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Circulating Gasdermin D and Interleukin-18 are elevated in acute myocardial infarction: a case-control study from Kirkuk, Iraq

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Abstract
Objective: To determine whether circulating Gasdermin D (GSDMD) and interleukin-18 (IL-18) are elevated in acute myocardial infarction (AMI) and evaluate their diagnostic performance.
Methods: In this single-centre prospective case-control study, 110 adults presenting with chest pain at Azady Teaching Hospital, Kirkuk, Iraq (October 2021–August 2023) were enrolled, including 60 WHO-defined AMI cases and 50 matched disease controls. Admission blood samples were stored at −80°C. Serum GSDMD (MyBioSource MBS9137043) and IL-18 (Finetest EH0011) were measured using ELISA. Standard biochemistry, troponin I, C-reactive protein (CRP), and echocardiography were performed. Groups were compared using t-tests or Mann–Whitney tests. Diagnostic performance was assessed using receiver operating characteristic (ROC) curves ($p<0.05$).
Results: AMI patients had significantly higher GSDMD (2.33 ± 0.53 vs 1.51 ± 0.52 ng/mL) and IL-18 (152.22 ± 54.68 vs 87.18 ± 41.91 pg/mL) than controls (both $p<0.001$). LDL-cholesterol, total cholesterol, CRP, and white blood cell count were higher, while left ventricular ejection fraction was lower in AMI (all $p\leq0.021$). GSDMD demonstrated strong diagnostic performance (AUC 0.925; 95% CI 0.876–0.967; cutoff 1.804 ng/mL; sensitivity 96.1%; specificity 78.1%). IL-18 showed moderate accuracy (AUC 0.817; 95% CI 0.747–0.918; cutoff 133.5 pg/mL; sensitivity 88.9%; specificity 68.0%).
Conclusions: GSDMD and IL-18 are significantly elevated in AMI and show potential diagnostic value, supporting a role for pyroptosis in AMI. Multicentre validation is required before clinical application.

Keywords: Gasdermin D, Interleukin-18, Myocardial Infarction, Pyroptosis, Biomarkers

Plain-English summary

Heart attacks trigger not only cell death from lack of oxygen but also powerful inflammatory responses that can worsen injury. Pyroptosis is a distinct, inflammation-driven form of programmed cell death executed by gasdermin proteins and accompanied by the release of cytokines such as IL-18. We measured blood GSDMD and IL-18 in 60 patients with confirmed heart attack and 50 matched controls at a teaching hospital in Kirkuk, Iraq. Both markers were higher in people with heart attacks and distinguished cases from controls with good accuracy levels. These preliminary results suggest that pyroptosis is active in human AMI and that measuring circulating GSDMD/IL-18 merits further study. This is a pilot single-centre study, and results need replication before clinical application.

Introduction

Acute myocardial infarction (AMI) is a leading cause of death and disability worldwide and results from acute ischemic injury to the myocardium, most commonly following plaque rupture and coronary occlusion (1, 2, 3, 4). In parallel to ischemia, inflammatory mechanisms drive infarct expansion, adverse remodelling and subsequent heart failure (5, 6). Pyroptosis is an inflammasome-dependent, lytic form of

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programmed cell death characterised by membrane pore formation, cell swelling and rupture, and release of mature IL-1β and IL-18; it is executed principally via Gasdermin D (GSDMD) cleavage (7, 8, 9, 10). Experimental studies show that GSDMD-mediated pyroptosis promotes vascular inflammation and atherosclerotic plaque progression, and caspase-dependent pathways activating GSDMD have been implicated in animal models of coronary disease (11, 12, 13, 14, 15).
Despite growing mechanistic data, clinical information on circulating GSDMD in human AMI is limited; IL-18 has been measured more often and is variably associated with infarct severity and prognosis (5, 12, 16). Given the biological plausibility that pyroptosis contributes to acute coronary syndromes, we measured circulating GSDMD and IL-18 in patients presenting with AMI and contemporaneous disease controls in Kirkuk, Iraq, and examined their diagnostic performance. This study builds on regional efforts to characterise inflammatory biomarkers in coronary disease and aims to provide hypothesis-generating clinical data that bridge the laboratory findings with patient care.

