IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 20, Issue 9 Ser.15 (September. 2021), PP 01-05
www.iosrjournals.org

To study the correlation of body mass index on random blood sugar and uric acid in the leprosypatients belonging to Bundelkhand region: A pilot study

[1-] Dr.Prabhakar Singh Bais , *Asso. Prof. –Department of Biochemistry MLB Medical college –Jhansi-284128(U.P)*
[2-] Dr. Loveleena, *Professor (Microbiology)-Prasad Institute of Medical Sciences-Lucknow(U.P)*
[3-] Dr. Amitabh Agarwal, *Asso. Prof. (Physiology)–Autonomous State Medical College Etah(U.P)*
[4-] Dr. Priyanka Chauhan , *Professor (Microbiology) -K.D.Dental college –Mathura(U.P)*
Corresponding Author: Dr. Priyanka Chauhan , Professor (Microbiology) -K.D.Dental college –Mathura(U.P)

Abstract:
Insulin resistance and relative lack of insulin factors lead to persistence of increased blood sugar level which contributes to the causation of diseases including cardiovascular diseases, stroke, chronic kidney diseases, and peripheral artery diseases.However, according to our knowledge, there is a lack of sample study in Bundelkhand region regarding the relationship of Body Mass Index (BMI) with uric acid and Random Blood Sugar (RBS).We have taken the criteria of WHO to divide the subjects into groups. Therefore, in the present study, we have taken the opportunity to investigate the relationship of BMI with uric acid and also with RBS in this pilot study. We observed marked significant difference in the mean values of BMI, RBS, and uric acid when compared between leprosy subjects and control subjects. Comparison of Ideal BMI with either overweight BMI or obese BMI participants of the controls group, we observed an insignificant in their RBS levels in the present study. Overweight and obese BMI subjects belonging to leprosy subjects showed a drastic increase in the RBS levels when compared with Ideal BMI subjects respectively.Our pilot study indicates that there is a strong association between overweight BMI and RBS and uric acid respectively among leprosysubjects in Bundelkhand region.In addition we also conclude that uric acid may potentially serve as a novel indicator for identifying patients with hyperglycemia.

Date of Submission: 18-09-2021 Date of Acceptance: 03-10-2021

I. Introduction:

Leprosy is a chronic, progressive bacterial infection caused by the bacterium Mycobacterium leprae [1]. It primarily affects the nerves of the extremities, the skin, the lining of the nose, and the upper respiratory tract [1,2]. Insulin resistance factor leads to persistence of increased blood sugar level which contributes to the causation of diseases including cardiovascular diseases, stroke, chronic kidney diseases, and peripheral artery diseases [2-4]. About three percent deaths in India in 2016 are as result of diabetes, which is up from one percent of all deaths in 1990 [5]. Apart from hereditary factors, research through the decades have accounted high Body Mass Index (BMI) [6,7], dietary factors due to industrialization [8], and tobacco use [9] were the most important risk factors for the development of leprosy. Moreover, reports have also demonstrated that individuals who reduced body weight through exercise had a better glycemic control during the period of study [10,11].

Physiologically uric acid is a catabolic product of purine nucleotides and is excreted in urine [12]. The other causes for high uric acid (hyperuricemia) are high fructose diet intake, fasting or rapid loss of weight, and while correcting low zinc by supplementation [12-14]. Increase in the concentration of uric acid has a close correlation to the mechanism of underlying renal function [12,13].According to a report, it has been reported that excessive uric acid was observed due to the accumulated fat molecules in adipose tissues in overweight and obese individuals [15-18]. These studies [12-18] may provide a possible mechanism for the association between BMI and uric acid. So far studies have shown the association between BMI and uric acid is wellestablished, BMI is an important modifiable risk factor for hyperuricemia in developed countries[15-18]. Lately, in some reports it has been demonstrated that uric acid is also said to be the risk factor for the initiation of leprosy [19,20]. Though the literature has been shown the relationship between BMI and uric acid, but it has clearly mentioned whether increase in uric acid occurs first or the initiation of leprosy. Interestingly, Bedir et al says that leptin hormone might be the regulator of uric acid concentrations in humans [21]. Studies have also shown

that the possible association of leptin towards the development of overweight and further obesity in an individual [22,23]. Leptin is also shown to be a risk factor the development of leprosy in humans [24,25]. Considering the connecting from the studies [19-25] it is not coincidental to say that BMI, uric acid, and leprosy are inter-related to each other.

