CODEN [USA]: IAJPBB ISSN: 2349-7750

INDO AMERICAN JOURNAL OF
PHARMACEUTICAL SCIENCES
http://doi.org/10.5281/zenodo.3358932

Available online at: http://www.iajps.com *Research Article*

SILENT MYOCARDIAL INFARCTION AND DIABETIC CARDIOVASCULAR RENAL DYSFUNCTIONS IN YOUNG WOMEN

Dr Affaf Shaukat[1], Dr Sidra Naeem[2], Dr Ayesha Hanif[3], Dr Imran Waheed[3]
[1]Mayo Hospital Lahore, [2]Sharif Medical and Dental College Lahore, [3]Allama Iqbal Memorial Teaching Hospital, Sialkot.

Article Received: June 2019 **Accepted:** July 2019 **Published:** August 2019

Abstract:
Diabetes Mellitus (DM) as ominous presumptive diagnostic of chronic kidney disease (CKD) worsening to end- stage renal damage (ESRD) is one of the spontaneous elicit in general health affair. Diabetes in young age firmly invokes the exceptional cardiovascular risk factors incidence and the progression of diabetic micro and macro angiopathies. Chronic heart failure and myocardial infraction is strongly provoked by latent pathophysiological mechanisms by the potential contributor of hyperglycemia in the hemodynamic derangements with history of diabetes as a vulnerable silent symptom.

Corresponding author:
Dr. Affaf Shaukat,
Mayo Hospital Lahore.

QR code

Please cite this article in press Affaf Shaukat et al., ***Silent Myocardial Infarction And Diabetic Cardiovascular Renal Dysfunctions In Young Women.,*** *Indo Am. J. P. Sci, 2019; 06[08].*

INTRODUCTION:

One of the leading causes affecting the young population around the world is cardiovascular disease which is responsible for leading global mortality [1]. The annual experience of silent MI in clinical entity CHD approximately 9.8 million per year predicting the unrecognized symptoms related to the occurrence of either ambulatory myocardial ischemia or sudden death with 60-70% cases asymptomatically [2]. The kidney disease outcomes initiative on clinical practice guidelines proves the threshold of ischemic chest pain is a powerful venue of cardiovascular morbidity and all cause-mortality [3] with the classification of hemo dynamic modifications independently estimate glomerular filtration rate (eGFR) in the scheme of arbitrary renal stages altering albuminuria, glomerular hyperfiltration and hyperglycemia in renal damage. Consequently, CKD as a clinical syndrome equivalent to CHD investigate the urinary excretion proteins and TGF-beta 1 initially in nephropathy as a diagnostic value in clinical parameters of previous MI in diabetic individuals [4].

In the Framingham heart study, the estimation of silent ischemia detection on large scale misinterpret by atypical angina following normal ECG in the undetectable prevalence of general population <45 years young men and women including the illness of metabolic impairments, eGFR fall, advanced CKD on dialysis, chronic inflammations and prothrombic complications in the acceptable clinical autopsy studies [5]. Additionally, diabetic kidney disease progress with the albuminuria status and serum creatinine measurements on screening at initial conventional method in pubertal diabetic interval challenging hyperglycemia on clinical nephropathy [6].

According to the Epidemiological studies, poor prognosis of CHD in CKD cases at less ratios make the diagnostic differences by the interpretation of ECG changes, risk factor profiles, pain perception, cardiac biomarkers and MI perfusions evaluation in the objective ofstable angina culminate the presentation of constant changes in troponin values presenting the attributes of premature CHD with the conclusion of progressive atheromatous plaques and calcifications on the likelihood of sepsis, anemia, platelet aggressiveness, nitric oxide abnormal metabolism, arterial stiffness, calcium-phosphate homeostasis, endothelial dysfunctions, recreational drugs, history of multiple traumas and surgeries. Classically, we present the case of inferior MI in young women with type 2 diabetes evolutions of 10 years and previous heart failure risk to death prolonging the complications of dyslipidemia progress to the advancement of diabetic nephropathy accelerating membrano-proliferative glomerulonephritis.

