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Therapeutic Patient Education and Short-Term Clinical Outcomes in Chronic Heart Failure: A Prospective Cohort Study at the University Hospital of Brazzaville

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Abstract

Background: Heart failure carries a poor short-term prognosis in sub-Saharan Africa, with high rates of post-discharge mortality partly attributable to modifiable behavioural determinants of decompensation. Therapeutic patient education (TPE) has been proposed as a strategy to reduce these avoidable precipitants, yet evidence on its direct impact on clinical trajectory in African settings remains scarce. **Objective:** To evaluate the effect of a structured TPE programme on all-cause mortality and cardiac decompensation at three months in patients with chronic heart failure at the University Hospital of Brazzaville. **Methods:** Prospective cohort study (February-August 2020). Patients were allocated to an educated group (n = 51) receiving a five-session multidisciplinary TPE programme or to a control group (n = 87) receiving usual care. Primary outcomes: all-cause mortality and cardiac decompensation (rehospitalisation) at three months. Secondary outcomes: NYHA functional class progression, blood pressure and heart rate control, lower limb oedema, body weight trajectory, cardiovascular risk factor management, and decompensation triggers. Effect measures included risk ratios (RR), odds ratios (OR), absolute risk reduction (ARR), and number needed to treat (NNT). **Results:** Mortality was 3.9% in the educated group versus 19.5% in controls (RR = 0.20 [95% CI 0.05 - 0.83]; ARR = 15.6%; NNT = 6.4; p = 0.01). Cardiac decompensation occurred in 7.8% versus 22.9% (RR = 0.34 [0.12 - 0.94]; NNT = 6.6; p = 0.02). Progression to NYHA class III - IV was observed in 13.6% of educated patients versus 36.7% of controls (RR = 0.37 [0.18 - 0.78]; NNT =

4.3; p = 0.03). Tachycardia occurred in 26.5% versus 71.4% (RR = 0.39 [0.24 - 0.61]; NNT = 2.2; p < 0.001). Blood pressure dysregulation affected 22.5% versus 48.3% respectively (RR = 0.46 [0.26 - 0.81]; NNT = 3.9; p < 0.001). Body weight remained stable in the educated group (mean change 0.0 ± 1.2 kg; p = 0.74) but increased significantly in controls (+1.5 ± 2.8 kg; p < 0.001). Good cardiovascular risk factor management was achieved in 75.5% of educated patients versus 18.1% of controls (p < 0.001). **Conclusion:** Structured therapeutic education was associated with substantial reductions in mortality, cardiac decompensation, functional deterioration, and haemodynamic instability. The consistently low NNT values (2 - 7) across all clinical outcomes support TPE as a high-impact, resource-efficient intervention warranting systematic integration into heart failure management in sub-Saharan Africa.

Keywords

Heart Failure, Therapeutic Patient Education, Mortality, Cardiac Decompensation, NYHA Functional Class, Haemodynamics, Number Needed to Treat, Sub-Saharan Africa

1. Introduction

Heart failure (HF) is a progressive clinical syndrome associated with one of the highest mortality burdens among cardiovascular conditions. In Western settings, five-year survival following an initial diagnosis of HF is estimated at approximately 50%, a figure that compares unfavourably with many malignancies [1]. In sub-Saharan Africa, available evidence suggests that this prognosis may be substantially worse. Hospital-based studies consistently report case fatality rates exceeding 10% during the index admission alone, reflecting delayed presentation, limited access to evidence-based therapies, and the predominance of hypertensive aetiology associated with advanced structural disease at diagnosis [2] [3]. At the University Hospital of Brazzaville, heart failure accounts for more than half of all cardiology admissions [4], placing a considerable burden on a healthcare system with constrained human and material resources.

A characteristic feature of heart failure in this context is the high frequency of potentially avoidable decompensation episodes. Studies in both high- and low-income settings demonstrate that more than one-third of rehospitalisations for heart failure are directly attributable to modifiable behavioural factors, principally treatment discontinuation and dietary non-compliance with sodium and fluid restriction [5]. These precipitants are not primarily pharmacological problems but rather consequences of insufficient patient knowledge, inadequate self-monitoring skills, and poor disease understanding. In Congo-Brazzaville, the high prevalence of unemployment and low socioeconomic status constitute additional barriers to sustained adherence and early recognition of warning signs [4].

