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Short-Term Outcome of Acute Heart Failure in Diabetic and Non-Diabetic Patients

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ABSTRACT

Background: The prevalence of both heart failure (HF) and diabetes is rising, with diabetes affecting up to 45% of acute HF patients. As a result of its effects on cardiac anatomy and function, diabetes is considered a more serious condition than HF alone.

Objective: The purpose of this research was to describe the clinical manifestations, echocardiographic findings, and short-term cardiovascular prognosis of acute HF in individuals with and without diabetes.

Patients and Methods: This observational, hospital based, single center study was carried out on 70 patients who were admitted to the Coronary Care Unit with acute HF, new onset HF and decompensated chronic HF, aged ≥ 18 years, both sexes. Group A consisted of 47 individuals with diabetes and acute HF, while Group B included 23 individuals without diabetes and acute HF. Every patient underwent a thorough evaluation that included electrocardiograms (ECGs), echocardiograms, complete clinical exams, laboratory testing, and a thorough history taking.

Results: Serum creatinine, glomerular filtration rate, C-reactive protein, sodium, potassium, serum random glucose, glycated haemoglobin, early diastolic velocity of the mitral annulus (E), systolic velocity of the mitral annulus (S′), and E over E-prime (E/e') were identified as independent predictors of acute HF in both diabetic and non-diabetic patients ($P<0.05$). Acute HF in both diabetic and non-diabetic individuals was independently predicted by glomerular filtration rate, NT pro-BNP, CRP, sodium, serum random glucose, HbA1c, E, and E/e', but not by serum creatinine, potassium, or S′, according to multivariate regression.

Conclusions: Patients with diabetes mellitus (DM) had inferior short-term clinical outcomes after acute HF. E, S′, sodium, potassium, serum glucose, low-density lipoprotein (LDL), CRP, and short-term HF outcomes were independently predicted by both diabetes and non-diabetic individuals. Patients with HF with DM had reduced orthopnea, heart rates, and E/e' compared to those without diabetes. Along similar lines, this group exhibited reduced levels of LDL, left atrial volume index, E′, and S′.

Keywords: Acute HF, Diabetic and Non-Diabetic Patients, Cardiovascular Diseases, Echocardiography.

INTRODUCTION

The prevalence of HF is on the rise due to variables such as longer life expectancy, more risk factors, and better outcomes for patients with cardiovascular disorders. Approximately 26 million individuals worldwide are affected by this condition. HF accounts for the vast majority of hospitalisations in Egypt for patients younger than 65. High rates of mortality and readmission are linked to heart failure hospitalisation. The overall clinical results are worsened by the presence of comorbidities, which effect around 75% of HF patients [1].

HF and diabetes (DS) have both been on the rise in recent decades and are projected to remain so in the years to come. There is likely to be a dramatic increase in the prevalence of DS as a result of the rising incidence of HF. DS may affect as many as 45 percent of patients experiencing acute HF, according to some registries. Diabetes must be considered as more than simply a co-occurring illness in HF patients because of the direct effect it has on the anatomy and function of the heart [2].

DS is one of the risk factors that might lead to heart failure on its own. It has also been shown that this risk changes depending on a person's age and gender. Women with diabetes have a fourfold increased risk of HF while males with diabetes have a twofold increased risk compared to non-diabetic individuals. Maybe the younger generation is more aware of these links [3,4].

In addition, individuals with acute HF who have diabetes tend to stay in the hospital for longer and are more likely to be readmitted. Importantly, it has been proven that diabetes increases cardiovascular morbidity and death in HF patients. It is still debatable, though, whether diabetes has any predictive value for both short-term and long-term mortality in individuals with acute HF [5].

Patients with chronic HF today have a greater chance of survival since conventional treatment programs include many new forms of therapy. Patients with acute HF still do not know whether DS improved their prognosis [6,7].

In this paper, the researchers at Sohag University Hospital set out to document the symptoms, echocardiographic results, and first cardiovascular prognosis of acute HF in both diabetic and non-diabetic individuals.

PATIENTS AND METHODS

This observational, hospital based, single center study was carried out on 70 patients who were admitted to the Coronary Care Unit with acute HF, new onset HF

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and decompensated chronic HF, aged ≥ 18 years, both sexes (38 males and 32 females).

