Serum Uric Acid in Smokers

Bassam E. Hanna, Jamal M. Hamed, Luma M. Touhala

Abstract

Objectives: To demonstrate the possible effect of smoking on serum uric acid.

Methods: Subjects enrolled in study were divided into two groups; nonsmokers and smokers, each with 60 male volunteers of the same social class and dietary habit without history of alcohol consumption, diabetes mellitus, hyperuricemia and gout, renal, joint, lung or heart diseases. Fasting blood and random urine samples were obtained from both groups for measurement of uric acid and creatinine. Calculation of both urine uric acid/urine creatinine ratio and fraction excretion of uric acid were done. The results were statistically evaluated by standard statistical methods.

Results: No significant differences in the age, serum creatinine, spot urine uric acid/urine creatinine ratio and fraction excretion of uric acid between the two groups, serum uric acid was significantly lower in smokers. In smokers there was significant negative correlation of smoking status (average number of cigarette smoked/day, duration of smoking and cumulative amount of smoking) with serum uric acid.

Conclusion: After exclusion of other factors affecting uric acid level, the significant low serum uric acid level in smokers was attributed to reduce endogenous production as a result of chronic exposure to cigarette smoke that is a significant source of oxidative stress. As this reduction is proportionate with smoking status and predisposes to cardiovascular disease, it is, therefore, recommended for smokers to stop or reduce smoking and introduce serum uric acid estimation as routine test since its cheap and simple to reflect their antioxidant level.

Keywords: Smokers; Uric acid; CVD.

Received: 25 July 2008
Accepted: 19 Sept 2008
From the Department of Medicine, Nineveh college of medicine Biochemistry, Mosul, IRAQ
Address Corresponding and reprint request to: Dr. Bassam E. Hanna, Department of Medicine, Nineveh college of medicine Biochemistry, Mosul, IRAQ
Email: bassamhanna2001@yahoo.com

Introduction

Cigarette-smoking is a well-known risk factor for atherosclerosis development and its complications including cerebral and cardiovascular diseases(CVD)[1,2] through vascular endothelial damage[3] that possibly occurs through oxygen free radicals production as superoxide radicals, hydrogen peroxide and hydroxyl radicals.[4,5] Several enzymes capable of producing oxygen free radicals including xanthine oxidase, NADPH oxidase, myeloperoxidase, and endotoxin.[4,5]

As cigarette smoke contains superoxide and reactive nitrogen species that readily react with various biomolecules,[1,6-9] it has been hypothesized that some of the adverse effects of smoking may result from oxidative damage to endothelial cells, which results in nitric oxide (NO) shortage.[1,10,11] (NO) shortage regulates vascular tone that accelerates insufficiency of coronary artery and vasoconstriction in many different tissues.[3,12] Therefore imbalance between oxidants and antioxidants may play an important role in the susceptible smoker.[13,14] In addition cigarette smokers have increased inflammatory responses that further enhance their oxidative stress.[8,9]

Since in humans, uric acid is the most abundant aqueous antioxidant, accounting for up to 60% of serum free radical scavenging capacity[15] and is an important intracellular free radical scavenger during metabolic stress including smoking,[16,17] therefore, measurement of its serum level reflects the antioxidant capacity.[15]

The aim of this study is to demonstrate the possible effect of smoking on serum uric acid concentration.

Methods

The study was conducted during the period from March to June 2008, the subjects enrolled in this study were divided into two groups (group I and group II).

Group I, considered as a control group, was composed of 60 apparently healthy nonsmoker male volunteers, their ages ranged from 20 to 69 years.

Group II, considered as a smoker group composed of 60 cigarette-smoker male volunteers, their ages ranged from 16 to 71 years.

A complete record of history was obtained, including name, age, average number of daily cigarettes smoked daily, duration of smoking, dietary habit, alcohol consumption , social class, past medical and drug history.

Members of both groups were within the same social class and dietary habit with no history of alcohol consumption, diabetes mellitus, hyperuricemia and gout, renal, joint, lung or heart diseases.

