The severity features of artharagra clinical course in metabolic syndrome

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Abstract – *A few studies have been devoted to the metabolic syndrome and insulin resistance study in artharagra, which showed the syndrome presence and its characteristic insulin resistance in the overwhelming patients' number. Only a few studies have reported on the insulin resistance effect and hyperinsulinemia on the artharagra course, in particular on the articular syndrome and other clinical disease manifestations. Nevertheless, they showed a high incidence of metabolic syndrome and its characteristic insulin resistance in the vast majority patients with artharagra.*

A direct relationship was revealed between the individual metabolic syndrome components, in particular, insulin resistance and hyperinsulinemia, and the hyperuricemia severity, which is accompanied by a more severe artharagray arthritis course in patients with artharagra.

Key words: *metabolic syndrome, artharagra severity index, hyperglycemia.*

1. Introduction

Purpose of the work: To study the severity features of clinical manifestations, data from instrumental and laboratory studies in patients with artharagra occurring in combination with metabolic syndrome.

Study object. Two samples were examined:

1. A representative the unorganized population sample at age 40 to 69 in 1335 people.
2. Patients sample with artharagra that underwent treatment at the Republican rheumatological center department of Tashkent medical academy in 120 people at age 40 - 69.

Study subject. The clinical course features of artharagra in MS were studied;

2. Research results.

To assess the severity characteristics of the artharagra clinical course, the artharagra severity index (ASI) was used, developed at Rheumatology institute of Russian medical sciences academy (Table 1). This index includes a number of parameters characterizing the clinical artharagra severity; the artharagra severity index was calculated taking into account the presence and the articular syndrome severity, the uric acid level and the reIG tenological picture.

The artharagra severity index calculation was carried out on the basis of I.A. Yakunina (2006) recommendations according to the following formula:

Tophus (0-no, 1-present)
+ number of tofus/40
+ number of affected joints at examination/28
+ number of affected joints during the entire period of illness/28
+ number of exacerbations in the last year/12
+ duration of the last exacerbation (in weeks)/52
+ patient's age (number of full years)/65
+ uric acid level (μmol / l)/420 = IT**.**

Table1.

Artharagra severity index for different age groups in impaired glucose tolerance (IGT)

Observations number (n)

Age	Normoglycemia	IGT	The overall result
40-49 years old	18	22	40
50-69 years old	15	65	80
The overall result	33	87	120

Average values (M+m)

Age	Normoglycemia	IGT	The overall result
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 *
The overall result	3,21±0,05	3,61±0,04	3,49±0,03

Note: *) - the table shows the reliability of the differences in age groups,

#) – differences between groups with normoglycemia and hyperglycemia.

According to the data obtained, the artharagra severity index at age 50-69 is significantly more common than at age 40-49. At the same time, the artharagra severity levels index among persons with hyperglycemia in the younger age group (40-49 years old) were significantly higher than among persons with normal glycemic levels.

From the data obtained, it can be concluded that hyperglycemia significantly aggravates the artharagra clinical course. However, hyperglycemia plays this role to a greater extent before at age 50. At the same time, a significantly artharagra severity higher level index with normal glycemia among people at age 50-69 years indicates that age is a risk factor for a more severe artharagra course, impaired glucose tolerance independent.

Considering that one of the objectives of this study was to study various hyperglycemia categories role, and then the artharagra severity indices index among people with impaired fasting glycemia and post-load hyperglycemia were studied (Table 2).

Table 2.

Artharagra severity index in different age groups with different glycemia categories

Observations number (n)

Age	Norm	Fasting hyperglycemia	Hyperglycemia after 2 hours	The overall result
40-49 years old	18	8	14	40
50-69 years old	15	3	62	80
The overall result	33	11	76	120

Average values (M+m)

Age	Norm	Fasting hyperglycemia	Hyperglycemia after 2 hours	The overall result
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 *
The overall result	3,21±0,07	3,78±0,10	3,57±0,07	3,49±0,08

Note: *) - the table shows the reliability of differences in age groups,

#) – differences between groups with normoglycemia and hyperglycemia ±

The findings indicate that both fasting and post-load hyperglycemia contribute to the worsening of the artharagra clinical course. At the same time, fasting hyperglycemia contributes more to the increase in the artharagra severity index than post-load hyperglycemia.

