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Clinical diagnosis of acute heart failure

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Abbreviations
PND: paroxysmal nocturnal dyspnea
VAS: visual analog scales
LVEF: left ventricular ejection fraction
AHF: acute heart failure
NYHA: New York Heart Association
PCWP: pulmonary capillary wedge pressure
JVP: jugular venous pressure
HJR: hepatojugular reflux
ED: Emergency Department

ABSTRACT
Deep knowledge of the relevance of signs and symptoms of acute heart failure (AHF) would lead to better diagnostic and decision-making strategies. The signs and symptoms of congestion are easily assessed bedside. It is interesting to analyze the sensitivity, specificity and the importance of each of them in the diagnosis of AHF. More specific signs (jugular venous pressure) are more difficult to determine, and the agreement among physicians examining the same patient tends to be poor. Both the symptoms and the classic signs of AHF can have a high sensitivity (dyspnea) or specificity (in the case of orthopnea, paroxysmal nocturnal dyspnea), but not both at the same time. Given the limitations of these findings, it is necessary to find support on other studies such as imaging tests and biomarkers for diagnostic confirmation.
Keywords: Acute heart failure, Signs, Symptoms, Diagnosis

Diagnóstico clínico de la insuficiencia cardíaca aguda

RESUMEN
El conocimiento profundo de la relevancia de los signos y síntomas de insuficiencia cardíaca aguda (ICA) daría lugar a mejores estrategias de diagnóstico y toma de decisiones. Los signos y síntomas de congestión son fácilmente evaluables a pie de cama. Es interesante analizar la sensibilidad, la especificidad y el valor de cada uno de ellos en el diagnóstico de la ICA. Los signos más específicos (presión venosa yugular) son más difíciles de determinar y la concordancia entre médicos que examinan el mismo paciente tiende a ser deficiente. Tanto los síntomas como los signos clásicos de ICA pueden tener una alta sensibilidad (disnea) o especificidad (en el caso de la ortopnea, disnea paroxística nocturna), pero no ambas a la vez. Las limitaciones de estos hallazgos hacen que para la confirmación diagnóstica haya que apoyarse en otros estudios como son las pruebas de imagen y los biomarcadores.
Palabras clave: *Insuficiencia cardíaca aguda, Signos, Síntomas, Diagnóstico*

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INTRODUCTION

Acute heart failure (AHF) is defined as the appearance or worsening of signs and symptoms of heart failure that require urgent treatment[1]. It constitutes one of the main causes of morbidity and mortality in the world, and it is a global health problem with more than one million admissions per year in Eu-

rope and the USA[2].

Heart failure affects approximately 1-2% of the population in developed countries and up to 10% of the elderly, who represent a significant proportion of the population in hospital Emergency Departments (ED)[3]. In the last decade, the flow of patients over 70 years of age has progressively increased and half of them are frail, which is why they must be approached carefully[4]. Frailty is a state of vulnerability associated with an increase in mortality, hospitalization, functional deterioration, institutionalization, and a worsening of quality of life in the face of a stressful situation. This frailty is especially present in older subjects with AHF and its presence increases the risk of poorer outcomes[5,6].

There are different classifications of AHF, including the phenotypic classification based on the state of congestion and perfusion, which divides it into four clinical categories[7]. Despite the considerable variation in clinical profiles and the substantial heterogeneity of the underlying causes, the vast majority of patients with AHF present signs and symptoms of pulmonary and systemic congestion rather than low cardiac output[8]. This classification is very useful in the initial evaluation at the bedside, in patients with AHF who are seen in the ED, without the need to spend a lot of time and invasive measures or techniques that require prior training. In the ED, the predominant group is the congestive and well-perfused (warm-wet) group, who are discharged without the need for admission from the ED in 82%[9]. Various studies, conducted both in hospitalized patients with AHF and in EDs, show that congestion is present in more than 90% of cases, regardless of the left ventricular ejection fraction (LVEF)[9]. In fact, patients with heart failure who do not respond satisfactorily to decongestant treatment during admission or who maintain some degree of congestion after discharge have a poorer prognosis, with an increase in adverse events during follow-up, including the need for new hospitalization or death[10,11].

