**THE DUAL ROLE OF LIVER ENZYMES IN THE DIAGNOSIS AND MONITORING OF NAFL**

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| **Abstract** Non-alcoholic fatty liver disease (NAFLD) has emerged as a major global health concern, often leading to significant morbidity and mortality. Liver enzymes, including alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP), are routinely measured in clinical practice. While these enzymes are traditionally used to assess liver function, their role in diagnosing, monitoring, and prognosis NAFLD is increasingly recognized. The results showed that there is a significant difference between the mean patient age of the two groups studied (control group and patient groups), the patient group was significantly excellent in the mean patient age which recorded 61.83 years as compared with 44.20 years for healthy persons in the control group. The results showed that the age groups above 70 years significantly outperformed the rest of the age groups in the prevalence of the disease, reaching 36%, followed by the age group 60-69 years, which recorded 30%, while the age group less than 30 years has not recorded any infection with the disease is 0%. The results presented that the average cumulative glucose concentration (HbA1C) achieved a significant increase in the patient group, amounting to 6.37% compared to 4.96% for the control group. The results indicated that the patient group was significantly superior to the control group in terms of a significant increase in the ALT level (67.11 vs. 34.60) U/L. The results revealed that the patient group was significantly superior to the control group in raising the average AST value to 39.22 U/L compared to 16.82 U/L for the control group. Data showed that there is a significant difference in the values of ALP levels between the two groups studied, but the patient group was significantly superior in raising the ALP values to 77.50 U/L compared to 49.60 U/L for the control group. |
| Keywords: NAFLD, ALT, AST, ALP |

1. **Introduction (Times New Roman 12-Bold)**

Our lifestyle has become more sedentary due to industrialization, urbanization, and modernity. More and People who use very little to no alcohol most commonly suffer from nonalcoholic fatty liver disease (NAFLD), a condition that affects the liver. In this disease, an excessive amount of fat accumulates in the liver. Overweight or obese individuals account for the majority of cases. As the number of individuals who are obese continues to climb, nonalcoholic fatty liver disease is becoming increasingly prevalent all over the globe, particularly in Western and Middle Eastern countries. About 25 percent of the world's population is affected by this kind of chronic liver disease, which is the most prevalent form. NAFLD affects around 100 million individuals in the United States of America [1]. Nonalcoholic steatohepatitis, often known as NASH, may develop in certain individuals who have nonalcoholic fatty liver disease. Because of the accumulation of fat in the liver, nonalcoholic fatty liver disease is a severe type of fatty liver disease that causes the liver to enlarge and cause damage to the liver. There is a possibility that NASH could worsen, which might result in cirrhosis, significant scarring of the liver, and perhaps liver cancer. High alcohol consumption induces harm comparable to this damage [2].

Now, there is an effort being made to alter the nomenclature of the condition that is now known as nonalcoholic fatty liver disease to metabolic dysfunction-associated steatotic liver disease (MASLD). Specialists have proposed MASH, an acronym for metabolic dysfunction-associated steatohepatitis, as a replacement for the term "nonalcoholic steatohepatitis." To come up with a full treatment plan for people with NAFLD, it is important to diagnose NAFLD (at least basic steatosis) and severe hepatic fibrosis as soon as possible. Note that you can reverse non-alcoholic fatty liver disease, especially in its early stages. In certain high-risk individuals, the failure to recognize the illness at an early stage may have severe clinical consequences [3].

However, early detection of the disease could lead to adverse clinical effects. A liver biopsy, by its very nature, is an intrusive diagnostic, inherently linked to adverse clinical outcomes, and ineffective for some individuals. Furthermore, the process consumes a substantial amount of resources, operates under the guidance of ultrasonography, and frequently requires a day-case admission. Additionally, it is a very expensive operation [4]. These non-invasive liver tests (NILTs) are now used in clinical practice because imaging tests can diagnose and even stage non-alcoholic fatty liver disease and fibrosis without the problems that come with a biopsy and blood biomarkers that show how well the liver is working. The availability of the tests and their integration into clinical practice vary significantly among NHS providers. As a result of these factors, the GDG placed a high priority on conducting an original economic analysis for the review of issues that examine objective diagnostic methods for the diagnosis of non-alcoholic fatty liver disease and advanced fibrosis, as well as the individuals who should be provided with such testing [5].

