A CLINICAL PROFILE ON STUDY OF CORONARY ARTERY DISEASE IN FEMALES

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Abstract:

As per the per the Study CAD in young women is increasing. Occupational stress, family stress and psychosocial factors are important risk factors in young MI. Obesity and metabolic syndrome including (PCOS) polycystic ovarian syndrome are noted as risk factors as per our study. The anginal symptoms of CAD are lacking in women with diabetes as typical symptoms of CAD are lacking, they presented in evolved stage of MI. Systemic hypertension and diabetes increase CAD mortality and morbidity. In our study low HDL and high LDL are noted as CAD risk factors. Early menopause in women had contributed to the development of CAD. Congestive cardiac failure was common in those women who presented late to the hospital.

Keywords: CAD, PCOS, MI, HDL, LDL,

Introduction

Cardiovascular disease remains the leading cause of death in women regardless of race or ethnicity. More women than men have died yearly from cardiovascular disease. It accounts for 1 in 3 women death. Mortality rates for CAD decreases for both men andwomen and rate of decrease is less in women than men. A greater proportion of women of 52 % and men of 42 % with ACS died of sudden death before they reaching the hospital. The worldwide INTERHEART study has revealed that women develop MI 10 years later than men, but mortality among women is greater[1,2].

Cardiovascular mortality has decreased in women similar to men since 1980s. The importance of coronary artery disease and its prevention in women is receiving increased physician attention. Exploration of sex differences also increased. Evidence based guidelines has been updated with expert panel review for prevention of CAD in women. Death among women is higher than men due to CAD. There was substantial ethnic variation among responses of women in 2005 survey[3,4].

CAD risk in Indian Women

Throughout the world half of the all MI occur under the age of 50 years and 25 % under the age 40. Excess burden of premature CAD is due to genetic susceptibility which is mediated by elevated level of Lipoprotein-a (LPa) and smaller caliber of the coronary arteries. The CAD mortality among Indians is greater among women than men. Three vessel disease on angiography is seen in one third of premenopausal women. The CAD mortality in women between 45 -64 years of age is double than in whites. In Singapore, CAD mortality among Asian Indian women 30-39 yearsof age is 8-fold higher than Chinese women of same age[5,6].

Angina pectoris due to reversible myocardial ischemia is caused by obstructive CAD that limits blood flow during myocardial oxygen demand. This syndrome will not affect women until they became elderly with the exception of diabetic women[7,8].

CAD in India Statistics[9,10]:

12 % in urban, 4 times more than USA, 6 times more than European, 20 times more than Japanese.

- 10 years earlier 53 Vs 63
- Virulent Progression
- Rural also increasing
- 50 % MI occur below 50 years
- Death due to MI is 2 -3 times greater

As Per the Review of Literature

Cardiovascular disease is the leading cause of death among women. This accounts for more death than from stroke, lung cancer, COPD and breast cancer combined. About half of these deaths results from coronary heart disease. Mortality rate decreases if modifiable risk factors like hypertension, cholesterol, smoking, physical inactivity are modified[11].

Mortality rate of STEMI among women is higher than men. Thesymptoms of ACS are atypical like epigastric discomfort, giddiness, and tiredness and also they present late to the hospital. The first presentation of CAD among women is 10 years later than among men and most commonly after menopause. The onset of CAD is earlier in developing countries. Mortality rate is higher among women than men in both developed and developing countries[12].

CAD

Acute coronary syndrome is a term representing a common end result, acute myocardial ischemia. Acute ischemia is usually, but not always caused by atherosclerotic plaque rupture, fissuring, erosion or a combinationwith superimposed intracoronary thrombosis and is associated with an increased risk of cardiac death and myonecrosis. It includes STEMI, NSTEMI, and unstable Angina. ST-segment elevation myocardial Infarction (STEMI) represents the most lethal from of ACS in which totally occluding thrombus results in total cessation of coronary blood flow in the territory of occluded artery and results in ST elevation in ECG[13].

STEMI:

Stages of STEMI

1. Early hyperacute phase - within hours
2. Fully Evolved acute phase - within days
3. Fully Evolved subacute phase - within weeks
4. Chronic stabilized phase - within months

The different modalities of management are planned according to the time of infarction and so it is essential to not only diagnose AMI, but also toidentify the stage of AMI[14,15].

Anatomy of Coronary Circulation

The Coronaries are the branches from the Right Coronary artery which runs in the right AV groove and divided into posterior descending artery and posterior left ventricle branches. The artery that gives rise to

posterior descending branch is the dominant coronary artery. The left main trunk is 1 cm to 4 cm in length and divides into left anterior descending artery and left circumflex artery. The LAD supplies the Ventricular septum and anterolateral wall. The LCX supplies the Lateral wall of LV and LA[16,17].

