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GLOBAL CARDIOVASCULAR HEALTH (GY BUKHMAN, SECTION EDITOR)

Diagnosis and Management of Acute Heart Failure in Sub-Saharan Africa

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Abstract

Purpose of Review Acute heart failure (AHF) is a common emergency presentation in Sub-Saharan Africa (SSA). In the current review, we present the most recent data on the epidemiology of AHF in SSA and discuss recommended approaches to management in resource-limited settings, with a particular focus on primary and secondary facilities (e.g., health centers and district hospitals), where these patients often present.

Recent Findings AHF in SSA is most often due to hypertension, cardiomyopathies, and rheumatic heart disease. The etiology of AHF may be different in rural as compared with urban settings. Diagnostic tools for AHF are often lacking in SSA, especially at the first-level facilities. Point-of-care ultrasound (POCUS) and biomarker tests, such as brain natriuretic peptide (BNP), offer promise in helping to mitigate diagnostic challenges. POCUS can also help distinguish among types of heart failure and prompt the correct treatment strategy. Many of the drugs and equipment commonly used to treat AHF in resource-rich settings are lacking in SSA. However, some adaptations of commonly available materials may provide temporary alternatives.

Summary The epidemiology of AHF in SSA differs from that of high-income settings. Management of AHF at the first-level facility in SSA is an important and understudied problem. Simplified diagnostic and treatment algorithms rooted in knowledge of the local epidemiology should be developed and tested as part of broader efforts to combat cardiovascular disease in SSA.

Keywords Acute heart failure · Sub-Saharan Africa · Echocardiography

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Introduction

Acute heart failure (AHF) is a major public health problem in Sub-Saharan Africa (SSA), where the disease tends to strike patients during their most productive years of life and thus causes heavy social and economic burden on families and society [1, 2]. Although much has been published on how to diagnose and treat AHF in the high-income setting, there is little consensus around best practices within the context of SSA epidemiology and resource constraints. Here, we aim to provide an overview of the epidemiology of AHF presentations in SSA and its current management in resource-limited environments, with a particular focus on the advances made in the past 10 years and their possible application to care at primary and secondary-level facilities.

Epidemiology of Acute Heart Failure Presentations in Sub-Saharan Africa

Although the data is scant, AHF appears to be a common presentation in the SSA emergency setting. A recent meta-analysis reported between 9.4 and 42.5% of adult medical inpatient admissions in SSA is attributed to AHF [3••]. The mean age of these patients was between 36.5 and 61.5 years. Another recent survey of illnesses presenting to an emergency department in a regional referral center in Tanzania showed that almost 2% of cases were AHF and 3.2% were hypertensive emergencies [4•]. Mortality for these patients is also high, especially accounting for their relatively young age. In the THESUS registry, 17.8% of patients died at 180 days [5]. However, most of these patients were treated at referral-level facilities and thus this may not be reflective of the situation at first-level facilities. A recent registry of patients admitted for AHF to a rural Ugandan district hospital found a much higher 6-month mortality of 43% [6••]. This wide range of mortality rate is reflected a recent systemic review, which found 180-day mortality ranging from 15 to 57.8% among 13 SSA AHF prevalence studies [3••].

Most evidence suggests that the epidemiology of AHF in SSA differs from that of high-income settings, especially among the rural poor. Compared with patients from North America and Europe, patients from SSA tend to be about 20 years younger and are less likely to have evidence of underlying coronary artery disease. A recent systematic review and meta-analysis found that ischemic heart disease accounted for 7.2% of cases of AHF in SSA [3••]. Some registries do suggest increasing contributions of ischemic heart disease, especially in urban areas, but at levels far below those seen in North America and Europe [7–11]. Instead, hypertension, cardiomyopathy, and rheumatic heart disease seem to cause most cases of AHF in SSA [3••, 12, 13•, 14•]. Several studies further describe the rheumatic heart disease burden by lesion and these suggest that most patients have primary regurgitant lesions, while 20–30% have mitral stenosis [13•, 14•, 15]. Tuberculosis pericarditis, cor pulmonale, HIV-related cardiomyopathy, endomyocardial fibrosis, and congenital lesions also contribute significant burden [3••, 14•, 16].