The use of genetics, molecular biology, mass spectrometry, and other fundamental scientific tools in the analysis of samples from individuals with NAFLD and murine models of the condition has led to a deeper understanding of the processes that underlie the start and progression of illness in NAFLD. New information has led to the discovery of several different possible drug targets that could help prevent, slow down, or reverse the hepatic inflammation and fibrosis that can happen with non-alcoholic fatty liver disease. Peroxisomal proliferator-activated receptor gamma (PPAR γ) or insulin-sensitizing medicines like glitazones have been areas of great attention up to this point [6].

Additionally, medications with anti-inflammatory or hepatoprotective effects, like vitamin E, have also been of significant interest. Recently, there has been a growing acknowledgment on a worldwide scale of the existing and possible future burden of non-alcoholic fatty liver disease. This has brought to light the significant need for effective pharmaceutical therapies for the illness, which is currently unfulfilled. Over the previous ten years, research in this field has advanced from pre-clinical investigations to randomized clinical trials. The use of appropriate histology and surrogate end-points in clinical trials of new therapies for non-alcoholic fatty liver disease is growing on a global scale. This has helped improve the quality of trial design across the scientific community [7].

To systematically review and meta-analyze the existing literature on the diagnostic accuracy of liver enzymes (ALT, AST, ALP) in detecting non-alcoholic fatty liver disease (NAFLD). To evaluate the prognostic significance of liver enzymes in predicting the progression of NAFLD to non-alcoholic steatohepatitis (NASH) and fibrosis. To investigate the potential role of liver enzymes as biomarkers for monitoring the response to NAFLD treatment interventions.

1. **Materials and Methods**

In this study, a random sample of 150 people was selected for patients visiting health institutions in the city of Baghdad / Iraq for the period from January 2 to June 30, 2024, to investigate the dual effect of liver enzymes in early diagnosis and detection of non-alcoholic fatty liver disease, thus facilitating control of the disease and reducing health harms and to prevent complications resulting from this disease. The random sample was divided into two groups as follows: The first group consisted of 50 healthy people who visited health institutions and did not suffer from non-alcoholic fatty liver disease. The second group consisted of 100 patients who visited health institutions and were diagnosed as suffering from non-alcoholic fatty liver disease through the diagnosis of a specialist doctor through a clinical examination and symptoms of the disease, which include: The patient suffers from type 2 diabetes, high blood pressure, high lipid levels, etc. Blood samples were drawn from patients, amounting to 5 ml for each patient, using a blood drawing device, and then the patient’s information was taken, which included name, age, gender, weight, and medical history, and then the blood samples were transferred to the specialized laboratory to conduct the required tests. The cumulative HbA1C blood sugar level was measured for the patients under study using the Que-Test HbA1C analyzer device produced by EKF Diagnostics, where the barcode is first read, and then blood is taken from the patient through finger pricking, and the blood is transferred into the cartridge, then the cartridge is inserted into the analyzer device, and then I pushed the slide in, closed the door, and started the test. The device will read the analysis within 5 minutes. This test is based on the ALT enzyme transferring the amino group from alanine to alpha-oxoglutarate to form pyruvate and glutamate. In the next step, pyruvate carries out a reductive reaction with NADH with the help of the lactate dehydrogenase enzyme to produce lactate and NAD+. The decrease in absorbance is due to the consumption of NADH, measured at 380 nm, equivalent to the activity of the ALT enzyme in the sample. The principle of this test is for the AST enzyme to reduce the transfer of the amino group from aspartate to oxoglutarate, with the production of glutamate and oxaacetate, and the latter reduces malate with the help of the MDH enzyme in the presence of reduced NADH. The reaction is measured kinetically at a temperature of 340 nm through the rate of decrease in absorbance resulting from the process of oxidizing NADH to NAD+ which is equivalent to the activity of the AST enzyme present in the sample. First, all reagents, samples, and controls must be incubated at the incubation temperature, then the photometer is set to zero absorbance using distilled water, and then placed in cuvette 1 of each of the working reagents at 37 and 30 degrees Celsius, then we add 50 microliters of the sample or control at 37 and 100 microliters of the model or control at 30 degrees Celsius and mix well, then insert the cuvette into the cell holder and incubate for 60 seconds. The initial absorbance reading is recorded, and the absorbance is re-read after 1, 2, and 3 minutes have passed and the amount of difference between the absorbance and the rate of change in absorbance per minute is calculated (∆A/ minute). The ALP enzyme catalyzes the hydrolysis of p-nitrophenylphosphate to a yellow product, which is nitrophenol and phosphate. This reaction occurs in a basic medium (10.3), and the rate of change in absorbance is measured at 405 nm. This change in absorbance is equivalent to the amount of activity of the ALP enzyme in the sample. Incubate the working reagent at 37°C, then add 20 microliters of the sample with 1 mL of the working reagent, then incubate at 37°C for 1 minute, read the amount of change in absorbance every minute for 3 minutes, and then calculate the rate of change in absorbance for every 1 minute. The statistical analysis of the variables in this study was conducted according to the statistical program SPSS, version 22.0, produced by the American company IBM. The average values between the tested groups were compared, and the standard deviation and standard error were extracted, while the Pearson correlation between the different values was studied.

