[35]

The great majority of patients (approximately 77%) with AHF present with a low or low-intermediate risk with the absence of decompensated cardiovascular comorbidities. These patients can be treated in hospital observation units, with the possibility of 50% of these patients being discharged after checkup without the need for admission and up to 80% being discharged within 72 hours.[36]

The observation unit may be structured in any physical hospital unit and conveys clinical benefits that involve reducing the hospitalization time with clinical safety demonstrated by the low rate of in-hospital complications and hospital readmission in 30 days, with a favorable cost-effectiveness profile.[37,38]

HOSPITALIZATION

Upon the admission of patients with the clinical suspicion of acute HF, it is important to identify whether the patient is in immediate risk of in-hospital mortality. Causal factors should be assessed, including respiratory failure, acute coronary syndrome, acute pulmonary edema, cardiogenic shock, arrhythmias, and

Table 4. ADHERE risk scale of in-hospital mortality.

Risk profile	BUN (mg/dL)	SBP (mmHg)	Mortality (%)
Low	≤ 43	≥ 115	2.14
Intermediate 1	≤ 43	≤ 115	5.49
Intermediate 2	≥ 43	≥ 115	6.4
Intermediate 3	≥ 43 (Cr< 2.7)	≤ 115	12.28
High	≥ 43 (Cr ≥ 2.7)	≤ 115	21.9

BUN, blood urea nitrogen; SBP, systolic blood pressure; Cr, creatinine.

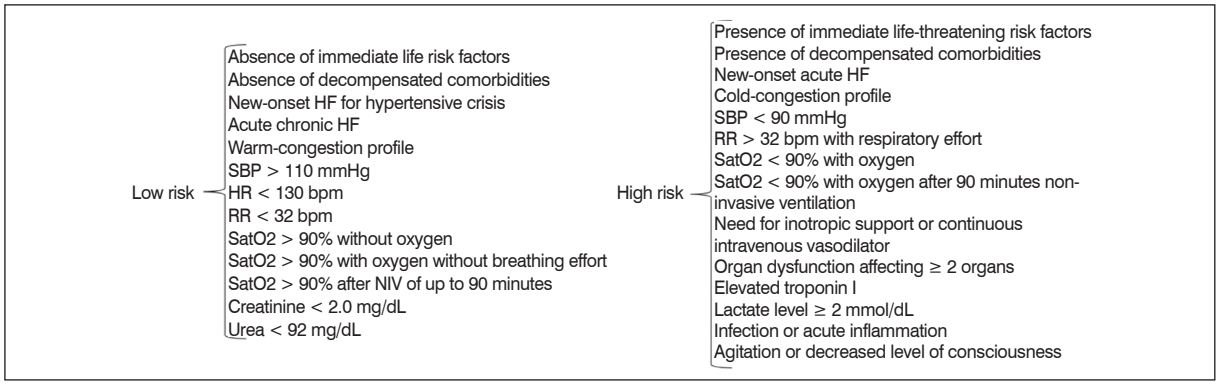

Figure 6. Indicators of clinical risk profile on admission for acute heart failure (HF). SBP, systolic blood pressure; HR, heart rate; RR, respiratory rate; SatO2, oxygen saturation; NIV, non-invasive ventilation.

stroke. These patients should be examined and treated as soon as possible, ideally within the first 30 minutes of admission. An early specific therapeutic intervention is the most important factor in the in-hospital prognosis evolution of these patients.[5,7,44]

There are four main steps in the patient's admission that should be followed to achieve clinical benefits: (1) early diagnosis and early and intense treatment to reduce the congestion associated with acute HF, as the main therapeutic target in acute HF is the reduction of congestion present in about 85% of patients,[39,40,5] (2) clinical monitoring of HR, RR, BP, ECG, and SatO2, control of diuresis, and laboratorial exams in a systematic and frequent way so that all changes are detected and corrected early to achieve clinical and hemodynamic improvement; (3) therapeutic orientation and pre-discharge life habits; and (4) clinical and laboratory reassessment within 7 days after hospital discharge.[41-43]

The risk profile of these patients should be reassessed frequently, as they can evolve with worsening of risk, indicating the need for change in the therapeutic strategy.

