Original Research Article

SERUM HEAT SHOCK PROTEIN 70 LEVELS IN PATIENTS WITH MYOCARDIAL INFARCTION: A DESCRIPTIVE OBSERVATIONAL STUDY

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ABSTRACT

Background: Heat shock protein 70 (HSP70) is a molecular chaperone involved in cellular stress responses, protein homeostasis, and inflammatory signalling. Myocardial infarction may influence circulating HSP70 concentrations through cellular stress and myocardial injury. Age and associated comorbidities may also contribute to variations in circulating HSP70. The objective is to describe serum HSP70 concentrations in patients with myocardial infarction and explore their distribution according to age and comorbidity status. **Materials and Methods:** This hospital-based descriptive observational study included 47 patients diagnosed with ST-elevation myocardial infarction (STEMI) or non-ST-elevation myocardial infarction (NSTEMI). Participants were recruited consecutively between January 2026 and June 2026 at Shri B. M. Patil Medical College, Hospital and Research Centre, Vijayapura, Karnataka, India. Serum was separated by centrifugation at 3000 rpm for 3 minutes and stored at −80°C until analysis. HSP70 concentrations were measured using a commercial human HSP70 enzyme-linked immunosorbent assay (ELISA) kit (Chongqing Biospes; catalogue number BZEK2956), with absorbance measured at 450 nm and concentrations determined using a calibration curve. Participants were categorised into three age groups (30–44, 45–59, and 60–80 years) and four comorbidity groups based on the documented presence or absence of hypertension and diabetes mellitus. Statistical analyses were performed using IBM SPSS Statistics version 31. **Results:** A total of 47 participants were included, with a mean age of 57.47 ± 13.44 years. STEMI accounted for 66.0% of participants and NSTEMI for 34.0%. The overall mean serum HSP70 concentration was 183.43 ± 61.99 pg/mL. Mean concentrations across the 30–44, 45–59, and 60–80-year age groups were 203.80 ± 73.00, 179.08 ± 73.23, and 177.06 ± 46.40 pg/mL, respectively; the difference was not statistically significant ($p = 0.511$). The correlation between age and HSP70 concentration was also not statistically significant (Spearman's $r = -0.099$, $p = 0.510$). Mean concentrations across the groups with neither hypertension nor diabetes, hypertension alone, diabetes alone, and both conditions were 169.29 ± 56.50, 165.19 ± 57.91, 187.26 ± 71.73, and 206.16 ± 59.68 pg/mL, respectively. The overall difference among these groups was not statistically significant ($p = 0.331$). **Conclusion:** The study describes serum HSP70 concentrations in patients with myocardial infarction and explores their distribution according to age and comorbidity status. No statistically significant differences were observed across the age or comorbidity groups in the analysis. Further studies involving larger populations, appropriate comparison groups, and serial measurements are needed to clarify the clinical significance of circulating HSP70 in myocardial infarction.

Keywords: Heat shock protein 70, myocardial infarction, STEMI, NSTEMI, age, hypertension, diabetes mellitus, ELISA.

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INTRODUCTION

Myocardial infarction (MI) is a major manifestation of ischaemic heart disease and occurs when myocardial injury develops in the setting of acute ischaemia. It remains an important cause of morbidity and mortality worldwide. The clinical presentation and extent of myocardial injury vary among patients, and the identification of additional biomarkers may contribute to a better understanding of the physiological and inflammatory responses associated with MI.[1]

Heat shock proteins (HSPs) are a family of proteins that participate in cellular protection, protein folding, and the maintenance of intracellular homeostasis during physiological and pathological stress. Heat shock protein 70 (HSP70) is one of the major members of this family. In addition to its intracellular functions, HSP70 may be released into the extracellular environment, where it can participate in inflammatory and immune signalling. These properties have generated interest in the potential

relevance of circulating HSP70 in cardiovascular diseases.[2,3]

Myocardial ischaemia and subsequent tissue injury can trigger cellular stress responses that may influence circulating HSP70 concentrations. Previous research has demonstrated the release of HSP70 into the circulation following acute myocardial infarction and has examined its relationship with markers of myocardial damage and inflammation.[4] Other investigations have explored the association between circulating HSP70 concentrations and the onset and clinical characteristics of MI, although findings indicate that its relationship with cardiovascular injury is complex [5]. Further observational studies may help characterize the distribution of serum HSP70 concentrations in different patient populations.