Therapeutic patient education (TPE) was defined by the World Health Organ-

ization as a structured, continuous process designed to help patients acquire and maintain the competencies necessary to manage their chronic condition [6]. In heart failure, TPE is assigned a class I, level A recommendation by both the European Society of Cardiology and the American Heart Association/American College of Cardiology [7] [8]. Landmark studies by Rich *et al.* and Krumholz *et al.* established that nurse-led educational interventions following hospitalisation for decompensated HF significantly reduced readmission rates and associated costs [9] [10].

Despite this international evidence, the prognostic impact of TPE on short-term clinical outcomes—specifically all-cause mortality, incidence of decompensation, and functional trajectory—has not been evaluated in the African context. The number needed to treat (NNT) provides a clinically intuitive measure of impact directly relevant to health planning in resource-limited settings. Adherence and quality-of-life outcomes from this cohort have been reported elsewhere [11]. The present study evaluates the effect of TPE on hard clinical endpoints including mortality, cardiac decompensation, NYHA functional class progression, and haemodynamic parameters.

2. Patients and Methods

2.1. Study Design and Setting

This was a prospective comparative cohort study conducted over a six-month period from 15 February to 28 August 2020, in the Department of Cardiology and Internal Medicine of the University Hospital of Brazzaville (CHU-B), Republic of Congo. CHU-B is the national reference centre for cardiovascular care. The cardiology department has a total capacity of 44 beds, including a four-bed cardiac intensive care unit, and is equipped with standard diagnostic facilities.

2.2. Study Population and Allocation

Eligible patients were aged 15 years or older, hospitalised for chronic heart failure, and regularly attending the outpatient cardiology clinic. Patients were allocated at discharge in a 1:2 ratio to an education group or usual care. Allocation was based on availability of upcoming group sessions; patients discharged between sessions were assigned to control. This non-randomised process introduces potential selection bias, as discussed in Section 4.6. A total of 192 patients were initially recruited. Following application of exclusion criteria and losses to follow-up (partly attributable to the COVID-19 pandemic context), 138 patients completed the three-month evaluation: 51 in the educated group and 87 in the control group.

2.3. Therapeutic Education Intervention

The TPE programme comprised five collective, interactive sessions of one to two hours each, delivered over five consecutive weeks in groups of approximately ten patients. The multidisciplinary team included cardiologists, cardiology residents, nurses, a social worker, and a nutritionist. Educational content covered heart fail-

ure pathophysiology, dietary management, physical activity, pharmacological treatment, and water and salt intake. Following completion of the five educational sessions, educated group patients received structured telephone follow-up for three months. Calls were made by a cardiology nurse at weeks 2, 4, 8, and 12 post-discharge. Each call followed a standardised protocol covering: 1) symptom screening (dyspnoea, oedema, weight gain); 2) medication adherence verification; 3) dietary compliance with sodium and fluid restrictions; and 4) scheduled appointment confirmation. Control group patients did not receive any equivalent telephone contact; they attended scheduled outpatient clinic visits only (at one and three months), consistent with standard institutional practice. The difference in contact frequency between groups is acknowledged as a limitation: the observed benefit may reflect additional surveillance and support rather than educational content alone (see Section 4.6). Control group patients received standard discharge counselling without formalised educational sessions.

2.4. Outcome Measures

Primary outcomes: all-cause mortality and cardiac decompensation (rehospitalisation for worsening heart failure) at three months. Outcomes were ascertained from hospital records, scheduled follow-up (including telephone at 1 and 3 months), and caregiver reports when needed; out-of-hospital deaths were identified via family report. Assessors were not blinded to group allocation, introducing potential ascertainment bias.

Secondary outcomes: progression of NYHA functional class to stage III - IV; blood pressure dysregulation (elevated ≥ 140/90 mmHg or low < 90/60 mmHg); tachycardia (heart rate ≥ 100 bpm); bradycardia (heart rate < 60 bpm); development of lower limb oedema; body weight variation from baseline; cardiovascular risk factor control; and identification of precipitating decompensation factors. Cardiovascular risk factor control at 3 months was defined as achieving all of the following: BP < 140/90 mmHg, heart rate 60 - 99 bpm, absence of clinical oedema, and no missed medication doses in the prior 2 weeks (self-reported). Assessment was performed by the cardiologist using examination and structured interview; no patient-reported instrument was used.

Baseline measurements (NYHA class, body weight, blood pressure, heart rate, and oedema status) were recorded just prior to hospital discharge.

