**Research Article**

# Prognosis and Complications of Hyperuricemia Patients Undergoing Isolated Carotid Endarterectomy

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## Summary

**Background:** Despite the association of hyperuricemia with cardiovascular risk factors, it has remained controversial as to whether uric acid is an independent predictor of cardiovascular disease, with many studies in favor and others against. The results of this study are expected to enhance our knowledge regarding the role of uric acid level on outcomes of patients with carotid artery endarterectomy and thus can be used by clinician in determining patient's prognosis.

**Methods:** Patients evaluation were performed by collecting prospective data on patients undergoing CEA at our hospital from September 2012 to May 2019, primary CEA was performed on 120 consecutive patients in the same surgical groups. From this sample, patients who underwent isolated CEA were selected, and this population was then divided into those with hyperurisemia (Group I) and those without hyperurisemia (Group II).Among the clinical complications that occurred following CEA, the following variables were analyzed: mortalite, x-clamp time, perioperative ICU (intensive care unit) times, entubation time, reentubationratio, hospitalization time, rehospitalization ratio, neurological complications, infectious complications, wound infection failure occurring within 30 days after the surgery.

**Results:** The two groups were similar according to clinical characteristics such as age, risk factors, gender, smoking, using alcohol, BMI, previous myocardial infarction, dyslipidemia, previous cerebrovascular accident. There was longer carotid x-clamp time in group I versus group II. Twenty-seven patients in Group I and 53 patients in Group II had normal blood pressure on antihypertensive medication.In group 1, neurological complications were observed only in one patient in the form of a transient ischemic attack. Coronary artery lessions have more affected Group I than Group II and number of coronary arterydiseasehavemoreGroup I ratherthanGroup II. Aditionaly periferic artery lessions have more affected Group I than Group II and number of periferic artery disease have more Group I rather than Group II. There have wound enfections in 3 patients group I and 2 patients in Group II but there haven't statistical diferrantation.

**Conclusion:** Hyperuricemia, which has effects on kidney, liver, coronary vessels, carotid and other peripheral vessels, should be considered a metabolic disease, such as a Diabetes Mellitus.

**Keywords:** Hyperurisemia; carotid; endarterectomy; prognosis; complication

## Introduction

Hyperuricemia is associated with deleterious effects on endothelial dysfunction, oxidative metabolism, platelet adhesiveness, hemorheology, and aggregation. The atherosclerotic plaque contains a considerable amount of uric acid which may increase platelet adhesiveness and potentiate thrombus formation [1].There is also increasing evidence that elevated serum uric acid levels may predict the development of type 2 diabetes [2]. Despite the association of hyperuricemia with cardiovascular risk factors, it has remained controversial as to whether uric acid is an independentpredictor of cardiovascular disease,with many studies in favour [3-5] and others against [6,7]. Serum concentrations of uric acid, the product of the metabolism of purine compounds, above 7 mg/dL result in hyperuricemia, causally associated with gout as evidenced in randomized clinical trials using urate-lowering therapies [8]. One of the major causes of hypertension and negative effects at the same time during childhood is a disease in which the multiplication of those provided in the etiology of

Diabetes mellitus is present. It is possible to speculate that the effects of the early onset of the early onset have serious side effects, in particular cardiovascular system, hypertension, kidney function disorder, diabetes mellitus, and calcium turnover problems. There is insufficient study of the effects of hyperuricemia on carotid artery disease especially surgical carotid artery disease. The aim of our study was therefore to assess the operative and short -term prognosis of hyperurisemic who had Carotid Artery Surgery comparing non-hyeprurisemic patients. The results of this study are expected to enhance our knowledge regarding the role of uric acid level on outcomes of patients with carotid artery endarterectomy(CEA)and thus can be use by clinician in determining patient's prognosis. To our knowledge, this is the first community-based study that explores the relationship between serum uric acid (SUA) levels and carotid endarterectomy surgical procedure.