Diagnosis of Acute Heart Failure

The diagnosis of heart failure proves challenging in many acute care settings in SSA [17••]. First, most patients with AHF will present with undifferentiated dyspnea or other symptoms that can mimic common and more high-profile diseases, such as tuberculosis or pneumonia. Most rural health institutions in SSA are staffed by non-physician health workers who may not have adequate training and experience in the diagnosis and management of AHF. Providers trained in these settings may lack a high index of suspicion for heart failure particularly given it was not historically emphasized as an important cause of dyspnea [18]. As a result, differentiating more prevalent conditions like tuberculosis and other chest infections from AHF may prove challenging. Moreover, mimicking conditions, such as tuberculosis, pneumonia, and anemia, may coexist with AHF or exacerbate it. For instance, one recently published registry of patients in Nigeria found that acute chest infection was the most common precipitant for AHF [1]. Another recent case series from Uganda found 64.3% of patients with AHF were also anemic [19].

Second, even at the level of institutions staffed with junior doctors/medical officers, the basic tools commonly used to diagnose AHF in high-income settings are scarce or non-existent, leaving providers to rely solely on clinical examination. A recent survey of East African facilities, including referral centers, found that chest x-ray was only available in 54% and 36% of hospitals in Kenya and Uganda, respectively. Ultrasound of any kind was available in 38% of Kenyan hospitals and in 45% of Ugandan hospitals [17••]. Even where some of these instruments are available, necessary skills to utilize them may not exist.

Point-of-care ultrasound (POCUS) has been shown to help distinguish AHF from other causes of dyspnea and can guide effective treatment of those patients in resource-limited settings. POCUS can reliably show signs of pulmonary edema, venous congestion, presence of pericardial effusion, and/or reduced ejection fraction, profoundly aiding in disease diagnosis [20•, 21•, 22•]. The use of a combined lung and cardiac ultrasound (LuCUS) protocol has been shown to increase diagnostic accuracy of AHF in the emergency setting [23], [24•], and POCUS evaluation of the inferior vena cava (IVC) may help determine a patient's volume status [23, 25, 26]. Moreover, POCUS can guide ongoing AHF management dynamically, as sonographic B-lines suggestive of pulmonary edema have been shown to rapidly decrease after diuresis in the emergent setting [27•].

In addition to the evidence showing its clinical efficacy, there is evidence showing POCUS can be successfully implemented in clinical settings in SSA. Brief training interventions have proven effective in teaching these point-of-care ultrasound skills to generalist physicians and mid-level providers in SSA [28–30, 31••, 32••]. POCUS allows immediate access to imaging data for acquisition and potential interpretation by clinicians at the bedside. It may also be less vulnerable to the shortages of staffing and consumables and the mechanical failures that frequently affect x-ray or laboratory testing in SSA [28, 33, 34]. Telemedicine interventions also show promise in helping to support use of POCUS in these settings [35].

Biomarkers may offer another promising diagnostic tool for AHF in resource poor settings [36, 37]. Natriuretic peptide tests such as BNP or proBNP have been shown to be helpful in confirming or ruling out heart failure as a cause of acute dyspnea and may be helpful in guiding AHF management [38–41]. To our knowledge, implementation of natriuretic peptides has not yet been studied in SSA. Some have pointed to the success of point-of-care testing for HIV and malaria as models for use of such point-of-care biomarker testing [37]. However, establishing cost-effectiveness of point-of-care testing in resource-limited settings is challenging. Proper analysis would have to consider not only the clinical usefulness of the test but also the cost of the test per unit, the number needed to test per case found, and the cost to the system of integrating it into a larger diagnostic strategy [42]. Furthermore, while biomarkers can help to suggest or exclude the general diagnosis, they cannot differentiate among the major causes of acute heart failure.

Current Debate and Evidence for Management of Acute Heart Failure and Adaptations for the Resource-Limited Setting

There is agreement in the literature that not all AHF exacerbations call for the same treatment. Indeed, effective therapies for some types of heart failure may be ineffective or dangerous when used for other types. Instead, effective management of AHF relies on a basic understanding of the underlying pathology and the factors driving the acute exacerbation [43••, 44, 45••]. At the most basic level, the provider needs to distinguish AHF patients with predominant hypertension from those with low to normal blood pressures. Most studies suggest that in SSA the latter group will be largely comprised of cardiomyopathies and regurgitant valvular lesions, both of which call for a similar acute treatment strategy of diuresis and afterload reduction. However, some common causes of AHF, such as mitral stenosis or pericardial disease, may be worsened by these interventions [46–48]. Thus, implementing POCUS can be particularly useful to differentiate among these important categories of AHF. Here, we outline treatment considerations for the most commonly encountered types of AHF in the SSA setting.