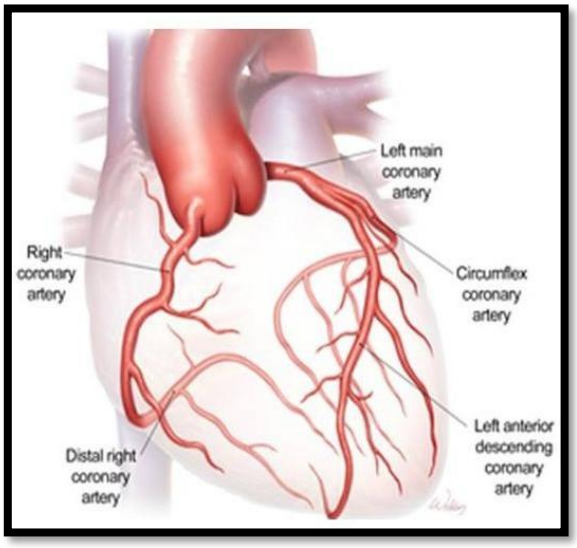


Fig No-01- Arteries in Heart

CAD risk factors and risk factor Modification

The classic risk factors for CAD are divided into those that are potentially modifiable (like diabetes, hypertension, hyperlipidemia, cigarette smoking, obesity and sedentary life style) and those that are not modifiable (age and family history)[18].

Lifestyle risk factors

Overweight is defined as BMI > 25 Kg/m^2. Obesity in women increases cardiovascular mortality. 35 % of women are obese[16]. 65% women are never engaged in vigorous physical activity. only 10 % of women engaged in vigorous activity of 5 days or more per week. Family and occupational stress also contributes CAD risk among women. Smoking among women increases CAD events.

Ethnic and racial differences in obesity occur. Obesity is linked to multiple cardiac risk factors including insulin resistance, diabetes, hypertension and hyperlipidemia and is independently associated with coronary events. The pattern of weight distribution also predicts CV events. Apple shape obesity has more risk than pear shape obesity who have weight on the hips and buttocks. A greater waist circumference increases health risk regardless of BMI[18].

Menopause and hormonal Therapy

Women with early menopause after gynecologic surgery are facing more CAD risk because of low hormone exposure. Estrogen alone contraindicated in women with uterus because of endometrial cancer[19].

Diagnosis of coronary artery disease in Women

CAD is often diagnosed with careful clinical history noninvasive stress testing aids in the treatment of individuals with intermediate risk for CAD. Unfortunately, each noninvasive technique has limitations in women.

Exercise stress testing is noninvasive way to assess CAD risk. Women has lower sensitivity and specificity with exercise stress testing than men due to low ECG voltage. Women have more frequent ST – T wave abnormalities. Negative stress testing reduces the need for catheterization. Stress imaging tests are used for assessing CAD severity. Nuclear stress perfusion testing with technetium is preferred for assessing CAD severity. Angina symptoms are less predictive of abnormal coronaries in women than men. Early multi detector CT Angiography reveals important sex differences in specific type of coronary lesions[20].

Congestive Heart failure

Women with CHF tend to have preserved systolic function and are older with hypertension. Although use of ICDs have increased among patients with low systolic EF. Sudden death occurs more often in men than women. Arrythmia are more common in women than men. Aggressive management and secondary prevention for documented CAD will improve symptoms[21].

Material & Methods

Materials:

Source of Data:

This study data was collected at Govt hospital. Guntur, Andhra Pradesh in collaboration with the department of Cardiology during the period of January 2022 to January 2023.

Study Design:

A prospective observationalUnicentric study

Study Duration:

12 months

Inclusion Criteria:

Study was conducted on 80 patients ranging in women aged 38 to 80 years. They were grouped in to two.

Group A - Premenopausal women

Group B - Postmenopausal women

Female patient who are admitted with symptoms and ECG or ECHO evidence of CAD.

All the particulars were inquired by a questionnaire containing their history, personal h/o, family h/o.

Exclusion Criteria: OP patients

Consent:

An informed consent was obtained from all the patients.

Methods of collection of data:

Blood sample collection

Nearly 8 ml of blood was collected in the fasting state and the below mentioned factors have been analyzed. Fasting blood sugar, lipid profile, are taken in empty stomach. Hb_1AC, Urine PCR, Urea creatinine were estimated in another sample.

Blood Pressure recording:

BP was recorded in sitting position with sphygmomanometer. Based on Korotkoff sounds BP was recorded. BP at which the sounds started to appear had been taken as systolic value of BP and that when sound disappears had been taken as diastolic pressure. The size of the BP cuff used was 12 X 22 cm. Calculation of body mass index:

Anthropometric measurements:

Height was recorded with tape to the nearest one centimeter. Subjects were instructed to stand upright without shoes with theirback against the wall, foot together and eyes directed forward.