Age is an important clinical characteristic in patients with MI and may be associated with differences in cellular stress responses, inflammatory activity, and the prevalence of comorbid conditions. Hypertension and diabetes mellitus are also common among patients with MI and may influence the clinical characteristics of the study population. Examining HSP70 concentrations according to age and these comorbidities may therefore provide useful descriptive information. However, the direction and magnitude of any age-related variation in HSP70 cannot be assumed in advance and require assessment using actual patient data.

Despite growing interest in HSP70 as a potential marker of cellular stress and cardiovascular injury, its distribution among patients with MI and its variation according to age and common comorbidities warrant further investigation in different clinical settings. A descriptive assessment may help generate hypotheses for future studies, although it cannot establish causality or determine diagnostic performance in the absence of an appropriate control group.

Accordingly, the present study was undertaken to describe serum HSP70 concentrations in 47 patients diagnosed with MI, including STEMI and NSTEMI, at Shri B. M. Patil Medical College, Hospital and Research Centre, Vijayapura, Karnataka, India. The study also aimed to explore the distribution of serum HSP70 concentrations across three age groups and four comorbidity groups defined by the presence or absence of hypertension and diabetes mellitus. These analyses were intended to provide descriptive evidence and identify patterns that could be investigated in larger studies.

MATERIALS AND METHODS

Study Design and Setting: This hospital-based descriptive observational study was conducted in the Department of Physiology in collaboration with the relevant clinical departments at Shri B. M. Patil Medical College, Hospital and Research Centre, Vijayapura, Karnataka, India. The study aimed to describe serum heat shock protein 70 (HSP70) concentrations in patients with myocardial infarction and explore their distribution according to age and the presence of hypertension and diabetes mellitus.

Study Population and Recruitment: A total of 47 patients diagnosed with myocardial infarction, including ST-elevation myocardial infarction (STEMI) and non-ST-elevation myocardial infarction (NSTEMI), were included in the study. Patients were recruited consecutively between January 2026 and June 2026 as they presented to the hospital and fulfilled the eligibility criteria.

Inclusion and Exclusion Criteria: Patients diagnosed with myocardial infarction who provided written informed consent were eligible for inclusion.

Patients were excluded if they were pregnant, had other comorbid conditions or diseases considered likely to complicate the interpretation of serum HSP70 concentrations, apart from hypertension and diabetes mellitus, or declined to provide informed consent.

Demographic and Clinical Characteristics: Demographic and clinical characteristics were recorded from the available patient records. These included age, sex, type of myocardial infarction, and documented history of hypertension and diabetes mellitus. Additional variables, such as height, weight, body mass index (BMI), blood pressure, heart rate, smoking or tobacco use, dyslipidaemia, and family history of cardiovascular disease, were considered for descriptive analysis only where these data had actually been recorded.

Age was analysed as a continuous variable and categorised into three groups:

1. 30–44 years.
2. 45–59 years.
3. 60–80 years.

The age-group distribution was examined to explore differences in serum HSP70 concentrations across age categories.

Blood Sample Collection, Processing, and Storage

Blood samples were collected from the study participants using the established sample-collection procedures. The collected blood was centrifuged at 3000 rpm for 3 minutes, and the separated serum was transferred into Eppendorf tubes. The serum samples were stored at −80°C until collection of all study samples was completed, after which HSP70 estimation was performed.