Case Buildup and Presentation: A 32 year old female presented to Emergency Department with severe chest pain, palpitation and vomiting for 3 days. Her previous diagnosis suggested that she had had episodes of heart failure and traditional risk factors of CAD in medical history. She described her chest pain with tightness and flank dull pain at both the areas of kidneys with back pain.

On physical examination, Cardiac sounds were normal on auscultation with no tenderness on palpation, no intra-abdominal rebound masses, no neck stiffness, no jugular vein enlargement, no dysmenorrhea, no clubbing, no family history of CAD and no hypertension. She was sweating profoundly and described weight loss, urine retention, fatigue and restlessness since last week. At the time of review current medications included Aspirin, Statin, Metformin, Insulin, Diuretics and Omeprazole.

On Admission, blood pressure was 85/60 mmHg and heart rate 66 bpm. ECG showed normal sinus rhythm with ST elevation in leads II, III and avf with the reciprocal of ST segment depression in leads V1-V6 (Figure 1A). To rule out myocardial injury, clinical diagnosis began with measuring cardiac enzymes (Table 1). The primary assessment of troponin elevation and NT- pro BNP impairment assesses the specificity and sensitivity limitations on trans-thoracic echocardiography revealing hyperkinesia with an LVEF of 48% motion index on homogenous contrast reflecting MI tension on inferior wall suspecting intracardiac thrombosis and pulmonary embolism.

Table 1: Clinical values of Combined Detection of 5 Indicators in the Diagnosis of Acute MI.

DAYS	CK-MB	MYO	cTnI	Glu	NT-ProBNP
Admission	13.66 ng/mL (0-5)	122.63 U g/L (0-110)	27.16 ng/mL (0-0.78)	15.60 mm/L (3.5-6.1)	4302.00 pg/mL
Day 2	8.45 ng/mL (0-5)	72.43 U g/L (0-110)	21.45 ng/mL (0-0.78)	17.16 mm/L (3.5-6.1)	11380.00 pg/mL
Day 4	2.73 ng/mL (0-5)	63 U g/L (0-110)	18.46 ng/mL (0-0.78)	15.05 mm/L (3.5-6.1)	15030.00 pg/mL
Day 6	1.42 ng/mL (0-5)	47.05 U g/L (0-110)	2.54 ng/mL (0-0.78)	10.15 mm/L (3.5-6.1)	9002.00 pg/mL
Day 8	1.31 ng/mL (0-5)	39.43 U g/L (0-110)	0.67 ng/mL (0-0.78)	10.03 mm/L (3.5-6.1)	289200 pg/mL

In the suspection of fibrinolysis and thrombo embolism, thoracic ultrasonography TUS certainly performed prior to the normal chest imaging previously and false positive predictive value in D-dimer test (Table 2). On the emergency based history of angina, bilateral thoracic probe examine the presence of the left sided non-specific pleural lesion of more than 5 mm on screening. It provokes the follow up of thrombolytic with the association of hypotension. Therefore, anticoagulation includes low-molecular- weight heparin therapy (LMWH) and tPA produce successful reperfusion within 12 h non-invasively.

Table 2: Quantitative D-dimer Assay for Pulmonary Embolism Diagnostic Test.

Lab test	Result	Standard range	Significance
PT	11.10 sec	(9-13) sec	Normal
APTT	28.20 sec	(20-40) sec	Normal
Fibrinogen	5.31 g/L	(2-4) ng/mL	Elevated
TAT	53.60 ng/mL	(0-4) ng/mL	Elevated
D-dimer	1.91 mg/L	(0-0.55) mg/L	Elevated

In regards with Gastrointestinal aspects, the alarming signs of dehydration, nausea, vomiting, fatigue and back pain warrant the examination of a comprehensive metabolic panel and amylase, lipase testing for the consideration of gastroenteritis or acute pancreatitis. The normal values result self-limiting bacterial infections by the management of fluid replacement, Calcitonin and supportive care.

