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Case Report Open Access

Case Report on the Occurrence of Silent Myocardial Infarction and Cardiovascular and Renal Dysfunctions in Young Women who have Diabetes

Glory Coppe*

Department of Cardiovascular medicine, Florida State University, United States

Abstract

Diabetes Mellitus (DM) is an alarming presumptive diagnosis of chronic kidney disease (CKD) progressing to endstage renal damage (ESRD). Diabetes at a young age strongly inspires the appearance of extraordinary cardiovascular risk factors and the initial evolution of diabetic micro and macro angiopathies. The potential contributor of hyperglycemia in the hemodynamic derangements complicating the natural course of diabetes with susceptible silent symptoms is extremely provoking of chronic heart failure and myocardial infarction through the hidden pathophysiological processes.

Keywords: Diabetes Mellitus; Chronic kidney disease; Myocardial infarction; Cardiovascular; Pain

Introduction

Cardiovascular disease is the most common primary cause of mortality in the world's young population [1]. At clinical entity silent MI, about 9.8 million people each year encounter CHD, predicting undiagnosed symptoms associated to the emergence of either ambulatory myocardial ischemia or sudden death, with 60-70% of instances occurring asymptomatically [2]. The kidney failure consequences approach on clinical practise guidelines shows that the limit of ischemic heart problems is a potent venue of cardiovascular events and all cardiovascular death [3], with the categorization of hemodynamic adjustments individually estimating glomerular filtration rate (eGFR) in the scheme of arbitrary renal stages changing albuminuria, glomerular hyperfiltration, and hyperglycemia in renal damage. As a result, CKD, which is a clinical condition comparable to CHD, analyses urine excretion proteins and TGF-beta 1 as a diagnostic value in clinical parameters of antecedent MI in diabetic individuals [4]. As in Framingham heart investigation, the prediction of passive ischemic stroke diagnosis on a wide scale is misinterpreted by anomalous chest pain and after regular ECG in the unidentifiable incidence of overall population 45 years young men and women including the illness of metabolic impairments, eGFR fall, advanced CKD on dialysis, chronic inflammatory diseases, and prothrombotic comorbidities in approved clinical autopsy research findings [5]. Moreover, diabetic kidney damage progresses with the albuminuria status and serum creatinine measures on monitoring in the pubescent diabetic interval confronting hyperglycemia on clinical nephropathy [6]. As per observational studies, poor prognosis of CHD in CKD case scenarios at lower ratios make screening disparities by interpreting ECG changes, risk factor characteristics, pain threshold, cardiac inflammatory markers, and MI perfusions assessment in the goal of stable 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. In the traditional sense, we describe a scenario where young women with a 10-year history of type 2 diabetes and a previous risk of heart failure are experiencing inferior myocardial infarction. This is leading to complications from dyslipidemia and the acceleration of membrano-proliferative glomerulonephritis, which is further prolonging the risk of death.

Case presentation

A woman who is 32 years old arrives at the Emergency Department with symptoms of severe chest pain, palpitations, and vomiting that have been ongoing for three days. She has a medical history of previous episodes of heart failure and has traditional risk factors for coronary artery disease. The patient describes her chest pain as a feeling of tightness, and also experiences dull pain in both kidney areas, accompanied by back pain. During the physical examination, there were no abnormalities detected in the patient's cardiac sounds or tenderness found during palpation. Additionally, no intra-abdominal rebound masses, neck stiffness, jugular vein enlargement, dysmenorrhea, clubbing, family history of coronary artery disease or hypertension were observed. However, the patient was experiencing symptoms of sweating, weight loss, urine retention, fatigability, and restlessness for one week. At the time of the examination, the patient was taking a combination of medications, which included aspirin, statin, metformin, insulin, diuretics, and omeprazole. Upon admission, the patient had a blood pressure of 85/60 mmHg and a heart rate of 66 beats per minute. The electrocardiogram (ECG) showed normal sinus rhythm, but there was ST elevation in leads II, III, and avf, with corresponding ST segment depression in leads V1-V6. To determine if the patient had suffered a myocardial injury, the first step in clinical diagnosis is to measure cardiac enzymes. Additionally, a primary assessment of troponin elevation and NTproBNP impairment was performed to evaluate the specificity and sensitivity limitations of trans-thoracic echocardiography. This test revealed hypokinesia and a left ventricular ejection fraction (LVEF) of 48%, as well as motion index on homogenous contrast reflecting myocardial infarction tension on the inferior wall, which raised suspicion for intracardiac thrombosis and pulmonary embolism.