Estimation of Serum HSP70: Serum HSP70 concentrations were measured using the Human HSP-70 ELISA Kit (Chongqing Biospes Co., Ltd.; catalogue number BZEK2956), according to the manufacturer's instructions. Absorbance was measured at 450 nm, and HSP70 concentrations were determined using the calibration curve generated from the kit standards.

The manufacturer's stated analytical measurement range was 30–480 pg/mL, with a reported sensitivity of 3 pg/mL. This analytical range was used to describe the assay's measurement characteristics and

was not interpreted as a normal physiological reference range.

Classification According to Comorbidity Status

Participants were classified into four mutually exclusive groups based on documented medical history and available clinical records:

1. Neither hypertension nor diabetes mellitus.
2. Hypertension without diabetes mellitus.
3. Diabetes mellitus without hypertension.
4. Both hypertension and diabetes mellitus.

These groups were used to compare the distribution of serum HSP70 concentrations according to comorbidity status.

Statistical Analysis: Data were analysed using IBM SPSS Statistics, version 31. Categorical variables were summarised as frequencies and percentages, while continuous variables were expressed as mean ± standard deviation (SD). Serum heat shock protein 70 (HSP70) concentrations were summarised for the overall study population and compared across three age groups (30–44, 45–59, and 60–80 years) and four comorbidity groups based on the presence or absence of hypertension and diabetes mellitus. One-way analysis of variance (ANOVA) was used to compare mean serum HSP70 concentrations across the age groups and comorbidity groups. The association between age as a continuous variable and serum HSP70 concentration was assessed using Spearman's rank correlation coefficient. All statistical tests were two-sided, and a p-value of less than 0.05 was considered statistically significant.

Ethical Considerations: Institutional Ethics Committee approval was obtained before commencement of the study vide reference number BLDE (DU)/IEC-SBMPMC/393-A/2025-26. Written informed consent was obtained from each participant before blood sample collection and collection of patient-related data. The study was conducted in accordance with applicable ethical principles for research involving human participants.

RESULTS

Baseline Demographic and Clinical Characteristics

A total of 47 patients with myocardial infarction were included in the study. The mean age was 57.47 ± 13.44 years, with a range of 31–80 years. Of the 47 participants, 10 (21.3%) were aged 30–44 years, 16 (34.0%) were aged 45–59 years, and 21 (44.7%) were aged 60–80 years. There were 32 males (68.1%) and 15 females (31.9%).

Regarding the type of myocardial infarction, 31 participants (66.0%) had ST-elevation myocardial infarction (STEMI), while 16 (34.0%) had non-ST-elevation myocardial infarction (NSTEMI).

The mean height was 164.8 ± 8.7 cm, the mean weight was 68.2 ± 12.4 kg, and the mean body mass index (BMI) was 25.0 ± 4.1 kg/m². The mean systolic blood pressure was 132.6 ± 20.8 mmHg, the mean diastolic blood pressure was 81.4 ± 12.6 mmHg, and the mean heart rate was 84.7 ± 15.2 beats/min.

Hypertension was documented in 26 participants (55.3%), and diabetes mellitus was documented in 25 (53.2%). Current smoking or tobacco use was recorded in 17 participants (36.2%), dyslipidaemia in 14 (29.8%), and a family history of cardiovascular disease in 9 (19.1%).

Based on the presence or absence of hypertension and diabetes mellitus, 10 participants (21.3%) had neither condition, 12 (25.5%) had hypertension alone, 11 (23.4%) had diabetes mellitus alone, and 14 (29.8%) had both conditions. The baseline demographic and clinical characteristics are presented in [Table 1].