Ethical considerations:

This study was carried out from April to October 2024 with the blessing of the Ethical Committee at Sohag University Hospitals in Sohag, Egypt. It was registered at the clinical trials registry (Soh -Med-24-05-13 MS). All the participants gave their written consent. The study adhered to the Helsinki Declaration throughout its execution.

People who had chronic HF were not included.

We classified the patients into two groups: those with diabetes and acute HF (Group A) and those without diabetes and acute HF (Group B).

Tests for glycated haemoglobin (HbA1C), electrocardiograms (ECGs), echocardiograms, lipid profiles, liver and kidney enzyme assays, complete blood counts (CBCs), and N-terminal pro-B-type natriuretic peptide (NT pro-BNP) were routinely performed on all patients.

Individuals were considered to have diabetes if they had ever taken insulin or an oral antidiabetic drug prior to their admission. A haemoglobin A1c reading of 6.5% or higher was used to define diabetes. We determined the HbA1C using ion exchange high performance liquid chromatography equipment and a Bio-Rad D-10 analyser, which is manufactured by BioRad, which is located in Porto, Portugal.

The following formula was used to determine the average blood glucose concentration from haemoglobin A1c values given during hospitalisation: To calculate eAG, take 28.7 times haemoglobin A1c and divide it by 46.7 [8]. There was a qualitative grading system for left ventricular ejection fraction (LVEF), with three possible categories: preserved, reduced, and mildly reduced. The quantitative characteristics of left ventricular end-filler function (LVEF) were classified as follows: intact LVEF > 50%, diminished LVEF 40-49%, and slightly diminished LVEF < 40%. Ischaemic heart failure and nonischaemic heart failure are the two most common forms of this condition [8].

Echocardiography:

All of the echocardiographic images were taken using the field-issued ultrasonic apparatus (System 6, GE Vingmed, Horten, Norway) with a 2.5 MHz probe. The LVEF was calculated using a biplane Simpson's technique, the apical 4-chamber and 2-chamber perspectives. They found three groups of heart failure patients: those with intact ejection fraction (HF with LVEF ≥ 50%), those with reduced ejection fraction (HF with LVEF < 40%), and those with mid-range ejection fraction (HF with LVEF < 40- 49%). Less than 30% LVEF was indicative of severe systolic dysfunction [9].

Statistical analysis

We used IBM SPSS Statistics, a statistical program developed and published in Chicago, IL, USA, by SPSS, Inc., to analyse and correlate the data that we gathered. The quantitative data were shown as the mean plus or minus the SD, and to find differences in the dichotomous variables, which were presented as frequency and percentage, a Pearson X^2 test or Fisher exact test was utilised. Using the Shapiro-Wilk test, we verified that the variables were regularly distributed or not. Parametric two numerical variables were compared using the t-test and nonparametric variables were compared using Mann-Whitney U test. The results were considered statistically significant for all analyses, where the corresponding P-value was less than 0.05. With each prediction test, we calculate a 95% confidence interval.

RESULTS

Regarding demographic data and baseline clinical data; including LL oedema, orthopnea, paroxysmal nocturnal dyspnea, jugular venous pressure >6 cm, third heart sound, pulmonary crackles-bibasilar, current smokers, hypertension, atrial fibrillation, cerebrovascular disease, chronic lung, kidney diseases, systolic and diastolic blood pressure; there were insignificant differences between both groups **(Table 1)**.

At the three-month follow-up, neither group showed any statistically significant differences in terms of LL oedema, PND, dyspnea, third heart sound, jugular venous pressure > 6 cm, or pulmonary crackles-bibasilar. The non-DM group had lower IHD and HR values compared to the DM group, which had considerably higher values (P<0.05). At the 3-month follow-up, there was a statistically significant difference in the prevalence of orthopnea between the DM and non-DM groups (P= 0.039). **(Table 1)**.