Fasting blood and random urine samples were obtained from both groups for colorimetric measurement of uric acid and creatinine that were done by Jaffe's reaction kinetic method[18] using a kit supplied by Biolabo Company and by uricase method[18] using a kit supplied by Biomeriux Company respectively by obtaining absorbencies using Cecil spectrophotometer-CE 1011 at the Biochemistry Laboratory of Nineveh College of Medicine in Mosul, Iraq.

From the data of serum and urine uric acid and creatinine the calculation of both urine uric acid/urine creatinine (Uuric acid/Ucreatinine) ratio[19] and fraction excretion of uric acid (FEuric acid)[19,20] were done in both groups, calculation of FEuric acid was done by the following formula:[19,20]

FEuric acid = (Uuric acid*Screatinine/Suric acid* Ucreatinine)*100

The results were statistically evaluated by standard statistical methods including mean, standard deviation (SD) range (minimum-maximum), Linear regression analysis (Pearson correlation coefficient r), student's t-test[21,22] with computer software programs including Microsoft Excel 2003 and SPSS 11.5[23] to evaluate the relation between different parameters of both groups. Differences between observations were considered not significant at $p > 0.05$.

Results

No statistical significant differences were noted in the age, serum creatinine, spot Uuric acid/Ucreatinine ratio and FEuric acid between two groups (P>0.05, Table I), whereas serum uric acid was significantly lower in group II than group I (P<0.001, Table 1, Fig.1).

The coefficient of variations of serum uric acid and creatinine were 17.68 and 18.46 in group I and 32.14 and 32.46 in group II respectively.

Table 2 demonstrates the mean ± SD and the range of smoking status in group II including the average number of cigarettes smoked/day, duration of smoking and cumulative amount of smoking (calculated by multiplying the number of cigarettes smoked/day with the duration of smoking).

In group II, a statistical significant negative correlations were noted between smoking status parameters and serum uric acid (P<0.001, Fig. 2, Fig. 3, Fig. 4), no such correlations with serum creatinine (P>0.050).

Table 1: Comparison between parameters of both groups; Values are presented as mean ± SD

Parameter	Group I (Control) n = 60	Group II (Smokers) n = 60	P-Value
Age (years)	33.50 ± 11.80	36.10 ± 12.00	>0.05
Serum creatinine (µmol/L)	96.82 ± 17.87	89.84 ± 29.16	>0.05
Serum uric acid (mmol/L)	0.27± 0.05	0.22 ± 0.07	<0.001
Uuric acid/Ucrearitine	0.43 ± 0.11	0.41 ± 0.17	>0.05
FEuric acid (%)	10.21 ± 2.38	11.85 ± 6.16	>0.05

Table 2: Smoking status in group II; Values are presented as mean ± SD

Parameter	Mean ± SD	Range
Number of cigarette/day	26.75 ± 16.41	5 - 80
Duration of smoking (Years)	12.75 ± 8.86	1.5 -35
Cumulative amount of smoking	432.63 ± 499.58	7.5 - 2100

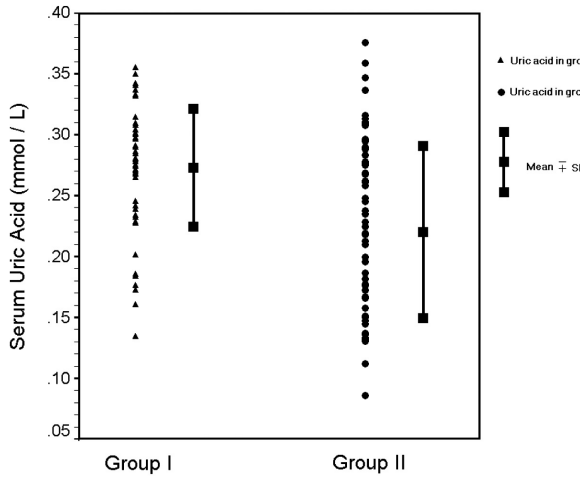

Figure 1: Spread of uric acid levels in group I and group II

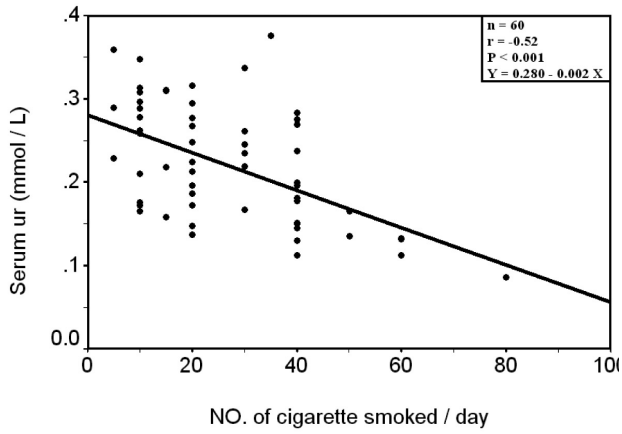

Figure 2: Correlation between serum uric acid and number of cigarette smoked/day in group II