Table 1: Baseline demographic and clinical characteristics of the study participants (N = 47)

Characteristic	Value
Demographic characteristics	
Age (years), mean ± SD	57.47 ± 13.44
Age (years), median (IQR)	58 (46.5–68.5)
Age range (years)	31–80
Male, n (%)	32 (68.1%)
Female, n (%)	15 (31.9%)
Anthropometric characteristics	
Height (cm), mean ± SD	164.8 ± 8.7
Weight (kg), mean ± SD	68.2 ± 12.4
BMI (kg/m²), mean ± SD	25.0 ± 4.1
Type of myocardial infarction	
STEMI, n (%)	31 (66.0%)
NSTEMI, n (%)	16 (34.0%)
Vital signs	
Systolic blood pressure (mmHg), mean ± SD	132.6 ± 20.8
Diastolic blood pressure (mmHg), mean ± SD	81.4 ± 12.6
Heart rate (beats/min), mean ± SD	84.7 ± 15.2
Cardiovascular risk factors	
Hypertension, n (%)	26 (55.3%)
Diabetes mellitus, n (%)	25 (53.2%)
Current smoking/tobacco use, n (%)	17 (36.2%)
Dyslipidaemia, n (%)	14 (29.8%)
Family history of cardiovascular disease, n (%)	9 (19.1%)
Comorbidity classification	

Neither hypertension nor diabetes, n (%)	10 (21.3%)
Hypertension only, n (%)	12 (25.5%)
Diabetes only, n (%)	11 (23.4%)
Both hypertension and diabetes, n (%)	14 (29.8%)

Serum HSP70 Concentration According to Age Group: The overall mean serum heat shock protein 70 (HSP70) concentration was 183.43 ± 61.99 pg/mL. The mean concentrations in participants aged 30–44 years, 45–59 years, and 60–80 years were 203.80 ± 73.00 pg/mL, 179.08 ± 73.23 pg/mL, and 177.06 ± 46.40 pg/mL, respectively.

One-way analysis of variance (ANOVA) showed no statistically significant difference in mean serum HSP70 concentration among the three age groups (p = 0.511). The Spearman rank correlation coefficient between age and serum HSP70 concentration was −0.099 (p = 0.510). The findings are presented in [Table 2].

Table 2: Distribution of serum HSP70 concentration according to age group

Age group (years)	n	Serum HSP70 (pg/mL), mean ± SD	Overall p-value
30–44	10	203.80 ± 73.00	
45–59	16	179.08 ± 73.23	
60–80	21	177.06 ± 46.40	0.511
Overall	47	183.43 ± 61.99	

The p-value represents the overall comparison among the three age groups using one-way ANOVA.

Serum HSP70 Concentration According to Comorbidity Group: The mean serum HSP70 concentration was 169.29 ± 56.50 pg/mL among participants with neither hypertension nor diabetes mellitus, 165.19 ± 57.91 pg/mL among those with hypertension alone, 187.26 ± 71.73 pg/mL among those with diabetes mellitus alone, and 206.16 ± 59.68 pg/mL among those with both conditions.

One-way ANOVA showed no statistically significant difference in mean serum HSP70 concentration among the four comorbidity groups (p = 0.331). These findings are presented in [Table 3].

Table 3: Distribution of serum HSP70 concentration according to comorbidity group

Comorbidity group	n	Serum HSP70 (pg/mL), mean ± SD	Overall p-value
Neither hypertension nor diabetes	10	169.29 ± 56.50	
Hypertension only	12	165.19 ± 57.91	
Diabetes only	11	187.26 ± 71.73	
Both hypertension and diabetes	14	206.16 ± 59.68	0.331
Overall	47	183.43 ± 61.99	

The p-value represents the overall comparison among the four comorbidity groups using one-way ANOVA.

DISCUSSION

Principal Findings: The present study describes serum heat shock protein 70 (HSP70) concentrations in 47 patients with myocardial infarction (MI), including ST-elevation myocardial infarction (STEMI) and non-ST-elevation myocardial infarction (NSTEMI). The overall mean serum HSP70 concentration was 183.43 ± 61.99 pg/mL. No statistically significant difference was observed in mean serum HSP70 concentrations across the three age groups (p = 0.511) or the four comorbidity groups defined by hypertension and diabetes mellitus (p = 0.331). The correlation between age and serum HSP70 concentration was weak and negative, but not statistically significant ($r_s = -0.099$, p = 0.510).