Table (1): Demographic data, clinical presentation, at baseline, follow-up after 3 months, comorbidities and vital signs of the studied groups

		DM group (n=47)	Non-DM group (n=23)	P value
Demographic data of the studied groups				
Age (years)		59.49 ± 7.89	59 ± 10	0.824
Sex	**Male**	28 (59.57%)	10 (43.48%)	0.204
	Female	19 (40.43%)	13 (56.52%)	
Weight (Kg)		71.32 ± 6.72	69.74 ± 7.98	0.389
Height (m)		167.49 ± 7.4	167.17 ± 5.93	0.859
BMI (kg/m^2)		25.54 ± 2.96	25.12 ± 3.93	0.613
NYHA classification	**III**	35 (74.47%)	17 (73.91%)	0.960
	IV	12 (25.53%)	6 (26.09%)	
Baseline clinical presentation				
LL oedema		35 (74.47%)	16 (69.57%)	0.883
Orthopnea		33 (70.21%)	14 (60.87%)	0.609
Paroxysmal nocturnal dyspnea		27 (57.45%)	14 (60.87%)	0.988
Jugular venous pressure > 6 cm		21 (44.68%)	10 (43.48%)	0.924
Dyspnea		47 (100%)	23 (100%)	1
Third heart sound		14 (29.79%)	7(30.43%)	0.955
Pulmonary crackles-bibasilar		29 (61.7%)	12 (52.17%)	0.615
Clinical presentation at follow-up after 3 months				
LL oedema		29 (61.7%)	10 (43.48%)	0.201
Orthopnea		33 (70.21%)	10 (43.48%)	**0.031***
Paroxysmal nocturnal dyspnea		24 (51.06%)	13 (56.52%)	0.799
Jugular venous pressure > 6 cm		17 (36.17%)	10 (43.48%)	0.607
Dyspnea		22 (46.81%)	13 (56.52%)	0.610
Third heart sound		10 (21.28%)	6 (26.09%)	0.883
Pulmonary crackles-bibasilar		28 (59.57%)	8 (34.78%)	0.090
Comorbidities				
Current smokers		22 (46.81%)	7(30.43%)	0.294
Hypertension		38 (80.85%)	17(73.91%)	0.723
Ischemic heart disease		24 (51.06%)	3(13.04%)	**0.002***
Atrial fibrillation		13 (27.66%)	7(30.43%)	0.809
Cerebrovascular disease		6 (12.77%)	1(4.35%)	0.413
Chronic lung disease		8 (17.02%)	1(4.35%)	0.254
Chronic kidney disease		8 (17.02%)	1(4.35%)	0.254
Vital signs				
HR (beats/min)		95.94 ± 5.55	93.13 ± 3.42	**0.03***
Systolic blood pressure (mmHg)		131.91 ± 31.18	127.39 ± 22.81	0.538
Diastolic blood pressure (mmHg)		83.4 ± 15.5	82.17 ± 13.13	0.745

Data are presented as mean ± SD or frequency %, *Significant as P value ≤0.05, BMI: Body mass index, NYHA: New York heart association, DM: diabetes mellitus, LL: lower limb edema, HR: Heart rate.

The levels of serum creatinine, BUN, NT pro-BNP, CRP, potassium, serum random glucose, and HbA1c were significantly different between the groups with and without diabetes mellitus at both the baseline and follow-up (P<0.001). The non-diabetic group had greatly elevated sodium levels and glomerular filtration rate at baseline and follow-up assessments, while the diabetes group demonstrated markedly decreased levels (P<0.05). When comparing the two groups' LDL values at the outset, there was no statistically significant difference. However, during follow-up, the DM group demonstrated significantly lower LDL levels (P=0.019) in comparison to the non-DM group. There was no statistically significant difference in the readmission rates of the two groups. **(Table 2)**.

Table (2): Laboratory investigations at baseline and at follow-up after 3 months and rehospitalization

