

Coronary artery bypass grafting in a patient with gout arthritis

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ABSTRACT

Ischemic heart disease is accepted as the most common cause of mortality and morbidity nearly all over the world. Gout disease is the most common condition of inflammatory arthritis among the adult population. Literature includes limited information about the treatment strategies when both the conditions coexist. In this report, we present the case report of a 63 year old male patient with the diagnosis of Gout arthritis who underwent a coronary artery bypass grafting procedure successfully.

Key words: Coronary artery bypass grafting, gout arthritis, hyperuricemia, ischemic heart disease

INTRODUCTION

Cardiovascular disorders are one of the the leading causes of mortality and morbidity today all over the world. They are endemic, and have been seen to increasingly affect a considerable number of people. They are also responsible for a major financial burden on the community and cause healthcare expenses. Although ischemic heart disease can be seen in different age groups, usually it is a disease of the aging population in whom related or un-related systemic disorders may also co-exist. On the other hand, other than the well known risk factors such as genetic inheritance, hyperlipidemia, cigarette smoking etc, it is well known that some of these co-existing systemic medical problems may also predispose to coronary artery disease.^[1]

Gout disease is a metabolic arthritis. It is characterized with deposition of crystals of monosodium urate due to

the increased circulating levels of uric acid in the blood.^[2] Literature includes scant reports about the co-existence of Gout arthritis and ischemic heart disease, or hyperuricemia related coronary artery disease.^[1,3-13] In this report, we present pre-, peri-, and post-operative strategies in a 63-year-old male patient with the history of Gout disease, and who underwent a successful surgical treatment for coronary artery disease.

CASE REPORT

The patient was a 63-year-old white Turkish male living in Istanbul, Turkey who presented to the clinic with increasing chest pain on exertion, which had progressed in the last six months. He was 169 cm tall and weighed 74 kg. He had been a smoker for more than 45 years, and on an average smoked 1-1.5 packs per day. His family history was negative for cardiovascular disorders. He was not hypertensive (blood pressure of 128/76 mmHg on left arm and 132/77 mmHg on right arm at rest) and did not have diabetes mellitus (overnight fasting blood glucose of 98 mg/dL, normal range: < 100 mg/dL). Blood lipid profile indicated total cholesterol and low density lipoprotein levels of 193 mg/dL (normal range: < 200 mg/dL) and 124 mg/dL (normal range: < 130mg/dL), respectively. Cardiologic check up protocol was considered and the patient underwent stress

Access this article online	
Quick Response Code:	Website: www.jcdronline.com
	DOI: 10.4103/0975-3583.91591

electrocardiography which resulted positive. Myocardial thallium perfusion scintigraphy indicated ischemic zones at the inferior, inferolateral and inferoapical zones of the heart. According to the results, coronary angiography was performed and it revealed triple vessel disease with subtotal occlusion of left anterior descending artery (LAD) at the midportion, midportion 80% stenosis at the circumflex coronary artery (Cx) and 70% stenosis at the right coronary artery (RCA). Echocardiography was normal with preserved left ventricular functions (65% ejection fraction) and left ventricular hypertrophy. Following the consent of the patient, coronary artery bypass grafting (CABG) was planned.

During the preparatory phase for surgery, the patient complained of acute first toe pain. The first toe looked swollen, hyperemic and painful on palpation. Laboratory findings including complete blood count, blood lipid profile, renal and hepatic enzymes, clotting times, were normal except increased serum uric acid levels (10.74 mg/dl, Normal: 3.4-7 mg/dl) and slightly elevated C-reactive protein (6.72 mg/L, Normal: 0-5 mg/L). He was consulted with internal medicine department and diagnosed as Gout arthritis. He was prescribed allopurinol 300 mg twice daily and scheduled for surgery when the circulating uric acid levels returned to normal. Following four days of treatment, uric acid levels returned to normal limits, and he underwent triple vessel CABG (Left internal mammarian artery to LAD, aorta-first obtuse marginal artery and aorta-posterior descending branch of the right coronary artery bypasses with separate saphenous veins) with the use of cardiopulmonary bypass on arrested heart at 33-34°C. Cross clamp and cardiopulmonary bypasses were 35 minutes and 47 minutes, respectively. After the operation, the patient was transferred to the intensive care unit and extubated there on the post-operative 4th hour. He was taken to the ward the next day and discharged on the 6th day. Blood and blood product usage was 1 units of erythrocyte suspension and 2 units of fresh frozen plasma in total.