Acute Heart Failure in Patients with Hypertension and Pulmonary Edema

Patients with AHF caused by very high blood pressures and pulmonary edema, also known as flash pulmonary edema or sympathetic crashing acute pulmonary edema or (SCAPE), are caught in a cycle of hypertension causing pulmonary edema causing further increases in sympathetic output and diversion of splanchnic blood flow to the pulmonary bed, further worsening pulmonary edema. Prompt initiation of blood pressure reduction with nitrates and provision of non-invasive positive pressure ventilation (NIPPV) can rescue most patients and break this cycle [43••, 44, 49].

Nitrates, namely intravenous nitroglycerin, are commonly used to achieve rapid vasodilation and reversal of pulmonary edema and hypertension but are not widely available in SSA. Nitrates were used in less than 10% of AHF in SSA in the THUESUS registry [5]. Intravenous nitroglycerin is not on the World Health Organization's Essential Drug List and is therefore unlikely to be available at first-level hospitals [50]. Furthermore, IV pumps and the electricity to run them are scarce, and unmonitored gravity infusions are vulnerable to dosing errors which could be dangerous with nitrate infusions [51, 52]. Development and introduction of low cost, electricity-independent drip meters may help this problem [53]. There is also evidence that high-dose bolus dosing of IV nitroglycerin can alleviate the need for an unregulated infusion and be safe and effective [54•].

Sublingual nitrates are on the WHO's Essential Drug List for use in acute coronary syndromes [50]. They have also been endorsed as a temporizing measure prior to initiating IV nitrates [55]. Older literature described the efficacy of sublingual nitroglycerin at doses two to six times higher than typically used to treat anginal chest pain (0.8–2.4 mg) in order to rapidly reverse pulmonary edema [56]. A recent Swiss study found a non-invasive high-dose nitrate strategy using a combination of 1.6 mg sublingual and 20–40 mg of transdermal nitrates was safe and resulted in a larger and faster decrease in BNP than standard treatment with diuretics [57].

In the past decade, non-invasive positive pressure ventilation (NIPPV) has become standard of care in resource-rich settings for patients presenting with pulmonary edema [38, 43••, 45••]. However, NIPPV is frequently not available in SSA settings. Weingert and Levitan have described a technique using a bag valve mask combined with a positive end-expiratory pressure (PEEP) valve and a nasal cannula to provide the equivalent of CPAP to a patient with spontaneous respirations; this technique could be applicable to settings without access to NIPPV [58]. Milliner et al. described an improvised continuous positive airway pressure CPAP (iCPAP) technique using high-flow oxygen and a water seal which might also be applicable to some settings which lack NIPPV, though it has not yet been tested in a clinical setting [59•]. It is worth noting that oxygen itself may be a limited resource at many first-level hospitals but is a clear priority for health system improvement [60, 61].

In resource-rich settings, angiotensin-converting enzyme inhibitors (ACE-I) are generally recommended as an adjunct to reduce afterload in AHF after initial NIPPV and nitrate application [43••, 38]. Older studies show their benefit in acute pulmonary edema prior to routine application of NIPPV, which may be relevant to SSA first-level hospitals without NIPPV [62]. ACE inhibitors are included in the WHO's Essential Drug List for medicine used in heart failure, although their availability at first-level hospitals is variable [50]. A recent survey found ACE inhibitors were only available in 51% of Kenyan and 79% of Ugandan hospitals and even then, supplies were often interrupted by stock outs [17••].

The role of diuresis in treatment of the hypertensive pulmonary edema patient is a subject of debate. Diuretics remain the cornerstone of treatment for pulmonary edema [38, 39]. The REALITY-AHF prospective observational study of heart failure patients in the USA showed that faster initiation of diuresis improved patient outcomes, giving rise to the concept of a "door-to-furosemide-time" [63••]. The study did not report the difference in diuresis effect between patients with normal and high blood pressure. However, the benefit seems to have been less significant among patients with a lower Get with the Guidelines Score, in which higher blood pressures result in a lower mortality score [63••, 64]. Indeed, there is a growing evidence that early diuresis in patients with acute pulmonary edema and hypertension may be harmful, as many patients are euvolemic, with symptoms due to fluid maldistribution rather than fluid overload [45••, 49, 65••]. The risks of inappropriate diuresis are theoretically larger in many SSA settings, as access to timely electrolyte measurement such as creatinine is also often lacking, even at referral centers [66].