		DM group (n=47)	Non-DM group (n=23)	P value
Kidney function test	**Serum creatinine (mg/dL)**	1.6 ± 0.22	1.37 ± 0.13	**<0.001***
	BUN (mg/dL)	28 ± 1.07	22.37 ± 1.26	**<0.001***
	Glomerular Filtration rate (ml/min)	62.17 ± 1.9	74.04 ± 3.77	**<0.001***
Lipid profile	**LDL (mg/dL)**	148.74 ± 25.72	154.48 ± 4.73	0.481
Proteins	**NT pro-BNP (pg/mL)**	1231.83 ± 22.17	1162.48 ± 8.17	**0.012***
	CRP (mg/dL)	3.06 ± 0.44	2.29 ± 0.37	**<0.001***
Electrolytes	**Sodium (mmol/L)**	135.34 ± 5.91	139.57 ± 7.72	**0.014***
	Potassium (mmol/L)	4.76 ± 0.87	4.24 ± 0.84	**0.021***
Follow-up after 3 months				
Kidney function test	**Serum creatinine (mg/dL)**	1.46 ± 0.17	0.98 ± 0.19	**<0.001***
	BUN (mg/dL)	26.03 ± 0.59	20.66 ± 0.96	**<0.001***
	Glomerular Filtration rate (ml/min)	57.87 ± 3.25	74.96 ± 4.29	**<0.001***
Lipid profile	**LDL (mg/dL)**	137.06 ± 25.31	156.96 ± 38.98	**0.019***
Proteins	**NT pro-BNP (pg/mL)**	1305.98 ± 105.99	1051.65 ± 7.47	**<0.001***
	CRP (mg/dL)	2.53 ± 0.4	1.82 ± 0.4	**<0.001***
Electrolytes	**Sodium (mmol/L)**	127.28 ± 7.37	149.22 ± 11.31	**<0.001***
	Potassium (mmol/L)	4.36 ± 0.96	3.78 ± 0.78	**0.015***
Serum random glucose (mg/dL)		226 ± 55.97	110.65 ± 18.7	**<0.001***
HbA1c (%)		6.66 ± 1.07	2.96 ± 0.72	**<0.001***
Rehospitalization		7(14.9%)	3(13%)	1

Data are presented as mean ± SD, *****Significant as P value ≤0.05, DM: Diabetes mellitus, BUN: Blood urea nitrogen, LDL: Low-density lipoprotein, NT pro-BNP: N Terminal PRO–B-type natriuretic peptide, CRP: C-reactive protein, HbA1c: Glycated hemoglobin.

At the baseline, in terms of RVSP, LVESD, LVEF, and LVEDD, there was no statistically significant difference between the categories. If diastolic dysfunction is present, the DM group had noticeably reduced values for LAVI, E′, and S, the early diastolic velocity of the mitral annulus, in comparison to the non-DM group (P <0.05). The E/e' ratio was significantly larger in the DM group compared to the non-DM group (P <0.001). Regarding LVEDD, LVESD, and RVSP, no statistically significant differences were seen between the two groups at the follow-up. The values of LVEF, LAVI, E, and systolic velocity of the S′ were considerably lower in the DM group at follow-up compared to the non-DM group (P<0.05). At follow-up, the DM group had a significantly higher E/e ratio than the non-DM group (P <0.001). **(Table 3)**.

Table (3): Echocardiographic findings of the studied groups at baseline and at follow-up after 3 months

	DM group (n=47)	Non-DM group (n=23)	P value
LVEDD (mm)	56.89 ± 11.77	60.17 ± 12.65	0.289
LVESD (mm)	45.53 ± 14.12	45.35 ± 13.55	0.959
LVEF (%)	51.4 ± 9.15	53.26 ± 8.49	0.417
LAVI (mL/m^2)	55.68 ± 2.53	66.65 ± 1.61	**<0.001***
E′ (cm/s)	5.68 ± 2.3	7.96 ± 2.67	**<0.001***
S′ (cm/s)	4.6 ± 2.04	5.83 ± 2.26	**0.026***
E/e'	21.69 ± 1.22	19.6 ± 1.11	**<0.001***
RVSP	42.85 ± 15.96	39.66 ± 9.39	0.379
Follow-up after 3 months			
LVEDD (mm)	51.77 ± 11.8	57.57 ± 12.45	0.062
LVESD (mm)	48.21 ± 14.7	46.13 ± 12.47	0.561
LVEF (%)	42.94 ± 9.73	48.35 ± 10.91	**0.039***
LAVI (mL/m^2)	56.89 ± 2.5	69.04 ± 2.87	**<0.001***
E′ (cm/s)	5.68 ± 2.3	7.96 ± 2.67	**<0.001***
S′ (cm/s)	5.09 ± 2.19	6.54 ± 2.09	**0.01***
E/e'	19.13 ± 1.11	16.92 ± 1.45	**<0.001***
RVSP	37.65 ± 11.32	39.37 ± 11.66	0.556

Data are presented as mean ± SD, *Significant as P value ≤0.05, DM: Diabetes mellitus, LVEDD: Left ventricular end-diastolic diameter, LVESD: Left ventricular end-systolic diameter, LVEF: Left ventricular ejection fraction, LAVI: Left atrial volume index, RVSP: Right ventricular systolic pressure, E′ (cm/s) early diastolic velocity of the mitral annulus, S′ (cm/s) –systolic velocity of the mitral annulus, E/e′: This ratio is a key parameter in assessing left ventricular filling pressures and diastolic function.