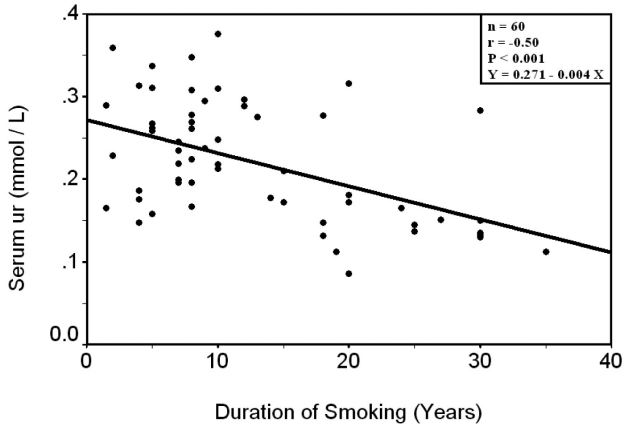

Figure 3. Correlation between serum uric acid and duration of smoking in group II

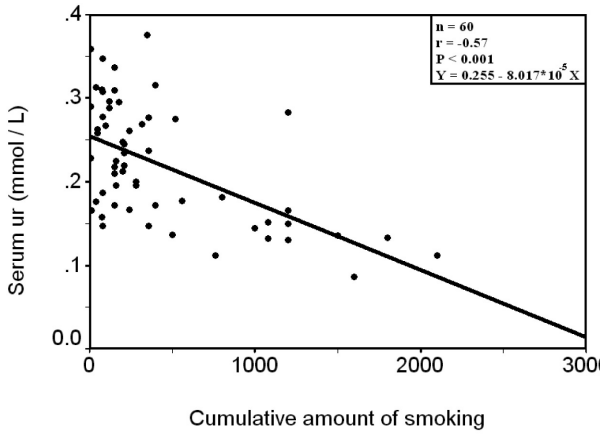

Figure 4. Correlation between serum uric acid and cumulative amount of smoking in group II

Discussion

Many but not all epidemiological studies have suggested that high serum uric acid is a risk factor for CVD[24-28] and warranted to evaluate its prognostic implications and potential utility in the monitoring of therapy.[29] This raised level of serum uric acid parallel to an increased risk of CVD could be either primary or secondary to underlying causes of CVD.[30-35] However, the specific role of serum uric acid in this constellation remains uncertain[36] although may be involved in platelet adhesiveness, aggregation or inflammation[37-39] and may be implicated in the genesis of hypertension.[40]

In contrast , there is some evidence suggesting that the increase of serum uric acid is protective against CVD since uric acid acts as an endogenous antioxidant[41-43] and the higher serum uric acid levels found in CVD patients suggests that any protective antioxidant effect which uric acid has is overwhelmed by other negative effects on pathogenesis.[44] Recently the viability of administering uric acid in solution has been established[41]even they do not specifically address the question of a biological link between uric acid and mechanisms of endothelial dysfunction or atherosclerosis since inhibition of xanthine oxidase by allopurinol is likely to reduce the production of hydrogen peroxide and thereby ameliorate oxidative stress even in smokers[45] independent of its effects on uric acid.[46] Furthermore, allopurinol has antioxidant properties.[47] Therefore, raising serum uric acid concentrations protects against acute oxidative damage as in smoking.[48]