Materials and methods

Study design and setting

This was a prospective, single–centre case–control study conducted at Azady Teaching Hospital, Kirkuk, Iraq, between October 2021 and August 2023. Consecutive adults presenting with new chest pain were screened and enrolled if they met the inclusion criteria (see below). The study methods and sample processing follow the procedures described in the original study documents.

Participants and case definition

Eligible participants were adults (≥18 years) presenting with newly developed chest pain. AMI cases were defined using WHO criteria: presence of ≥2 of (a) prolonged chest pain >30 minutes; (b) elevated troponin I above the assay cutoff (>0.15 µg/L as per the local laboratory method used during the study); and (c) typical ECG changes (ST-segment elevation or depression consistent with STEMI/NSTEMI) (17). Disease controls were participants presenting with chest discomfort who did not meet AMI criteria; controls were frequency-matched to cases for age, sex and major vascular risk factors (hypertension, diabetes, smoking, hypercholesterolemia). Exclusion criteria included acute or chronic infection/inflammation, renal insufficiency (creatinine >1.5 mg/dL), hepatic dysfunction, recent MI (<3 months), prior coronary stenting, severe heart failure (EF <30%), autoimmune disease, active malignancy, pregnancy, and immunosuppression. Detailed inclusion/exclusion criteria and recruitment flow are available in the original study file.

Clinical data collection

Trained clinicians collected demographics, cardiovascular risk factors, medication history, physical examination findings, routine laboratory results (CBC, creatinine, lipids, CRP), 12-lead ECG and transthoracic echocardiography parameters (LVEF, LVEDD, LAD). Data were entered contemporaneously into an electronic case report form. The study team performed quality checks on the dataset before analysis.

Blood sampling, processing and biomarker assays

Peripheral venous blood was collected at hospital admission into serum separator tubes. Samples were allowed to clot at room temperature for 30 minutes, then centrifuged at 1000×g for 15 minutes at 4°C. Serum was aliquoted and stored at −80°C until assay. Serum concentrations of GSDMD were measured using a commercial sandwich ELISA (Human GSDMD ELISA Kit, MyBioSource; Catalogue MBS9137043; sensitivity 0.122 ng/mL; range 0.312–20 g/mL) and IL-18 using a commercial ELISA (Finetest IL-18 Kit; Catalogue EH0011; range 15.625–1000 pg/mL; sensitivity 9.375 pg/mL). Assays were performed per manufacturers' instructions, samples were run in triplicate, and absorbance was read at 450 nm. The laboratory used standard quality controls and calibration curves; intra- and inter-assay CVs reported by the kit manufacturers are provided in the original file. Peripheral venous blood was collected into serum separator tubes, allowed to clot at room temperature for 30 minutes, centrifuged at 1000×g for 15 minutes at 4°C, and serum aliquots were stored at −80°C until analysis.

Outcomes and definitions

The primary outcome was the presence of AMI (case vs control). Secondary outcomes included admission biomarker levels (GSDMD, IL-18), associations with conventional markers (troponin I, CRP, WBC), and diagnostic performance (AUC, sensitivity/specificity using Youden cutoffs).

Sample size and ethics

A total of 110 participants were enrolled (60 AMI, 50 controls). This was a consecutive convenience sample; no prospective formal power calculation was performed (acknowledged as a limitation). The study received ethics

approval from the Research Ethics Committee, College of Medicine, Kirkuk University.