Weight was measured with weighing machine using spring balance that was kept on firm horizontal surface. The scale was checked on daily basis and calibration was done with known weights. Weight was recorded to the nearest 0.5 Kg.

The formula given below has been used to calculate BMI:

BMI = Weight in KG / (Height in Meters)

Analysis of data:

The information collected regarding all the cases were tabulated in a master chart. Data was analyzed with help of statistical software tool epidemiological information package. Using this software range, frequencies, percentages, means, standard deviations, and p values were calculated.

Results:

In the present study the data collected was given the different parameters about the various age groups of females

Descriptive Statistics **Table no: 01 Descriptive Statistics**

S. No	Item	Min.	Max	Mean	S. D
1	Age	38	80	55.29	10.994
2	weight	46	80	63.28	7.144
3	Height	149	180	162.97	7.567
4	BMI	17.36	35.56	23.9367	3.33910
5	BP	110/70	180/110	145/90	4.68
6	TC	115	261	186.59	24.474
7	TGL	106	310	179.93	40.873
8	HDL	30	44	38.45	2.490
9	LDL	28	190	112.54	24.720
10	VLDL	21	62	35.99	8.175
11	EF	42	70	59.77	7.741
12	Bld. Urea	14	42	25.95	6.601
13	S. Creat	.60	1.60	.9875	.25364
14	HB	6.00	11.50	9.3363	1.15969
15	Hb_1AC	4.6	9.5	7.5	1.5

Statistical Graph

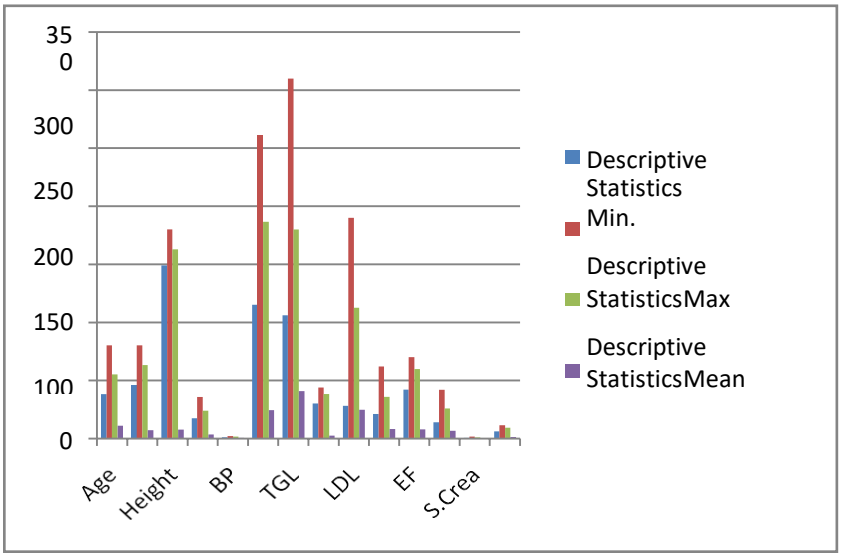
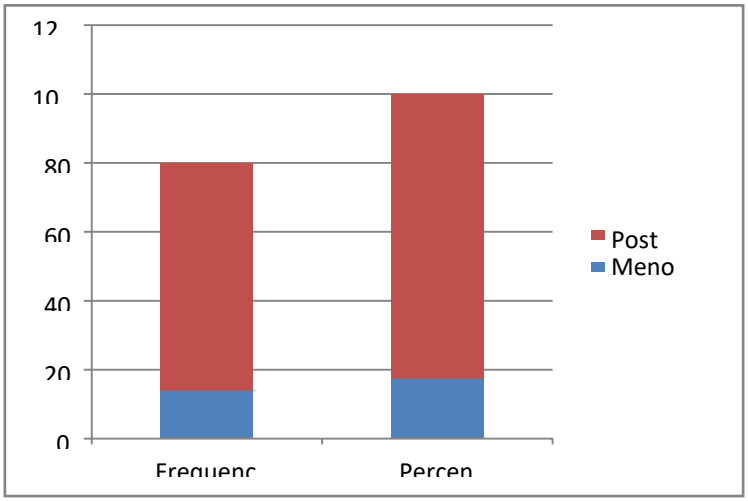


Figure No:01 Descriptive Statistics

Sample

Particulars	Frequency	Percent
Pre Meno	14	17.5
Post Meno	66	82.5

Table No: 02 Sample Particulars



Post Meno & Pre Meno-Graph

Figure No:02 Post Meno & Pre Meno-Graph

Age

Particulars	Frequency	Percent
Below 45yrs	14	17.5
46 to 59yrs	41	51.3
60yrs & above	25	31.3