Sodium, potassium, HbA1c, E, S′, and E/e' were independent predictors in both diabetic and non-diabetic patients with acute HF in univariate regression, along with serum creatinine, glomerular filtration rate, NT pro-BNP, CRP, sodium, and serum random glucose. Acute HF in both diabetic and non-diabetic individuals was independently predicted by glomerular filtration rate, NT pro-BNP, CRP, sodium, serum random glucose, HbA1c, E, and E/e', but not by serum creatinine, potassium, or S′, according to multivariate regression. **(Table 4)**.

Table (4): Univariate and multivariate regression of various variables of short-term outcomes of acute (HF) in diabetic and nondiabetic patients

	Univariate regression			Multivariate regression		
	Odds ratio	**95% CI**	**P**	**Odds ratio**	**95% CI**	**P**
Serum creatinine (mg/dL)	487.7	17.11813898	**<0.001***	364.37	0.004 - 27645015.7	0.303
BUN (mg/dL)	11.3		0.998			
Glomerular filtration rate (ml/min)	0.368	0.1799 - 0.7531	**0.006***	0.3834	0.1864 - 0.7886	**0.009***
NT pro-BNP (pg/mL)	1.0068	1.0012-1.0124	**0.017***	1.0105	1.0017- 1.0193	**0.019***
CRP (mg/dL)	160.09	12.65- 2025.21	**<0.001***	197.5306	12.5981 - 3097.1502	**<0.001***
Sodium (mmol/L)	0.906	0.83- 0.983	**0.017***	0.9142	0.8401 -0.9949	**0.037***
Potassium (mmol/L)	2.0068	1.08 -3.69	**0.025***	1.8451	0.9842 - 3.4591	0.056
Serum random glucose (mg/dL)	1.1060	1.04- 1.17	**0.001***	1.1104	1.0232 - 1.205	**0.012***
HbA1c (%)	6.788	2.418-19.05	**<0.001***	8.288	1.7623 - 38.986	**0.007***
LAVI (mL/m2)	0.0002	--	0.998	---	----	--
E′(cm/s)	0.684	0.540- 0.86	**0.001***	0.656	0.4598 -0.9382	**0.02***
S′(cm/s)	0.758	0.590-0.973	**0.03***	0.7042	0.4944 -1.0029	0.051
E/e'	3.8909	2.027 7.467	**<0.001***	3.269	1.7438 - 6.1302	**<0.001***

Data are presented as *Significant P value ≤0.05, BUN: Blood urea nitrogen , CI: Confidence interval, NT pro-BNP: N Terminal PRO–B-type natriuretic peptide, CRP: C-reactive protein, HbA1c: Glycated hemoglobin, LVEF: Left ventricular ejection fraction, LAVI: Left atrial volume index , E′ (cm/s) early diastolic velocity of the mitral annulus ,S′ (cm/s) –systolic velocity of the mitral annulus , E/e′: This ratio is a key parameter in assessing left ventricular filling pressures and diastolic function.

DISCUSSION

HF can occur from a variety of structural or functional cardiac problems; the clinical state is complex, but systolic or diastolic myocardial dysfunction is the underlying structural basis. This illness is the culmination of a series of heart and metabolic cardiovascular function abnormalities; it mostly impacts the left ventricle [10].

In our study, regarding baseline and follow up clinical presentation, LL oedema, orthopnea, PND, jugular venous pressure >6 cm, dyspnea, third heart sound and pulmonary crackles bibasilar were insignificantly different between both groups. While orthopnea was significantly higher in DM group than non-DM group at follow up.

Supporting our findings, **Izraiq *et al.*** [11] found that a significant difference was observed at the follow-up between the non-DM and DM groups with respect to LVEF, LAVI, E, and systolic velocity of the mitral annulus (S′) ($P<0.05$). The E/e ratio was significantly greater in the DM group ($P <0.001$) as compared to the non-DM group at follow-up.

Also, **Raharinavalona *et al.*** [12] found no correlation between the presence or absence of type 2 diabetes and the following symptoms: left ventricular oedema, high jugular venous pressure, pulmonary crepitations, or third heart sound.

In the current study, in terms of hypertension, chronic renal disease, cerebrovascular illness, atrial fibrillation, or current smoking, there was no significant difference between the two groups. The major reason the DM group had a much higher incidence of IHD than the non-DM group was due to the DS.