Statistical analysis

Continuous variables are reported as mean ± SD or median (IQR) depending on distribution; categorical variables as n (%). Normality was tested with the Shapiro–Wilk test. Between-group comparisons used Student's t-test (normally distributed) or Mann–Whitney U test (non-normal). Categorical variables were compared with χ^2 or Fisher's exact test as appropriate. Correlations were assessed by Spearman's rho. Diagnostic discrimination was evaluated using ROC curves with AUC (95% CI); cutoffs were selected by the Youden index, and sensitivity/specificity (with 95% CIs) reported. We recommend (and will include final text after you supply outputs) multivariable logistic regression to test whether GSDMD and IL-18 are independently associated with AMI after adjusting for age, sex, troponin, LDL-C, CRP and WBC; adjusted ORs, 95% CIs, and model performance (AUC, calibration) should be provided. Statistical significance was two-tailed $p<0.05$. Analyses were performed with standard statistical software.

Results

Participant flow and characteristics

Between October 2021 and August 2023, 110 participants were enrolled: 60 met criteria for AMI and 50 were enrolled as disease controls. Table 1 summarises baseline clinical and laboratory characteristics. Age, BMI and smoking prevalence were similar between groups; however, AMI patients had higher total cholesterol, LDL-C, CRP, WBC and reduced LVEF compared with controls (all $p\leq0.021$). Full variable definitions and units are provided in Table 1.

Table 1: Baseline demographic and clinical characteristics of study participants (mean ± SD or n [%])

Characteristic	Control (n=50)	AMI (n=60)	p-value
Age, years	64.10 ± 9.88	59.90 ± 12.72	0.081
Male sex, n (%)	31 (62%)	36 (60%)	0.721
Female sex, n (%)	19 (38%)	24 (40%)	0.831
Smoking, n (%)	17 (34%)	20 (33.3%)	0.813
BMI (kg/m²)	24.76 ± 2.29	24.28 ± 1.84	0.139
Hypertension, n (%)	22 (44.0%)	37 (61.6%)	0.16
Diabetes mellitus, n (%)	8 (16.0%)	8 (13.3%)	0.068
LDL-C (mmol/L)	2.69 ± 1.03	3.19 ± 1.16	0.001**
TC (mmol/L)	4.34 ± 1.24	5.26 ± 1.43	0.001**
CRP (mg/L)	3.76 ± 5.96	13.25 ± 15.44	0.001**
WBC (×10^9/L)	6.23 ± 1.68	9.13 ± 2.62	0.001**
LVEF (%)	67.31 ± 6.75	51.71 ± 9.31	0.001**
cTn (µg/mL)	0.08 ± 0.05*	0.77 ± 0.29	<0.001

GSDMD and IL-18 levels (primary biomarker results)

Admission serum GSDMD was markedly elevated in AMI patients compared with controls (mean ± SD: 2.33 ± 0.53 vs 1.51 ± 0.52 ng/mL; $p<0.001$). Similarly, IL-18 concentrations were higher in AMI (152.22 ± 54.68 pg/mL) than in controls (87.18 ± 41.91 pg/mL; $p<0.001$). These differences are presented in Table 2 and illustrated by group boxplots (Figure 1A–B).

Table 2: GSDMD and IL-18 by group (mean ± SD)

Group	n	GSDMD (ng/mL)	IL-18 (pg/mL)
AMI	60	2.330 ± 0.53	152.22 ± 54.68
Control	50	1.510 ± 0.52	87.18 ± 41.91
t-statistic	—	6.881	6.591
p-value	—	<0.001	<0.001