2.5. Statistical Analysis

Data were entered using Epi Info version 3.5.4 and analysed with R version 3.6. Categorical variables were expressed as frequencies and percentages; continuous variables as means ± standard deviations. Between-group comparisons used the chi-square test (or Fisher's exact test for sparse cells) and Student's t-test (Welch correction). Risk ratios (RR) and odds ratios (OR) with 95% confidence intervals were calculated from 2 × 2 contingency tables. Absolute risk reduction (ARR) was defined as the between-group difference in event rates; NNT was calculated as the

reciprocal of ARR. Ninety-five percent confidence intervals for proportions used the Wilson score method.

Given the limited number of events, particularly for mortality (n = 19 total), the events-per-variable (EPV) rule precluded multivariable logistic regression with more than one covariate [12]. Primary analyses are therefore unadjusted; confounding by age and socioeconomic status is addressed in the discussion. A two-sided p-value < 0.05 was considered statistically significant.

3. Results

3.1. Baseline Characteristics

Baseline characteristics are summarised in Table 1. Detailed sociodemographic and pharmacological data have been reported in the companion publication [11]. Briefly, the educated and control groups differed significantly at inclusion with respect to mean age (52.4 ± 12.9 vs 59.5 ± 16.5 years; p = 0.01), employment status (p = 0.01), education level (p = 0.03), and socioeconomic status (p < 0.001). Cardiovascular risk factor profiles, NYHA class, type of heart failure, left ventricular ejection fraction categories, and prescribed pharmacological treatment did not differ significantly between groups (all p > 0.05). All patients were in NYHA class I at inclusion with no lower limb oedema at baseline.

Table 1. Summary of baseline characteristics.

Variable	Educated (n = 51)	Control (n = 87)	p
Mean age ± SD (years)	52.4 ± 12.9	59.5 ± 16.5	0.01
Female, n (%)	30 (58.8)	47 (54.0)	NS
Unemployed, n (%)	17 (33.3)	54 (62.1)	0.01
Low socioeconomic status, n (%)	22 (43.1)	59 (67.8)	<0.001
Hypertension, n (%)	23 (45.1)	28 (32.2)	0.13
Reduced LVEF (<40%), n (%)	42 (82.3)	72 (82.8)	0.05
NYHA class I at inclusion, n (%)	51 (100)	87 (100)	1.00
Mean baseline weight ± SD (kg)	64.4 ± 10.5	65.3 ± 11.9	NS

LVEF = left ventricular ejection fraction; SD = standard deviation; NS = not significant. Full baseline data in companion publication [11].

3.2. Primary Outcomes: Mortality and Cardiac Decompensation

At three-month follow-up, all-cause mortality was 3.9% in the educated group (2/51) versus 19.5% in controls (17/87), corresponding to a risk ratio of 0.20 (95% CI 0.05 - 0.83), an odds ratio of 0.17 (95% CI 0.04 - 0.76), an ARR of 15.6%, and a NNT of 6.4 (p = 0.01). Cardiac decompensation occurred in 4 of 51 educated patients (7.8%) versus 20 of 87 controls (22.9%; RR = 0.34 [95% CI 0.12 - 0.94]; OR = 0.29 [0.09 - 0.89]; ARR = 15.1%; NNT = 6.6; p = 0.02). These data are presented in Table 2.

Table 2. Primary and secondary binary clinical outcomes at three-month follow-up.

Outcome	G1 n/N	G1 %	G2 n/N	G2 %	RR [95% CI]	OR [95% CI]	ARR	NNT/p
All-cause mortality	2/51	3.9	17/87	19.5	0.20 [0.05 - 0.83]	0.17 [0.04 - 0.76]	15.6%	6.4/0.01
Cardiac decompensation	4/51	7.8	20/87	22.9	0.34 [0.12 - 0.94]	0.29 [0.09 - 0.89]	15.1%	6.6/0.02
Lower limb oedema	4/51	7.8	23/87	26.4	0.30 [0.11 - 0.81]	0.24 [0.08 - 0.73]	18.6%	5.4/0.01
Dietary non-compliance	4/51	7.8	41/87	47.1	0.15 [0.05 - 0.39]	0.10 [0.03 - 0.29]	39.3%	2.5/ < 0.001

RR = risk ratio; OR = odds ratio; ARR = absolute risk reduction; NNT = number needed to treat. OR calculated as $(a \times d)/(b \times c)$; 95% CI: $\exp[\ln(OR) \pm 1.96 \times \sqrt{(1/a + 1/b + 1/c + 1/d)}]$.

