

The Risk Probability of Fasting Ramadan for Patients with CABG

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Abstract: The effect of Fasting Ramadan on high risk patients with coronary artery disease (CAD) who had coronary artery bypass grafting (CABG) is a difficult question to answer. Some doctors advise these patients to break fasting, but many patients keep fasting with no medical problems or if present not recorded. Patients with CABG who persist to fast Ramadan give us the opportunity to do our study. We studied 42 patients as regard the effect of Ramadan fast on their clinical status including chest pain (angina), blood pressure, cardiac enzymes, ECG and echocardiography. We also, study the effect of fasting on serum cholesterol, triglycerides and Malondialdehyde (MDA) as the end product of lipid peroxidation, Ultrasensitive CRP (Hs-CRP) as a predictive value of the inflammatory process and the osmolarity of the blood during fasting and compared to the non-fasting. The results showed three patients developed chest pain on day 17, 20, 24 of fasting due to chest infection and pleurisy with no other abnormalities detected. Two other patients developed elevated systolic blood pressure due to stoppage of diuretics without replacement with other drug during fasting. Two patients of the cigarette smokers were frequently complained of chest pain before fasting they become symptoms free during fasting which may be due to cessation of smoking which was of great benefit for these patients to release this risk factor. There was significant decrease of Hs-CRP during fasting which reflects improvement of the inflammatory process thus releasing one of the risk factors for CAD. There was no significant difference between fasting and non-fasting cholesterol and triglycerides levels. MDA showed significant increase during fasting reflecting increased lipid peroxidation. There was also sig increased osmolarity during fasting with hem concentration. We conclude that the patient can fast Ramadan safely with proper medical supervision for control of hypertension and if diuretic was withdrawn it must be replaced with other modality of treatment during fasting with proper hydration. Also, the patient must take anti-oxidant during fasting.

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1. Introduction

Every year Muslims undergo Ramadan fasting all over the world in different circumstances. The annual Ramadan fast is not obligatory for all Muslims but patients with chronic illness and those who cannot bear fasting Ramadan with hem concentration as a result of no water intake have the right to be excused. In CHD there is mostly or completely block of the flow of oxygen rich blood to heart muscle. CABG is a surgery in which the grafted artery or vein bypass the blocked portion of coronary artery (CA). Doctors advise patients to break their fasting especially in obese hypertensive patients, with ischemic heart disease or those with CABG as the incidence of angina may increase (1). Some patients follow this medical excuse to break fasting but other persist for fasting Ramadan and usually they have no problems (2,3) To find a scientific answer for our patients about fasting Ramadan we take the opportunity to do our study on those patients with high risk factors with CABG who persist fasting. We study the effect of fasting on their clinical status, lipid profile; serum cholesterol and triglycerides (TG),

MDA, hem concentration, osmolarity, Hs-CRP, and its clinical impacts.

Aim of Study:

To answer the question of high risk patients who had underwent CABG "if I can fast Ramadan", or if fasting cause deterioration of their illness.

2. Patients & methods:

42 patients 18 female and male 24 had CABG with high risk factors; 16 patients hypertensive, 15 patients diabetic, 9 patients' cigarette smokers and 18 had high cholesterol and TG level. They fast Ramadan (30 days).

The patients were clinically examined before, during (2 weeks after fasting), and after (and one month after Ramadan) fasting. Follow up of the patients for cardiac enzymes; CK-MB, CK, LDH, AST was done. The clinical state of the patients was monitored with ECG and Echocardiography. Patients were examined repeatedly for any chest pain episode; with any chest pain episode ECG, cardiac enzymes, and echocardiography was repeated.

Table no (1) Risk Factors

Risk factors	No of cases
Hypertension controlled before start fasting	16
Diabetics Controlled during study (90-180)	15
Smokers Cessation of smoking during fasting	9
Hypercholesterolemia before fasting, with failed medical ttt.	18

Two Blood samples were collected from every patient; the first sample was taken two weeks after fasting, and the second was one month after the end of Ramadan. We use the serum of each sample to determine its; cholesterol, triglycerides, Malondialdehyde, hs-CRP and calculated osmolarity from sodium, potassium, glucose and urea. Serum cholesterol, triglycerides, urea, glucose, CK, CK-MB, LDH and AST were done on auto analyzer (BIOLYIS 240). Sodium and potassium were measured in the serum by ISE on (OMNI C). Hs-CRP was assayed by a micro-plate immunozymomemtic assay provided from Accu-Bind ELISA Micro-wells. Malondialdehyde content of samples was determined by the thiobarbituric acid (TBA) activity by using the method recommended by Esterbaur and Cheesman (4). MDA of the serum sample reacts with TBA to form a colored pigment, the absorption of which is measured by spectrophotometer at 535nm.