At a time when diagnostic tools are constantly being developed, in-depth knowledge of the relevance of data and clinical histories would lead to better diagnostic strategies[12]. The symptoms and signs of congestion are easily assessable at the bedside, regardless of the care settings in which the patient with AHF is seen: primary care outpatient clinic or other specialties, prehospital emergency services, EDs, or during hospitalization. They only require an optimal medical history and a careful physical examination. Currently, the large number of complementary examinations that support clinical diagnosis (radiology, biomarkers or ultrasound) seem to have displaced the importance of clinical semiology[12-14], which has the advantage of its accessibility. Furthermore, it is possible that some of these symptoms and signs of congestion may be associated with a poor outcome. Although this has not been evaluated, particularly in the patient treated in the emergency room. There is room for improvement in EDs in terms of their diagnostic-therapeutic capacities for patients with AHF, and opportunities are being detected to ensure more effective continuity of care[15].

The clinical history should include cardiovascular risk factors, unhealthy lifestyles, precipitating factors, which include dietary and treatment transgressions, and non-cardiac diseases. Special care must be taken to know exactly the patient's symptoms, as well as a thorough physical examination. Nor should regional variability be forgotten, since there are differences both in the baseline characteristics and in the clinical behavior of heart failure episodes[16].

Given that the clinical history and physical examination are tools available to everyone and immediately implemented, it is interesting to analyze the sensitivity, specificity, and predictive value of each of the symptoms and signs in the diagnosis of AHF.

METHOD

A search for articles was carried out in PubMed and Google Scholar with the keywords *"acute heart failure"* and *"emergency"*. Retrospective and prospective studies, systematic reviews and meta-analyses, clinical guidelines and narrative reviews focused on the diagnosis of AHF, including clinical history and physical examination were included. The literature review was limited to studies published in English and Spanish.

Emergency physicians, experts in AHF and with experience in the critical appraisal of the literature, reviewed all the articles and decided which studies to include for the review, by consensus. Special attention was paid to articles relevant to emergency medicine. 45 articles were selected for inclusion in this review.

Data regarding: dyspnea, orthopnea, paroxysmal nocturnal dyspnea (PND), rales, peripheral edema, bendopnea, body weight, jugular venous pressure

(JVP), hepatojugular reflux (HJR), and third heart sound were analyzed.

SYMPTOMS AND SIGNS

Dyspnea

Dyspnea, defined as a sensation of difficulty or discomfort in breathing, and which includes the physician's perception of this difficulty by the patient, is one of the most frequent symptoms in the presentation of AHF. The prevalence of heart failure in patients who consult for dyspnea ranges from 25 to 59%, depending on the studies[17-20]. This symptom is the one that most frequently leads patients to go to the ED, and it is also an important marker to evaluate the reduction of congestion[21]. However, by themselves, or combined with other signs or symptoms of congestion, they are insufficient to obtain a reasonable diagnostic value[20,21]. The sensitivity and specificity of this symptom are 0.89 and 0.51 respectively, according to the meta-analysis conducted by Renier *et al.*[17].

Acute dyspnea is one of the leading causes of hospitalization among adults, especially in elderly patients. The subjective experience is not always consistent with the physical findings[20]. Currently, there is no validated method to evaluate it, although a consensus has been proposed to facilitate the standardization of the measurement. Both the Likert scale and the visual analog scales (VAS) can be used for this[21]. URGENT-DYSPNEA was designed to describe the symptomatic response to initial conventional therapy in patients with AHF, very early in their hospital course. The study presents a high degree of concordance between a 5-point Linkert scale, a VAS, and a 7-point Linkert, in dyspneic patients in the sitting position[22]. The VAS measures the absolute severity of the symptoms and also measures them at baseline. This scale may be more sensitive to subtle changes in symptoms. On the other hand, the Likert scale seems to be closer to daily clinical practice, where symptoms are evaluated quite roughly and mainly in comparison with the baseline level. The differences between the two methods explain why the Likert scale was more sensitive to early changes in symptoms, while the VAS was able to show a persistent improvement, even at the end of hospitalization[23].

