Chapter Three

Results

A total of 100 Sudanese subjects were enrolled in this study, 50 of them were diabetic patients with CVDs and 50 healthy individuals as a control group. The mean age of patients was 62.64 years and of controls was 57.24 years (Table3.1).

Table 3.1 Comparison of age in patients and controls.

Variables	**Age (years)**	
	Mean	**SD**
patients	62.64	8.778
Controls	57.24	17.699

Twenty seven (54%) of patients were females and 23(46%) were males, while 21(42%) of controls were females and 29(58%) were males (Figure 3.1). There was no statistically significant difference in gender according to genotypes (*P. value* 1.23).

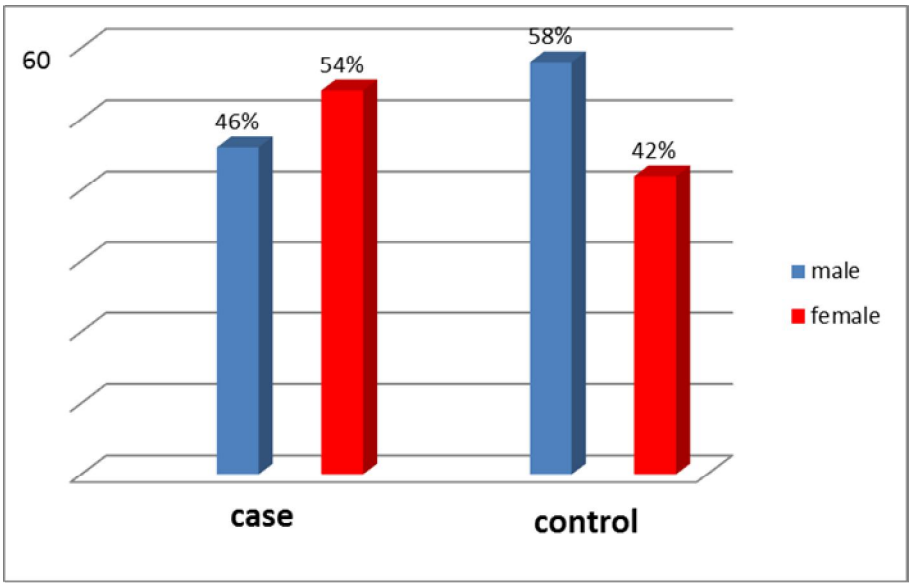

Figure 3.1 Distribution of gender in patients and controls.

AS(atherosclerosis), CM(cardiomyopathy), MI(myocardial infarction), AF(atrial fibrillation), MVR(mitral valve regurgitation), AVR(aortic valve regurgitation), MYO(myocardial hyperatrophy).

Twenty eight (56%) of diabetic patients were also hypertensive (Figure 3.2).

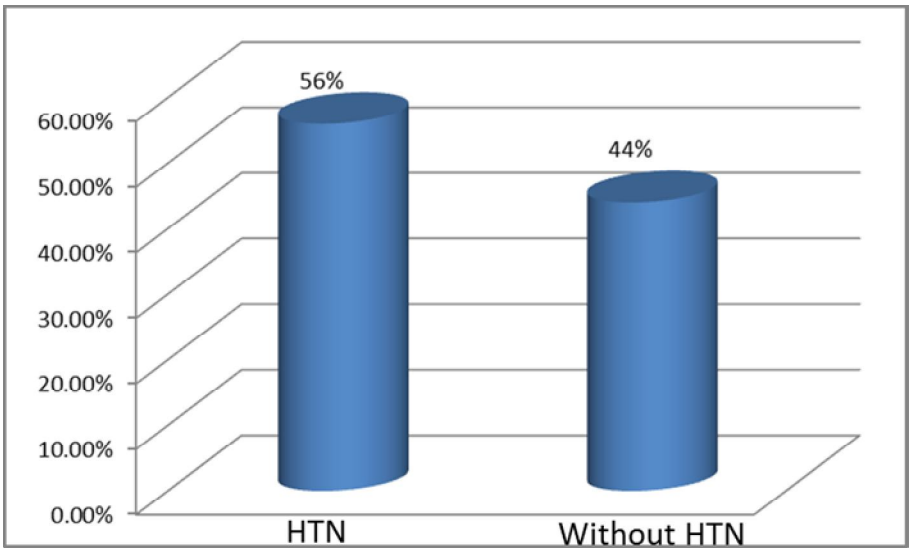

Figure 3.2 Frequency of hypertension among patients group.

The DD genotype was the most frequent in both patients and control groups, followed by ID genotype, while II genotype was totally absent. There was no statistically significant difference in genotype distribution when compared in patients and control groups (Table3.2).

Table 3.2 Genotypes distribution in patients and control groups

Variables	Genotypes distribution		Total	*P. value*
	DD	ID		
Patients	36 (72%)	14 (28%)	50(100%)	0.646
Control	37 (74%)	13 (26%)	50(100%)	

There was no statistically significant difference in mean of disease duration and mean age according to genotype (Table3.3).

Table 3.3 Comparison of mean duration of diabetes mellitus and mean age of patients according to genotypes

Patients	Mean ±SD		*P. value*
	DD	ID	
Age	62.75±8.453	62.36±9.896	0.889
Duration	12.28±8.410	12.21±8.894	0.981

Table 3.4 Genotypes distribution in patients with and without HTN

Patients	**DD**	**ID**	**Total**	***P. value***
HTN	21(42%)	7(14%)	28(56%)	0.59
Without HTN	15(30%)	7(14%)	22(44%)	
Total	36(72%)	14(28%)	50(100%)	

There was no statistically significant association between ACE I/D polymorphic genotypes and types of CVDs (Table3.5).

Table 3.6 Genotypes distribution in CVDs types

Patients	DD	ID	Total	*P. value*
AS	11(22%)	3(6%)	14(28%)	
CM	8(16%)	1(2%)	9(18%)	
Angina	1(2%)	1(2%)	2(4%)	
MI	4(8%)	2(4%)	6(12%)	
AF	2(4%)	1(2%)	3(6%)	
MVR	2(4%)	3(6%)	5(10%)	
AVR	3(6%)	1(2%)	4(8%)	
CM,AF	1(2%)	0(0%)	1(2%)	0.49
MVR,AF	1(2%)	0(0%)	1(2%)	
AS,CM	0(0%)	1(2%)	1(2%)	
AS,MVR	0(0%)	1(2%)	1(2%)	
Ischemia	1(2%)	0(0%)	1(2%)	
Myocardial hypertrophy	1(2%)	0(0%)	1(2%) 1(2%)	
Arrhythmia	1(2%)	0(0%)	1(2%)	
Total	36(72%)	14(28%)	50(100%)	

The logistic regression showed no interaction between ACE genotypes and other known MI risk factors (Table 3.7).

Table 3.6 Interaction between ACE I/D polymorphism and other risk factors in diabetic patients with CVDs

Variables	OR	95%C.I.		*p. value*
		Lower	Upper	
Age	0.995	0.927	1.068	0.886
Duration	0.999	0.928	1.076	0.981
Types of CVDs	1.075	0.922	1.254	0.389
Other chronic diseases	0.714	0.207	2.467	0.595

ACE: angiotensin converting enzyme; I: insertion; D: deletion; OR: odds ratio; CI: confidence interval.

The frequency of D allele was 0.86 in patients and 0.87 in control group, while the frequency of I allele was 0.14 in patients and 0.13 in control group. The association between D and I allele was not statistically significant. No deviation from Hardy Weinberg equilibrium was observed (X^2= 1.33, df=1, P>0.067).