3. Results:

Clinical events, three patients developed chest pain on day17, 20, 24, and ask for medical advice, blood samples were taken for lipid profile and cardiac enzymes, then ECG, and transthoracic echocardiography were done with no abnormality detected, one of these patients continued fasting and the other two patients break fasting for 3 days to receive medical ttt for chest infection and pleurisy. Other two patients had elevated systolic blood pressure during fasting compered to blood pressure readings before and after Ramadan. They were advised from their clinician during fasting to stop diuretic ttt for fear of hem concentration, without substitution for it which may explain the elevated systolic blood pressure.

Table (2) Clinical Events during fasting

Clinical events during fasting	No. of cases	Percent
Chest pain due to chest infection	3	7.1%
Hypertensive episodes due to diuretic stoppage	2	4.8%
Patients with no complaint	37	88.1%

Table (3) Descriptive Statistics

	N	Mean	Std. Deviation
Fasting Malondialdehyde	42	9.56	±2.139
Non- Fasting Malondialdehyde	42	8.08	±2.433
Fasting High-sensitive CRP	42	43.86	±37.789
Non- Fasting High-sensitive CRP	42	132.67	±74.047
Fasting Cholesterol	42	164.7	±59.63
Non- Fasting Cholesterol	42	157.5	±60.47
Fasting Triglycerides	42	105.9	±51.18
Non- Fasting Triglycerides	42	101.5	±50.10
Fasting Osmolarity	42	285.5	±25.49
Non- Fasting Osmolarity	42	275.4	±23.82

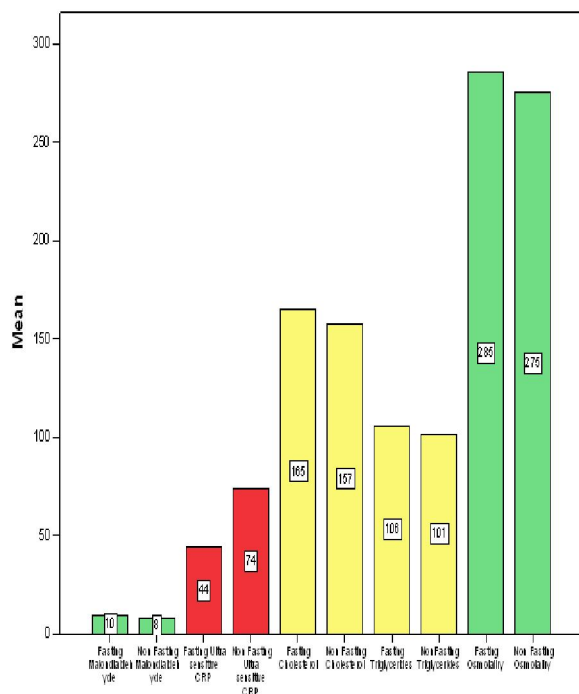
Table (4) Paired Samples T-Test between fasting and non-fasting samples

		t	D f	Sig. (2 tails)
Pair 1	Fasting Malondialdehyde & Non- Fasting Malondialdehyde	3.864	41	.000**
Pair 2	Fasting Hs-CRP & Non-fasting Hs-CRP	-4.522	41	.000**
Pair 3	Fasting Cholesterol & Non-fasting Cholesterol	0.572	41	.570
Pair 4	Fasting Triglycerides & Non-fasting triglycerides	0.387	41	.701
Pair 5	Fasting Osmolarity & Non- Fasting Osmolarity	2.039	41	.048*

*Significant at level .05

** Significant at level .01

In our study (42 CABG patients); we found that fasting Hs-CRP is significantly lower than non-fasting Hs-CRP ($P < 0.01$). Fasting MDA are significant higher than non-fasting MDA ($P < 0.01$). Fasting Osmolarity are significant higher than non-fasting Osmolarity ($P < 0.05$). There is no significant difference between fasting and non-fasting serum cholesterol and triglycerides.



Graph (1) Comparison between fasting and non-fasting means.