In this study, although serum uric acid level in smokers did not reach the lower reference range (0.12 mmol/L)[49] however it is significantly lower than the nonsmoker group (P<0.001) and has significant negative correlation with smoking status including the average number of cigarettes smoked/day, duration of smoking

and cumulative amount of smoking. This finding is in agreement with other studies that showed low serum uric acid in regular smokers[1,50] and reduction of antioxidants including uric acid in smokers[51,52] indicating that oxidative stress increases everytime a cigarette is smoked.[1] Other studies proved that even nonsmokers exposed to cigarette smoke have a significantly lower plasma antioxidant status than unexposed nonsmokers do, independent of differences in dietary antioxidant intakes.[51] It even proved that administration of uric acid raises circulating antioxidant defenses and allows restoration of endothelium-dependent vasodilation.[11,52] Therefore, high serum uric acid concentrations might be protective in situations characterized by increased cardiovascular risk and oxidative stress as smoking,[11] and by reducing its level it increases susceptibility to oxidative damage and accounts for the excessive free radical production.[53] Therefore, the possibility that uric acid confers protection against the development of atherosclerosis, in view of its antioxidant properties, has been recognized.[54,55]

In this study, serum creatinine, FEuric acid and Uuric acid/Ucreatinine ratio are not significantly differ between two groups, in addition to that FEuric acid and Uuric acid/Ucreatinine ratio values lie within the mean ± SD observed in the control group by other studies.[19,56-58] Since these tests have been reported to be useful to evaluate renal handling of uric acid[59-63] and as serum uric acid concentrations are highly dependent on endogenous production as well as renal excretion.[64,65] Therefore, low serum uric acid level in smokers is attributed to reduction of endogenous production. As result of smoking, oxidative stress consumption rather than increased renal excretion after exclusion of other factors that affect its level as all members of groups I and II are subjected to the same preanalytical and postanalytical factors and all of them are within the same social class, dietary habit with no history of alcohol consumption in addition to standardization of laboratory uric acid measurement to avoid analytical variations.

This finding is in agreement with other studies that proved that reduction of antioxidants including uric acid in smokers[66] is due to both chronic exposure to cigarette smoke that is a significant source of oxidative stress[8,53] and low intake of dietary antioxidants.[67]

Conclusion

After exclusion of other factors affecting uric acid level, the significant low serum uric acid level in smokers was attributed to reduce endogenous production as a result of chronic exposure to cigarette smoke that is a significant source of oxidative stress. As this reduction is proportionate with smoking status and predisposes to cardiovascular disease, it is recommended for smokers to stop or reduce smoking and introduce serum uric acid estimation as routine test since its cheap and simple to reflect their antioxidant level.