According to the Brazilian Guidelines published in 2018,[44] Figure 7 shows the initial approach to patients with acute HF, while Figure 8 shows the therapeutic approach in accordance with the clinical classification of patients with acute HF.

For some aspects that exceed the scope of the current review, such as indications for hospitalization and invasive monitoring, cardiogenic shock, and circulatory assistance using devices, the 2018 Guideline[44] can also be used.

CONFLICTS OF INTEREST

The author declares that he has no conflicts of interest in this work.

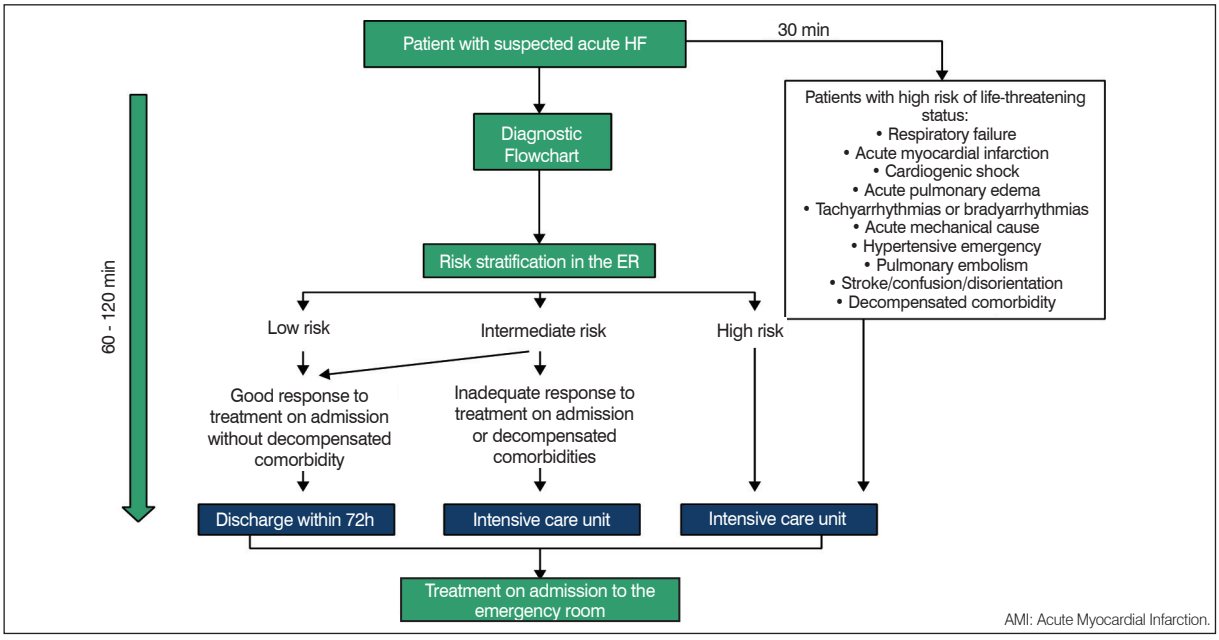

Figure 7. Flowchart of approach to admission for acute heart failure (HF) in the emergency room (ER).