3.3. Secondary Outcomes: Functional Class, Haemodynamics, and Weight

Table 3 presents the complete evolution of functional and haemodynamic parameters at three-month follow-up.

NYHA functional class. At three months, NYHA class I was maintained in 33 educated patients (64.7%) versus 38 controls (43.7%). Progression to NYHA class III - IV was observed in 7 educated patients (13.6%) versus 32 controls (36.7%), corresponding to a risk ratio of 0.37 (95% CI 0.18 - 0.78), an ARR of 23.1%, and a NNT of 4.3 ($p = 0.03$). This represents a 63% relative risk reduction in severe functional deterioration attributable to the educational intervention.

Blood pressure. Normal blood pressure was maintained in 40 educated patients (77.5%) versus 45 controls (51.7%). Blood pressure dysregulation—defined as elevated (≥140/90 mmHg) or low (<90/60 mmHg)—was present in 11 educated patients (22.5%) versus 41 controls (47.1%), yielding a risk ratio of 0.46 (95% CI 0.26 - 0.81), an ARR of 25.5%, and a NNT of 3.9 ($p < 0.001$). Low blood pressure was particularly marked in the control group (23.4%, n = 20) versus the educated group (4.1%, n = 2), reflecting a higher burden of advanced decompensation and circulatory failure among non-educated patients. Elevated blood pressure affected 18.4% of educated patients (n = 9) versus 24.7% of controls (n = 21).

Heart rate. Normal heart rate was maintained in 37 educated patients (73.5%) versus 24 controls (27.3%). Tachycardia (heart rate ≥ 100 bpm) was observed in 14 educated patients (26.5%) versus 62 controls (71.4%), representing a risk ratio of 0.39 (95% CI 0.24 - 0.61), an ARR of 44.9%, and a NNT of 2.2 ($p < 0.001$)—the lowest NNT of all measured outcomes. No bradycardia was recorded in the educated group, compared with 1 case (1.3%) in controls.

Body weight. Body weight remained stable in the educated group at three months (64.4 ± 10.2 kg versus 64.4 ± 10.5 kg at inclusion; mean change 0.0 ± 1.2 kg; $p = 0.74$). In contrast, mean body weight increased significantly in the control group (66.8 ± 11.9 kg versus 65.3 ± 11.9 kg); mean change (+1.5 ± 2.8 kg; $p < 0.001$), consistent with progressive fluid retention. The between-group difference in absolute weight at three months did not reach statistical significance ($p = 0.25$).

Cardiovascular risk factor management. Good management of cardiovascular risk factors at three months was observed in 75.5% of educated patients (37/49) versus 18.1% of controls (13/72; ARR = 57.4%; NNT = 1.7; $p < 0.001$).

Table 3. Haemodynamic and functional parameters at three-month follow-up.

Parameter	Educated group (n = 51) n (%)	Control group (n = 87) n (%)	p-value	RR [95% CI] NNT/ARR
A. NYHA Functional Class at M3				
NYHA class I	33 (64.7)	38 (43.7)		
NYHA class II	11 (21.6)	17 (19.5)		
NYHA class III	3 (5.8)	12 (13.8)		
NYHA class IV	4 (7.8)	20 (22.9)		
NYHA III - IV combined	**7 (13.6)**	**32 (36.7)**	**0.03**	**RR 0.37 [0.18 - 0.78] NNT 4.3/ARR 23.1%**
B. Blood Pressure at M3				
Normal BP	40 (77.5)	45 (51.7)*		
Elevated BP ≥ 140/90	9 (18.4)	21 (24.7)		
Low BP < 90/60	2 (4.1)	20 (23.4)		
Any BP dysregulation	**11 (22.5)**	**41 (47.1)***	**<0.001**	**RR 0.46 [0.26 - 0.81] NNT 3.9/ARR 25.5%**
C. Heart Rate at M3				
Normal HR (60 - 99 bpm)	37 (73.5)	24 (27.3)		
Tachycardia ≥ 100 bpm	14 (26.5)	62 (71.4)		
Bradycardia < 60 bpm	0 (0.0)	1 (1.3)		
Tachycardia	**14 (26.5)**	**62 (71.4)**	**<0.001**	**RR 0.39 [0.24 - 0.61] NNT 2.2/ARR 44.9%**
D. Body Weight at M3				
Mean weight ± SD (kg)	64.4 ± 10.2	66.8 ± 11.9	0.25 (NS)	
Intra-group weight change	0.0 ± 1.2 kg	+1.5 ± 2.8 kg		
Intra-group p (paired test)	p = 0.74	p < 0.001	—	
E. Cardiovascular Risk Factor Management at M3 †				
Good management	37 (75.5)	13 (18.1)	<0.001	RR 4.17 [2.49 - 6.99] NNT 1.7/ARR 57.4%
Poor management	12 (24.5)	59 (81.9)	<0.001	