1. **Results and Discussion**
	1. **The Age of Patients of Groups Studied**

The results of Table 1 showed that there is a significant difference between the mean patient ages of the two groups studied (control and patients’ groups), The patient group was significantly excellent in the mean patient age which recorded 61.83 years as compared to with 44.20 years for healthy persons in control group. The standard deviation of the patients group recorded 12.13 while the control group recorded 8.29.

**Table 1.** The age of patients in the groups studied

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Group studied | Mean Year | N | Std. Deviation | Std. Error of Mean |
| Control | 44.2000 | 50 | 8.29310 | 1.17282 |
| Patients | 61.8300 | 100 | 12.13315 | 1.21331 |
| Total | 55.9533 | 150 | 13.78275 | 1.12536 |

**Figure 1.** The age of patients in the groups studied

The results presented in Table 2 showed that the age groups above 70 years significantly outperformed the rest of the age groups in the prevalence of the disease, reaching 36%, followed by the age group 60-69 years, which recorded 30%, while the age group less than 30 years has not recorded any infection with the disease is 0%. [8] found that the age group most affected by the disease was 56-60 years. Many studies indicated that the age group most affected by the disease was the age group over 65 years [9]. [10] also showed that older patients were more sensitive to contracting the disease in addition to other risk factors such as high blood pressure, diabetes, obesity, and high ALT values. Table 4.3 showed that there is a strong positive significant correlation between the patient’s age and the tested groups, as it recorded 0.605 at the 0.01 level.

**Table 2.** Age period of patient group

|  |  |
| --- | --- |
| Age period | Patients group N (%) |
| <30 Years | 0(0.0) |
| 30-39 Years | 4(4.0) |
| 40-49 Years | 19(19.0) |
| 50-59 Years | 11(11.0) |
| 60-69 Years | 30(30.0) |
| >70 Years | 36(36.0) |
| Total | 100(100.0) |

1. **Conclusion**

**Figure 2.** Number of patients infected with non-alcoholic fatty liver disease

**Table 3.** Correlation between the age of patients and groups studied

|  |  |  |
| --- | --- | --- |
|  | Group studied | Age |
| Group studied | Pearson Correlation | 1 | .605\*\* |
| Sig. (2-tailed) |  | .000 |
| N | 150 | 150 |
| Age | Pearson Correlation | .605\*\* | 1 |
| Sig. (2-tailed) | .000 |  |
| N | 150 | 150 |

\*\*Correlation is significant at the 0.01 level (2-tailed)

**3.2. HbA1C level of groups studied**

The results presented in Table 4 show that the average cumulative glucose concentration (HbA1C) achieved a significant increase in the patient group, amounting to 6.37% compared to 4.96% for the control group. Explained that insulin resistance is one of the influential factors that contribute to the development of non-alcoholic fatty liver disease. [11] found that patients with non-alcoholic fatty liver disease had elevated HbA1c concentrations ranging from 5.7 to 6.5%. [12] also showed that HbA1C levels are significantly associated with the presence of non-alcoholic fatty liver disease and concluded that HbA1C can be used as a biomarker to predict the disease.

Table 5 revealed that there is a strong positive correlation between HbA1C level, and the groups studied which recorded 0.756 at a level of 0.01.

**Table 4.** The HbA1C level of groups studied

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Group studied | Mean % | N | Std. Deviation | Std. Error of Mean |
| Control | 4.9622 | 50 | .42863 | .06062 |
| Patients | 6.3740 | 100 | .64112 | .06411 |
| Total | 5.9034 | 150 | .88285 | .07208 |

**Table 5.** Correlation between HbA1C level and groups studied

|  |  |  |
| --- | --- | --- |
|  | Group studied | HbA1C |
| Group studied | Pearson Correlation | 1 | .756\*\* |
| Sig. (2-tailed) |  | .000 |
| N | 150 | 150 |
| HbA1C | Pearson Correlation | .756\*\* | 1 |
| Sig. (2-tailed) | .000 |  |
| N | 150 | 150 |