Acute Heart Failure in Patients with Low to Normal Blood Pressure

Recent expert recommendations emphasize the importance of distinguishing between hypertensive AHF patients and patients with blood pressure at or below their baseline [43••, 44, 45••]. The latter have a different pathophysiology contributing to their heart failure and require a different treatment approach. In SSA, this group will likely include those with cardiomyopathy and decompensated regurgitant valvular lesions, as well as those with pericardial disease.

Cardiomyopathies and Decompensated Regurgitant Valvular Lesions

Using a simple echocardiographic approach, generalist and mid-level providers can be taught to identify both of these conditions in their most general form, the former by low ejection fraction and the latter by a combination of normal ejection fraction and loud murmur [14•, 29]. These patients are more likely to be fluid overloaded and to require diuresis as well as gentle afterload reduction [43••].

Acute Decompensated Heart Failure Due to Mitral Stenosis

Rheumatic mitral stenosis seems to be a less common, but still important cause of AHF in SSA and its diagnosis and treatment may pose a challenge to clinicians in austere environments. These patients can be harmed by treatments beneficial to other heart failure patients (such as diuresis) and, in contrast, helped by those treatments not emphasized in other etiologies (such as aggressive rate control and anticoagulation) [46]. However, mitral stenosis cannot be reliably detected on physical exam, given that the murmur is often quite soft. Rheumatic mitral stenosis has a characteristic appearance on ultrasound, but POCUS algorithms developed for high-income regions with low mitral stenosis prevalence will not likely address its identification [24•, 67, 68, 69]. Several training interventions designed for high-prevalence regions have been able to teach its identification to medical students, generalist physicians, or mid-level providers and may show promise for wider implementation [29, 31••, 70].

Acute Decompensated Heart Failure Due to Pericardial Disease

AHF from pericardial disease may present with low to normal blood pressure or in shock. A recent review found most pericardial disease in SSA is due to tuberculosis and that almost 80% of these patients present with pericardial effusion [71••]. POCUS can rapidly aid in the diagnosis of pericardial effusion [28]. POCUS is also an important adjunct in helping to diagnose and initiate management for pericardial manifestations of extra-pulmonary tuberculosis [72, 73]. Not all patients will require pericardiocentesis, but this can be life-saving if the patient presents in tamponade. Successful pericardiocentesis using POCUS and a central line kit has been described in resource-limited settings [74] [75]. However, given that safe pericardiocentesis is unlikely available at first-level hospitals in SSA, the WHO's training for first-level emergency providers emphasizes temporizing measures such as fluid administration to help the patient with tamponade reach definitive care [76].

Acute Heart Failure Presenting with Hypotension

Hypotension and cardiogenic shock portend a poor prognosis in any setting [77••]. Some patients may benefit from a small fluid bolus if they are dehydrated or have a pre-load dependent lesion. However, many patients will be volume overloaded and require diuresis combined with ionotropic support [43••]. While the choice of inotropic agent has been a source of debate in North America and Europe, availability of any kind of inotropic support is a more relevant issue in SSA [66].

Conclusion

Acute heart failure is a vexing problem in the best resourced settings and is particularly challenging in the austere environment of the typical SSA first-level hospital. However, effective strategies to diagnose and treat heart failure in its acute form are a necessary component of any effort to improve heart failure diagnosis and care. These strategies require adaptation of accepted diagnostics and treatments to the local epidemiology and resource constraints. Point-of-care testing for biomarkers such as BNP may have applications in these settings to help diagnose AHF in the undifferentiated dyspneic patient. POCUS offers a relatively accessible and effective way to diagnose and categorize AHF, and reports suggest that first-level hospital staff can be taught to differentiate among the important categories of heart failure and tailor initial therapy accordingly. Some innovations introduced in high-resource settings, such as adaptions to create NIPPV without a ventilator, may be useful in managing AHF in low-resource settings. Management of acute heart failure should be taken into account when designing essential drug lists and first-level hospital training modules.

Compliance with Ethical Standards

Conflict of Interest Alice Kidder Bukhman, Vizir Jean Paul Nsengimana, Mindy C. Lipsitz, Endale Tefera, Shada A. Rouhani, Damas Dukundane, and Gene Y. Bukhman declare that they have no conflict of interest.

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