In agreement with our findings, **Cho *et al.*** [13] found that neither the non-DM nor the DM group differed significantly with respect to smoking, CVD, or chronic CKD. Compared to the non-DM group, the DM group had a much greater incidence of IHD.

Also, **Kong *et al.*** [14] discovered no significant difference between the diabetes and non-diabetic groups with regard to the prevalence of chronic obstructive pulmonary disease and current smoking. There was a statistically significant difference in the incidence of IHD between the groups with and without diabetes mellitus. There were some shared symptoms between the two groups, but the diagnoses of hypertension, atrial fibrillation, and cerebrovascular illness were very different. The difference could be due to different study objectives and sample numbers.

The significant difference in HR between the DM and non-DM groups may be due to cardiac autonomic neuropathy. When we looked at diastolic and systolic blood pressure, statistical analysis did not show that the two groups were different.

Supporting our findings, **Mebazaa *et al.*** [15] discovered that compared to the non-diabetic group, the diabetic group had noticeably higher HR. There was no statistically significant difference in DBP between the diabetic and non-diabetic groups.

Also, **Kristensen *et al.*** [16] discovered that the non-DM group's HR were noticeably lower than the diabetic group's. There was no significant difference in SBP between the diabetic and non-diabetic groups.

In disagreement with our findings, **Kong *et al.*** [14] discovered that the groups without diabetes had similar HR. The diabetes group had significantly greater SBP compared to the control group. Compared to the non-DM group, the DM group had a reduced DBP.

The non-DM group had significantly lower levels of serum glucose, HbA1c, pro-BNP, CRP, potassium, and BUN compared to the DM group both at baseline and after follow-up laboratory testing. In comparison to the non-diabetic group, the diabetic group had substantially reduced glomerular filtration rate and sodium levels. The LDL levels of the diabetic group were substantially lower than those of the non-DM group during follow-up, despite the fact that both groups' levels were comparable at baseline. Supporting our findings, in the study of **Kong *et al.*** [14] the diabetes group exhibited significantly lower levels of sodium compared to the non-diabetic group, while the diabetic group exhibited significantly higher levels of creatinine, BUN, pro-BNP, CRP, potassium, HbA1c, and blood glucose in terms of baseline laboratory testing.

Also, **Farkouh *et al.*** [17] reported that LDL at follow up was lower in patients with coronary heart disease with T2DM.

The two groups did not differ significantly from one another in terms of baseline echocardiographic results, including LVEF, RVSP, LVEDD, and LVESD. The DM group had substantially reduced LAVI, E′, and S′ compared to the non-DM group. Compared to the non-DM group, the DM group had a substantially higher E/e'. When comparing the two groups at follow-up, there was no statistically significant difference in LVEDD, LVESD, or RVSP. The DM group had much decreased LVEF, LAVI, E, and S′ compared to the non-DM group.

Supporting our findings **Akashi *et al.*** [18] found that the DM group had considerably greater E/e′ than the non-DM group at both the baseline and mid-term follow-up measurements.

Also, **Swiatkiewicz *et al.*** [19] reported that in diabetic patients, elevated E/E’ are the most prevalent In this study, we used the univariate regression method to predict the short-term prognosis of HF in both diabetic and non-diabetic patients based on sodium, potassium, CRP, E, S′, and E/e'. While serum creatinine, LDL, C-reactive protein, potassium, and blood glucose were not independent predictors of the short-term outcome of HF in both diabetic and non-diabetic patients, LVEF, E, S′, and E/e' were in multivariate regression.

Supporting our findings, **Wan *et al.*** [20] reported that LVEF and E/e' were independent predictors of outcome of HF in diabetic and non-diabetic patients.

Also, **Kong *et al.*** [14] determined the short-term fate of HF from the patient's serum creatinine concentration and LVEF, regardless of diabetes status.

Limitations: Single center study that may result in different findings than elsewhere, small sample size and limited follow-up period.

CONCLUSION

Individuals with acute HF and diabetes exhibited less favourable short-term clinical outcomes. E, S, E/e', LVEF, sodium, potassium, CRP, LDL, and HF short-term outcomes were independently predicted in diabetic and non-diabetic patients. Low LDL VLDL, E′, and S′ levels were associated with HF and DM, but orthopnea, HR, and E/e' were higher in the diabetic group.

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