Table (5) Group Statistics according to blood pressure

Group Statistics					
	Hypertension	N	Mean	Std. Deviation	Std. Error Mean
Fasting Manlondialdehyde	no	26	9.2277	2.01475	.39513
	hypertensive	16	10.0925	2.28957	.57239
Fasting Ultra sensitive CRP	no	26	40.4792	39.14921	7.67779
	hypertensive	16	49.3506	36.01007	9.00252
Fasting Triglycerides	no	26	101.38	54.378	10.664
	hypertensive	16	113.19	45.938	11.484
Fasting Cholesterol	no	26	153.23	30.226	5.928
	hypertensive	16	183.38	87.160	21.790
Fasting Osmolality	no	26	281.6385	20.78327	4.07594
	hypertensive	16	291.7519	31.43376	7.85844
Non Fasting Manlondialdehyde	no	26	8.5069	2.34168	.45924
	hypertensive	16	7.3825	2.49131	.62283
Non Fasting Ultra sensitive CRP	no	26	66.2235	38.62093	7.57419
	hypertensive	16	86.7613	35.59324	8.89831
Non Fasting Cholesterol	no	26	165.23	66.406	13.023
	hypertensive	16	144.88	48.711	12.178
NonFasting Triglycerides	no	26	101.58	55.602	10.904
	hypertensive	16	101.31	41.316	10.329
Non Fasting Osmolality	no	26	274.6231	21.64901	4.24572
	hypertensive	16	276.5388	27.69154	6.92288

Table (6) Group Statistics according to sex

Group Statistics					
	sex	N	Mean	Std. Deviation	Std. Error Mean
Fasting Manlondialdehyde	male	24	8.9475	1.98592	.40538
	female	18	10.3700	2.11435	.49836
Fasting Ultra sensitive CRP	male	24	39.2363	39.10573	7.98242
	female	18	50.0222	36.11856	8.51323
Fasting Cholesterol	male	24	161.38	32.903	6.716
	female	18	169.17	84.111	19.825
Fasting Triglycerides	male	24	98.71	45.067	9.199
	female	18	115.44	58.089	13.692
Fasting Osmolality	male	24	289.6642	22.73181	4.64011
	female	18	279.9272	28.46342	6.70889
Non Fasting Manlondialdehyde	male	24	8.0404	2.17568	.44411
	female	18	8.1294	2.80499	.66114
Non Fasting Ultra sensitive CRP	male	24	58.4571	38.91986	7.94448
	female	18	94.8344	26.59037	6.26741
Non Fasting Cholesterol	male	24	160.67	69.704	14.228
	female	18	153.22	47.041	11.088
NonFasting Triglycerides	male	24	109.25	63.847	13.033
	female	18	91.11	18.394	4.336
Non Fasting Osmolality	male	24	268.1108	9.05659	1.84867
	female	18	285.0089	32.92784	7.76117

Table (7) Group Statistics according to smoker.

Group Statistics					
	Smoker	N	Mean	Std. Deviation	Std. Error Mean
Fasting Manlondialdehyde	non smoker	33	9.3836	2.05901	.35843
	smoker	9	10.1933	2.42950	.80983
Fasting Ultra sensitive CRP	non smoker	33	47.4242	38.03565	6.62116
	smoker	9	30.7856	35.87214	11.95738
Fasting Triglycerides	non smoker	33	106.52	53.903	9.383
	smoker	9	103.56	41.711	13.904
Fasting Cholesterol	non smoker	33	154.06	31.850	5.544
	smoker	9	203.78	109.460	36.487
Fasting Osmolality	non smoker	33	284.8327	24.53829	4.27157
	smoker	9	287.9056	30.20359	10.06786
Non Fasting Manlondialdehyde	non smoker	33	8.1252	2.38463	.41511
	smoker	9	7.9078	2.74793	.91598
Non Fasting Ultra sensitive CRP	non smoker	33	77.6991	35.72240	6.21847
	smoker	9	60.6578	46.88008	15.62669
Non Fasting Cholesterol	non smoker	33	155.03	65.787	11.452
	smoker	9	166.44	36.270	12.090
NonFasting Triglycerides	non smoker	33	100.85	48.825	8.499
	smoker	9	103.78	57.608	19.203
Non Fasting Osmolality	non smoker	33	276.2961	26.09650	4.54282
	smoker	9	271.8944	12.87335	4.29112

4. Discussion:

Cardiovascular diseases are the leading cause of death all over the world. (5) Fasting Ramadan may be risky in patient with CAD (5). Gumaa et al., 1978(1) reported that fasting Ramadan increased the incidence of angina. On the contrary, Khafaji et al., 2012(6) reported that fasting Ramadan in stable cardiac patients has no effect on their clinical status. He found that 71.4% had no change in their symptoms during fasting while 28.6% felt better. These results confirmed by Mohsen Nematy et al 2012(7) who found that there is significant improvement in 10 years CAD risk based on Framingham risk score. So there is an argument about fasting Ramadan of high risk CHD patients especially after CABG. Some doctors advise them to break fasting for fear of deterioration of their illness. Others concluded that fast has no adverse effect on the health of these patients. In spite of all we find many patients keep fasting with no medical problems or if presents not recorded or studied. Forty two stable cardiac patients who had CABG and refused to break fasting Ramadan gave us the opportunity to study the effect of fasting on them. In our study, we found that 88% of the patients had no change in their symptoms and were clinically stable during fasting Ramadan. Our results go hand in hand with Khafaji et al.2012(6)and Mohsen Nematy et al.,2012. (7)