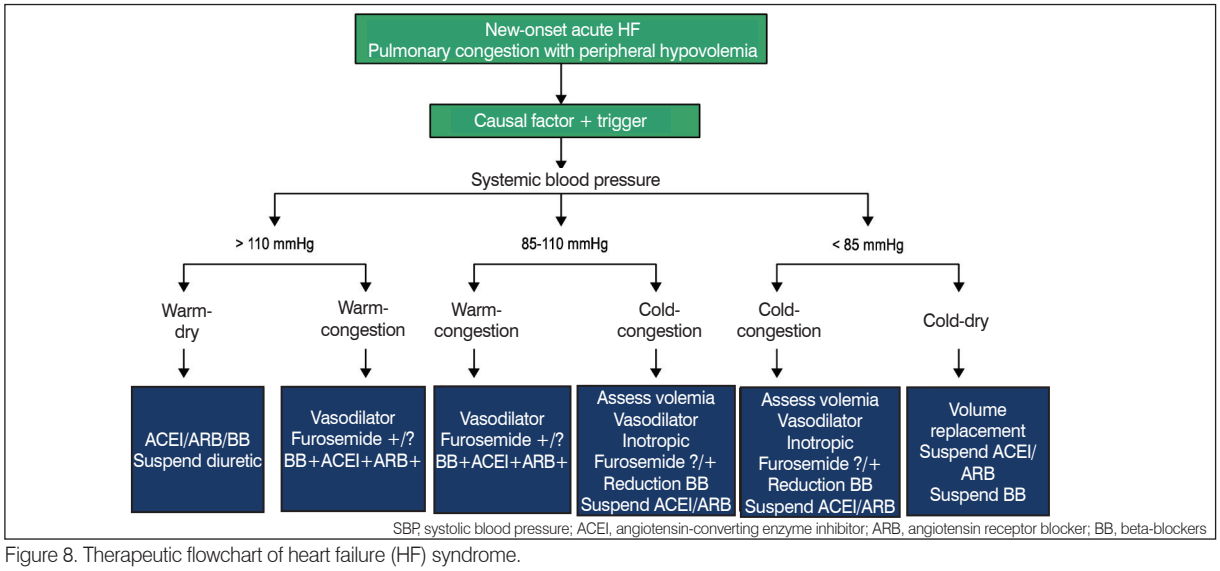

Figure 8. Therapeutic flowchart of heart failure (HF) syndrome.