References

1. Tsuchiya M, Asada A, Kasahara E, Sato EF, Shindo M, Inoue M. Smoking a single cigarette rapidly reduces combined concentrations of nitrate and nitrite and concentrations of antioxidants in plasma. Circulation 2002 Mar;105(10):1155-1157.
2. Celermajer DS, Sorensen KE, Georgakopoulos D, Bull C, Thomas O, Robinson J, et al. Cigarette smoking is associated with dose-related and potentially reversible impairment of endothelium-dependent dilation in healthy young adults. Circulation 1993 Nov;88(5 Pt 1):2149-2155.
3. Benzuly KH, Padgett RC, Kaul S, Piegors DJ, Armstrong ML, Heistad DD. Functional improvement precedes structural regression of atherosclerosis. Circulation 1994 Apr;89(4):1810-1818.
4. Calver A, Collier J, Moncada S, Vallance P. Effect of local intra-arterial NG-monomethyl-L-arginine in patients with hypertension: the nitric oxide dilator mechanism appears abnormal. J Hypertens 1992 Sep;10(9):1025-1031.
5. Kanani PM, Sinkey CA, Browning RL, Allaman M, Knapp HR, Haynes WG. Role of oxidant stress in endothelial dysfunction produced by experimental hyperhomocyst(e)inemia in humans. Circulation 1999 Sep;100(11):1161-1168.
6. Karnath B. Smoking cessation. Am J Med 2002 Apr;112(5):399-405.
7. Pryor WA. Cigarette smoke radicals and the role of free radicals in chemical carcinogenicity. Environ Health Perspect 1997 Jun;105(Suppl 4):875-882.
8. Alberg A. The influence of cigarette smoking on circulating concentrations of antioxidant micronutrients. Toxicology 2002 Nov;180(2):121-137.
9. Brown KM, Morrice PC, Duthie GG. Erythrocyte vitamin E and plasma ascorbate concentrations in relation to erythrocyte peroxidation in smokers and nonsmokers: dose response to vitamin E supplementation. Am J Clin Nutr 1997 Feb;65(2):496-502.
10. Node K, Kitakaze M, Yoshikawa H, Kosaka H, Hori M. Reversible reduction in plasma concentration of nitric oxide induced by cigarette smoking in young adults. Am J Cardiol 1997 Jun;79(11):1538-1541.
11. Waring WS, McKnight JA, Webb DJ, Maxwell SR. Uric acid restores endothelial function in patients with type 1 diabetes and regular smokers. Diabetes 2006 Nov;55(11):3127-3132.
12. Schectman G, Byrd JC, Gruchow HW. The influence of smoking on vitamin C status in adults. Am J Public Health 1989 Feb;79(2):158-162.
13. van der Vaart H, Postma DS, Timens W, ten Hacken NH. Acute effects of cigarette smoke on inflammation and oxidative stress: a review. Thorax 2004 Aug;59(8):713-721.
14. Polidori MC, Mecocci P, Stahl W, Sies H. Cigarette smoking cessation increases plasma levels of several antioxidant micronutrients and improves resistance towards oxidative challenge. Br J Nutr 2003 Jul;90(1):147-150.
15. Maxwell SR, Thomason H, Sandler D, Leguen C, Baxter MA, Thorpe GH, et al. Antioxidant status in patients with uncomplicated insulin-dependent and non-insulin-dependent diabetes mellitus. Eur J Clin Invest 1997 Jun;27(6):484-490.
16. Mathru M, Dries DJ, Barnes L, Tonino P, Sukhani R, Rooney MW. Tourniquet-induced exsanguination in patients requiring lower limb surgery. An ischemia-reperfusion model of oxidant and antioxidant metabolism. Anesthesiology 1996 Jan;84(1):14-22.
17. Hellsten Y, Tullson PC, Richter EA, Bangsbo J. Oxidation of urate in human skeletal muscle during exercise. Free Radic Biol Med 1997;22(1-2):169-174.