To investigate the possibility of fibrinolysis and thromboembolism, thoracic ultrasonography (TUS) was performed before conducting a

***Corresponding author:** Glory Coppe, Department of Cardiovascular medicine, Florida State University, United States, E-mail: glorycop@yahoo.com

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normal chest imaging examination and to avoid false positive predictive value of D-dimer test. During the emergency assessment of the patient's angina, a bilateral thoracic probe was used to examine the presence of a non-specific pleural lesion measuring over 5 mm on screening of the left side. This prompted follow-up treatment with thrombolytic therapy, which was associated with hypotension. As a result, anticoagulation therapy was initiated using low-molecular-weight heparin (LMWH) and tissue plasminogen activator (tPA) to achieve successful reperfusion noninvasively within 12 hours. Regarding gastrointestinal symptoms, the presence of dehydration, nausea, vomiting, fatigue, and back pain suggest the need for a comprehensive metabolic panel and amylase/lipase testing to evaluate the possibility of gastroenteritis or acute pancreatitis. If the results of these tests indicate normal values, it may suggest self-limiting bacterial infections that can be managed through fluid replacement, administration of Calcitonin, and supportive care.

Given the patient's history of type 2 diabetes, it was deemed necessary to perform a urinalysis, bladder palpation, and evaluate oliguria. Quantitative measurements of total protein and positive test results, combined with a high-standard exercise electrocardiogram (ECG) review, confirmed the presence of reciprocal changes in pathologic Q waves and hyper-acute T waves during a non-fatal angina attack, reflecting preload independently. In addition to evaluating the patient's renal profile, further globulin tests were conducted based on laboratory evidence. A decline in estimated glomerular filtration rate (eGFR), leukocytosis, and elevated cholesterol levels suggest the pathogenesis of contrast-induced nephropathy in association with nephrotoxic drugs, resulting in advanced stages of kidney damage beyond glomerulonephritis and residual renal dysfunctions. The ANA negative investigation ruled out the presence of certain autoantibodies, but monoclonal immunoglobulin IgG testing indicated a pre-malignancy in renal insufficiency, which increases the risk of developing multiple myelomas. In cases of proteinuria and myeloma-related diseases, Bence Jones test may produce false negative results when conducted on concentrated urine samples. It is worth noting that subclinical vitamin K deficiency in chronic kidney disease (CKD) is associated with arterial calcification and increased severity of atherosclerosis. This represents a significant limitation for independent peritoneal dialysis, which may compromise the ability to maintain adequate nutrition in young CKD patients with less comorbidity.

Treatment

The management approach for type 2 diabetes involves the use of long-lasting insulin therapy in combination with Sulfonylurea and Metformin to manage hyperglycemia. Diuretics are used to restore electrolyte balance, and Vitamin C is prescribed to alleviate nausea. Diazepam is administered orally to treat anxiety, and Cedilanid is given to ensure hemodynamic stability. Dopamine hydrochloride is used to improve cardiac function, while Hydroxylamine and MgSO4 are given to control frequent arrhythmias. The patient is prescribed Clopidogrel (150 mg) and Aspirin (100 mg) along with LMWH heparin therapy to prevent heart failure and recurrent myocardial infarction. In cases of hyperkalemia, IV Sodium bicarbonate, insulin, and 50% Dextrose are administered, and Atorvastatin is prescribed orally at a dose of 20 mg/day to lower LDL levels. On day 9, ECG modifications displayed ST segment resolution and T wave inversion after the administration of medication. Calcium gluconate was also given to improve cardiac function, and dialysis may be necessary in cases of renal failure and severe hypomagnesaemia. Finally, IV human albumin infusion was used as a therapeutic plasmapheresis for hypovolemic shock.

Discussion

The DIAD study (Ischemia detection in asymptomatic Diabetics) [7] is significant due to the higher incidence of occlusion in arteries in long-standing type 2 diabetes mellitus, and the lack of scientific data supporting the use of anti-ischemic medications in frequent CAD cases. Thus, there is a need for judicious analysis to determine the consequences of intermediate interventions in screening programs. The American Diabetes Association (ADA) recommends beta blockers or revascularization medical therapy in aggressively treated cases, and annual reviews of abnormal resting ECGs to intervene in lesser degrees of ischemia to improve the prognosis of cardiovascular events. Non-traditional factors such as hyper coagulation and clotting mediators [8] increase the risk of thrombotic events and complications of CKD, leading to unclear etiologies such as congestive heart failure, ESRD, hemorrhagic stroke, and a relative risk of peripheral artery disease that is proportional to sudden cardiac death. Therefore, an appropriate medical management strategy is necessary to prevent incidental risk factors in adult-onset diabetes [9].