As the patient on type 2 diabetes expansion on clinical estimation follow urinalysis on palpation of the bladder and oliguria. The exercising ECG reviewed verify the reciprocal changes in pathologic Q waves and hyper acute T waves in nonfatal angina attack, according to the quantitative measurements on total protein positive test, reflect preload independently as shown in Figure 1B Apart from the renal profile, further globulin tests were progressed on the basis of laboratory evidences as shown in Tables 3 and 4 decline in eGFR, leucocytosis and elevated cholesterol conclude the pathogenesis of contrast induced nephropathy in association of nephrotoxic drugs eliminating the advanced staging of kidney damage other than glomerulonephritis and residual renal dysfunctions.

Table 3: Comprehensive Metabolic Panel with eGFR Blood Test.

Lab test	Result value	Standard range	Significance
ALB	**22.72 g/L**	**40-55 g/L**	**Low**
GLB	**19.13 /L**	**20-40 g/L**	**Low**
Urea	**13.80 mmol/L**	**2.6-7.5 mmol/L**	**High**
Creatinine	**78U mmol/L**	**41-73 mmol/L**	**High**
K	**5.74 mmol/L**	**3.5-5.3 mmol/L**	**High**
Na	**134 mmol/L**	**137-147 mmol/L**	**Low**
Cl	**106.00 mmol/L**	**99-110 mmol/L**	**Normal**
Ca (adj)	**3.20 mmol/L**	**2.10-2.37 mmol/L**	**CKD**
Mg	**0.66 mmol/L**	**0.75-1.02 mmol/L**	**Low**
eGFR	**117.40 mL/min**	**>90 mL/min**	**High**

Table 4: Complete Blood Count Test Results

Laboratory test	Result values	Reference range
WBC	14.9610^7 /L	3.5-9.5 /L
PLT	413.00 x 10^9 /L	125-350 /L
AST	45.00 U/L	13-35U /L
LDL-Ch	5.04 mmol/L	1.9-3.1 /L
TG	8.11 mmol/L	3.1-5.2 /L
Hb	136.00 g/L	115-150 g/L
Hct	13%	37-48%
Glu	15.60 mmol/L	3.5-6.1 mml/L
TP	41.90 Ug/L	65-85U g/L

On the basis of ANA negative investigation, monoclonal immunoglobulin IgG determines the pre-malignancy in renal insufficiency with plasmapheresis at high risk of multiple myelomas (Table 5). Here in the diagnosis of proteinuria and myeloma related diseases, Bence Jones test reveal false negative results in concentrated urine. Vitamin K status in CKD sub clinically link to the formation of arterial calcification in the high moderations of atherosclerosis constitute the notable limitations on independent peritoneal dialysis to maintain the equivalent nutrition at the less co- morbidity of young age in CKD according to the results.

Table 5: Serum Protein Electrophoresis to diagnose M protein.

Fractions	%	Reference %
Albumin	34.1	(48.1-59.5)
Alpha	5.5	(2.3-4.9)
Alpha 2	19.7	(6.9-13.0)
Beta	20.6	(13.8-19.7)
Gamma	20.1	(10.1-21.9)

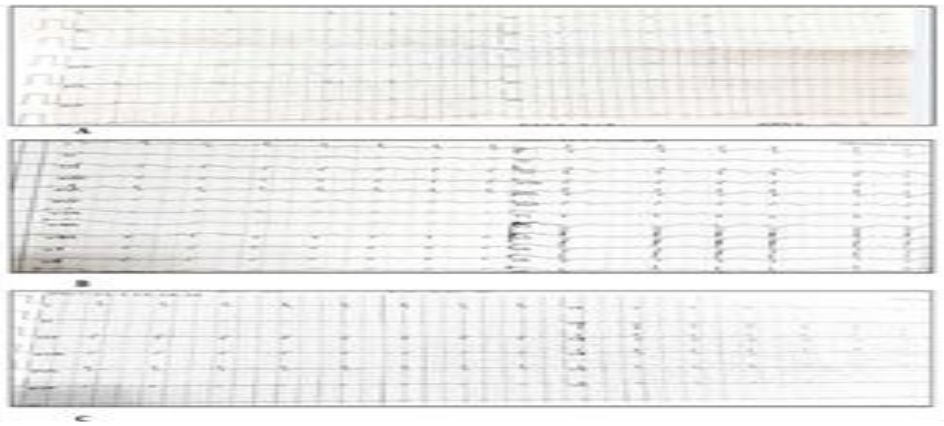

Figure 1: (A) Initially ECG shows ST elevation at inferior leads with the reciprocal of ST depression in avR. (B) No simultaneous changes in right ventricular MI on various segments of ECG. (C) New ST depression in the leads of II. III, and avF after following fibrinolysis in 12 lead ECG.