SD = standard deviation; BP = blood pressure; HR = heart rate; RR = risk ratio; NNT = number needed to treat; ARR = absolute risk reduction; NS = not significant; M3 = three-month assessment. BP normal category calculated within the control group (n = 87). Cardiovascular risk factor management available for n = 72.

3.4. Decompensation Triggers

In the educated group, treatment non-adherence was the sole decompensation trigger (4 patients, 7.8%). In the control group, three categories of triggers were identified: isolated treatment non-adherence (8 patients, 9.2%), isolated dietary non-compliance (7 patients, 8.0%), and combined non-adherence and dietary non-compliance (5 patients, 5.7%). The between-group comparison was statistically significant (p = 0.04), demonstrating that TPE eliminated dietary non-compliance entirely as a decompensation precipitant in educated patients. Decompensation triggers differed significantly between groups, as shown in Table 4.

Table 4. Precipitating factors of cardiac decompensation at M3.

Decompensation trigger	Educated n (%)	Control n (%)	p
Treatment non-adherence (isolated)	4 (7.8)	8 (9.2)	
Dietary non-compliance (isolated)	0 (0.0)	7 (8.0)	
Combined (non-adherence + dietary)	0 (0.0)	5 (5.7)	
Total decompensation events	**4 (7.8)**	**20 (22.9)**	**0.04**

No complications were reported in either group during the three-month follow-up period.

4. Discussion

4.1. Mortality Reduction: Magnitude and Clinical Significance

The 80% relative reduction in three-month all-cause mortality (3.9% vs 19.5%; RR = 0.20; NNT = 6.4; p = 0.01) represents the most clinically compelling finding of this study. A NNT of 6.4 compares favourably with pharmacological interventions in heart failure, where ACE inhibitors and beta-blockers achieve NNTs of approximately 14 - 22 for mortality reduction over 12 - 24 months [7]. This difference should be interpreted in light of the high baseline event rate in the control group—a setting in which the potential for absolute risk reduction, and therefore a low NNT, is inherently greater. This mortality difference should, however, be interpreted cautiously given significant baseline imbalances: control patients were seven years older (p = 0.01) and more frequently in low socioeconomic status (67.8% vs 43.1%; p < 0.001)—established predictors of adverse outcomes [13].

4.2. Cardiac Decompensation: Pathophysiological Mechanisms

The 66% relative risk reduction in cardiac decompensation (7.8% vs 22.9%; RR = 0.34; NNT = 6.6; p = 0.02) can be attributed to the elimination of modifiable behavioural triggers. In the control group, decompensation arose from isolated dietary non-compliance (8.0%), treatment non-adherence (9.2%), and their combination (5.7%). In the educated group, dietary non-compliance was entirely absent as a decompensation trigger. This complete elimination is mechanistically consistent with the educational curriculum, which included dedicated sessions on sodium and fluid management, early warning sign recognition, and daily weight self-monitoring. The progressive weight gain observed in the control group (+1.5 kg, p < 0.001) provides objective haemodynamic confirmation of this process [14].

4.3. Functional Trajectory: NYHA Class and Haemodynamic Control

The novel quantification of functional trajectory in this study reveals a striking divergence between groups. Progression to NYHA class III - IV occurred in only 13.6% of educated patients versus 36.7% of controls (RR = 0.37 [0.18 - 0.78]; NNT = 4.3; p = 0.03), representing a 63% relative risk reduction in severe functional deterioration. NYHA class III - IV is associated with substantially increased symptom burden, reduced exercise tolerance, and elevated short-term mortality risk

[8]. The prevention of functional deterioration at this threshold therefore represents a clinically meaningful outcome with direct implications for patient autonomy and healthcare utilisation.

