The Severity of The Clinical Course of Gout in Metabolic Syndrome.

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ABSTRACT

The metabolic syndrome and insulin resistance in gout have been studied in only a few studies, showing the presence of the syndrome and its characteristic insulin resistance in the vast majority of patients the effects of insulin resistance and hyperinsulinemia on gout, in particular on joint syndrome and other clinical manifestations, have only been reported in a few studies. However, they have shown a high incidence of metabolic syndrome and its characteristic insulin resistance in an overwhelming number of gout patients

A direct link has been found between certain components of the metabolic syndrome, in particular insulin resistance and hyperinsulinemia, and the severity of hyperuricaemia, which is associated with a more severe course of gouty arthritis in gout patients.

Keywords: Metabolic syndrome, gout index severity (IS), hyperglycaemia.

Purpose of work:

To study the severity of clinical manifestations, instrumental and laboratory findings in patients with gout combined with metabolic syndrome.

Subject of study. Two samples were studied:

1. A representative sample of the unorganised population aged between 40 and 69, numbering 1,335 people.
2. A sample of 120 gout patients treated in the department of the Republican Rheumatology Centre of Tashkent Medical Academy, aged 40-69.=.

Subject of research. The clinical course of gout in MS has been studied;

Research results

The Gout Severity Index (GSE), developed at the Institute of Rheumatology, Russian Academy of Medical Sciences, was used to assess the severity of the clinical course of gout (table.1.). This index includes a number of parameters characterising the clinical severity of gout severity index was calculated taking into account the presence and severity of joint syndrome, uric acid levels and the radiological picture..

The gout severity index was calculated based on the recommendations of I.A. Yakunina (2006) using the following formula:
Tophus(0-no, 1-yes)
+ number of tophuses/40
+ the number of joints affected on examination. /28
+ the number of joints affected over the course of the illness /28
+ the number of exacerbations in the last year /12
+ Length of last exacerbation (in weeks))/52
+ the age of the patient (number of whole years)/65
+ uric acid levels (μmol/l)/420 **= IS.**

Table1.
Gout severity index in different age groups with impaired glucose tolerance (IGT)

Number of observations (n)

Age	**Normoglycaemia**	**IGT**	**Total**
40-49 years old	18	22	40
50-69 years old	15	65	80
Total	33	87	120

Average values (M±m)

Age	**Normoglycaemia**	**IGT**	**Total**
40-49 years old	2,75±0,07	3,49±0,06 #	3,16±0,5
50-69 years old	3,74±0,08 *	3,68±0,05	3,69±0,04 *
Total	3,21±0,05	3,61±0,04	3,49±0,03

Comment: *) - the table shows the significance of differences in the age groups,
(#) - differences between normoglycaemic and hyperglycaemic groups

According to the findings, the gout severity index in the 50-69 age group is significantly more common than in the 40-49 age group. At the same time, gout severity index levels among those with hyperglycaemia in the younger age group (40-49 years) were significantly higher than among those with normal glycaemia levels.

It can be concluded from the findings that hyperglycaemia significantly aggravates the clinical course of gout. However, this role is played by hyperglycaemia to a greater extent before the age of 50. However, the significantly higher level of gout severity index at normal glycaemia among the 50-69 age group indicates that age is a risk factor independent of impaired glucose tolerance for a more severe course of gout.

Given that one of the aims of this study was to investigate the role of different categories of hyperglycaemia, gout severity index scores among those with impaired fasting glycaemia and post-load hyperglycaemia were further investigated (Table.2).

Table 2.
Gout severity index in different age groups in different glycaemic categories

Number of observations (n)

Age	Norm	Hyperglycaemia on an empty stomach	Hyperglycaemia after 2 hours	Total
40-49 years old	18	8	14	40
50-69 years old	15	3	62	80
Total	33	11	76	120

Average values (M±m)

Age	Norm	Hyperglycaemia on an empty stomach	Hyperglycaemia after 2 hours	Total
40-49 years old	2,75±0,08	3,83±0,11 #	3,26±0,09 #	3,16±0,07
50-69 years old	3,74±0,09 *	3,66±0,14	3,68±0,08	3,69±0,06 *
Total	3,21±0,07	3,78±0,10	3,57±0,07	3,49±0,08

Comment: *) - the table shows the significance of differences in the age groups,
(#) - differences between normoglycaemic and hyperglycaemic groups ±

The findings suggest that both fasting and post-load hyperglycaemia contribute to a worsening of the clinical course of gout. However, fasting hyperglycaemia contributes more to the increase in gout severity index than does post-load hyperglycaemia.