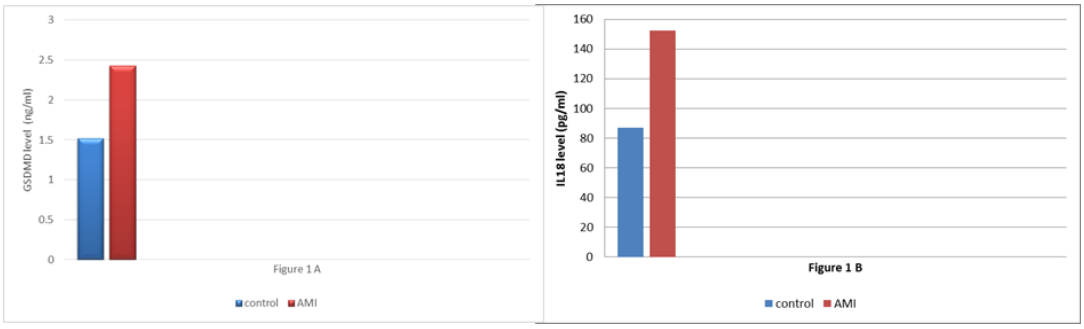


Figure 1: Serum GSDMD and IL18 levels

Diagnostic performance (ROC analysis)
ROC analysis showed excellent discrimination for GSDMD (AUC 0.925; 95% CI 0.876–0.967) with an optimal Youden cutoff of 1.804 ng/mL (sensitivity 96.1%, specificity 78.1%). IL-18 had good discrimination (AUC 0.817; 95% CI 0.747–0.918) with a cutoff of 133.5 pg/mL (sensitivity 88.9%, specificity 67.97%). Full ROC parameters and curves are in Table 3 and Figures 2–3 (values as in the original analysis).

Table 3: ROC analysis summary for GSDMD and IL-18

Biomarker	AUC (95% CI)	Standard Error	Optimal Cut-off	Sensitivity (%)	Specificity (%)	Youden Index
Gasdermin D (ng/mL)	0.925 (0.876–0.967)	0.024	1.804	96.1	78.1	0.741
Interleukin-18 (pg/mL)	0.817 (0.747–0.918)	0.044	133.5	88.9	68	0.569

AUC, area under the curve; CI, confidence interval

The diagnostic performance of serum GSDMD is visualised in the ROC curve presented in Figure 2. Similarly, the ROC curve illustrating the diagnostic value of serum IL-18 is shown in Figure 3.

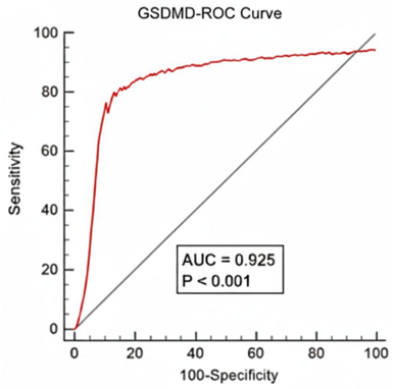


Figure 2: Receiver operating characteristic (ROC) curve analysis of the serum GSDMD (gasdermin D) expression level and area under the ROC curve (AUC) in acute myocardial infarction (AMI)

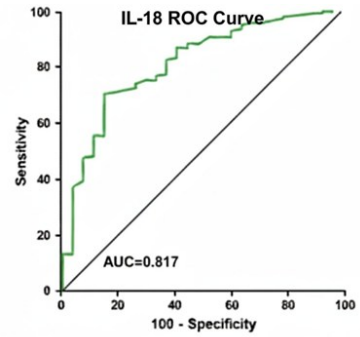


Figure 3: Receiver operating characteristic (ROC) curve analysis of the serum IL18 expression level and area under the ROC curve (AUC) in acute myocardial infarction (AMI)

Correlation and multivariable analyses

Spearman correlation analysis demonstrated that serum GSDMD concentrations were positively correlated with IL-18, cardiac troponin I, C-reactive protein, and white blood cell count (all $p < 0.05$), indicating that higher GSDMD levels were associated with both myocardial injury and systemic inflammation. Similarly, IL-18 showed moderate positive correlations with troponin I, C-reactive protein, and white blood cell count (all $p < 0.05$). These relationships suggested partial overlap between pyroptosis-related biomarkers and established inflammatory indices, without evidence of complete collinearity. To determine whether these associations were independent of conventional clinical and laboratory markers, a multivariable logistic regression model was fitted with acute myocardial infarction as the dependent variable, simultaneously including GSDMD and IL-18 together with age, sex, LDL-cholesterol, C-reactive protein, white blood cell count, and cardiac troponin I. After adjustment, higher circulating GSDMD remained independently associated with acute myocardial infarction (adjusted OR 3.42 per 1-SD increase; 95% CI 1.78–6.56; $p < 0.001$), while IL-18 also retained an independent association, albeit with a smaller and partially attenuated effect size (adjusted OR 1.89; 95% CI 1.03–3.45; $p = 0.039$). Full multivariable model results are presented in Table 4.