In our study, there were three patients developed chest pain on day 17, 20, 24 of fasting and they asked for medical advice. We examined their blood samples for lipid profile and cardiac enzymes, also ECG and echocardiography were done. There were no abnormalities denoting ischemia or recent infarction was detected in those three patients. One of them continued fasting and the other two patients break fasting for 3 days to receive medical treatment for chest infection and pleurisy. Our result is confirmed by Dubai study; concluded that fasting reduces the risk of heart attack. (8) In our study, 9 patients were cigarette smokers and obliged to stop smoking during fasting. This released one of influencing risk factors of cardiac diseases. Two patients of them were frequently complained of chest pain before fasting they become symptoms free during fasting which may be due to cessation of smoking. Cigarette smoking is one of the most important exogenous factors, which cause 3-fold higher incidence of myocardial infarction. Smoking influences the prevalence of myocardial infarction by means of several mechanisms, including atherosclerotic injury, increase in Platelet aggregation, and increase in the levels of adhesion molecules and fibrinogen and vasoconstriction. (9,10)

On other aspect; as regard blood pressure, Saleh et al. 2005 (11) reported that there is no change in the

mean blood pressure and neither resting systolic pressure nor diastolic pressure changes during the fast. We found that systolic and diastolic blood pressure of the patients was stable during fasting except in two patients. They had elevated systolic blood pressure during fasting compared to their blood pressure before and after fasting. This may be occurred as they stopped diuretic treatment without replacement with other antihypertensive drugs during fasting. These patients followed the advice of their clinician to avoid hem concentration during fasting. Another opinion may explain this increased of systolic (but not diastolic) pressure by what happened during steady-state of aerobic exercise during Ramadan. (12) Also with our study as there was significant increase of calculated osmolarity during fasting so the clinician has to adjust the hydration of the patient for proper hemodynamics and avoid the use of diuretic during fasting. So we advise to stop diuretics for fear of hem concentration and to replace it with another drug to control blood pressure during fasting as this practice was confirmed

The most common risk factors associated with increased risk of atherosclerotic heart disease or strokes are abnormalities in plasma lipids, hypertension and smoking. One of the most important risk factors for CHD is hypercholesterolemia and high-fat diet. (13) Many studies were conducted to study the effect of fasting on this risk factor. Some of them showed that there is no change or slight decrease in the concentration of total cholesterol and triglycerides during Ramadan fasting. (14, 15) Another studies concluded that lipids increased with increased (HDL) especially in diabetics. (16-18) Others studies showed decreased levels of lipid profile (19). Adlouni et al 1997reported that Ramadan fasting diminishes the risk of atherosclerosis as it increase HDL cholesterol, and lowering LDL and increase some of Apo lipoprotein fractions associated with decrease risk of atherogenesis.(3) In our study there was no significant difference between fasting and non-fasting serum cholesterol and triglycerides levels. Thus, we concluded that their load on patients suffering from CHD with CABG does not changed by fasting. We measured MDA which is the breakdown product of lipid peroxidation and the marker of oxidative activity of free radicals in a biological system (20). In our study, we demonstrated a significant increased level of MDA during fasting. The cause for this may be the major dependence of patients on oxidation of lipid for producing energy. Elevated levels of MDA indicate increase in the level of production of oxygen free radicals which may cause harm for our patients. This drawback effect of fast can be justified by the use of antioxidants during fasting.

Inflammation is a major factor in atherothrombotic disease (21). Ultra-sensitive C-reactive protein (Hs-CRP) is a marker of systemic inflammation and a mediator of atherothrombotic disease. Measurement of the inflammatory biomarker (Hs-CRP) is advocated for CHD risk assessment; its level has been shown to correlate with cardiovascular disease risk. (22) In our study there was significant decrease in Hs-CRP during Ramadan fasting which means improvement in patients' inflammatory mediator. The decrease of the inflammatory process may be able to improve the clinical outcome due to the release of one of the risk factors of CAD. Ramadan fasting in patients with CABG has no deteriorating effects but may improve their clinical status.

Conclusion:

We concluded that fasting Ramadan of patients with CABG caused no change of their clinical status. Fasting improved their inflammatory process and did not change their lipid profile. Fasting removed more than one of the risk factors for patients with CABG. Also, the patients benefit from cut off smoking. The patient can fast Ramadan safely with proper medical supervision in order to control hypertension before start fasting. During fasting, the clinician has not to use diuretics and to adjust the hydration of the patient to avoid hem concentration for proper hemodynamics. These patients must take anti-oxidants during fasting to overcome the effect of increased lipid oxidation.

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