REFERENCES

1. Metra M, Felker GM, Zacà V, Bugatti S, Lombardi C, Bettari L, et al. Acute heart failure: multiple clinical profiles and mechanisms require tailored therapy. Int J Cardiol. 2010;144(2):175-9.
2. Filippatos G, Zannad F. An introduction to acute heart failure syndromes: definition and classification. Heart Fail Rev. 2007;12(2):87-90.
3. DataSUS. Internações por insuficiência cardíaca em 2017. Acesso em 6 2018. Disponível em: http://datasus.saude.gov.br/informacoes-de-saude/tabnet.
4. Albuquerque DC, Neto JD, Bacal F, Rohde LE, Bernardez-Pereira S, Berwanger O, et al. I Brazilian Registry of Heart Failure - Clinical Aspects, Care Quality and Hospitalization Outcomes. Arq Bras Cardiol. 2015;104(6):433-42.
5. Mebazaa A, Yilmaz MB, Levy P, Ponikowski P, Peacock WF, Laribi S, et al. Recommendations on pre-hospital & 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. Eur J Heart Fail. 2015;17(6):544-58.
6. Peacock WF, Emerman C, Costanzo MR, Diercks DB, Lopatin M, Fonarow GC. Early vasoactive drugs improve heart failure outcomes. Congest Heart Fail. 2009;15(6):256-64.
7. Ponikowski P, Voors AA, Anker SD, Bueno H, Cleland JGF, Coats AJS, 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 Heart J. 2016;37(27):2129-200.
8. Ponikowski P, Jankowska EA. Pathogenesis and clinical presentation of acute heart failure. Rev Esp Cardiol (Engl Ed). 2015;68(4):331-7.
9. Rudiger A, Harjola VP, Müller A, Mattila E, Säila P, Nieminen M, et al. Acute heart failure: clinical presentation, one-year mortality and prognostic factors. Eur J Heart Fail. 2005;7(4):662-70.
10. Metra M, Felker GM, Zacà V, Bugatti S, Lombardi C, Bettari L, et al. Acute heart failure: multiple clinical profiles and mechanisms require tailored therapy. Int J Cardiol. 2010;144(2):175-9.
11. Miró Ò, Rossello X, Gil V, Martín-Sánchez FJ, Llorens P, Herrero-Puente P, et al. Predicting 30-Day Mortality for Patients With Acute Heart Failure in the Emergency Department: A Cohort Study. Ann Intern Med. 2017;167(10):698-705.
12. Taylor DM, Fui MN, Chung AR, Gani L, Zajac JD, Burrell LM. A comparison of precipitants and mortality when acute decompensated heart failure occurs in the community and hospital settings. Heart Lung Circ. 2012;21(8):439-43.
13. Mentz RJ, Kelly JP, von Lueder TG, Voors AA, Lam CS, Cowie MR, et al. Noncardiac comorbidities in heart failure with reduced versus preserved ejection fraction. J Am Coll Cardiol. 2014;64(21):2281-9.
14. Nohria A, Tsang SW, Fang JC, Lewis EF, Jarcho JA, Mudge GH, et al. Clinical assessment identifies hemodynamic profiles that predict outcomes in patients admitted with heart failure. J Am Coll Cardiol. 2003;41(10):1797-804.
15. Nohria A, Mielniczuk LM, Stevenson LW. Evaluation and monitoring of patients with acute heart failure syndromes. Am J Cardiol. 2005;96(6A):32G-40G.
16. Almeida Júnior GL, Esporcatte R, Rangel FO, Rocha RM, Gouvêa e Silva GeM, Tura BR, et al. [Therapy of advanced heart failure adapted to hemodynamic objectives acquired by invasive hemodynamic monitoring]. Arq Bras Cardiol. 2005;85(4):247-53.
17. Behnes M, Brueckmann M, Ahmad-Nejad P, Lang S, Wolpert C, Elmas E, et al. Diagnostic performance and cost effectiveness of measurements of plasma N-terminal pro brain natriuretic peptide in patients presenting with acute dyspnea or peripheral edema. Int J Cardiol. 2009;135(2):165-74.
18. Mueller C, Scholer A, Laule-Kilian K, Martina B, Schindler C, Buser P, et al. Use of B-type natriuretic peptide in the evaluation and management of acute dyspnea. N Engl J Med. 2004;350(7):647-54.
19. McCullough PA, Nowak RM, McCord J, Hollander JE, Herrmann HC, Steg PG, et al. B-type natriuretic peptide and clinical judgment in emergency diagnosis of heart failure: analysis from Breathing Not Properly (BNP) Multinational Study. Circulation. 2002;106(4):416-22.
20. Maisel AS, Krishnaswamy P, Nowak RM, McCord J, Hollander JE, Duc P, et al. Rapid measurement of B-type natriuretic peptide in the emergency diagnosis of heart failure. N Engl J Med. 2002;347(3):161-7.
21. Moe GW, Howlett J, Januzzi JL, Zowall H, Investigators CMIMoPWCH-FI-CS. N-terminal pro-B-type natriuretic peptide testing improves the management of patients with suspected acute heart failure: primary results of the Canadian prospective randomized multicenter IMPROVE-CHF study. Circulation. 2007;115(24):3103-10.
22. Januzzi JL Jr, Chen-Tournoux AA, Moe G. Amino-terminal pro-B-type natriuretic peptide testing for the diagnosis or exclusion of heart failure in patients with acute symptoms. Am J Cardiol. 2008;101(3A):29-38.