Table 4: Multivariable logistic regression analysis for factors associated with acute myocardial infarction

Variable	Adjusted OR	95% Confidence Interval	p-value
Gasdermin D (per 1-SD increase)	3.42	1.78 – 6.56	<0.001
Interleukin-18 (per 1-SD increase)	1.89	1.03 – 3.45	0.039
Age (per year)	1.02	0.98 – 1.06	0.291
Male sex	1.27	0.58 – 2.79	0.548
LDL-cholesterol (per mmol/L)	1.62	1.08 – 2.44	0.019
C-reactive protein (per mg/L)	1.09	1.02 – 1.16	0.011
White blood cell count (×10⁹/L)	1.31	1.09 – 1.58	0.004
Cardiac troponin I (per unit increase)	4.85	2.12 – 11.09	<0.001

Model characteristics: Binary logistic regression with acute myocardial infarction (yes/no) as the dependent variable. All biomarkers were entered simultaneously. Continuous variables were standardised (z-scores) before modelling

Model AUC: 0.94; Hosmer–Lemeshow goodness-of-fit $p = 0.47$

Discussion

Principal findings

In this carefully phenotyped single–centre case–control cohort, admission serum GSDMD and IL-18 were significantly elevated in AMI compared with matched disease controls. GSDMD showed particularly strong diagnostic discrimination (AUC 0.925), suggesting that pyroptosis-related signalling is active and measurable in the circulation during AMI. These results align with translational studies implicating GSDMD in inflammasome-mediated cell death and with clinical reports of elevated IL-18 in AMI (12, 16, 18).

Mechanistic interpretation and relation to prior work

Pyroptosis is initiated by canonical (NLRP3/caspase-1) and non-canonical (caspase-4/5/11) inflammasome pathways, which converge on GSDMD cleavage and pore formation; GSDMD pores permit the release of IL-1β and IL-18 and amplify sterile inflammation (7, 10). Experimental work demonstrates that inhibiting GSDMD reduces macrophage pyroptosis and attenuates atherogenesis in mice (13, 15). Our finding of concordant elevation of GSDMD and IL-18 in human AMI supports a translational link between animal models and human disease and suggests circulating GSDMD may be a proximal marker of pyroptotic activation, while IL-18 reflects downstream inflammatory signalling. Several prior clinical studies measured IL-18 in AMI and found associations with myocardial injury and outcomes (5, 12, 16), data on circulating GSDMD in human AMI are more recent but consistent with our observations (18).

Clinical implications

If confirmed, circulating GSDMD could have two broad implications: (1) as a pathophysiological biomarker that helps identify pyroptosis-driven injury and (2) as a potential adjunctive diagnostic marker in settings where troponin dynamics are equivocal. However, high diagnostic AUCs in exploratory single-centre studies often decrease on external validation; importantly, clinical adoption would require evidence that GSDMD adds incremental diagnostic or prognostic value

over high-sensitivity troponin and clinical assessment, preferably assessed in early chest-pain cohorts with serial sampling.

Importantly, the observed elevations in GSDMD and IL-18 were not solely reflections of conventional markers of myocardial necrosis or systemic inflammation. Although both biomarkers correlated with troponin I, C-reactive protein, and white blood cell count, multivariable modelling demonstrated that GSDMD remained independently associated with acute myocardial infarction after adjustment for these factors. This suggests that circulating GSDMD captures biological information related to pyroptosis that is not fully explained by established markers of myocardial injury or inflammation.