TREATMENT:

Management is initiative with the long-lasting insulin therapy in type 2 diabetes with the combination of Sulfonylurea and Metformin to control hyperglycemia. Secondly use of diuretics to restore electrolyte imbalance and Vitamin C for the nauseate feeling. Thirdly Diazepam orally for the anxiety and cardiac therapy Cedilanid for hemodynamic stability, Dopamine hydrochloride for improving the cardiac functions, Hydroxylamine and $MgSO_4$ to control frequent arrhythmias, Clopidogrel 150 mg + Aspirin 100 mg with heparin therapy of LMWH in the preventions of heart failure and recurrent myocardial infarction. Lastly IV Sodium bicarbonate+ insulin+ 50% Dextrose for hyperkalemia and Atorvastatin of 20 mg oral/day for LDL reduction.

On the Ninth day, ECG changes (Figure 1C), ST resolution and T wave inversion after the pharmaceutical drugs. Additionally Calcium gluconate for cardiac improvement on stable renal function and timely dialysis may be required on renal failure and severity of hypermagnesemia. Lately the IV human albumin infusion as a therapeutic plasmapheresis in the indication of hypovolemic shock.

DISCUSSION:

The DIAD (Ischemia detection in asymptomatic Diabetics) [5,7] assumes the importance of greater incidence in long standing type 2 diabetes mellitus focus on the factor of occlusion in arteries on the possible analyses without support of sufficient scientific data in the management of anti-ischemic medications in frequently happening CAD cases. American Diabetes Association (ADA) recommends the measures of Beta blockers or re-vascularization medical therapy on aggressive intensive treated cases on investigating the annual review of abnormal resting ECG with the lesser degrees of ischemia intervention can improve the prognosis on cardiovascular events. The nontraditional factors of hyper coagulation and clotting mediators [8] pronounce the elevation of high risk on thrombic events statistically with the complications of CKD underlying the unclear etiology predominantly result congestive heart failure, ESRD, hemorrhagic stroke and relative risk of peripheral artery disease proportional to sudden cardiac death. Thus, an appropriate medical therapeutic management needed in terms of risk factors incidental preventions in adult onset diabetes [9].

A study undertaken at Helsinki Heart Study [10] showed poor outcomes in Diabetic individuals with CHD identifying high risk of aggressiveness in dyslipidemia treatment, which is for the maintenance of LDL and Total protein. The General Practice Research thrombosis Prevention trial [11] on the secondary prevention confirms the benefit of Aspirin in the establishment of atherosclerotic disease in prospective trials reduced the risk of CHD and non-fatal events on the clinical recommendation of anti-platelet therapy can also be used as a preventive strategy to overt the nephropathy in <30 years age individuals. Therefore large phase prospective studies and trials are required to explain the issues of uncertain protein restriction in the adherence of management in routine setting care in diabetic nephropathy.

Observational Studies in the demonstration of direct effect on CVD risk factors deteriorate the kidney

functions in hyperglycemia. The Reduction of End points in Non-insulin-dependent Diabetes with the Angiotensin II Antagonist Losartan (RENAAL) and Irbesartan Diabetic Nephropathy Trial (IDNT) studies include the trial of Losartan and Iresartan as a renoprotective in the combination of Ramipril and Telmisartan initiate the defensive effect on proteinuria as compared to the therapy of (VA NEPHRON-D) study of Losartan and Lisinopril on macroalbuminuria >300 mg/day. Thus, the supportive directions on definite limitations of safety concerns utilize the consideration of Renin Angiotensin Aldosteron System (RAAS) lessens micoalbuminuria 30-300 mg/day in normic diabetes cases [12].