Table no: 03 Age Particulars

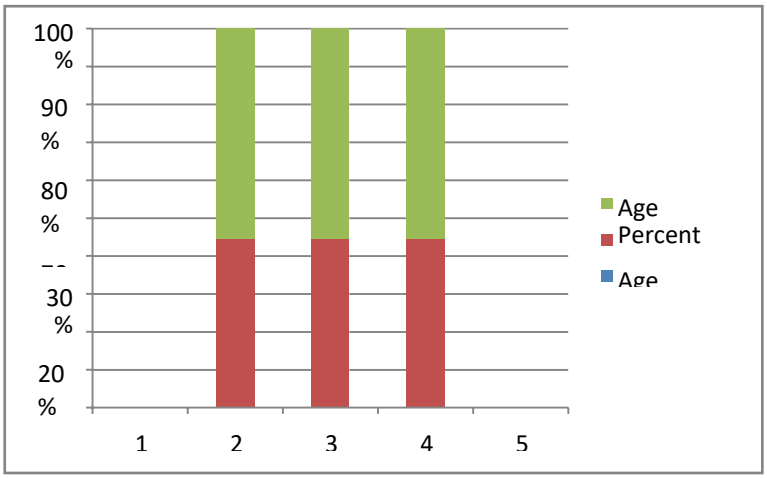


Figure No: 03 Age Percentage Frequency Graph

BP

Particulars	Frequency	Percent
High	30	37.5
Normal	50	62.5

Table No: 04 BP Particulars

Blood Pressure Graph

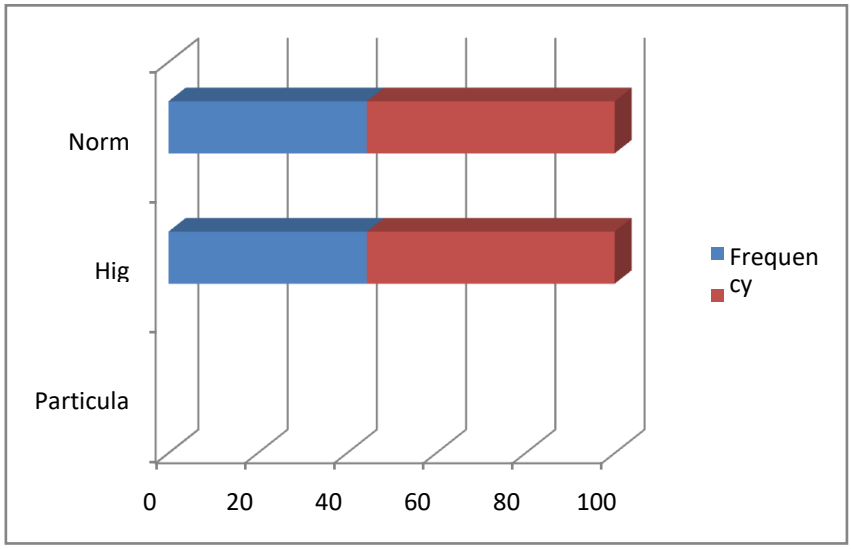


Figure No: 04 BP Graph

ECG

Particulars	Frequency	Percent
STEMI	33	41.3
NSTEMI	16	20.0
UNSTABLE ANGINA	31	38.8