In patients with dyspnea, one of the most useful complementary examinations for the diagnosis of AHF is cardiopulmonary ultrasound using the POCUS (point-of-care ultrasound) algorithm, which includes lung ultrasound and three echocardiographic measurements performed in an apical four-chamber plane, in addition to MAPSE (mitral annular plane systolic excursion) and Doppler mitral flow and tissue in the lateral mitral annulus[24].

Orthopnea

Orthopnea is a symptom with a low sensitivity (0.50), but is more specific (0.77)[12]. It is related to the pulmonary capillary wedge pressure (PCWP), which has a sensitivity of approximately 90%. The supine position involves the mobilization of fluid from the dependent venous reservoirs in the abdomen and lower limbs, which increases venous return (250-500 mL) to the thoracic compartment. As a result, pulmonary venous and capillary pressures increase, raising the already high filling pressures of both ventricles and can lead to interstitial pulmonary edema, reduced lung compliance, increased airway resistance, and dyspnea. Orthopnea can be assessed by asking the patient to remain in the supine position for a defined time (2 minutes), while respiratory rate and dyspnea are monitored[21]. Persistent orthopnea is associated with a longer hospital stay.

It has been hypothesized that postural changes may affect lung function and dyspnea in AHF, in relation to changes in pulmonary blood volume and its mechanical alteration. Small changes in blood flow and airway mucosal conductance are related to changes in lung function. The decrease in this lung function when going from the upright to the supine position has been demonstrated both in healthy subjects and in patients with heart failure. For the latter, postural changes can also affect the bronchial vasculature and alter the caliber of the airways. It has been suggested that, even in mild heart failure, excessive myocardial stretch may block bronchial vasoconstriction and produce congestion of the mucosa and bronchi[25].

Both dyspnea and orthopnea are symptoms that may or may not be cardiac in origin. Severity may reflect the rate of increase in PCWP as well as its absolute value. Functional lung capacity has been shown to predict mortality and hospitalizations. As expected, dyspnea on exertion is the most persistent and common symptom at discharge. Patients without locomotor problems should be able to walk for at

least 6 minutes on level ground without undue respiratory distress and complete at least 200 meters without postural symptoms of dizziness or lightheadedness[21]. Assessment of orthopnea is best done with the VAS compared to the 5-point Likert scale. The VAS may present greater precision for the changes in patients with AHF and dyspnea in the supine position[22].

Paroxysmal nocturnal dyspnea

It is a symptom that occurs during sleep, it is due to the nocturnal redistribution of liquids that causes an increase in left ventricular filling pressure and causes the patient to wake up with intense shortness of breath, which is relieved by sitting upright[12]. It is a major symptom that often precedes acute pulmonary edema by several nights/days. Symptoms similar to chronic obstructive pulmonary disease can occur[21]. Like orthopnea, it is a specific symptom (0.84), but not very sensitive (0.41)[12]. The values of sensitivity and specificity of this, and the other symptoms commented on, are shown in the **table**[26].

Subclassifying dyspnea at rest, immediately on mild exertion, orthopnea, and PND may improve the specificity of elevated filling pressures. An analysis of the EPICA registry identified the descriptors dyspnea at rest, orthopnea, and previous PND as highly specific (99%) for heart failure[27].

Rales or crackles

The presence of rales on pulmonary auscultation may indicate fluid overload, but it is also less reliable since its presence has a sensitivity of approximately 60% and a specificity close to 70%[28]. It is a sign of a high presence in patients with AHF. In the ADHERE and OPTIMIZE-HF registries, crackles were observed in approximately two-thirds of the patients[19,29].