It should be noted that both types of hyperglycaemia, as well as impaired glucose tolerance in general, have their predominantly negative effect on the clinical course of gout among those aged 40-49 years.

Note that impaired glucose tolerance is only one component of the metabolic syndrome. However, the aim of our study was to investigate the role of the metabolic syndrome in general on the clinical course of gout.

In this context, the average gout severity index in persons with metabolic syndrome with impaired glucose tolerance was considered.

Table 3.

Gout severity index among those with metabolic syndrome with impaired glucose tolerance

Number of observations (n)

Presence of metabolic syndrome	*Norm*	Impaired glucose tolerance .	*Overall result*
There is metabolic syndrome	13	87	100
No metabolic syndrome	20	-	20
Total	33	87	120

Average values (M±m)

Presence of metabolic syndrome	*Norm*	*Impaired glucose tolerance .*	*Overall result*
There is metabolic syndrome	3,38±0,10	3,61±0,07	3,58±0,8 *
No metabolic syndrome	3,08±0,09	-	3,08±0,09
Total	3,20±0,08	3,61±0,06	3,49±0,07

Comment: *) - the significance of the difference between the groups with and without MS,

As the data in Table 3 show, the severity index of gout is significantly higher among those with the metabolic syndrome than among those without the syndrome. This confirms the important role of the metabolic syndrome in the pathogenesis of gout.

However, according to the modern classification of metabolic syndrome, it can be fixed not only when all 4 of its components are present, but also when 3 components are present. This interpretation of the diagnosis of metabolic syndrome stems from the fact that in addition to impaired glucose tolerance, other components may also contribute to the formation and more severe course of gout.

Therefore, the average gout severity index among individuals with different numbers of individual components of the metabolic syndrome was analyzed further (Table 4.).

Table 4.

Gout severity index among individuals with different numbers of individual components of the metabolic syndrome in impaired glucose tolerance.

Number of observations (n)

Number of components	Normal glucose tolerance	Impaired glucose tolerance	Overall result
1	5	-	5
2	14	1	15
3	14	22	36
4 (total metabolic syndrome))	-	64	64
Total	33	87	120

Average values (M±m)

Number of components	Normal glucose tolerance	Impaired glucose tolerance	Overall result
1	2,41±0,18	-	2,41±0,18
2	3,26±0,09	3,55±0,0	3,28±0,09
3	3,39±0,10 *	3,62±0,08	3,54±0,09 *
4 (total metabolic syndrome))	-	3,60±0,06	3,59±0,06 *
Total	3,20±0,07	3,61±0,05	3,49±0,06

Comment: *) - significance of differences to the 1 component MS group is indicated

Conclusion:

Overall, according to the findings, there is an increase in the severity index of gout as the number of components of the metabolic syndrome increases. Moreover, the differences in gout severity index among individuals with a single component of the metabolic syndrome are significantly lower than those with 3 or 4 components of the metabolic syndrome.

In the group of individuals with normal glucose tolerance, there was also an increase in the severity index of gout. The difference in gout severity index between the groups with the 1st and 3rd components of the metabolic syndrome was statistically significant.

Thus, it can be concluded that in general, an increase in the number of components of the metabolic syndrome contributes to the aggravation of the clinical course of gout, as indicated by an increase in the gout severity index.

The gout severity index (GSI), reflecting the severity of the clinical course of the disease and uric acid levels, has a direct correlation with the glycaemic rate and an inverse relationship with the post-glycaemic rate, indicating that the clinical course of gout depends on the activity of the sympathoadrenal and vagoinsular phases of the glycaemic curve.