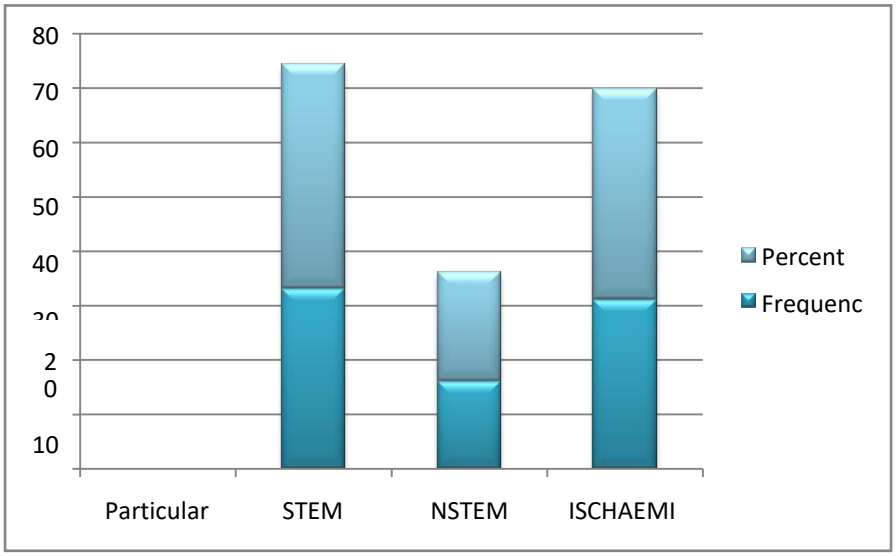


Table No: 05 ECG Particular

Figure No: 05 ECG Graph

ECHO

Particulars	Frequency	Percent
RWMA -	50	62.5
RWMA +	30	37.5

Table No: 06 ECHO Particulars

ECHO Graph

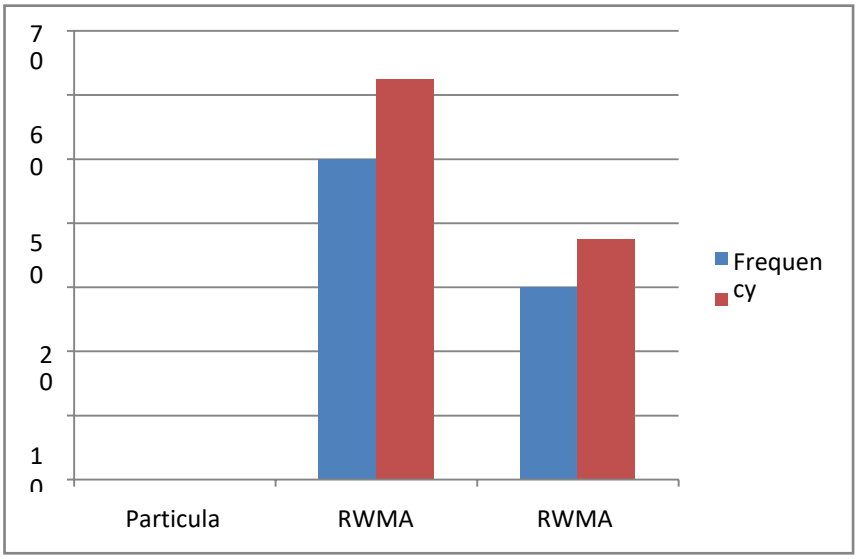


Figure No: 06 ECHO Graph

Discussion:

This study was conducted to find out risk factors and clinical features of coronary artery disease in women. The study population was 80 patients. They are divided into premenopausal and postmenopausal. 14 patients werein premenopausal group and 66 patients were postmenopausal group. Fasting lipid profile was measured using an auto analyzer after 12 hrs. of fasting. FBS, PPBS, Hb1AC, Urine PCR, BMI were also calculated. The results were statistically analyzed.

Age distribution:

The mean age for premenopausal group was 40 years. The young MI that involving in CAD is 38 years. CAD occurs between 46 to 59 years of age.

Distribution of BMI among the groups:

Mean BMI among premenopausal age was 26, S.D 4.79. ie., They are overweight. BMI among postmenopausal age was 23. S.D.2.671. We compared BMI between Pre and Postmenopausal age groups; the p value is significant (p 0.001), obesity plays significant role for causation of CAD among women.

Mean total cholesterol in premenopausal women is 184.57 SD 22.3. The mean total cholesterol among postmenopausal women 187.02, S.D.25.049. On statistical analysis we found that there is no significant alteration in cholesterol between premenopausal and postmenopausalwomen.

TGL:

We observed that TGL were significantly elevated in premenopausalwomen (mean 191.86) S.D 40.733.

For postmenopausal women mean was 177.39 with S.D of 40.76. There is no statistical significance between two groups.

HDL:

In our study the mean HDL among premenopausal women is 40.14, S.D 2.033, and among postmenopausal women 38.09, SD 2.441. This was found very significant on statistical analysis with a p value < 0.004.

LDL:

In our study mean LDL among premenopausal women was 106.06, S.D 18.607 and among postmenopausal women it was 113.91, S.D 25.739. We didn't find any statistically significant difference in LDL level betweenpremenopausal and postmenopausal women.

VLDL:

We observed that, there is no significant rise in serum VLDL level between pre and postmenopausal women (p value 0.23, 70.05). The mean value of VLDL among premenopausal women was 38.37; S.D 8.147 andamong postmenopausal women was 35.48, S.D 8.152.

BP:

There is no significant statistical difference between premenopausal and postmenopausal groups. We observed that 37.5 % of women having high BP and 62.5 % have normal BP.

FBS & PPBS:

FBS in premenopausal in an average was 142.93; S.D 27.647 and postmenopausal women it was 99.53, SD 24.99. There is significant statistical difference between premenopausal and postmenopausal women in FBS value. PPBS value also got significant difference between pre and postmenopausal women.

PPBS in premenopausal women was 257.36 S.D 66.89 and postmenopausal women it was 159.82, S.D 60.98.

Hb_1AC:

We observe that Hb_1AC were significantly elevated among premenopausal women 7.7143, S.D 1.442 and among postmenopausal women it was 6.01, S.D 1.69. This was found very significant on statistical analysis with a p value of <0.001

ECG:

ST segment elevation MI was observed in 33% of patients. 16% were NSTEMI and 31 % were unstable angina. Most of the patient present late to the hospital. In ECG Q was present in most of the patient present in evolved stage and most of them were lacking typical symptoms.