In the setting of AHF, crackles and wheezing are findings suggestive of fluid retention, elevated left ventricular filling pressures, and pulmonary edema. If they are not corrected, they can progress to the accumulation of fluids in the pleural space. These thoracic manifestations generally occur with other manifestations of fluid overload and congestion: dyspnea, presence of a third sound or gallop rhythm, loud second sound, and elevated JVP. Chest findings help assess the severity of fluid retention, the need for hospitalization, and more aggressive treatment. A pleural effusion, caused by heart failure, usually begins and is more prominent on the right side. Isolated left pleural effusion is unusual and justifies looking for other causes[30].

Peripheral edema

Edema in the lower limbs has a sensitivity of 0.5 and a specificity of 0.78[28]. It is more useful in men than in women to diagnose heart failure, its precise cause is unknown, although venous insufficiency accompanied by pelvic obstruction of venous blood flow is commonly blamed[31]. It is one of the signs most likely to cause a doctor to consider a diagnosis of heart failure. Edema is relatively common in decompensated patients and was present in two-thirds of the patients in the ADHERE[19] and OPTIMIZE-HF registries[29]. Its specificity as a sign increases in the presence of elevated JVP and, perhaps because it is so easily appreciated on physical examination, the severity of the edema on admission is one of the most predictive variables of the length of hospitalization for heart failure[32].

It is usually associated with elevated right atrial pressure, most often due to left heart failure. During hospitalization, it generally responds to diuretic treatment. It must be taken into account that the edema can be redistributed to dependent areas, such as the sacral region, a situation that can be overlooked.

Table. Sensitivity and specificity of the signs and symptoms of congestion[26].

Signs and symptoms	Sensitivity	Specificity
Dyspnea	0.31	0.70
Orthopnea	0.50	0.77
Paroxysmal nocturnal dyspnea	0.41	0.84
Elevated jugular venous pressure	0.39	0.92
Rales	0.60	0.78
Peripheral edema	0.50	0.78
Third heart sound or gallop rhythm	0.13	0.99
Hepatojugular reflux	0.24	0.96
Bendopnea	0.73	0.65

Apparent improvement without weight loss means fluid redistribution. One limitation in the evaluation of this sign is that it may not be related to high filling pressures but to extravascular volume changes resulting from low plasma oncotic pressure, high vascular permeability, or both. The specificity of edema improves when accompanied by increased JVP[21].

Bendopnea

Bendopnea, shortness of breath that occurs after forward flexion of the trunk during the first 30 seconds, for example, when putting on shoes or tying shoelaces; has been associated with advanced heart failure[33], with a sensitivity of 0.73 and a specificity of 0.65[26]. Patients with bendopnea have a hemodynamic profile characterized by increased left ventricular filling pressure and decreased cardiac index[34]. The anterior trunk flexion maneuver causes an increase in the filling pressures that these patients already have high at baseline. Patients with bendopnea have symptoms of right and left heart failure, including orthopnea, PND, oliguria, elevated JVP, and abdominal fullness (abdominal distension and pressure). Bendopnea is associated with increased short-term mortality in patients with advanced NYHA (New *York Heart Association)* functional class and causes moderate-severe limitation of quality of life[35].

Hemodynamically, this posture, like squatting or the Valsalva maneuver, increases left ventricular afterload, while increasing preload from increased abdominal pressure. For this reason, the Valsalva maneuver is of particular interest at the bedside as an aid in differentiating advanced stages of diseases due to left ventricular dysfunction[36].

When the different types of dyspnea are analyzed: exertional dyspnea, orthopnea, PND and bendopnea; the latter is the only one that is not related to the presence of respiratory disease or coronary disease, so it can be especially useful to differentiate heart failure from other groups of diseases that are also associated with dyspnea, and its presence is greater in patients with preserved LVEF than in those with a reduced LVEF (51.5 vs. 37.9%)[33,34].