## Patients and Methods

Patient evaluation was performed by collecting prospective data on patients undergoing CEA at ourhospitalfromSeptember 2012 to May 2019, primary CEA was performed on 120consecutivepatients in the same surgical groups. One senior nurse at the hospital was trained to collect these data using prespecified settings. From this sample, patients who underwent isolated CEAwere selected, and this population was then divided into those with hyperurisemia (group I) and those without hyperurisemia(group II). The excluded criteria;Patients were excluded if they had undergone any other cardiac surgery, Diabetes Mellitus Tip I, II, redo CEA, emergency cases, chronic obstructive lung disease, chronic renal disease, active gout attack and symptomatic patients (blood uric acid level ≥ 13 mg/dl). Patients have demographics, indications for surgery, severity of disease and clinical outcomes for the two groups were shown in Table 1. Surgical indication has shown Table 2. Serum samples were collected a median of 1 day (interquartile range 1–2 days)preoperatively, and uric acid measured using the ADVIA 1650 GeneralChemistry Analyzer (Siemens Diagnostics Solutions, Tarrytown, NY). Hyperuricemia was defined as 6.8 mg/dL in men and 6.0 mg/dL in women [7,8].

## Surgical Technique

### Standard CEA

The CCA, ICA, and ECA are exposed through an oblique incision parallel to the anterior border of the sternocleidomastoid muscle. Manipulation of the carotid body at the carotid bifurcation is avoided. After systemic heparinization, the CCA, ECA, and ICA are clamped. A longitudinal arteriotomy is made in the CCA and extended to the ICA distal to the end of the atherosclerotic plaque. This is followed by a meticulous CCA, ECA, and ICA endarterectomy. Based on the discretion of the surgeon, the ICA intima may be removed and/or a patch may be used for closure. After obtaining complete hemostasis, the incision is closed.

### Eversion CEA

The E-CEA technique involves an oblique transection of the ICA from the common carotid artery at the bulb. After division,

the ICA may appear redundant; a cephalad incision from the heel of the transected ICA may be used to shorten the artery. The ICA is everted over the atheroma core. It is important to remove the most external layers of the media when doing so to maintain eversion of the artery over the end of the atheroma. The endpoint is directly visualized and loose fragments are removed; 6-0 or 7-0 monofilament sutures can be placed distally, if necessary. After completion of the ICA endarterectomy, the arteriotomy can be extended to the common carotid artery to facilitate removal of the CCA and ECA plaque. The ICA can then be tailored and shortened if necessary for reanastomosis to the CCA. After obtaining complete hemostasis, the incision is closed.

## Follow-up

Postoperative follow-up of patients in the first week, first month were recorded by outpatient visits, phone calls and home visits.Among the clinical complications that occurred following CEA, the following variables were analyzed:mortalite, x-clamp time, perioperative ICU (intensive care unit) times, entubation time, reentubation ratio,,hospitalization time, rehospitalization ratio, neurological complications, infectious complications, wound infectionfailure occurring within 30 days after the surgery. Postoperative uric acid level measured first visit and second visit. Ethical approval for the study was granted by the local research ethics committee. All of these patients gave their appropriate informed consent and were studied under the approval of the institutional review boards. Our study was conducted in accordance with the principles of Declaration of Helsinki.

## Clinical and Uric Acid Data Collection

Data obtained from the medical records including history taking associated with carotid disease symptoms, head to toe physical examination, and laboratory examination. The laboratory examinations include peripheral blood examination, uric acid, blood urea nitrogen (BUN), creatinine, ALT, AST, random blood glucose, electrolyte, lipid profile. Other examinations assessed are electrocardiography, chest x-ray, echocardiography, and carotid digital angiography or conventional carotid anjiography. All the hospital's carotid surgery teams agreed to provide datafor the development of the database.