However, according to our knowledge, there is a lack of sample study in Bundelkhand region regarding the relationship of BMI with uric acid and RBS.Therefore, in the present study, we have taken the opportunity to investigate the relationship of BMI with uric acid and also with RBS in the pilot study.

II. Materials & Methods:

Our study consisted of fifty healthy subjects and fifty leprosy subjects from the out-patient department at the Department of Skin & V.D. of MLB medical college, Jhansi. All subjects completed the physical and blood examinations performed from March to April 2019.A physical examination was performed on all subjects by a qualified doctor per established standard methods. Weight and Height was obtained of the participants of the study after informed written consent from all the study group subjects. BMI was calculated by dividing body weight (kg) by the square of height (m^2). Using diagnostic criteria for obesity in BMI for Asian populations recommended by the WHO [26], we categorized BMI into three categories: Ideal BMI (18.5-24.9 kg/m^2), overweight (25-29.9 kg/m^2), and obese ($\geq$30 kg/m^2).Exclusion criteria were type 1 diabetes individuals, less than five years of known duration of leprosy, and with complications. Inclusion criteria for healthy controls were non-diabetic, not taking supplementations, and having no other complications.Fasting venous blood (5ml) were drawn into EDTA and plane vials with a disposable syringe & needle, under all aseptic conditions. Serum was separated by centrifuging the blood at 3000 rpm for 20 minutes. Samples were stored in aliquots at -20^o C until assayed. Blood sugar was estimated by glucose oxidase peroxidase end point method with the kit obtained from Transasia Biomedicals. Uric acid is estimated by uricase end point method with kit obtained from Autospan laborartories.

Statistical analysis:

All the biochemical parameters mentioned were measured in plasma and serum of leprosy and control subjects respectively. All the values obtained were expressed as mean ± S.D. The statistical analysis of the results was carried out by unpaired 't' test between two groups. One Way Analysis of variance (ANOVA) was used when the analysis was between more than two groups. Pearson correlation was used to test the correlation between two parameters. $P < 0.05$ was considered significant.

III. Results:

Table 1 shows the mean values of age, BMI, RBS, and uric acid in both the groups of the present study. We observed insignificant difference in the age when compared between leprosy subjects and control subjects. On the other hand, observed marked significant difference in the mean values of BMI, RBS, and uric acid when compared between leprosy and control subjects. Figure 1 shows the different RBS mean values observed in three different leprosy subjects and control subjects placed according to ideal BMI, overweight BMI, and obese BMI participants respectively. Comparison of Ideal BMI with either overweight BMI or obese BMI participants of the controls group, we observed an insignificant in their RBS levels in the present study. Overweight and obese BMI subjects belonging to leprosy subjects showed a drastic increase in the RBS levels when compared with Ideal BMI subjects respectively ($P<0.001$). Figure 2 shows the different uric acid mean values observed in three different leprosy subjects and control subjects placed according to ideal BMI, overweight BMI, and obese BMI participants respectively. Comparison of Ideal BMI with either overweight BMI or obese BMI participants of the control group, we observed an insignificant in their uric acid levels in the present study. Overweight and obese BMI subjects belonging to leprosy subjects showed a drastic increase in the uric acid levels when compared with Ideal BMI subjects respectively ($P<0.001$). In the leprosypatient group positive correlation was observed between overweight BMI and RBS of respective subgroup subjects (Table 2). Similar positive correlation was observed between overweight BMI and uric acid of respective subgroup subjects. In the control group patient group (Table 3) negative correlation was observed between obese BMI versus uric acid. Rest of the correlation we did not observe significant association between the variables of Ideal BMI and overweight BMI.