\*\*Correlation is significant at the 0.01 level (2-tailed)

**Figure 3.** The HbA1C level of groups studied

**3.3. ALT level of groups studied**

Table 6 indicated that the patient group was significantly superior to the control group in terms of a significant increase in the ALT level (67.11 vs. 34.60) U/L. [13] found that there was an increase in ALT and AST levels in patients with non-alcoholic fatty liver disease. While [14] indicated that there is an increase in the level of ALT by 19 and 30 times for women and men with the disease, respectively. While [15] indicated that ALT is a poor predictor of non-alcoholic fatty liver disease. However [16], showed that the ALT level can be used as a high-risk indicator of the severity of non-alcoholic fatty liver disease, especially in patients with moderate conditions. Confirmed that the use of ALT levels in the medical diagnosis of non-alcoholic fatty liver disease still needs additional tests and studies. While [17] found that non-alcoholic fatty liver disease was significantly associated with elevated ALT [18] found that people who drink small amounts of alcohol have elevated ALT levels.

**Table 6.** ALT level of groups studied

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Group studied | Mean U/L | N | Std. Deviation | Std. Error of Mean |
| control | 34.6000 | 50 | 9.68694 | 1.36994 |
| patients | 67.1112 | 100 | 8.28515 | .82851 |
| Total | 56.2741 | 150 | 17.68978 | 1.44436 |

The results of Table 7 showed that there is a positive strong significant correlation between ALT levels and the groups studied which recorded 0.869 at a level of 0.01.

**Table 7.** Correlation between ALT level and groups studied

|  |  |  |
| --- | --- | --- |
|  | Group studied | ALT |
| Group studied | Pearson Correlation | 1 | .869\*\* |
| Sig. (2-tailed) |  | .000 |
| N | 150 | 150 |
| ALT | Pearson Correlation | .869\*\* | 1 |
| Sig. (2-tailed) | .000 |  |
| N | 150 | 150 |

\*\*Correlation is significant at the 0.01 level (2-tailed)

**Figure 4.** ALT level of groups studied

**3.4. AST level of groups studied**

The results of Table 8 revealed that the patient group was significantly superior to the control group in raising the average AST value to 39.22 U/L compared to 16.82 U/L for the control group. [19] point out that liver enzymes are commonly used to evaluate people for a wide range of diseases. It is known that AST is an enzyme found mainly in the liver, in addition to its presence in other organs of the body. When liver cells are destroyed, this leads to the release of this enzyme into the blood, and its concentrations in the blood rise, this helps us in diagnosing this disease [20]. [21] show that a high AST level is associated with an increased risk of non-alcoholic fatty liver disease. While [22] showed that patients with alcoholic fatty liver disease have a high AST/ALT ratio.

**Table 8.** AST level of groups studied

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Group studied | Mean U/L | N | Std. Deviation | Std. Error of Mean |
| Control | 16.8200 | 50 | 2.12555 | .30060 |
| Patients | 39.2260 | 100 | 8.11866 | .81187 |
| Total | 31.7573 | 150 | 12.55352 | 1.02499 |

The results of Table 9 pointed that there is a positive strong significant correlation between AST level and groups studied which recorded 0.844 at a level of 0.01.

**Table 9.** Correlation between AST level and groups studied

|  |  |  |
| --- | --- | --- |
|  | Group studied | AST |
| Group studied | Pearson Correlation | 1 | .844\*\* |
| Sig. (2-tailed) |  | .000 |
| N | 150 | 150 |
| AST | Pearson Correlation | .844\*\* | 1 |
| Sig. (2-tailed) | .000 |  |
| N | 150 | 150 |

\*\*Correlation is significant at the 0.01 level (2-tailed)

**Figure 5.** AST level of groups studied

**3.5. ALP level of groups studied**

Data from Table 10 showed that there is a significant difference in the values ​​of ALP levels between the two groups studied, but the patient group was significantly superior in raising the ALP values ​​to 77.50 U/L compared to 49.60 U/L for the control group. [23] revealed that the ALP level was 220 U/L for patients with type 2 diabetes. While [24] found that the ALP levels of about 99.94% of the population in his study were within the normal range. While [25] confirmed a slight increase in ALP levels in patients with non-alcoholic fatty liver disease. [26] indicated that ALP can be used as a good indicator for predicting non-alcoholic fatty liver disease, especially in obese patients. [27] found that there is a significant correlation between ALP levels and changes in dependent fatty liver disease and that this hormone can be used to predict this disease.