## Statistical Analysis

Initially, all variables were descriptively analyzed. For quantitative variables, analyses were carried out by observing minimum and maximum values and calculating the mean and standard deviation. For qualitative variables, absolute and relative frequencies were calculated. To compare the averages of the two groups, we used Student's t test, and when the assumption of data normality was rejected, we used the nonparametric Mann-Whitney U test. To test the homogeneity between proportions, we used the chi-square test or the Fisher's exact test. Two-sided P values were always computed and differences were considered statistically significant at p ≤0.05. All analysis performed using satatistical package for social solution (SPSS) software version 17.

# Results

The two groups were similar according to clinical characteristics such as age, risk factors, gender, smoking, using alcohol, BMI, previous myocardial infarction, dyslipidemia, previous cerebrovascular accident, (Table1). There were longer carotid x-clamp time (mean±SD) in group I versus group II.( 12.4±8.0, 11.1 ±7.2 p≤0.05) (Table 3). The gender distribution was similar in both groups (group 1: men: 90.6%; group 2: men: 82.1%, respectively).Twenty-seven patients in group 1 (64.3%) and 53 patients in group 2 (68.2%) had normal blood pressure (systolic blood pressure <140 mm Hg and diastolic pressure <90 mm Hg) on antihypertensive medication (Table 4). During the first six hours in the recovery room and after step-down to the surgical ward until discharge, the need for intravenous and oral vasodilators in group 2 was significantly higher than in group 1 (p < 0.05).In group 1, neurological complications were observed only in one patient in the form of a transient ischemic attack. None of the patients with post-operative HTN requiring pharmacologic treatment had a cardiac complication. The other complications are summarized in Table 1. There was no difference in the length of hospitalization of patients in either group. Coronary artery lessions have more affected Group I than Group II and number of coronary artery disease have more Group I rather than Group II (Table 2). Coronary angiographic have been evaluating Group C lessions more affected group I than group II(p≤0.05) (Table 2) and aditionaly periferic artery lessions have more affected Group I than Group II and number of periferic artery disease have more Group I rather than Group II (Table 2). There have wound enfections in 3 patients group I and 2 patients in Group II but there haven't statistical diferrantation. All of the patients have heeling after 1 weeks postoperative period (Table 3). In relation to medical treatment, hyperurisemia had the highestrates of previous use of medications such as calcium-channel blockers (21.5% of patients with Group Ivs. 17.6% of Group IIpatients; P=0.043), angiotensin-converting enzyme inhibitors (52.6% of patients with Group Ivs. 47.4% of Group II; P=0.038) and angiotensin II receptor blockers (20.4% of patients with Group Ivs. 15.0% of Group II; P=0.048).

**Table 1:** Demographics and clinical data of patients undergoing CEA.

| Patients | Group 1 (n=42) | Group 2 (n=78) | P |
|---|---|---|---|
| Gender (M) | 36 (78.1%) | 64 (82.1%) | NS |
| Age (Mean ± SD, years) | 69.2 ± 9.2 | 66.2 ± 7.9 | NS |
| BSA | 25.2 | 26.1 | NS |
| Surgical site (left) | 34 (70.1%) | 53 (67.8%) | NS |
| **Surgical technique** | | | |
| e-CEA | 20 | 38 | NS |
| e-CEA | 22 | 40 | 0.04 |
| HTN | 35 (80.5%) | 35 (80.5%) | 0.04 |
| CAD | 12 (27.6%) | 12 (27.6%) | 0.03 |
| DM | 15 (37.5%) | 24 (30.8%) | 0.04 |
| HLP | 36 (78.1%) | 66 (85.7%) | NS |
| PAD | 4(10.5%) | 2 (2.5%) | 0.04 |
| COPD | 4 (10.5%) | 3 (7.5 %) | NS |
| C-rp (mg/dl) mean±SD | 1.2±0.6 | 0.8±0.6 | 0.34 |
| Nicotineuse | 20 (46.8%) | 39(50%) | NS |
| Alcoholuse | 6 (16.6%) | 8 (10.2%) | NS |
| Stenosis (% ± SS) | 82.1 ± 6.8 | 76.8 ± 5.5 | 0.06 |
| **Contralateral stenosis** | | | |
| 50-69% | 11(26.1%) | 18 (23.3%) | |
| 70-99% | 4(10.8%) | 7 (8.9%) | |
| Contralateral occlusion | 3(8.1%) | 4 (6.3%) | |
| **Complications** | | | |
| Mortality | 0 | 0 | NS |
| Stroke | 1 | 0 | NS |