23. Solomon SD, Zile M, Pieske B, Voors A, Shah A, Kraigher-Krainer E, et al. The angiotensin receptor neprilysin inhibitor LCZ696 in heart failure with preserved ejection fraction: a phase 2 double-blind randomised controlled trial. Lancet. 2012;380(9851):1387-95.
24. Thiele H, Zeymer U, Neumann FJ, Ferenc M, Olbrich HG, Hausleiter J, et al. Intraaortic balloon support for myocardial infarction with cardiogenic shock. N Engl J Med. 2012;367(14):1287-96.
25. Zannad F, Mebazaa A, Juillière Y, Cohen-Solal A, Guize L, Alla F, et al. Clinical profile, contemporary management and one-year mortality in patients with severe acute heart failure syndromes: The EFICA study. Eur J Heart Fail. 2006;8(7):697-705.
26. Chakko S, Woska D, Martinez H, de Marchena E, Futterman L, Kessler KM, et al. Clinical, radiographic, and hemodynamic correlations in chronic congestive heart failure: conflicting results may lead to inappropriate care. Am J Med. 1991;90(3):353-9.
27. Martindale JL, Wakai A, Collins SP, Levy PD, Diercks D, Hiestand BC, et al. Diagnosing Acute Heart Failure in the Emergency Department: A Systematic Review and Meta-analysis. Acad Emerg Med. 2016;23(3):223-42.
28. Moore CL, Rose GA, Tayal VS, Sullivan DM, Arrowood JA, Kline JA. Determination of left ventricular function by emergency physician echocardiography of hypotensive patients. Acad Emerg Med. 2002;9(3):186-93.
29. Gargani L, Frassi F, Soldati G, Tesorio P, Gheorghiade M, Picano E. Ultrasound lung comets for the differential diagnosis of acute cardiogenic dyspnoea: a comparison with natriuretic peptides. Eur J Heart Fail. 2008;10(1):70-7.
30. Liteplo AS, Marill KA, Villen T, Miller RM, Murray AF, Croft PE, et al. Emergency thoracic ultrasound in the differentiation of the etiology of shortness of breath (ETUDES): sonographic B-lines and N-terminal pro-brain-type natriuretic peptide in diagnosing congestive heart failure. Acad Emerg Med. 2009;16(3):201-10.
31. Hochman JS, Sleeper LA, Webb JG, Sanborn TA, White HD, Talley JD, et al. Early revascularization in acute myocardial infarction complicated by cardiogenic shock. SHOCK Investigators. Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock. N Engl J Med. 1999;341(9):625-34.
32. van Diepen S, Katz JN, Albert NM, Henry TD, Jacobs AK, Kapur NK, et al. Contemporary Management of Cardiogenic Shock: A Scientific Statement From the American Heart Association. Circulation. 2017;136(16):e232-e68.
33. Collins S, Storrow AB, Albert NM, Butler J, Ezekowitz J, Felker GM, et al. Early management of patients with acute heart failure: state of the art and future directions. A consensus document from the society for academic emergency medicine/heart failure society of America acute heart failure working group. J Card Fail. 2015;21(1):27-43.
34. Peacock F, Beckley P, Clark C, Disch M, Hewins K, Hunn D, et al. Recommendations for the evaluation and management of observation services: a consensus white paper: the Society of Cardiovascular Patient Care. Crit Pathw Cardiol. 2014;13(4):163-98.
35. Fonarow GC, Adams KF, Abraham WT, Yancy CW, Boscardin WJ, ADHERE Scientific Advisory Committee SuG, and Investigators. Risk stratification for in-hospital mortality in acutely decompensated heart failure: classification and regression tree analysis. JAMA. 2005;293(5):572-80.
36. Collins SP, Pang PS, Fonarow GC, Yancy CW, Bonow RO, Gheorghiade M. Is hospital admission for heart failure really necessary?: the role of the emergency department and observation unit in preventing hospitalization and rehospitalization. J Am Coll Cardiol. 2013;61(2):121-6.
37. Zuckerman RB, Sheingold SH, Orav EJ, Ruhter J, Epstein AM. Readmissions, Observation, and the Hospital Readmissions Reduction Program. N Engl J Med. 2016;374(16):1543-51.
38. Collins SP, Schauer DP, Gupta A, Brunner H, Storrow AB, Eckman MH. Cost-effectiveness analysis of ED decision making in patients with non-high-risk heart failure. Am J Emerg Med. 2009;27(3):293-302.
39. Filippatos G, Zannad F. An introduction to acute heart failure syndromes: definition and classification. Heart Fail Rev. 2007;12(2):87-90.
40. Wuerz RC, Meador SA. Effects of prehospital medications on mortality and length of stay in congestive heart failure. Ann Emerg Med. 1992;21(6):669-74.
41. Collins SP, Storrow AB. Acute heart failure risk stratification: can we define low risk? Heart Fail Clin. 2009;5(1):75-83, vii.
42. Peacock WF. Using the emergency department clinical decision unit for acute decompensated heart failure. Cardiol Clin. 2005;23(4):569-88, VIII.
43. Butler J, Hanumanthu S, Chomsky D, Wilson JR. Frequency of low-risk hospital admissions for heart failure. Am J Cardiol. 1998;81(1):41-4.
44. Diretriz Brasileira de Insuficiência Cardíaca Crônica e Aguda. Arq Bras Cardiol 2018;111(3):436-539.