**Table 10**. ALP-level of groups studied

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Group studied | Mean U/L | N | Std. Deviation | Std. Error of Mean |
| Control | 49.6000 | 50 | 4.52657 | 0.64015 |
| Patients | 77.5040 | 100 | 9.41900 | 0.94190 |
| Total | 68.2027 | 150 | 15.48792 | 1.26458 |

The results of Table 11 revealed that there is a positive significant correlation between ALP level and the groups studied which recorded 0.852 at a level of 0.01.

**Table 11.** Correlation between ALP level and groups studied

|  |  |  |
| --- | --- | --- |
|  | Group studied | ALP |
| Group studied | Pearson Correlation | 1 | .852\*\* |
| Sig. (2-tailed) |  | .000 |
| N | 150 | 150 |
| ALP | Pearson Correlation | .852\*\* | 1 |
| Sig. (2-tailed) | .000 |  |
| N | 150 | 150 |

\*\* Correlation is significant at the 0.01 level (2-tailed)

**Figure 6.** ALP-level of groups studied

1. **Conclusion**

The results showed that there is a significant difference between the mean patient age of the two groups studied (control and patient groups), the patient group was significantly excellent in the mean patient age which recorded 61.83 years as compared with 44.20 years for healthy persons in the control group. The results showed that the age groups above 70 years significantly outperformed the rest of the age groups in the prevalence of the disease, reaching 36%, followed by the age group 60-69 years, which recorded 30%, while the age group less than 30 years has not recorded any infection with the disease is 0%. The results presented that the average cumulative glucose concentration (HbA1C) achieved a significant increase in the patient group, amounting to 6.37% compared to 4.96% for the control group. The results indicated that the patient group was significantly superior to the control group in terms of a significant increase in the ALT level (67.11 vs. 34.60) U/L. The results revealed that the patient group was significantly superior to the control group in raising the average AST value to 39.22 U/L compared to 16.82 U/L for the control group. Data showed that there is a significant difference in the values of ALP levels between the two groups studied, but the patient group was significantly superior in raising the ALP values to 77.50 U/L compared to 49.60 U/L for the control group.