| | | | |
|---|---|---|---|
| Dysesthesiawoundareas | 8 | 4 | 0.03 |
| Cranialnerveinjury | 0 | 0 | NS |
| Neckhematoma | 2 | 1 | NS |
| Shunting | 8 (18.4%) | 14 (21.5%) | NS |

CAD*: Non-Surgicalcoronaryarterydisease; c-CEA, Conventionalcarotidendarterectomy;

COPD: Chronicobstructivepulmonarydisease; DM: Diabetesmellitus; e-CEA, Eversioncarotidendarterectomy; HLP: Hyperlipoproteinemia; HTN: Hypertension; PAD: Peripheralarterydisease; BSA: Body surfacearea, C-rp: c-Rekatif protein, SD: Standart Deviation

**Table 2:** Indication for surgery.

| Surgical indication | Group I | Group II | p value |
|---|---|---|---|
| Patients | 42 | 78 | |
| Symptom free | 26(61.2%) | 42(56.7%) | 0.072 |
| Symptomatic patients | 18(39.8%) | 31(38.6%) | 0.078 |
| TIA | 9(21.9%) | 16(20.2%) | 0.082 |
| Stroke | 5(11.7%) | 8 (10.3%) | 0.065 |
| Vertebrobasilar Symptoms | 2(5.0%) | 3(3.84%) | 0.067 |
| **Preoperative ultrasound** | | | |
| ICA velocity (cm/sec) | 410±120 | 405±110 | 0.098 |

**Table 3:** Operative variables results.

| Variables | Group I (n=42) | Group II ( n=78) | p |
|---|---|---|---|
| Cross-clamp time(min) ± SD | 12.4±8.0 | 11.1±7.2 | 0.034 |
| Time spent in theintensivecareunit± SD | 1.6±0.9 | 1.6±1.4 | ns[4] |
| Prolongedmechanicalventilation n,% | 1 (2.3) | none | 0.806[1] |
| Orotrachealreintubationduringhospitalization,n,% | 1 (2.3 ) | none | <0.001[2] |
| Readmissiontotheintensivecareunit n,% | 1 (2.3) | 1(2.1) | 0.856[2] |
| Bleedinghemovacdrainagetubes(mL) ± SD | 150±80 | 130±60 | 0.032[4] |
| Time of hospitalization(days) ± SD | 3.9±2.7 | 3.8±2.6 | 0.088[4] |
| Cerebrovascularaccident, n,(%) | 1 (0.5) | none | 0.623[3] |
| Acute renal failure n,(%) | 1 (0.9) | none | 1.000[3] |
| Dialysis n,(%) | 1 (0.5) | none | [3] |
| Infections (wound) n,(%) | 3 (6.5) | 2(7.4) | 0.705[2] |
| Urinarytractin fectionnonenone | | | |
| Pneumonianonenone | | | |
| Arrhythmia n, (%) | 2 (4.6) | 3 (3.75) | 0.892[2] |
| Intraoperative death | none | none | |
| Death within 30 daysnonenone | | | |

[1]Descriptive level of probability by the Student's t-test.

[2]Descriptive level of probabilitybythechi-square test.

[3]Descriptive level of probability by the Fisher' sexact test.

[4]Descriptive level of probability by the nonparametric Mann-Whitney U-test.

**Table 4:** Arterial pressures averaged at baseline, and postoperatively.

| | Pre-Operative [median (IQR)] | Post-Operative 0-6 h [median (IQR)] | Post-Operative 6-24 [median (IQR)] | Post-Operative 72 h [median (IQR)] |
|---|---|---|---|---|
| MAP | | | | |
| Group 1 | 94.87 ( 75.46-106.64) | 85.46 (80.32-98.68) | 89.42 (80.44-103.96) | 93.1 (73.64-104.04) |
| Group 2 | 92.54 (80.42-99.66) | 89.66 (79.42-102.84) | 98.68 (82.86-110.28) | 96.46 (69.78-108.54) |
| p | NS | 0.03 | 0.04 | 0.02 |

MAP: Mean arterial pressure.