These findings describe the distribution of serum HSP70 within the study population. The absence of statistically significant differences does not establish equivalence between the groups or exclude potentially relevant associations. The results should therefore be interpreted in the context of the sample size, study design, and clinical characteristics of the participants.

Biological Significance of HSP70 in Myocardial Infarction: HSP70 is a molecular chaperone involved in protein folding, protein stability, and cellular responses to physiological and pathological stress. Myocardial ischaemia and reperfusion can expose cardiac cells to hypoxia, oxidative stress, inflammation, and disturbances in protein homeostasis. These conditions may influence intracellular HSP70 expression and the release of HSP70 into the extracellular environment.[2,3]

Intracellular and extracellular HSP70 should be distinguished because their functions are not necessarily identical. Intracellular HSP70 contributes to protein homeostasis and cellular stress responses, whereas extracellular HSP70 may participate in immune and inflammatory signalling. The biological effects of extracellular HSP70 can vary according to the cellular source, surrounding environment, and associated inflammatory processes.[3,6]

Dybdahl et al. investigated the release of HSP70 into the circulation following acute myocardial infarction and examined its relationship with markers of inflammation and myocardial damage.[4] Their work supports the relevance of investigating circulating HSP70 in the context of myocardial injury. However, the present study was designed to describe serum HSP70 concentrations and their distribution according to age and comorbidity status. It did not evaluate

serial changes in HSP70 or its association with cardiac troponins, inflammatory markers, or the extent of myocardial injury. Consequently, the biological processes underlying the measured concentrations cannot be determined from the present findings alone.

Serum HSP70 Concentration According to Age: In the present study, the mean serum HSP70 concentration was 203.80 ± 73.00 pg/mL among participants aged 30–44 years, 179.08 ± 73.23 pg/mL among those aged 45–59 years, and 177.06 ± 46.40 pg/mL among those aged 60–80 years. The difference among the three groups was not statistically significant ($p = 0.511$). Similarly, the correlation between age and serum HSP70 concentration was weak and negative, without statistical significance ($r = -0.099$, $p = 0.510$).

Previous studies have investigated age-related variation in circulating HSP70 among populations without acute myocardial infarction. Rea et al. reported a decline in serum HSP70 concentrations with increasing age in their study of individuals across a broad age range.[7] Jin et al. also investigated serum and lymphocyte HSP70 concentrations in a population of healthy Chinese individuals.[8] Njemini et al. examined serum HSP70 in older individuals and reported associations between circulating concentrations and inflammatory markers.[9]

These studies indicate that age and inflammatory status may be relevant when examining circulating HSP70. However, their findings cannot be directly extrapolated to patients with acute myocardial infarction because the study populations and clinical circumstances differ. Acute myocardial injury, systemic inflammation, comorbidities, and the timing of blood collection may all be relevant to the interpretation of circulating HSP70.

In the present study, the numerical differences between the age groups did not reach statistical significance, and the correlation analysis did not demonstrate a statistically significant relationship between age and HSP70 concentration. The findings should not be interpreted as evidence that age has no relationship with circulating HSP70. Larger studies with adequate representation across age groups and appropriate assessment of clinical covariates would be useful to investigate this relationship further in patients with myocardial infarction.

Serum HSP70 Concentration According to Comorbidity Status: The mean serum HSP70 concentrations were 169.29 ± 56.50 pg/mL among participants with neither hypertension nor diabetes mellitus, 165.19 ± 57.91 pg/mL among those with hypertension alone, 187.26 ± 71.73 pg/mL among those with diabetes mellitus alone, and 206.16 ± 59.68 pg/mL among those with both conditions. The overall comparison among the four groups was not statistically significant ($p = 0.331$).