**References**

1. Powell, E. E., Wong, V. W. S. and Rinella, M. 2021. Non-alcoholic fatty liver disease. *The Lancet*, *397*(10290): 2212-2224.
2. Neuschwander-Tetri, B. A. 2017. Non-alcoholic fatty liver disease. *BMC medicine*: *15*, 1-6.
3. Choudhari, S. and Jothipriya, M. A. 2016. Non-alcoholic fatty liver disease. *Research Journal of Pharmacy and Technology*, *9*(10): 1782-1785.
4. Bellentani, S. 2017. The epidemiology of non‐alcoholic fatty liver disease. *Liver international*, *37*: 81-84.
5. Maurice, J. and Manousou, P. 2018. Non-alcoholic fatty liver disease. *Clinical medicine*, *18*(3): 245.
6. Calzadilla Bertot, L. and Adams, L. A. 2016. The natural course of non-alcoholic fatty liver disease. *International journal of molecular sciences*, *17*(5): 774.
7. Bedossa, P. 2017. Pathology of non‐alcoholic fatty liver disease. *Liver International*, *37*: 85-89.
8. Summart, U., Thinkhamrop, B., Chamadol, N., Khuntikeo, N., Songthamwat, M. and Kim, C. S. 2017. Gender differences in the prevalence of nonalcoholic fatty liver disease in the Northeast of Thailand: A population-based cross-sectional study. *F1000Research*, *6*.
9. Lonardo, A., Nascimbeni, F., Ballestri, S., Fairweather, D., Win, S., Than, T. A. and Suzuki, A. 2019. Sex differences in nonalcoholic fatty liver disease: state of the art and identification of research gaps. *Hepatology*, *70*(4): 1457-1469.
10. Frith, J., Day, C. P., Henderson, E., Burt, A. D. and Newton, J. L. 2009. Non-alcoholic fatty liver disease in older people. *Gerontology*, *55*(6): 607-613.
11. Chen, C., Zhu, Z., Mao, Y., Xu, Y., Du, J., Tang, X. and Cao, H. 2020. HbA1c may contribute to the development of non-alcoholic fatty liver disease even at normal-range levels. Biosci Rep., 40(1): BSR20193996.
12. Masroor, M. and Haque, Z. 2021. HbA1C as a biomarker of non-alcoholic fatty liver disease: comparison with anthropometric parameters. *Journal of Clinical and Translational Hepatology*, *9*(1): 15.
13. Bayard, M., Holt, J. and Boroughs, E. 2006. Nonalcoholic fatty liver disease. *American Family Physician*, *73*(11): 1961-1968.
14. Hadizadeh, F., Faghihimani, E. and Adibi, P. 2017. Nonalcoholic fatty liver disease: Diagnostic biomarkers. *World journal of Gastrointestinal Pathophysiology*, *8*(2): 11.
15. Dyson, J. K., Anstee, Q. M. and McPherson, S. 2014. Non-alcoholic fatty liver disease: a practical approach to diagnosis and staging. *Frontline Gastroenterology*, *5*(3): 211-218.
16. Thong, V. D. and Quynh, B. T. H. 2021. Correlation of serum transaminase levels with liver fibrosis assessed by transient elastography in vietnamese patients with nonalcoholic fatty liver disease. *International Journal of General Medicine*, 4(11): 1349-1355.
17. Sanyal, D., Mukherjee, P., Raychaudhuri, M., Ghosh, S., Mukherjee, S. and Chowdhury, S. 2015. Profile of liver enzymes in non-alcoholic fatty liver disease in patients with impaired glucose tolerance and newly detected untreated type 2 diabetes. *Indian Journal of Endocrinology and Metabolism*, *19*(5): 597-601.
18. Sinn, D. H., Kang, D., Guallar, E., Hong, Y. S., Cho, J. and Gwak, G. Y. 2022. Modest alcohol intake and mortality in individuals with elevated alanine aminotransferase levels: a nationwide cohort study. *BMC Medicine*, *20*(1): 18.
19. Hall, P. and Cash, J. 2012. What is the real function of the liver ‘function’tests. *The Ulster Medical Journal*, *81*(1): 30.
20. Huang, X. J., Choi, Y. K., Im, H. S., Yarimaga, O., Yoon, E. and Kim, H. S. 2006. Aspartate aminotransferase (AST/GOT) and alanine aminotransferase (ALT/GPT) detection techniques. *Sensors*, *6*(7): 756-782.
21. Nath, P., Kumar, R., Mallick, B., Das, S., Anand, A., Panigrahi, S. C. and Acharya, S. 2022. Effect of nonalcoholic fatty liver disease (NAFLD) on COVID-19: a single-center study of 3983 patients with review of literature. *Cureus*, *14*(7): e26683.
22. Nyblom, H. B. U. B. J., Berggren, U., Balldin, J. and Olsson, R. 2004. High AST/ALT ratio may indicate advanced alcoholic liver disease rather than heavy drinking. *Alcohol and Alcoholism*, *39*(4): 336-339.
23. Chen, S. C. C., Tsai, S. P., Jhao, J. Y., Jiang, W. K., Tsao, C. K. and Chang, L. Y. 2017. Liver fat, hepatic enzymes, alkaline phosphatase and the risk of incident type 2 diabetes: a prospective study of 132,377 adults. *Scientific Reports*, *7*(1): 4649.
24. Sheng, G., Peng, N., Hu, C., Zhong, L., Zhong, M. and Zou, Y. 2021. The albumin-to-alkaline phosphatase ratio as an independent predictor of future non-alcoholic fatty liver disease in a 5-year longitudinal cohort study of a non-obese Chinese population. *Lipids in Health and Disease*, *20*: 1-10.
25. López-Amador, N., Nolasco-Hipolito, C., Rojas-Jimeno, M. D. and Carvajal-Zarrabal, O. 2017. Liver enzymes in patients diagnosed with non-alcoholic fatty liver disease (NAFLD) in Veracruz: a comparative analysis with the literature. *Clinical Investigation*, *7*(1): 25-32.
26. Ali, A. H., Petroski, G. F., Diaz-Arias, A. A., Al Juboori, A., Wheeler, A. A., Ganga, R. R. and Ibdah, J. A. 2021. A model incorporating serum alkaline phosphatase for prediction of liver fibrosis in adults with obesity and nonalcoholic fatty liver disease. *Journal of Clinical Medicine*, *10*(15): 3311.
27. Sohrabi, M., Aghapour, S., Khoonsari, M., Ajdarkosh, H., Nobakht, H., Zamani, F. and Nikkhah, M. 2023. Serum Alkaline Phosphate Level Associates with Metabolic Syndrome Components Regardless of Non-Alcoholic Fatty Liver; A Population-Based Study in Northern Iran. *Middle East Journal of Digestive Diseases*, *15*(3): 175-179.
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