Hyperglycemia as a therapeutic potent in diabetes, the epidemiological early analysis illustrate the fundamental controversy of minimal outcomes in macrovascular hazards can ascend the occasion of CVD risk factors, extravagant mortality rates and vigorous symptoms with the median of HbA1c%. Hyperglycemia controlled tightly is permeable in converting high risk of hyperfiltration and glomerular hypertrophy partially on HbA1c <7% and apparently controls normal ranges with the treatment of insulin in the maintenance of proteinuria on reduced value.

According to the American Heart Association guidelines, the pharmacotherapy in CKD associated with CVD risk factors include the counsel use of Fibrinolytic, Antiplatelet, Glycoprotein II b/III a receptor antagonist, Anti-coagulants, Beta blockers, ACEIs/ARBs, Aldosterone blocker and Statin can assess the randomized controlled trials of efficacy and welfare to diminish the vascular events in non- chronic dialysis patients. The another Study of Heart and Renal Protection (SHARP) involve the substantial results in combined therapy composite to the dominance in controlling the major atherosclerotic relative risks, intracranial hemorrhage, left ventricular hypertrophy and STEMI intimated the remarkable decline in hospital death and sudden cardiac arrest for least 1 year. Ultimately, pharmacokinetic studies in renal dysfunction require essential regulations for the clinical controlled trials further on extensive population with distinct precise dosing in terminating the predictable adverse outcome pathways.

REFERENCES:

1. Department of Health (2000) National Service Framework for Coronary Heart Disease.
2. Oberman A, Kouchoukos NT, Holt JH, Russell RO (1977) Long-term results of the medical treatment of coronary artery disease. Angiology 28: 160-168.
3. Nesto RW, Phillips RT, Kett KG, Hill T, Perper E, et al. (1988) Angina and Exertional myocardial ischemia in diabetic and nondiabetic patients: assessment by exercise thallium scintigraphy. Ann Intern Med 108: 170-175.
4. Ishii H, Jirousek MR, Koya D, Tagaki C, Xia P, et al. (1996) Amelioration of vascular dysfunction in diabetic rats by an oral PKC inhibitor. Science 272 : 728-731.
5. Kannel WB, Abbott RD (1984) Incidence and prognosis of unrecognized myocardial infarction. An update on the Framingham study. N Engl J Med 311: 1144-1147.
6. American Diabetes Association (2004) Nephropathy in diabetes. Diabetes Care 27: S79-83.
7. Bansal S, Wackers FJT, Inzucchi SE, Chyun DA, Davey JA, et al. (2011) Five-year outcomes in high-risk participants in the Detection of Ischemia in Asymptomatic Diabetics (DIAD) study: a post hoc analysis. Diabetes Care 34: 204-209.
8. Baber U, Bander J, Karajgikar R, Yadav K, Hadi A, et al. (2013) Combined And independent impact of diabetes mellitus and chronic kidney disease on residual platelet reactivity. Thromb Haemost 110: 118-123.
9. Seshasai SR, Kaptoge S, Thompson A, Di Angelantonio E, Gao P, et al. (2011) Diabetes mellitus, fasting glucose, and risk of cause-specific death. N Engl J Med 364: 829-841.Koskinen P, Manttari M, Manninen V, Huttunenctk, Heinnonen OP, et al. (1992) Coronary heart disease incidence in NIDDM patients in the Helsinki Heart Study. Diabetes Care 15: 820-825.
10. The Medical Research Council's General Practice Research Framework (1998) Thrombosis prevention trial: randomised trial of low-intensity oral anticoagulation with warfarin and low-dose aspirin in the primary prevention of ischaemic heart disease in men at increased risk. Lancet 351: 233-241.
11. Hirst JA, Taylor KS, Stevens RJ, Blacklock CL, Roberts NW, et al. (2012) The impact of renin-angiotensin-aldosterone system inhibitors on Type 1 and Type 2 diabetic patients with and without early diabetic nephropathy. Kidney Int 81: 674-683.