The systematic review and meta-analysis by Pranata *et al.*[37] concluded that bendopnea is associated with dyspnea, orthopnea, PND, elevated JVP, and abdominal fullness. NYHA IV functional class was more prevalent in patients with bendopnea, whereas classes I, II, and III were inversely correlated. This may explain why, although it may be present in all stages of heart failure, it is more specific for advanced heart failure, and is associated with increased mortality[37].

Body weight

This is one of the major Framingham criteria, specifically a weight loss of more than 4.5 kg with treatment.

The increase in body weight is a manifestation derived from the retention of sodium and water, and more specifically from their daily variation, so a sharp change in body weight is a reasonable indicator of a disturbance in fluid balance. The measurement should be done in the most consistent manner, by placing the scale on a smooth, solid surface. Variations are not always due to intravascular volume changes; since, in the EVEREST trial, the patients who received tolvaptan lost weight, but the edema was still present. The degree of absolute weight loss may not be as important in patients with hypertensive AHF, since some may be euvolemic with pulmonary congestion, but not systemic[32].

Good discharge planning requires taking into account a target weight (dry weight), and even below it. Reducing congestion with the use of diuretic treatment involves monitoring renal function and the hemodynamic situation. With adequate diuresis, worsening and hypotension can be prevented[21]. It must be taken into account that worsening renal function in the first 48 hours is associated with higher mortality, and this increased risk is concentrated during the first trimester[38]. In this sense, patients with worsening renal function in the context of adequate decongestion and clinical improvement do not have an adverse prognosis[39].

The relationship between increased body weight and the presence of an exacerbation of heart failure is not entirely clear. There are studies that show little daily correlation between responses to symptom questionnaires and self-recorded daily weights, with a modest incidence (25-40%) of weight gain in the weeks prior to admission for AHF. In contrast, in a case-control study of NYHA III heart failure patients whose weight was measured daily, hospitalization for heart failure was associated with a daily increase in body weight during the week prior to hospitalization[40]. In the WHARF trial (Weight Monitoring in Heart Failure)[41], NYHA III and IV patients with reduced LVEF were randomly assigned to conventional therapy or to follow-up with data transmitted by telephone, which included daily weight measurements

and questions about heart failure symptoms. The investigators concluded that daily electronic weight monitoring may reduce mortality and warrants further study. In turn, the Telephone Intervention in Chronic Heart Failure randomized trial (DIAL[42]) demonstrated a significant improvement in the primary combined endpoint of all-cause mortality or hospital admission for heart failure, mainly due to the reduction in hospitalizations.

Instead, the quantum of weight loss during an AHF hospitalization and the relationship of weight loss to subsequent outcomes is an area of growing interest. In the ADHERE[29] registry, a substantial percentage of hospitalized patients lost less than 5 pounds (2.3 kilograms) of weight or gained weight during hospitalization. In the ESCAPE study, weight was measured at enrollment and at discharge. The median weight loss was 2.8 kilograms and 17% of the patients increased or did not lose weight[29,32]. The results of the EVEREST trial, which analyzed the use of tolvaptan in patients with depressed LVEF, showed a significantly greater loss of body weight on days 1 and 7, or at discharge, in the tolvaptan group, which was associated with improvements in dyspnea on day 1 and edema on day 7[32].

Jugular venous pressure and hepatojugular reflux

The HJR and the elevated JVP have a specificity of 0.93 and 0.87; and a sensitivity of 0.14 and 0.37 respectively[28].

JVP reflects right atrial pressure, its presence is specific and sensitive to elevated PCWP in patients with heart failure[43]. If performed correctly and by experienced clinicians, the JVP estimate is extremely accurate[21]. Although the JVP directly represents the filling pressure of the right cavities, the right atrial pressure often reflects the filling pressures of the left ventricle in patients with chronic heart failure[32]. The finding of an elevated JVP on physical examination provides important and independent prognostic information in patients with heart failure and is associated with an increased risk of hospitalization[44].