18. Whelton A, Watson JA, Robert RC. Nitrogen metabolism and renal function. In: Tietz fundamentals of clinical chemistry (Burtis CA, Ashwood ER, eds). 4th ed. Saunders com 1996; 578-580.
19. Perez-Ruiz F, Calabozo M, Erauskin GG, Ruibal A, Herrero-Beites AM. Renal underexcretion of uric acid is present in patients with apparent high urinary uric acid output. Arthritis Rheum 2002 Dec;47(6):610-613.
20. Shichiri M, Hirata Y. Serum uric acid level and fractional excretion of urate in fluid and electrolyte disturbances. Rinsho Byori 1999 May;47(5):417-423.
21. Chap TLE. Introductory biostatistics. Wiely interscience 2003; 283 – 292.
22. Jones D. Pharmaceutical statistics. Pharmaceutical press 2002.
23. Landau S, Everitt BS. A Handbook of Statistical Analyses using SPSS. A CRC Press Company USA 2003.
24. Freedman DS, Williamson DF, Gunter EW, Byers T. Relation of serum uric acid to mortality and ischemic heart disease. The NHANES I Epidemiologic Follow-up Study. Am J Epidemiol 1995 Apr;141(7):637-644.
25. Culleton BF, Larson MG, Kannel WB, Levy D. Serum uric acid and risk for cardiovascular disease and death: the Framingham Heart Study. Ann Intern Med 1999 Jul;131(1):7-13.
26. Rich MW. Uric acid: is it a risk factor for cardiovascular disease? Am J Cardiol 2000 Apr;85(8):1018-1021.
27. Fang J, Alderman MH. Serum uric acid and cardiovascular mortality the NHANES I epidemiologic follow-up study, 1971-1992. National Health and Nutrition Examination Survey. JAMA 2000 May;283(18):2404-2410.
28. Meisinger C, Koenig W, Baumert J, Döring A. Uric acid levels are associated with all-cause and cardiovascular disease mortality independent of systemic inflammation in men from the general population: the MONICA/KORA cohort study. Arterioscler Thromb Vasc Biol 2008 Jun;28(6):1186-1192.
29. Ioachimescu AG, Brennan DM, Hoar BM, Hazen SL, Hoogwerf BJ. Serum uric acid is an independent predictor of all-cause mortality in patients at high risk of cardiovascular disease: a preventive cardiology information system (PreCIS) database cohort study. Arthritis Rheum 2008 Feb;58(2):623-630.
30. Whitelaw DC, O'Kane M, Wales JK, Barth JH. Risk factors for coronary heart disease in obese non-diabetic subjects. Int J Obes Relat Metab Disord 2001 Jul;25(7):1042-1046.
31. Johnson RJ, Kang DH, Feig D, Kivlighn S, Kanellis J, Watanabe S, et al. Is there a pathogenetic role for uric acid in hypertension and cardiovascular and renal disease? Hypertension 2003 Jun;41(6):1183-1190.
32. Johnson RJ, Feig DI, Herrera-Acosta J, Kang DH. Resurrection of uric acid as a causal risk factor in essential hypertension. Hypertension 2005 Jan;45(1):18-20.
33. Wheeler JG, Juzwishin KD, Eiriksdottir G, Gudnason V, Danesh J. Serum uric acid and coronary heart disease in 9,458 incident cases and 155,084 controls: prospective study and meta-analysis. PLoS Med 2005 Mar;2(3):e76.
34. Ruggiero C, Cherubini A, Ble A, Bos AJ, Maggio M, Dixit VD, et al. Uric acid and inflammatory markers. Eur Heart J 2006 May;27(10):1174-1181.
35. Kawamoto R, Tomita H, Oka Y, Ohtsuka N. Relationship between serum uric acid concentration, metabolic syndrome and carotid atherosclerosis. Intern Med 2006;45(9):605-614.
36. Dzielak DJ, Kivlighn SD. Emerging concepts in cardiovascular disease: should elevated serum uric acid be considered a risk factor? Expert Opin Investig Drugs 1998 Jan;7(1):85-89.
37. Mustard JF, Murphy EA, Ogryzlo MA, Smythe HA. Blood coagulation and platelet economy in subjects with primary gout. Can Med Assoc J 1963 Dec;89:1207-1211.
38. Newland H. Hyperuricemia in coronary, cerebral and peripheral arterial disease: an explanation. Med Hypotheses 1975 Jul-Aug;1(4):152-155.
39. Selby JV, Friedman GD, Quesenberry CP Jr. Precursors of essential hypertension: pulmonary function, heart rate, uric acid, serum cholesterol, and other serum chemistries. Am J Epidemiol 1990 Jun;131(6):1017-1027.
40. Nicholls A, Snaith ML, Scott JT. Effect of oestrogen therapy on plasma and urinary levels of uric acid. Br Med J 1973 Feb;1(5851):449-451.
41. Waring WS, Webb DJ, Maxwell SR. Systemic uric acid administration increases serum antioxidant capacity in healthy volunteers. J Cardiovasc Pharmacol 2001 Sep;38(3):365-371.
42. Maxwell AJ, Bruinsma KA. Uric acid is closely linked to vascular nitric oxide activity. Evidence for mechanism of association with cardiovascular disease. J Am Coll Cardiol 2001 Dec;38(7):1850-1858.
43. Schretlen DJ, Inscore AB, Jinnah HA, Rao V, Gordon B, Pearlson GD. Serum uric acid and cognitive function in community-dwelling older adults. Neuropsychology 2007 Jan;21(1):136-140.
44. Torun M, Yardım S, Simsek B, Burgaz S. Serum uric acid levels in cardiovascular diseases. J Clin Pharm Ther 1998 Feb;23(1):25-29.
45. Guthikonda S, Sinkey C, Barenz T, Haynes WG; American Heart Association Clinical Investigation and Reports. Xanthine oxidase inhibition reverses endothelial dysfunction in heavy smokers. Circulation 2003 Jan;107(3):416-421.
46. Desco MC, Asensi M, Márquez R, Martínez-Valls J, Vento M, Pallardó FV, et al. Xanthine oxidase is involved in free radical production in type 1 diabetes: protection by allopurinol. Diabetes 2002 Apr;51(4):1118-1124.
47. Ricardo SD, Bertram JF, Ryan GB. Podocyte architecture in puromycin aminonucleoside-treated rats administered tungsten or allopurinol. Exp Nephrol 1995 Sep-Oct;3(5):270-279.
48. Waring WS, Convery A, Mishra V, Shenkin A, Webb DJ, Maxwell SR. Uric acid reduces exercise-induced oxidative stress in healthy adults. Clin Sci (Lond) 2003 Oct;105(4):425-430.
49. Boon NA, Colledqe NR, Walker BR, Hunter J. Davidson's principle and practice of medicine. Churchill livinqstone 2006: 1320.
50. Timimi FK, Ting HH, Haley EA, Roddy MA, Ganz P, Creager MA. Vitamin C improves endothelium-dependent vasodilation in patients with insulin-dependent diabetes mellitus. J Am Coll Cardiol 1998 Mar;31(3):552-557.
51. Dietrich M, Block G, Norkus EP, Hudes M, Traber MG, Cross CE, et al. Smoking and exposure to environmental tobacco smoke decrease some plasma antioxidants and increase gamma-tocopherol in vivo after adjustment for dietary antioxidant intakes. Am J Clin Nutr 2003 Jan;77(1):160-166.
52. Goraca A, Skibska B. Plasma antioxidant status in healthy smoking and non-smoking men. Bratisl Lek Listy 2005;106(10):301-306.
53. Reilly M, Delanty N, Lawson JA, FitzGerald GA. Modulation of oxidant stress in vivo in chronic cigarette smokers. Circulation 1996 Jul;94(1):19-25.
54. Becker BF. Towards the physiological function of uric acid. Free Radic Biol Med 1993 Jun;14(6):615-631.
55. Nieto FJ, Iribarren C, Gross MD, Comstock GW, Cutler RG. Uric acid and serum antioxidant capacity: a reaction to atherosclerosis? Atherosclerosis 2000 Jan;148(1):131-139.
56. Pak CY. Renal Calculi. In: Cecil Textbook of Medicine, Volume 1 (Wyngaarden JB and Smith LH Jr). 18th ed. Philadelphia PA: WB Saunders com 988; 638-644.