IV. Discussion:

The present study showed a marked significant difference in uric acid levels of the leprosy subjects when compared with control subjects. In addition, the study also showed a positive correlation between uric acid and with the overweight BMI subjects. In the literature few studies have shown the association of uric acid with that of BMI corroborated with the present study results [13-16]. Numerous epidemiological studies have also shown a positive correlation between BMI and increased uric acid levels [27,28]. Studies reported that subjects

having overweight have shown increase in uric acid levels and the levels are strongly related overproduction and a decrease in uric acid excretion [18]. Another study inferred the increase in uric acid levels to elevated plasma free fatty acids induction into the liver and thus uric acid is produced in large quantities by activating the uric acid synthesis pathway [16,29,30]. Extrapolation of the data speculates on the possible mechanisms, which may explain the relationship between uric acid and BMI. Physiologically uric acid is a catabolic product of purine nucleotides and is excreted in urine [12]. Uric acid is increased during the destruction of cells generally. The other causes for hyperuricemia are high fructose diet intake, fasting or rapid loss of weight, and while correcting low zinc by supplementation [12-14]. Increase in the concentration of uric acid has a close correlation to the mechanism of underlying renal function [31]. Therefore, the increase in uric acid should involve the factors such as excessive production or deficient excretion. However, our thought differs from these studies [14,18,27,28,31]. It is clearly demonstrated in previous reports that hyperglycemia induces superoxide in the diabetes affected individual [32,33]. Superoxide radical inhibits glyceraldehydes-3-phosphate dehydrogenase enzyme, which leads to the accumulation of substrates above glyceraldehydes-3-phospate in the glycolysis pathway [34,35]. Therefore, glucose-6-phospahte that has been accumulated is converted to the ribose-5-phosphate through hexose monophosphate pathway [12,34]. This ribose-5-phosphate is the initial substrate for the formation of purine nucleotides. When the production increases irrespective of the need, then these excessively produced nucleotides catabolism to yield uric acid. Moreover, we consider the effect of significant interaction of overweight BMI with uric acid was a risk factor for elevated uric acid in overweight individuals but not a predictor among ideal and obese BMI participants.

Significant difference was observed in blood sugar levels when compared between ideal BMI and overweight BMI subjects and Ideal BMI and obese BMI subjects in leprosy group. It is evident that diabetes is a disorder characterized by hyperglycemia due to insulin resistance or relative lack of insulin in the body [1]. Insulin resistance is commonly seen in overweight and obese individuals even when they are not diabetic as such individuals develop enhanced oxidative stress due to hyperglycemia [36]. Oxidative stress release nuclear transcription factors that cause serine phosphorylation instead of tyrosine phosphorylation and induce insulin resistance [34].

The other important function of uric acid is its antioxidant property [12]. Antioxidants are the molecules that counteract the effects of free radicals which are mostly prominently produced during the metabolism of bio-molecules. Pertaining to the present study in the leprosypatient group positive correlation was observed between overweight BMI and RBS. In the control group patient group negative correlation was observed between obese BMI versus uric acid. At first it was seems to be contradictory but as it is evident in literature that hyperglycemia induces free radical production [12,31,33-36], to combat the free radicals, it is a compensatory mechanism to enhance the production of uric acid. It is therefore inferred that increase in uric acid is due to the increase in free radical production.

V. Conclusion:

Our pilot study indicates that there is a strong association between overweight BMI and RBS and uric acid respectively among leprosy subjects in Bundelkhand region.In addition we also conclude that uric acid may potentially serve as a novel indicator for identifying patients with hyperglycemia. Therefore, further studies are warranted to determine the different roles of BMI, uric acid, and hyperglycemia involving leprosy subjects on a large scale.

Conflict of interest:
None declared.

References:

[1]. Kadri SM, Petkovic M, Taj A, Brady AH. Leprosy in the Modern Era. Pathogenic Bacteria. 2020 Dec 9:157.
[2]. Gakhar A, Pethe SS, Bansal T, Rohilla D, Gakhar M. Lepromatous Leprosy with Erythema Nodosum Leprosum Masquerading as Quadriparesis. Journal of Evolution of Medical and Dental Sciences. 2021 Feb 8;10(6):387-91.
[3]. Molehin OR, Adefegha SA, Adeyanju AA. Role of oxidative stress in the pathophysiology of type 2 diabetes and cardiovascular diseases. InRole of oxidative stress in pathophysiology of diseases 2020 (pp. 277-297). Springer, Singapore.
[4]. Mancusi C, Izzo R, di Gioia G, Losi MA, Barbato E, Morisco C. Insulin resistance the hinge between hypertension and type 2 diabetes. High Blood Pressure & Cardiovascular Prevention. 2020 Sep 22:1-2.
[5]. Tripathy JP. Burden and risk factors of diabetes and hyperglycemia in India: findings from the Global Burden of Disease Study 2016. Diabetes, metabolic syndrome and obesity: targets and therapy. 2018;11:381.
[6]. Kasper P, Martin A, Lang S, Kütting F, Goeser T, Demir M, Steffen HM. NAFLD and cardiovascular diseases: a clinical review. Clinical Research in Cardiology. 2021 Jul;110(7):921-37.
[7]. Hsu CY, McCulloch CE, Iribarren C, Darbinian J, Go AS. Body mass index and risk for end-stage renal disease. Annals of internal medicine. 2006 Jan 3;144(1):21-8.
[8]. Doddigarla Z, Ahmad J, Parwez I. Effect of chromium picolinate and melatonin either in single or in a combination in high carbohydrate diet-fed male Wistar rats. Biofactors. 2016 Jan;42(1):106-14.
[9]. Chang SA. Smoking and type 2 diabetes mellitus. Diabetes & metabolism journal. 2012 Dec 1;36(6):399-403.