Strategies have been suggested to improve the measurement of PVY since it is often limited by phenotype and comorbidity. In addition, in some individuals with pulmonary hypertension (PH) or tricuspid regurgitation, a high JVP is required to maintain adequate filling pressures on the left side, so normalizing the JVP is neither indicated nor beneficial. The HJR can also be used to assess elevated pressure and is sensitive and reliable. It is a simple measure of congestion and is therefore a potential target for evaluating treatment[21].

Regarding the interpretation that JVP and HJR are related to central venous pressure (CVP), there are reasonable doubts. The *Basic in Acute Shortness of Breath review studies* substudy did not find this relationship. CVP was similar in patients with normal neck veins, with HJR, or with elevated JVP. Consequently, pathological examinations of the neck veins lacked sensitivity (68.5%) and specificity (28.5%) in the diagnosis of elevated CVP[45].

HJR is assessed by an increase in JVP induced by 10 seconds of continuous pressure on the abdomen. A positive HJR, in the absence of isolated right ventricular dysfunction, reliably predicts PCWP greater than 15 mmHg. Because the internal JVP is not known prior to examination, it is important to examine the patient in a 90° sitting position, in addition to examining both sides of the neck due to anatomical variations[32]. Volume overload in the setting of right ventricular dysfunction will result in liver enlargement and often the presence of a firm and tender liver edge. Along with JVP, liver size in heart failure is an indicator of intravascular volume status[30].

Third heart sound or gallop rhythm (S3)

The presence of an S3 is the sign with the highest specificity (0.97-0.99), but its sensitivity is low (0.13). In addition, it can be difficult to detect in the emergency setting and the inter-observer reliability can be poor[28]. Patients with heart failure may have S3 as a result of low ventricular compliance, increased filling pressures, or early diastolic filling velocity[34]. For its examination, the bell of the stethoscope should be placed on the point of maximum impulse, preferably with the patient in left lateral decubitus, which will increase the probability of detecting an S3[32].

The finding of elevated JVP or an S3 on physical examination conveys important prognostic information in patients with symptomatic heart failure. The presence of these signs is associated with the need for hospitalization and with an increased risk of heart failure progression. Patients with elevated JVP, S3, or both are also at increased risk of death from all causes, as found in the *Studies in Left Ventricular Dysfunction trial*[82]. These associations hold even after adjustment for many other markers of HF severity, including LVEF, NYHA functional class, and blood sodium. It is not known why elevated JVP or the pres-

ence of S3 is associated with an increased risk of heart failure progression and risk of hospitalization, but these findings may increase confidence that bedside assessment is relevant and may give trainees further impetus to hone their physical examination skills[44].

CONCLUSIONS

The approach to the diagnosis of suspected acute heart failure in the emergency room should be based on the patient's symptoms and signs since they are easily identifiable during the initial assessment. The limitations of these findings, in terms of sensitivity and specificity, make it necessary to rely on other studies for diagnostic confirmation, such as imaging tests and biomarkers.

Excellence in the practice of the emergency physician is based on a good diagnosis, which leads to the timely initiation of appropriate treatment for the patient and an appropriate estimate of the severity of the episode, which helps in making decisions about the discharge or hospitalization of the patient.

The inconsistency of the specificities of the signs and symptoms corresponds to the fact that many of the symptoms of heart failure are due to volume overload or an increase in pressure with vascular redistribution and, therefore, are not specific. More specific signs, such as jugular venous pressure, are more difficult to determine, and agreement between different clinicians examining the same patient tends to be poor.

In general terms, both the classic symptoms and signs of acute heart failure may have high sensitivity (dyspnea) or specificity (in the case of orthopnea and paroxysmal nocturnal dyspnea), but not both at the same time. To make it even more difficult, it is known that the degree of interobserver concordance regarding the presence or absence of clinical data on acute heart failure is low. For this reason, in practice, the objective information offered by various complementary examinations is necessary; among them, biomarkers and ultrasound.

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