## Discussion

Many studies have shown that elevated serum uric acid (SUA) levels are a component of metabolic syndrome and often accompanied by obesity, hypertension [9], hyperlipidemia [10], glucose intolerance [11], and cardiovascular risk factor clustering [12], all of which play a causal role in the pathogenesis of cardiovascular, carotid arterial and periferic arterial diseases. Otherwise Gout is a painful inflammatory arthritis which can eventually lead to the decrease in quality of life [1,2].The prevalence is reported to be increasing in many countries. In the USA the figure reached as much as 3.9 % (8.3 million inhabitants) during 2007-2008 [1] .Gout affects approximately 2% of men older than 30 years and 2% of women older than 50 years, and is the most common form of inflammatory joint disease in men older than 40 years. Serum uric levels are, on average, 0.5 to 1.0 mg/dL higher in men than women, making male sex a risk factor for hyperurisemia and gout. Lower serum uric levels in women are associated with the presence of estrogen, which is thought to act as an antihyperuricemic [3]. Theenzyme, xanthine oxidase, which is present in significant levels only in the liver and intestine, oxidizes hypoxanthine and xanthine to uric acid in the purine catabolic pathway. Xanthin oxidase inhibitorscould be used clinically to block the final step in uric acid synthesis, there by reducing the production of uric acid. To our knowledge, this is the first studies in a cohort carotid endarterectomy surgery patients admitted to assess whether high uric acid levels at admission might increase risk for major adverse events during hospitalization, operation variables, using survival approachto describe and analyze the free survivals between hyperurisemic and normourisemic groups.

There has not any study hiperurisemia and carotid enarterectomy in literaturea. There have a few research about hyperurisemia and coronary artery byapss surgery. One study, in 58 patients undergoing complicated cardiac surgery, found that preoperative uric acid >6.0 mg/dL was associated with a near¬ly 4-fold increased risk of Akut kidney ınjury(AKI) and a longer hospital stay than preoperative uric acid ≤6.0 mg/dL [13]. Another study, in 190 patients undergoing cardiovascular surgery, found that, after adjustment for confounders by multivariate logistic regression analysis, serum uric acid concentrations ≥7.0 mg/dL were as¬sociated with a 35-fold higher risk of AKI, and increased hospi¬tal stay and duration of mechanical ventilation support, than serum uric acid concentrations <7.0 mg/dL [14]. Our previous study in patients undergoing cardiovascular surgery also found that preoperative hyperuricemia was an independent risk fac¬tor of postoperative AKI and was related to poor outcomes [15]. Our findings are in agreement with those of the above studies. At the same time intubation time, the proportion of reentubation time, time spent in the intensive care, prolonged mechanical ventilation, orotracheal reintubation during hospitalization, readmission to the intensive care unit more Group I than Group II. However, no statistical difference was found between the reasons for the low number of cases.

It is very well known that there are certainrisk factors such as genetic susceptibility, hypertension, hyperlipidemia, smoking, and diabetes mellitus, which may predispose an individual to 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 [1,3-16]. Several prospective studies have shown an association between hyperuricemia and cardiovascular diseases and death [1,5-7,13,15]. In this study, the number of carotid artery involvement in hyperuricemic patients and Type C lesion in coronary arteries were statistically significant. Asymptomatic long-term follow-up of hyperuricemia is considered to be a factor in this issue. Interestingly, moreprevalence of carotid artery disease was observed. However, velocityflowrates in the USG of preoperative carotid doppler were higher in group I.