[10]. Pi-Sunyer X, Blackburn G, Brancati FL, Bray GA, Bright R, Clark JM, Curtis JM, Espeland MA, Foreyt JP, Graves K, Haffner SM. Reduction in weight and cardiovascular disease risk factors in individuals with type 2 diabetes: one-year results of the Look AHEAD trial. Diabetes care. 2007 Jun;30(6):1374-83.
[11]. Boulé NG, Haddad E, Kenny GP, Wells GA, Sigal RJ. Effects of exercise on glycemic control and body mass in type 2 diabetes mellitus: a meta-analysis of controlled clinical trials. Jama. 2001 Sep 12;286(10):1218-27.
[12]. Vasudevan DM, Sreekumari S, Vaidyanathan K. Textbook of biochemistry for medical students. JP Medical Ltd; 2013 Aug 31.
[13]. Cirillo P, Sato W, Reungjui S, Heinig M, Gersch M, Sautin Y, Nakagawa T, Johnson RJ. Uric acid, the metabolic syndrome, and renal disease. Journal of the American Society of Nephrology. 2006 Dec 1;17(12 suppl 3):S165-8.
[14]. Angelopoulos TJ, Lowndes J, Zukley L, Melanson KJ, Nguyen V, Huffman A, Rippe JM. The effect of high-fructose corn syrup consumption on triglycerides and uric acid. The Journal of nutrition. 2009 Apr 29;139(6):1242S-5S.
[15]. Tsushima Y, Nishizawa H, Tochino Y, Nakatsuji H, Sekimoto R, Nagao H, Shirakura T, Kato K, Imaizumi K, Takahashi H, Tamura M. Uric acid secretion from adipose tissue and its increase in obesity. Journal of Biological Chemistry. 2013 Sep 20;288(38):27138-49.
[16]. Matsuura F, Yamashita S, Nakamura T, Nishida M, Nozaki S, Funahashi T, Matsuzawa Y. Effect of visceral fat accumulation on uric acid metabolism in male obese subjects: visceral fat obesity is linked more closely to overproduction of uric acid than subcutaneous fat obesity. Metabolism. 1998 Aug 1;47(8):929-33.
[17]. Lapidus L, Bengtsson C, Hällström T, Björntorp P. Obesity, adipose tissue distribution and health in women—results from a population study in Gothenburg, Sweden. Appetite. 1989 Aug 1;13(1):25-35.
[18]. Kim TH, Lee SS, Yoo JH, Kim SR, Yoo SJ, Song HC, Kim YS, Choi EJ, Kim YK. The relationship between the regional abdominal adipose tissue distribution and the serum uric acid levels in people with type 2 diabetes mellitus. Diabetology & metabolic syndrome. 2012 Dec;4(1):3.
[19]. Dehghan A, Van Hoek M, Sijbrands EJ, Hofman A, Witteman JC. High serum uric acid as a novel risk factor for type 2 diabetes. Diabetes care. 2008 Feb 1;31(2):361-2.
[20]. Kodama S, Saito K, Yachi Y, Asumi M, Sugawara A, Totsuka K, Saito A, Sone H. Association between serum uric acid and development of type 2 diabetes. Diabetes care. 2009 Sep 1;32(9):1737-42.
[21]. Bedir A, Topbas M, Tanyeri F, Alvur M, Arik N. Leptin might be a regulator of serum uric acid concentrations in humans. Japanese heart journal. 2003;44(4):527-36.