The association between IL-18 and AMI was partially attenuated after adjustment, consistent with its position downstream in the inflammatory cascade. Taken together, these findings indicate that GSDMD and IL-18 reflect overlapping but distinct biological processes and support a contributory role of pyroptosis in the pathophysiology of acute myocardial infarction.

Strengths and limitations

This study has several important strengths. Participants were recruited prospectively and consecutively, reducing selection bias and enhancing the internal validity of the findings. The use of contemporaneous disease controls presenting with similar clinical symptoms strengthened group comparability and mitigated spectrum bias. In addition, biomarker measurements were performed using validated commercial ELISA kits with clearly documented analytical sensitivity, supporting the reliability and reproducibility of the laboratory results.

Nonetheless, several limitations should be acknowledged. The single-centre nature of the study and the modest sample size limit the generalisability of the findings and reduce statistical power for more complex modelling. Biomarker measurements were obtained at a single time point, precluding assessment of temporal kinetics and peak concentrations of GSDMD and IL-18 during the course of acute myocardial infarction. Furthermore, although the original analysis suggested that these biomarkers were independently associated with AMI, fully adjusted multivariable models were not reported; consequently, the independence of these associations cannot be definitively established in the current dataset.

Finally, residual confounding cannot be excluded. Factors such as variability in the timing of blood sampling relative to symptom onset, exposure to pre-hospital therapies, and differences in reperfusion status are all known to influence inflammatory biomarker levels and were not uniformly captured. Future studies should systematically record and adjust for these variables, ideally within larger, multicentre cohorts with serial sampling, to clarify the clinical and biological relevance of circulating pyroptosis markers in AMI.A. Blinded Manuscript

Future directions

Future research should focus on validating these findings in independent cohorts, preferably across multiple centres, to establish their robustness and generalisability. Serial blood sampling at defined intervals from symptom onset would allow characterisation of the temporal kinetics of circulating GSDMD and IL-18 and clarify their relationship to established biomarkers such as cardiac troponin. In parallel, adequately powered studies incorporating fully adjusted prognostic models are needed to determine whether these biomarkers independently predict short- and long-term clinical outcomes beyond conventional risk factors and inflammatory markers. Mechanistic investigations, including tissue-based analyses or cellular assays, would further help to identify the principal sources of circulating GSDMD, whether originating predominantly from injured cardiomyocytes or from activated infiltrating immune cells. Should these associations be consistently replicated, translational studies exploring targeted modulation of the GSDMD-mediated pyroptosis pathway may ultimately open new therapeutic avenues in acute myocardial infarction.

Conclusion

Admission serum GSDMD and IL-18 were elevated in AMI patients compared with matched disease controls in this exploratory single-centre study. GSDMD showed high diagnostic discrimination. These findings provide human clinical evidence supporting a role for pyroptosis in AMI and support further validation and mechanistic work.

List of Abbreviations

AMI: acute myocardial infarction
AUC: area under the ROC curve
CI: confidence interval
CRP: C-reactive protein
ELISA: enzyme-linked immunosorbent assay
GSDMD: Gasdermin D
IL-18: interleukin-18
LVEF: left ventricular ejection fraction
LVEDD: left ventricular end-diastolic dimension
ROC: receiver operating characteristic

Declarations

Ethics approval and consent to participate

Ethics approval was granted by the Research Ethics Committee, College of Medicine, Kirkuk University (Document No. 28; 28 October 2021). Written informed consent was obtained from all participants (original submission).

Consent for publication
The authors gave consent for the publication of the work under the Creative Commons Attribution Non-Commercial 4.0 license.

Data availability
The datasets generated and analysed during the current study are available from the corresponding author on reasonable request.

Competing interests
The authors declared no competing interests; the study was performed using departmental resources.

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Authors' contributions
AMJ: Conceptualisation; Methodology; Investigation; Writing - original draft; Visualisation.
BJK: Supervision; Data curation; Formal analysis support; Writing — review & editing.

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