Otherwise Hyperurisemia patients present an increased risk for pre- postoperative hypertension. Untreated hypertension is generally considered / presumed to be one of the independent risk factors for poor outcome following CEA. While certain studies describe a positive relation between perioperative hypertension and the risk of poor neurological outcome and / or death after CEA, other studies have not identified such a relationship (Table 4) [17,18].

Due to the current discussions and uncertainty concerning the effect of preoperative and postoperative hypertension on postoperative outcomes, patients with uncontrolled preoperative hypertension were excluded from the study. Hyperurisemiapatients present an increased risk for postoperative infections due to depreciated host defense mechanisms, such as impaired as wound healing and granulocyte function, decreased cellular immunity, impaired complement function, and reduced immune response, which may be influenced by the gout control [19-23]. In this study,

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even if there was no statistical difference in wound healing, it was found to be worse in Group I. Hyperuricemia appears to be a factor in both wound infection and late closing of the wound site. However, more studies are needed on this subject. Another issue is the investigation of hyperuricemia on postoperative bleeding and on the coagulation cascade. In this study, postoperative bleeding rates, red packed and use of fresh frozen were found to be higher in the hyperuricemic group. The effects of hyperuricemia on kidney function, hypertension and vascular structure are known. The group was mostly bypassed and had a longer lesion length. These noted effects were effective on postoperative bleeding. However, we believe that this issue needs further investigation[23-25]. Hyperuricemia has a negative effect on the cardiovascular system as seen in this study and many other studies. Peripheral arterial disease and coronary heart disease rates were higher in preoperative examinations (Table I).

In addition, the complexity of lesions and lesion prevalence was found in the hyperuricemic group. Metabolic diseases such as Diabettes mellitus were more common in the hyperuricemic group. Webelievethatthisincreasestheimportance of thework. Wealsothinkthat hyperuricemia and cardivascular relationship should be investigated more. Another issue is the question of postoperative use of acetyl salicylic acid. As is known, the use of anti-aging is essential after carotid artery surgery. OtherwiseLow-dose asetil salisilic acid can precipitate gout attacks. In this case, it is necessary to use clopidogrel, which is another antiagregan in place of acetyl salicylic acid. However, the correlation between clopidogrel and hyperuricemia is not well researched. In this respect, it is also necessary to decide on the study and the application of an antiagregan protocol in postoperative hyperuricemic patients. Additionally, Gout is more common after surgery, trauma,and dehydration. Certain medications such as diuretics (commonly known as water pills), which treat high blood pressure, that raise the level of uric acid in the bloodstream are risks for gout. For this reason, the hyperuricemic values of patients receiving surgery should be closely monitored. In this study, blood uric acid levels of all patients after the early period were routinely monitored and, if necessary, patients were directed to rheumatology. Foods and drinks that contribute most to hyperuricemia and gout include: red meat, organ meat, seafood, beer, liquor, and sugar-sweetened items, especially with high fructose corn syrup. Hyperuricemic diet has been applied in all of your patients and diets have been adjusted by dieticians. As a result of this study, all cardiovascular and cardiologists should be more sensitive about hyperuricemic diet.

Health problems such as obesity, high blood pressure, high blood sugar, and high cholesterol may also lead to increased uric acid and the development of gout. And because some medications may also increase risk. Other causes of hyperuricemia and gout include kidney disease, exposure to lead, hypothyroidism, severe illness or stress, and extreme physical exertion [26-28]. As a result, hyperuricemia seems to be an issue that does not stop at all. Hyperuricemia, which has effects on kidney, liver, coronary vessels, carotid and other peripheral vessels, should be considered a metabolic disease, such as a Diabetes Mellitus. Patients with close follow-up and treatment should be informed about possible effects of hyperuricemia. We believe that there should be an entity that should be emphasized especially in terms of cardiovascular surgery.

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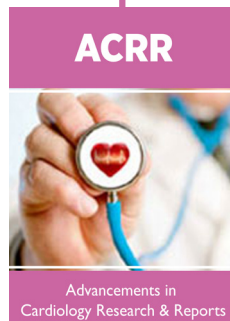
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