[22]. Paracchini V, Pedotti P, Taioli E. Genetics of leptin and obesity: a HuGE review. American journal of epidemiology. 2005 Jul 15;162(2):101-14.
[23]. Scarpace PJ, Zhang Y. Elevated leptin: consequence or cause of obesity. Front Biosci. 2007 May 1;12(1):3531-44.
[24]. Fischer S, Hanefeld M, Haffner SM, Fusch C, Schwanebeck U, Köhler C, Fücker K, Julius U. Insulin-resistant patients with type 2 diabetes mellitus have higher serum leptin levels independently of body fat mass. Acta diabetologica. 2002 Sep 1;39(3):105-10.
[25]. Widjaja A, Stratton IM, Horn R, Holman RR, Turner R, Brabant G. UKPDS 20: plasma leptin, obesity, and plasma insulin in type 2 diabetic subjects. The Journal of Clinical Endocrinology & Metabolism. 1997 Feb 1;82(2):654-7.
[26]. Gallagher D, Heymsfield SB, Heo M, Jebb SA, Murgatroyd PR, Sakamoto Y. Healthy percentage body fat ranges: an approach for developing guidelines based on body mass index. The American journal of clinical nutrition. 2000 Sep 1;72(3):694-701.
[27]. Choi HK, Atkinson K, Karlson EW, Curhan G. Obesity, weight change, hypertension, diuretic use, and risk of gout in men: the health professionals follow-up study. Archives of internal medicine. 2005 Apr 11;165(7):742-8.
[28]. Stamler J. Epidemiologic findings on body mass and blood pressure in adults. Annals of epidemiology. 1991 May 1;1(4):347-62.
[29]. Fox IH. Metabolic basis for disorders of purine nucleotide degradation. Metabolism. 1981 Jun 1;30(6):616-34.
[30]. Fabregat I, Revilla E, Machado A. Short-term control of the pentose phosphate cycle by insulin could be modulated by the NADPHNADP ratio in rat adipocytes and hepatocytes. Biochemical and biophysical research communications. 1987 Jul 31;146(2):920-5.
[31]. Tanaka K, Ogata S, Tanaka H, Omura K, Honda C, Hayakawa K. The relationship between body mass index and uric acid: a study on Japanese adult twins. Environmental health and preventive medicine. 2015 Sep;20(5):347.
[32]. Nishikawa T, Edelstein D, Du XL, Yamagishi SI, Matsumura T, Kaneda Y, Yorek MA, Beebe D, Oates PJ, Hammes HP, Giardino I. Normalizing mitochondrial superoxide production blocks three pathways of hyperglycaemic damage. Nature. 2000 Apr;404(6779):787.
[33]. Giacco F, Brownlee M. Oxidative stress and diabetic complications. Circulation research. 2010 Oct 29;107(9):1058-70.
[34]. Giacco F, Brownlee M. Mechanisms of hyperglycemic damage in diabetes. InAtlas of Diabetes 2012 (pp. 217-231). Springer, Boston, MA.
[35]. Yan LJ. Pathogenesis of chronic hyperglycemia: from reductive stress to oxidative stress. Journal of diabetes research. 2014;2014.
[36]. Hotamisligil GS, Shargill NS, Spiegelman BM. Adipose expression of tumor necrosis factor-alpha: direct role in obesity-linked insulin resistance. Science. 1993 Jan 1;259(5091):87-91.