The haemodynamic data provide further mechanistic insight. Blood pressure dysregulation affected only 22.5% of educated patients versus 47.1% of controls (RR = 0.46; NNT = 3.9; $p < 0.001$). Notably, low blood pressure—reflecting advanced decompensation and circulatory failure—was observed in 23.4% of controls versus only 4.1% of educated patients. This disparity likely reflects both the direct cardiovascular consequences of uncontrolled decompensation in the control group and the protective effect of adherence to diuretic and vasodilator therapy in the educated group.

The most pronounced haemodynamic difference was observed for tachycardia, which affected 71.4% of controls versus only 26.5% of educated patients (RR = 0.39 [0.24 - 0.61]; NNT = 2.2; $p < 0.001$). This NNT of 2.2—the lowest of all outcomes measured—indicates that educating two patients prevents one case of tachycardia within three months. Persistent tachycardia in heart failure reflects ongoing neurohormonal activation, inadequate beta-blocker adherence, or volume overload-driven sympathetic stimulation. Its prevention in educated patients likely reflects the combined effects of better medication adherence (particularly beta-blockade) and effective dietary sodium restriction limiting volume-driven adrenergic activation.

4.4. Cardiovascular Risk Factor Control

Good management of cardiovascular risk factors was achieved in 75.5% of educated patients versus only 18.1% of controls (NNT = 1.7; ARR = 57.4%; $p < 0.001$). In Congo-Brazzaville, where hypertension is the predominant aetiological factor following the epidemiological transition [3], uncontrolled blood pressure perpetuates ventricular afterload and accelerates remodelling. The TPE curriculum explicitly addressed blood pressure self-monitoring, salt restriction, and the rationale for antihypertensive medication, translating into measurable behavioural change with direct downstream haemodynamic consequences.

4.5. Public Health Implications in Sub-Saharan Africa

The NNT values across all outcomes (range 1.7 - 6.6) support a compelling public health case for TPE in this setting. A structured programme serving seven patients is expected to prevent one death, one rehospitalisation, one case of severe functional deterioration, and one episode of BP dysregulation within three months. In healthcare systems where inpatient cardiac care is costly and hospital beds are scarce, these numbers translate into resource savings that likely offset programme costs substantially [15]. The group-based format, use of existing hospital infrastructure, and involvement of family members—reflecting Congolese sociocultural norms—demonstrate that effective TPE is achievable without dedicated facilities or additional staffing budgets.

4.6. Methodological Limitations

Several limitations must be acknowledged. The non-randomised design introduces potential selection bias, with significant baseline differences in age and socioeconomic status representing major confounders of clinical outcomes, particularly mortality. The EPV rule limited the mortality model to a single predictor, precluding multivariable adjustment [12]. Losses to follow-up (21/70 in the educated group; 35/122 in controls) and the COVID-19 pandemic context may have introduced differential attrition bias. The three-month follow-up is insufficient to evaluate medium-term effects on ventricular remodelling and sustained NYHA class stability. The missing data for cardiovascular risk factor management in 15 control group patients (17.2%) limits precision for this outcome. Despite these limitations, the consistency of findings across multiple independent outcomes and their concordance with international literature support the overall validity of the results. Outcome assessors were not blinded to group allocation, introducing risk of differential ascertainment bias, particularly for softer endpoints such as identification of decompensation triggers. Mortality, a hard objective endpoint, is less susceptible to this bias. Out-of-hospital deaths were captured through family report and may be incompletely ascertained.

5. Conclusion

This prospective cohort study demonstrates that structured therapeutic patient education significantly modifies the short-term clinical trajectory of chronic heart failure in a sub-Saharan African setting. Beyond its established effects on adherence and quality of life [11], TPE was associated with substantial reductions in all-cause mortality (NNT = 6.4), cardiac decompensation (NNT = 6.6), NYHA class III - IV progression (NNT = 4.3), blood pressure dysregulation (NNT = 3.9), and tachycardia (NNT = 2.2). The consistently low NNT values—ranging from 1.7 to 6.6 across all clinical outcomes—indicate that TPE does not merely modify patient-reported outcomes but directly alters the hard clinical endpoint profile of heart failure at three months. These findings provide strong empirical support for the systematic integration of structured therapeutic education into heart failure management protocols across sub-Saharan Africa, with the urgency and resource allocation currently reserved for pharmacological therapies. Randomised controlled trials with extended follow-up are warranted to confirm the causal magnitude of these effects.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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