The Helsinki Heart Study [10] found unfavorable results in Diabetic patients with CHD, indicating the need for aggressive dyslipidemia treatment to maintain LDL and Total protein levels. Statins are the preferred pharmacological intervention for young patients with diabetic nephropathy. The General Practice Research thrombosis Prevention trial [11-15] showed that Aspirin treatment is beneficial for secondary prevention of atherosclerotic disease and reduces the risk of CHD and non-fatal events. Anti-platelet therapy can also be used as a preventive strategy to avoid nephropathy in individuals under 30 years of age. Therefore, large prospective studies and trials are necessary to clarify the uncertainties regarding protein restriction in routine management of diabetic nephropathy. The studies RENAAL and IDNT have shown that hyperglycemia can negatively affect kidney function and that Losartan and Irbesartan can protect against this, particularly when used in combination with Ramipril and Telmisartan. These drugs have been found to be more effective in reducing proteinuria compared to the use of Losartan and Lisinopril in the VA NEPHRON-D study for macroalbuminuria >300 mg/day. However, safety concerns and limitations need to be taken into consideration when using Renin Angiotensin Aldosteron System (RAAS) for cases of normoglycemia with microalbuminuria of 30-300 mg/day. The management of hyperglycemia in diabetes presents a therapeutic challenge due to its controversial minimal outcomes in macrovascular hazards, high mortality rates, and severe symptoms with a median of HbA1c%. The Action in Controlling Cardiac Risk factors in Diabetes (ACCORD) study confirms that delaying vascular complications related to CKD staging 3-4 can be achieved through optimal HbA1c and hypoglycemia control. Therefore, tight control of hyperglycemia can reduce the risk of hyperfiltration and glomerular hypertrophy, leading to better outcomes with HbA1c <7% and insulin therapy to maintain proteinuria at a reduced value. As per the guidelines from the American Heart Association, pharmacotherapy for individuals with CKD and CVD risk factors should include the cautious use of drugs such as Fibrinolytics, Antiplatelets, Glycoprotein II b/III a receptor antagonists, Anticoagulants, Beta blockers, ACE inhibitors/ARBs, Aldosterone blockers, and Statins. These drugs have been shown to be effective and safe in reducing vascular events in non-dialysis-dependent CKD patients in randomized controlled trials. The Study of Heart and Renal Protection (SHARP) demonstrated the significant benefits of combined therapy in controlling major atherosclerotic risks, intracranial hemorrhage, left ventricular hypertrophy, and STEMI, resulting in a remarkable reduction in hospital deaths and sudden

cardiac arrests for at least one year. However, pharmacokinetic studies are required to establish essential regulations for clinical controlled trials, especially in a broad population, with distinct and precise dosing, to terminate the predictable adverse outcome pathways.

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Not applicable.

Conflict of Interest

Author declares no conflict of interest.

References

1. Oberman A, Kouchoukos NT, Holt JH, Russell RO (1977) Long-term results of the medical treatment of coronary artery disease. Angiology 28:160-168.
2. 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.
3. 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.
4. 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.
5. American Diabetes Association (2004) Nephropathy in diabetes. Diabetes Care 27:79-83.
6. 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.
7. 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.
8. 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.
9. 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. Van Ark J, Hammes HP, Van Dijk MC, Lexis CP, Van Der Horst IC, et al. (2013) Circulating alpha-klotho levels are not disturbed in patients with type 2 diabetes with and without macrovascular disease in the absence of nephropathy. Cardiovasc Diabetol 12:116.
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.
12. Wang AY, Ho SS, Wang M, Liu EK, Ho S, et al. (2005) Cardiac valvular calcification as a marker of atherosclerosis and arterial calcification in end-stage renal disease. Arch Intern Med 165:327-332.
13. Hruska KA, Mathew S, Lund RJ, Menom I, Saab G, et al. (2009) The pathogenesis of vascular calcification in the chronic kidney disease mineral bone disorder: the links between bone and the vasculature. Semin Nephrol 29:156-165.
14. Berezin A, Zulli A, Kerrigan S, Petrovic D, Kruzliak P (2015) Predictive role of circulating endothelial-derived microparticles in cardiovascular diseases. Clin Biochem 48:562-568.
15. Mubarak NA, Roubin GS, Iyer SS, Gomez CR, Liu MW, et al. (2000) Carotid stenting for severe radiation-induced extracranial carotid artery occlusive disease. J Endovasc Ther 7:36-40.