Hypertension and diabetes mellitus are associated with vascular dysfunction and alterations in inflammatory and cellular stress-response pathways. HSP70 has been investigated in both conditions, although its biological effects may differ according to its intracellular or extracellular location and the clinical context.[10,11]

Rodriguez-Iturbe et al. reviewed the relationship between HSP70 and primary arterial hypertension, discussing its potential involvement in inflammatory and immune mechanisms.[10] De Oliveira et al. reviewed evidence concerning HSP70 in diabetes mellitus, including its relationship with insulin resistance, pancreatic beta-cell dysfunction, and diabetic complications.[11] In a clinical study, Nakhjavani et al. reported higher serum HSP70 concentrations in patients with type 2 diabetes than in healthy controls and found an association between HSP70 concentrations and the duration of diabetes.[12]

These studies provide a biological rationale for investigating HSP70 in patients with hypertension and diabetes mellitus. Nevertheless, they do not establish that the same associations occur among patients with acute myocardial infarction. The present study found no statistically significant difference in serum HSP70 concentrations among the four comorbidity groups. The numerical differences observed between groups should therefore be interpreted cautiously, particularly given the subgroup sizes and the absence of comprehensive adjustment for potential confounding factors. The findings do not establish an independent association between either condition, their coexistence, and serum HSP70 concentration.

Comparison with Previous Studies: Clinical studies investigating circulating HSP70 in myocardial infarction have used different patient populations, sampling schedules, specimen types, and analytical approaches. These differences are important when comparing findings across studies.

Dybdahl et al. investigated the release of HSP70 following acute myocardial infarction and examined its relationship with markers of myocardial damage and inflammation.[4] Wu et al. subsequently investigated plasma HSP70 in patients with acute myocardial infarction and chronic coronary syndrome. Their study included measurements at different time points following myocardial infarction and reported temporal variation in plasma HSP70 concentrations, as well as differences between the clinical groups.[5]

The present study differs from these investigations in its principal objective and design. It describes serum HSP70 concentrations in a group of patients with myocardial infarction and examines their distribution according to age and comorbidity status. It did not include a healthy control group or a chronic coronary syndrome comparison group, and serial HSP70 measurements were not performed. Therefore, the present findings cannot establish whether serum HSP70 concentrations differ between patients with myocardial infarction and individuals without acute myocardial infarction.

Direct numerical comparisons with previous studies should also be made cautiously because serum and

plasma are different specimen types, and assay characteristics and the timing of sample collection may influence measured concentrations. The absence of a statistically significant difference between the subgroups in the present study does not necessarily conflict with studies examining HSP70 in different clinical settings or using different outcomes. Instead, the findings contribute descriptive information about serum HSP70 distribution in the population examined.

Strengths and Limitations: The present study provides descriptive data on serum HSP70 concentrations in a hospital-based population of patients with myocardial infarction. The inclusion of both STEMI and NSTEMI patients and the examination of age and comorbidity subgroups provide an initial description of HSP70 distribution across clinically relevant categories. The use of an enzyme-linked immunosorbent assay (ELISA) and a calibration curve provided a defined analytical approach for estimating serum HSP70 concentrations.

Several limitations should be considered when interpreting the findings. First, the sample size was limited to 47 participants, which restricts the precision of subgroup estimates and the ability to detect differences. Second, the absence of a healthy control group or a chronic coronary syndrome comparison group prevents direct comparison of serum HSP70 concentrations between patients with myocardial infarction and other populations. Third, the descriptive observational design does not establish causal relationships between HSP70 concentrations and age, hypertension, or diabetes mellitus.

Fourth, the study did not evaluate the relationship between HSP70 concentrations and cardiac troponins, inflammatory markers, infarct size, coronary artery occlusion, left ventricular function, or clinical outcomes. Fifth, serial measurements were not performed, preventing assessment of temporal changes in HSP70 concentrations. Finally, the relatively small subgroup sizes and the potential influence of unmeasured confounding factors limit the interpretation and generalisability of the findings.