Table 1: Findings in patients with leprosy and healthy control groups

Variable	LEPROSY subjects (n=50)	Control subjects (n=50)	P- value
Age (years)	49.9±7.2	49±4.7	NS
BMI (kg/mt^2)	28.1±4.2	24.1±2.3	S
RBS (mg/dL)	189.7±9.4	119±6.8	S
UA (mg/dL)	8.1±1	5.3±0.6	S

Note: leprosy: Type 2 Diabetes Mellitus; BMI- body mass index, Random Blood Sugar (RBS), UA-Uric Acid, S-Significant (<0.05), NS-Not Significant (>0.05)

Table 2: Pearson correlation in three different subgroups of leprosy group

Parameter	Random Blood Sugar		Uric acid	
	Pearson r	*P value*	*Pearson r*	*P value*
Ideal BMI	0.1680	=0.690	-0.2576	=0.53
Overweight BMI	0.6199	<0.05	0.3251	<0.05
Obese BMI	0.2161	=0.50	0.2648	=0.18

Table 3: Pearson correlation in three different subgroups of control group

Parameter	Random Blood Sugar		Uric acid	
	Pearson r	P value	Pearson r	P value
Ideal BMI	0.1028	=0.13	-0.0791	=0.80
Overweight BMI	-0.2733	=0.21	0.2641	=0.23
Obese BMI	-0.3802	<0.05	0.3621	=0.36

Figure 1: Findings of RBS in different BMI in leprosy and control groups

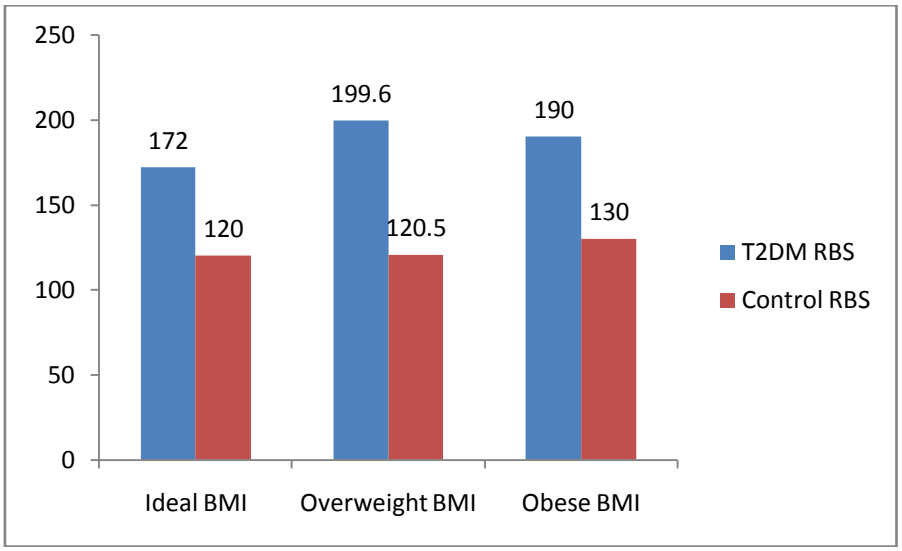
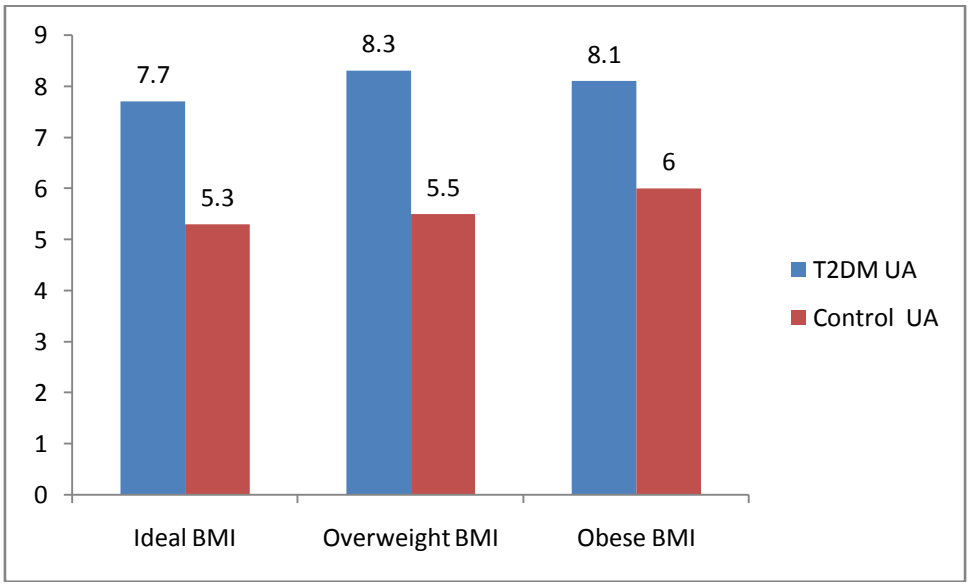


Figure 2: Findings of Uric acid in different BMI of leprosy and control groups



Dr. Priyanka Chauhan, et. al. "To study the correlation of body mass index on random blood sugar and uric acid in the leprosypatients belonging to Bundelkhand region: A pilot study." *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*, 20(9), 2021, pp. 01-05.