It should be noted that both hyperglycemia types, as well as impaired glucose tolerance in general, have their predominant negative effect on the clinical course of artharagra among people at age 40-49.

Note that impaired glucose tolerance is only one of the metabolic syndrome components. At the same time, the objectives of our study were to study metabolic syndrome role in general on the artharagra clinical course.

In this regard, the average indicators of the artharagra severity index in persons with metabolic syndrome, were considered occurring against the impaired glucose background tolerance.

Table 3.

Artharagra severity index among persons with metabolic syndrome and impaired glucose tolerance

Observations number (n)

Presence of metabolic syndrome	*Norm*	Impaired glucose tolerance	*The overall result*
Have 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	*The overall result*
Have 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

Note: *) - the reliability of the differences between the groups with and without MS is indicated,

As follows from the data shown in Table 3, the artharagra severity index level among people with metabolic syndrome is significantly higher than among people who do not have this syndrome. This confirms the important role of metabolic

syndrome in the pathogenesis of artharagra.

However, according to the modern classification of metabolic syndrome, it can be recorded not only when all 4 of its components are present, but also in the 3 presence components. This interpretation of metabolic syndrome diagnosis is due to the fact that, along with impaired glucose tolerance, other components can also contribute to the formation of a more severe course of artharagra.

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

Table 4.

Artharagra severity Index among individuals with different amounts
Individual components of the metabolic syndrome with impaired glucose tolerance

Observations number (n)

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

Average values (M+m)

Number of components	Normal glucose tolerance	Impaired glucose tolerance	The 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 (complete metabolic syndrome)	-	3,60±0,06	3,59±0,06 *
Total	3,20±0,07	3,61±0,05	3,49±0,06

Note: *) - the differences reliability for the group with 1 MS component is indicated

3. Conclusions:

According to the obtained data, in general, as the number of metabolic syndrome components increases, the artharagra severity index increases. Moreover, the differences in the value of the index of severity of artharagra among persons with one component of the metabolic syndrome are significantly lower than those with 3 or 4 components of the metabolic syndrome.

In the group of persons with normal glucose tolerance, an increase in the ASI level is also observed. At the same time, the differences in the artharagra severity index level between the groups with the 1st and 3rd components of the metabolic syndrome were statistically significant.

Thus, we can conclude that, in general, an increase in the number of metabolic syndrome components contributes to clinical course aggravation of artharagra, as evidenced by an increase in the artharagra severity index.

The artharagra severity index (ASI), which reflects the clinical course severity of this disease and the uric acid level, has a direct correlation with the glycemic and inverse relationship with the postglycemic coefficient, which indicates the artharagra clinical course dependence on the sympathoadrenal and vagoinsular phases activity of the glycemic curve.

References

1. Barskova V.G., Nasonova V.A. Arthragra and insulin resistance syndrome // Russian med. journal. 2003. T.P., № 23. p. 1299-1301.
2. Sukhikh J.L., Shtonda M.V., Arthragra - modern aspects of diagnosis and treatment. Clinical medicine,2011 №3
3. Arthragra. Clinical guidelines APP 2016.
4. Byrne, C. D. Themetabolicsyndrome / C. D. Byrne, S. H. Wild. Chichester, 2005.-418 p
5. Chen S.Y., Chen C.L., Shen M.L. et al. Trends in the manifestations of artharagra in Taiwan. Rheumatology (Oxford). 2003; 42:1529–1533.
6. Metabolic syndrom and ischemic heart disease in artharagra / J. Vazquez-Mellado et al. // J. Clin. Reumatol. 2004.

V. 10, 3. P. 105-109.
7. Miller, 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.