Immediately after the surgery, there was a slight increase in serum uric acid levels (7.4 mg/dl) detected in the blood drawn in the intensive care unit. As soon as the patient was extubated and oral intake was started, allopurinol was initiated at the same pre-operative dosage, and this returned the serum uric acid levels to normal. Post-operative course was otherwise uneventful. He has been followed symptom free for more than 9 months and is on allopurinol treatment in addition to anti-aggregant (aspirin, 100mg daily), anti-lipidemic (atorvastatin, 20mg daily) and low dose beta-blocker (metoprolol 25 mg daily) medications and a low calori diet.

DISCUSSION

Coronary artery disease is the leading cause of mortality and morbidity worldwide in the current decade. With the advances in medical knowledge about the different pathophysiologic events leading to ischemic heart disease, diagnostic methods and treatment strategies, various therapeutic measures are now-a-day performed to prevent, diagnose, treat and obtain deeper knowledge about the disease. It is very well known that there are certain risk factors such as genetic susceptibility, hypertension, hyperlipidemia, smoking, and diabetes mellitus, which may predispose an individual to coronary artery disease. Additionally, hyperhomocysteinemia and metabolic syndromes have been identified as other risk factors for the disease as well as predictors of the success of the treatment and survival of the patients.^[1] Recently, elevated serum uric acid level is identified as an independent risk factor for cardiovascular diseases.^[1,3-13] Several prospective studies have shown an association between hyperuricemia and cardiovascular diseases and death.^[1,5-7,10,11] In our case, the patient only had smoking as a major cardiovascular risk factor, and otherwise he was not hypertensive, hyperlipidemic, or diabetic, and did not define positive family history. We may speculate that in addition to the over 45 years of cigarettes smoking history, increased serum uric acid levels might have increased the risk of coronary artery disease or have exacerbated the symptoms of the patient in our case study.

Gout is a kind of acute inflammatory arthritis, and characterized by recurrent painful attacks. In more than half of the patients, the metatarsophalangeal joint is the most commonly affected region as well as the presenting symptom of the patients in the clinic. The pathology is caused by the crystalization, accumulation and deposition of elevated levels of uric acid of monosodium urate character in the blood, at the joints, tendons, cartilages, and the surrounding tissues. This leads to painful attacks. Additionally, the disease may also present as tophi, kidney stones, or urate nephropathy.^[2] It has been shown that chronic inflammatory disorders such as systemic lupus erythematosus, rheumatoid arthritis, ankylosing spondylitis, diabetes mellitus, etc are associated with shorter life expectancy in affected individuals as compared to the general population.^[1] On the other hand, hyperuricemia is associated with reduced survival due to increased tendency to obesity, hypertension, insulin resistance, diuretic usage and renal disease.^[1] The National Health and Nutrition Examination Survey I Follow-Up Study reported that serum uric acid was an independent predictor of cardiovascular

mortality in subjects above 45 years of age regardless of race, menopausal status, diuretic use, or presence of cardiovascular disease.^[6] The Framingham Study showed that serum urate levels were not independently associated with the risk of coronary heart disease; however, Gout was associated with a 60% increased risk of coronary artery disease.^[5] A recent study, based on the Multiple Risk Factor Intervention Trial reports that Gout may lead to upto 26% increased risk of acute myocardial infarction.^[3] Choi *et al.*,^[14] in their 12 years Health Professionals Follow-Up Study, which included 51,297 male participants, strongly support the link between hyperuricemia, Gout, and cardiovascular diseases.

Although a clear mechanism indicating the consequences of hyperuricemia on cardiovascular diseases and decreased survival could not be identified, a recent novel rodent model showed that that uric acid could cause renal afferent arteriopathy and tubulointerstitial disease, which leads to hypertension.^[15] When the serum uric acid levels are reversed, the renal lesions and hypertension were prevented.^[9,11,14] Another explanation for the increased cardiovascular disease rate in the presence of Gout may be the ongoing and relapsing inflammation due to hyperuricemia among patients with the disease, which might enhance and promote atherogenesis and thrombogenesis, as seen in other inflammatory rheumatic disorders.^[11,12]

In conclusion, patients with Gout have higher risks of cardiovascular disorders, myocardial infarction and death due to increased blood uric acid levels. As soon as hyperuricemia has been diagnosed, strict control of Gout should be aimed for, to prevent any systemic complications^[12,13] as well as cardiovascular consequences. This can be achieved with a multidisciplinary approach through the usage of well known cardioprotective medications such as aspirin, statins, beta-blockers when needed, and angiotensin converting enzyme inhibitors following coronary artery bypass grafting.

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How to cite this article: Ugurlucan M, Filizcan U, Caglar IM, Zencirci E, Kaya E, Es MU, *et al.* Coronary artery bypass grafting in a patient with gout arthritis. *J Cardiovasc Dis Res* 2012;3:52-4. Source of Support: Nil, **Conflict of Interest:** None declared.