ECHO:

On ECHO Regional wall motion abnormalities were present in 30 % of patients and it was absent in 50 % of

patients. Some present early in hyperacute phase itself . So myocardial salvage was good.

EF:

We observed statistically significant difference in ejection fraction between premenopausal and postmenopausal women (p 0.024 < 0.05). The average ejection fraction among premenopausal women was 64.00 S.D 5.491, and among postmenopausal women was 58.88, S.D 7.883. Women with CAD present late to the hospital.

CONCLUSION

REFERENCES

1. Thom T, Hasse N, Rosamond W, et al.Heart disease and stroke statistics- 2006 update. A report from the American Heart Associationstatistics.

2. Mosca I, Banka CI, Benjamin EJ, et al. Evidence based guidelines for cardiovascular disease prevention in women : 2007 update.

3. Shaw LJ, Bugiardini R, Merz NB, Women and Ischemic heart disease evolving knowledge J AM college of cardiol 2009.

4. Mosca L, Linfante A, Benjamin E, et al. National study of physician Awareness and adherence and cardiovascular disease prevention guidelines circulation 2005;

5. Arbustini E, Dal Bello B, Morbini P, et al: Plaque erosion is a major substrate for coronary thrombosis in AMI Heart 1999.

6. Enas EA, Senthilkumar A, Juturu V, Gupta R CAD in women Indian Heart J 2001.

7. Lloyd.Jones D, Adams R, Carnethon M, et al: Heart disease and stoke statistics – 2009 update A report from the American Heart Associationstatistics committee and stroke statistics subcommittee.

8. Yamada Y, Izawa H, I chihara S, et al: Prediction of the risk of myocardial infarction from polymorphisms in candidate genes ne jm 2002.

9. Age – specific relevance of usual blood pressure to vascular mortality. A meta -analysis of individual data for one million adults in61 prospective studies Lancet 2002; 360:1903.

10. Sagie A. The natural history of borderline isolated systolic hypertension. NEIM 1993.

11. Rigotti NA, Pipe AL, Benowirz, et al: Efficacy and safety of varenicline for smoking cessation in patients with cardiovascular disease; a randomized trial circulation 2010; 121:221-220.

12. Centers for disease control and prevention. Women and smoking a report of the surgeon

General (executive summary). MMWR mort mortal Wkly Rep 2002.

13. Schneider JG, Tompkins C, Blumenthal RS, et al: The metabolic syndrome in women. Cardio Rev, 2006.

14. Wild R, polycystic ovary syndrome : a risk for coronary artery disease? Am J obstet Gynecol 2002,

15. Ford ES, mokdad AH, Giles WH, Mensah GA, Serum total cholesterol concentration and awareness, treatment and control of hypercholesterolemia among Vs adults: Findings from the National health and Nutrition Examination survey, 1999 and 2000.

16. Ogden CL, Carrdl MD, Mc Dowell MA, Flegal KM: Obesity among adults in the united states : No statistically significant change since 2003-2004.

17. Figueredo VM. The time has come for physicians to take notice: The impact of psychosocial stressors on the heart. AM J med.2009.

18. Charney P.Future directions. In: Charney P. ed CAD in Women : prevention, Diagnosis and management, American College of Physicians; 1999.