57. Pesce AJ, Kaplan LA. Methods in Clinical Chemistry, St Louis, MO: Mosby-Year Book Inc, 1987, 32.
58. Katopodis KP, Elisaf MS, Pappas HA, Theodorou JC, Milionis HJ, Bourantas KL, et al. Renal abnormalities in patients with sickle cell-beta thalassemia. J Nephrol 1997 May-Jun;10(3):163-167.
59. Puig JG, Mateos FA, Jiménez ML, Ramos TH. Renal excretion of hypoxanthine and xanthine in primary gout. Am J Med 1988 Oct;85(4):533-537.
60. Nishida Y, Ito K. Decreased renal phosphate threshold in patients with gout. Nephron 1992;62(2):142-144.
61. Emmerson BT. The management of gout. N Engl J Med 1996 Feb;334(7):445-451.
62. Tykarski A. Evaluation of renal handling of uric acid in essential hypertension: hyperuricemia related to decreased urate secretion. Nephron 1991;59(3):364-368.
63. Perez-Ruiz F, Calabozo M, Herrero A, Ruiz-Lucea E, Alonso Ruiz A. Analysis of the methods for classifying gout according to renal excretion of uric acid. Rev Esp Reumatol 1998;25:335-339.
64. Leyva F, Wingrove CS, Godsland IF, Stevenson JC. The glycolytic pathway to coronary heart disease: a hypothesis. Metabolism 1998 Jun;47(6):657-662.
65. Quiñones Galvan A, Natali A, Baldi S, Frascerra S, Sanna G, Ciociaro D, et al. Effect of insulin on uric acid excretion in humans. Am J Physiol 1995 Jan;268(1 Pt 1):E1-E5.
66. Tomita M, Mizuno S, Yokota K. Increased levels of serum uric acid among ex-smokers. J Epidemiol 2008;18(3):132-134.
67. Bloomer RJ. Decreased blood antioxidant capacity and increased lipid peroxidation in young cigarette smokers compared to nonsmokers: Impact of dietary intake. Nutr J 2007;6:39.