References

1. Alnuvairakh A.A., Tyrenko V.V.,Tsygan E.N. Hyperuricemia and metabolic syndrome in gout patients. Bulletin of the Russian Military Medical Academy .№3 (35) 2011 75-78.
2. Barskova V. G. Metabolic syndrome and cardiovascular disorders in gout // Author's abstract. D. in medical sciences. M., 2006. P.39.
3. Barskova V.G., Ilinykh E.V., Nasonov E.L. Febuxostat - a new drug in gout therapy. Scientific and Practical Rheumatology. 2011;49(2)
4. Eliseev M.S., Barskova V.G. Modern principles of diagnosis and treatment of gout.//BC. - 2010. - volume 18, № 27
5. Eliseev M.S.,Chikalenkov N.A., Denisov I.S., Barskova V.G. Risk factors for gout. Scientific and practical rheumatology . 2011 (6) 28-31
6. Kayumov U.K. Metabolic disorders and new perspectives for their correction in

general medical practice.// Magazine «MedicalExpress».- 2010. - № 1.- p.22-24
7. New Acre guidelines for the management of gout 2020. Comments on some items.
8. Gout. APP clinical guidelines 2018. Association of Rheumatologists of Russia.
9. Sukhikh J.L.,Shtonda M.V., Gout - current aspects of diagnosis and treatment. Clinical medicine,2011 №3
10. Shalnova S.A., Deyev A.D., Artamonova G.V. et al. Hyperuricemia and its correlates in Russian population (results of an epidemiological study). *Rational pharmacotherapy in cardiology.* 2014;10(2):153-159.
11. Yakubova.S.P. Gout. New diagnostic and treatment options. *Therapeutic archive*. 2018;90(05):88-92.
12. Chen S. Y., Chen C. L., Shen M. L. Manifestations of metabolic syndrome associated with male gout in different age strata // Clin. Rheumatol. 2007. V. 26, 9. P. 453-457.
13. Hiroyasu, I. Metabolic syndrome and the risk of ischemic heart disease and stroke among Japanese men and women /1. Hiroyasu, S. Shinichi, A. Kitamura // Stroke. 2007. - № 38. - P. 1744-1751.
14. Insulin resistance syndrome in patients with gout and its influence on uric acid concentration and severity of arthritis /M.S. Eliseev, V.G. Barsko-va, V.A. Miller,
15. M. APOC3 promoter polymorphisms C-482T and T-455C are associated with the metabolic syndrome / M. Miller, J. Rliyne, H. Chen // Arch. Med. Res. 2007. - V. 38, № 4. - P. 444-451.
16. Dolgova L.N., Krasivina I.G., Dolgov N.V., Lugovkina D.G. Metabolic risks of hyperuricemia. Meditsinskiy sovet = Medical Council. 2019;(18):76-84. (In
17. Li Q., Li X., Wang J., Liu H. et al. Diagnosis and treatment for hyperuricemia and gout: a systematic review of clinical practice guidelines and consensus statements. *BMJ Open.* 2019;9(8):1-13.
18. Woyesa S.B., Hirigo A.T., Wube T.B. Hyperuricemia and metabolic syndrome in type 2 diabetes mellitus patients at Hawassa university comprehensive specialized hospital, South West Ethiopia. *BMC Endocr Disord*. 2017;17(1)
19. 18.Widecka K., Szymański F.M., Filipiak K.J. et al. Hyperuricemia and its treatment in patients with a high cardiovascular risk – experts opinion.*Arterial Hypertens*. 2017;21(1)
20. White W.B., Saag K.G., Becker M.A. et al. Cardiovascular Safety of Febuxostat or Allopurinol in Patients with Gout. *The New England Journal of Medicine*. 2018;378(13)
21. Yamanaka H., Tamaki S., Ide Y. et al. Stepwise dose increase of febuxostat is comparable with colchicine prophylaxis for the prevention of goutflares during the initial phase of urate-lowering therapy: results from FORTUNE-1, a prospective, multicentre randomised study. *Annals of the Rheumatic Diseases*. 2018;77(2)
22. Li Q., Li X., Wang J., Liu H. et al. Diagnosis and treatment for hyperuricemia and gout: a systematic review of clinical practice guidelines and consensus statements. *BMJ Open.* 2019;9(8)
23. Woyesa S.B., Hirigo A.T., Wube T.B. Hyperuricemia and metabolic syndrome in type 2 diabetes mellitus patients at Hawassa university comprehensive specialized hospital, South West Ethiopia. *BMC Endocr Disord*. 2017;17(1)