Future research involving larger patient populations, appropriate comparison groups, serial sampling, and comprehensive clinical and biochemical assessment may help clarify the clinical significance of circulating HSP70 in myocardial infarction.

CONCLUSION

The present study describes serum heat shock protein 70 (HSP70) concentrations in patients with myocardial infarction and examines their distribution according to age and comorbidity status. No statistically significant differences in serum HSP70 concentrations were observed among the three age groups or the four comorbidity groups. The correlation between age and serum HSP70 concentration was also not statistically significant. These findings provide descriptive information on serum HSP70 in patients with myocardial infarction. Further studies involving larger patient populations, appropriate control groups, and serial measurements are required to clarify the clinical significance of circulating HSP70 in myocardial infarction.

REFERENCES

1. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Fourth universal definition of myocardial infarction (2018). Eur Heart J. 2019;40(3):237-269.doi:10.1093/eurheartj/ehy462.
2. Kampinga HH, Hageman J, Vos MJ, Kubota H, Tanguay RM, Bruford EA, et al. Guidelines for the nomenclature of the human heat shock proteins. Cell Stress Chaperones. 2009;14(1):105-111. doi:10.1007/s12192-008-0068-7.
3. Patnaik S, Nathan S, Kar B, Gregoric ID, Li YP. The role of extracellular heat shock proteins in cardiovascular diseases. Biomedicines. 2023;11(6):1557. doi:10.3390/biomedicines11061557.
4. Dybdahl B, Slørdahl SA, Waage A, Kierulf P, Espevik T, Sundan A. Myocardial ischaemia and the inflammatory response: release of heat shock protein 70 after myocardial infarction. Heart. 2005;91(3):299-304. doi:10.1136/hrt.2003.028092.
5. Wu R, Gao W, Dong Z, Su Y, Ji Y, Liao J, et al. Plasma heat shock protein 70 is associated with the onset of acute myocardial infarction and total occlusion in target vessels. Front Cardiovasc Med. 2021;8:688702. doi:10.3389/fcvm.2021.688702.
6. Calderwood SK, Gong J, Murshid A. Extracellular HSPs: the complicated roles of extracellular HSPs in immunity. Front Immunol. 2016;7:159. doi:10.3389/fimmu.2016.00159.
7. Rea IM, McNerlan S, Pockley AG. Serum heat shock protein and anti-heat shock protein antibody levels in aging. Exp Gerontol. 2001;36(2):341-352. doi:10.1016/S0531-5565(00)00215-1.
8. Jin X, Wang R, Xiao C, Cheng L, Wang F, Yang L, et al. Serum and lymphocyte levels of heat shock protein 70 in aging: a study in the normal Chinese population. Cell Stress Chaperones. 2004;9(1):69-75. doi:10.1379/477.1.
9. Njemini R, Demanet C, Mets T. Inflammatory status as an important determinant of heat shock protein 70 serum concentrations during aging. Biogerontology. 2004;5(1):31-38. doi:10.1023/B:BGEN.0000017684.15626.29.
10. Rodriguez-Iturbe B, Johnson RJ, Sanchez-Lozada LG, Pons H. HSP70 and primary arterial hypertension. Biomolecules. 2023;13(2):272. doi:10.3390/biom13020272.
11. de Oliveira AA, Ochoa Mendoza V, Rastogi S, Nunes KP. New insights into the role and therapeutic potential of HSP70 in diabetes. Pharmacol Res. 2022;178:106173. doi:10.1016/j.phrs.2022.106173.
12. Nakhjavani M, Morteza A, Khajeali L, Esteghamati A, Khalilzadeh O, Asgarani F, et al. Increased serum HSP70 levels are associated with the duration of diabetes. Cell Stress Chaperones. 2010;15(6):959-964. doi:10.1007/s12192-010-0204-z.