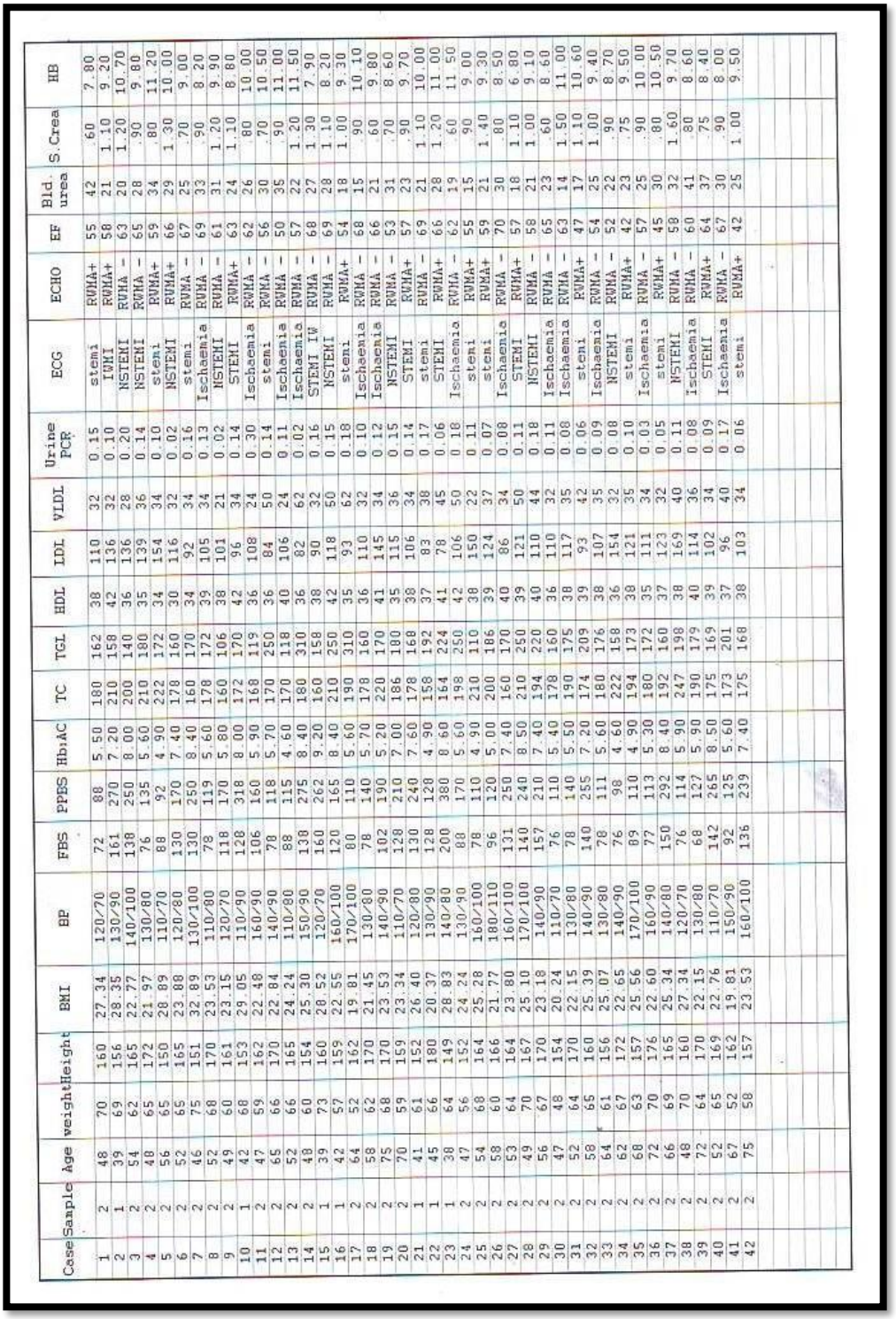
19. Yan LL, Liu K, Daviglus MI, et al; Education, 15 years risk factor progression, coronary artery calcium in young adulthood and early middle age. The coronary Artery risk development in young adults study JAMA 2006.

20. ANTMAN EM : STEMI : Management, in: P Libby, Ro Bonow, DLMann, DP zipes (eds), Braunwald's Heart Disease.

21. Braunwald E, ANTMAN EM: STEMI pathology, pathophysiology and clinical features in P Libby, Ro Bonow, DL Mann, DP zipes (eds)Braunwald's Heart disease.

APPENDIX

MASTER CHART



Case	Sample	Age	weight	Height	BMI	BP	FBS	PPBS	Hb1AC	TC	TGL	HDL	LDL	VLDL	Urine PCR	ECG	ECHO	EF	Bld. urea	S.Crea	HB
1	2	48	70	160	27.34	120/70	72	88	5.50	180	162	38	110	32	0.15	stemi	RVMA+	55	42	.60	7.80
2	1	39	69	156	28.35	130/90	161	270	7.20	210	158	42	136	32	0.10	IWMI	RVMA+	58	21	1.10	9.20
3	2	54	62	165	22.77	140/100	138	250	8.00	200	140	36	136	28	0.20	NSTEMI	RVMA -	63	20	1.20	10.70
4	2	48	65	172	21.97	130/80	76	135	5.60	210	180	35	139	36	0.14	NSTEMI	RVMA -	65	28	.90	9.80
5	2	56	65	150	28.89	110/70	88	92	4.90	222	172	34	154	34	0.10	stemi	RVMA+	59	34	.80	11.20
6	2	52	65	165	23.88	120/80	130	170	7.40	178	160	30	116	32	0.02	NSTEMI	RVMA+	66	29	1.30	10.00
7	2	46	75	151	32.89	130/100	130	250	8.40	160	170	34	92	34	0.16	stemi	RVMA -	67	25	.70	9.00
8	2	52	68	170	23.53	110/80	78	119	5.60	178	172	39	105	34	0.13	Ischaemia	RVMA -	69	33	.90	8.20
9	2	49	60	161	23.15	120/70	118	170	5.80	160	106	38	101	21	0.02	NSTEMI	RVMA -	61	31	1.20	9.90
10	1	42	68	153	29.05	110/90	128	318	8.00	172	170	42	96	34	0.14	STEMI	RVMA+	63	24	1.10	8.80
11	2	47	59	162	22.48	160/90	106	160	5.90	168	119	36	108	24	0.30	Ischaemia	RVMA -	62	26	.80	10.00
12	2	65	66	170	22.84	140/90	78	118	5.70	170	250	36	84	50	0.14	stemi	RVMA -	56	30	.70	10.50
13	2	52	66	165	24.24	110/80	88	115	4.60	170	118	40	106	24	0.11	Ischaemia	RVMA -	50	35	.90	11.00
14	2	48	60	154	25.30	150/90	138	275	8.40	180	310	36	82	62	0.02	Ischaemia	RVMA -	57	22	1.20	11.50
15	1	39	73	160	28.52	120/70	160	262	9.20	160	158	38	90	32	0.16	STEMI IW	RVMA -	68	27	1.30	7.90
16	1	42	57	159	22.55	160/100	120	165	8.40	210	250	42	118	50	0.15	NSTEMI	RVMA -	69	28	1.10	8.20
17	2	64	52	162	19.81	170/100	80	110	5.60	190	310	35	93	62	0.18	stemi	RVMA+	54	18	1.00	9.30
18	2	58	62	170	21.45	130/80	78	140	5.70	178	160	36	110	32	0.10	Ischaemia	RVMA -	68	15	.90	10.10
19	2	75	68	170	23.53	140/90	102	190	5.20	220	170	41	145	34	0.12	Ischaemia	RVMA -	66	21	.60	9.80
20	2	70	59	159	23.34	110/70	128	210	7.00	186	180	35	115	36	0.15	NSTEMI	RVMA -	53	31	.70	8.60
21	1	41	61	152	26.40	120/80	130	240	7.60	178	168	38	106	34	0.14	STEMI	RVMA+	57	23	.90	9.70
22	1	45	66	180	20.37	130/90	128	128	4.90	158	192	37	83	38	0.17	stemi	RVMA -	69	21	1.10	10.00
23	1	38	64	149	28.83	140/80	200	380	8.60	164	224	41	78	45	0.06	SIEMI	RVMA+	66	28	1.20	11.00
24	2	47	56	152	24.24	130/90	88	170	5.60	198	250	42	106	50	0.18	Ischaemia	RVMA -	62	19	60	11.50
25	2	54	68	164	25.28	160/100	78	110	4.90	210	110	38	150	22	0.11	stemi	RVMA+	55	15	.90	9.00
26	2	58	60	166	21.77	180/110	96	120	5.00	200	186	39	124	37	0.07	stemi	RVMA+	59	21	1.40	9.30
27	2	53	64	164	23.80	160/100	131	250	7.40	160	170	40	86	34	0.08	Ischaemia	RVMA -	70	30	.80	8.50
28	2	49	70	167	25.10	170/100	140	240	8.50	210	250	39	121	50	0.11	STEMI	RVMA+	57	18	1.10	6.80
29	2	56	67	170	23.18	140/90	157	210	7.40	194	220	40	110	44	0.18	NSTEMI	RVMA -	58	21	1.00	9.10
30	2	47	48	154	20.24	110/70	76	110	5.40	178	160	36	110	32	0.11	Ischaemia	RVMA -	65	23	.60	8.60
31	2	64	64	170	22.15	130/80	78	140	5.50	190	175	38	117	35	0.08	Ischaemia	RVMA -	63	14	1.50	11.00
32	2	52	65	160	25.39	140/90	140	255	7.20	174	209	39	93	42	0.06	stemi	RVMA+	47	17	1.10	10.60
33	2	64	61	156	25.07	130/80	78	111	5.60	180	176	38	107	35	0.09	Ischaemia	RVMA -	54	25	1.00	9.40
34	2	62	67	172	22.65	140/90	76	98	4.60	222	158	36	154	32	0.08	NSTEMI	RVMA -	52	22	.90	8.70
35	2	68	63	157	25.56	170/100	89	110	4.90	194	173	38	121	35	0.10	stemi	RVMA+	42	23	.75	9.50
36	2	72	70	176	22.60	160/90	77	113	5.30	180	172	35	111	34	0.03	Ischaemia	RVMA -	57	25	.90	10.00
37	2	66	69	165	25.34	140/80	150	292	8.40	192	160	37	123	32	0.05	stemi	RVMA+	45	30	.80	10.50
38	2	48	70	160	27.34	120/70	76	114	5.90	247	198	38	169	40	0.11	NSTEMI	RVMA -	58	32	1.60	9.70
39	2	72	64	170	22.15	130/80	68	127	5.90	190	179	40	114	36	0.08	Ischaemia	RVMA -	60	41	.80	8.60
40	2	52	65	169	22.76	110/70	142	265	8.50	175	169	39	102	34	0.09	STEMI	RVMA+	64	37	.75	8.40
41	2	67	52	162	19.81	150/90	92	125	5.60	173	201	37	96	40	0.17	Ischaemia	RVMA -	67	30	.90	8.00
42	2	75	58	157	23.53	160/100	136	239	7.40	175	168	38	103	34	0.06	stemi	RVMA+	42	25	1.00	9.50