SUPPLEMENTARY DATA

Methods of the supplementary data

Acquisition of cardiovascular magnetic resonance images: Images were acquired by a phased-array body surface coil during breath holds and were triggered by electrocardiography. Cine images were acquired in two-, three-, and four-chamber views, and in short-axis views using a steady-state free precession sequence (repetition time/echo time: 2.8/1.2 ms; flip angle: 58 degrees; matrix: 256 × 300; field of view: 320 × 270 mm; slice thickness: 7 mm).[1,2] Late gadolinium enhancement imaging was performed 10–15 min after administering 0.1 mmol/kg of gadolinium diethylenetriaminepentaacetic acid (Magnograf, Juste S.A.Q.F., Spain) in the same locations as in the cine images using a segmented inversion recovery steady-state free precession sequence (repetition time/echo time: 750/1.26 ms; flip angle: 45 degrees; matrix: 256 × 184; field of view: 340 × 235 mm; slice thickness: 7 mm). Inversion time was adjusted to nullify normal myocardium.[1,2] Black blood, T2-weighted short TI inversion recovery sequences were carried out in the same short-axis view as the cine sequences, all in mid-diastole. A half-Fourier acquisition single-shot turbo spin echo multisection sequence was used (recovery time: two R-R intervals; echo time: 33 ms; inversion time: 170 ms; slice thickness: 8 mm; interslice interval: 2 mm; flip angle: 160 degrees; matrix: 256 × 151; bandwidth: 781 Hz/pixel). Additionally, a segmented turbo-spin echo sequence was obtained with one slice per breath hold (recovery time: two R-R intervals; echo time: 100 ms; inversion time: 170 ms; slice thickness: 8 mm; interslice interval: 2 mm; flip angle: 180 degrees; matrix: 256 × 146; bandwidth: 235 Hz/pixel).[1,2]

Analysis of cardiovascular magnetic resonance indices: Left ventricular (LV) ejection fraction (%), LV end-diastolic volume index (ml/m2), LV end-systolic volume index (ml/m^2), and LV mass index (g/m^2) were calculated by manual planimetry of endocardial and epicardial borders in short-axis

view cine images.[1,2] Areas showing late gadolinium enhancement were visually quantified by manual planimetry. Infarct size (% of LV mass) was assessed as the percentage of LV mass showing late gadolinium enhancement. Edema, hemorrhage, and microvascular obstruction were defined as indices of reperfusion injury. Myocardial edema was regarded as areas of high T2 signal intensity. A core of low signal intensity surrounded by an area with high signal intensity indicated myocardial hemorrhage (included in the area of myocardial edema). For all sections, only the T2-weighted sequence with the highest image quality was used to analyze edema and hemorrhage. All short axis sections were separately analyzed, and the presence of signal intensity at least 2 standard deviations greater than in a remote non-infarcted area in the same section indicated edema.[1,2] The 17-segment model was applied, after which myocardial edema (% of LV) and myocardial hemorrhage (number of segments) were manually revised. Microvascular obstruction was defined visually as the number of segments showing a lack of contrast uptake in the core of tissue showing late gadolinium enhancement.[1,2] The myocardial salvage index was calculated by subtracting the mass of infarcted myocardium from myocardium showing edema and expressed as the percentage of LV mass with myocardial edema.[1,2]

Inter-observer variability for calculation of traditional cardiovascular magnetic resonance indexes used in the present study in our laboratory has been previously reported and is less than 5%.

Three-[4,5-dimethylthiazol-2-yl]-2,5 diphenyl tetrazolium bromide (MTT) assay: This method evaluates the function of mitochondrial succinate dehydrogenase in living cells. Human coronary artery endothelial cells (HCAEC) were incubated with 10µl of 5 mg/ml stock solution of 3-[4,5-dimethylthiazol-2-yl]-2,5-diphenyl tetrazolium bromide for 2h at 37ºC. Afterwards, culture medium was removed, 100µl of dimethyl sulfoxide was added and absorbance at

550nm was determined. Cell viability was expressed as the percentage of cells treated with control serum (100% viability).

Lactate dehydrogenase (LDH) release: LDH, a cytosolic enzyme, is released to the cell culture medium upon cell membrane damage. LDH released was quantified using the CyQUANT LDH cytotoxicity Assay Kit (#C20300, Invitrogen, Thermo Fisher Scientific, Madrid, Spain) following manufacturer protocol. Absorbance was measured at 490nm and 680nm (background signal from instrument). Afterwards, the percentage of cytotoxicity was calculated using the following formula: Cytotoxicity = (compound-treated LDH activity-spontaneous LDH activity (untreated cells)/ (spontaneous LDH activity (untreated cells).

Flow cytometry: Utilization of dual selectivity with annexin-V and propidium iodide (PI) by flow cytometry is a standard procedure to evaluate apoptosis and necrosis *in vitro.*[3] Briefly, once incubated, HCAEC were double-stained for FITC-annexin-V binding and PI using Apoptosis Detection Kit (Immunostep, Spain). Early apoptotic cells were defined as Annexin V-positive and PI-negative (Annexin V-fluorescein isothiocyanate $(FITC)^{+}/PI^{-}$), while late apoptotic cells were Annexin V/PI-double-positive (Annexin $V\text{-}FITC^{+}/PI^{+}$). In contrast, necrotic cells were selected as Annexin V-negative and PI-positive (Annexin $V\text{-}FITC^{-}/PI^{+}$). Percentage of apoptotic (early and late) and necrotic cells were evaluated using a BD LSR Fortessa X-20 cytometer (Beckman Coulter, Brea, United States), and a minimum of 10 000 events were acquired.

Caspase activity assay: Caspase 3 and 8 activity was quantified using colorimetric assay kits following the manufacturer's instructions (#ab39383 and #ab39534, Abcam, United Kingdom). Briefly, HCAEC cells were lysed in cell lysis buffer and incubated with the DEVED-AFC (for caspase 3) or IETD-AFC (for caspase 8) for 2 h at 37ºC. Samples were read at 450-nm using a microtiter plate reader. Fold increase in caspase activity was determined comparing the results

of cells treated with serum isolated from ST-segment elevation myocardial infarction patients with the level of those incubated with control serum. Each experiment was performed in duplicate.

Quantitative real-time polymerase chain reaction (PCR): mRNA expression of receptor interacting serine/threonine-protein kinase (RIPK) 1 and RIPK3 as markers of necroptosis was determined by real-time PCR. RNA was extracted using RNeasy Plus Mini Kit (QIAGEN GmbH, Hilden, Germany) following the manufacturer's instructions. RNA quantity and purity were determined using the Nanodrop ND-2000 (Nanodrop, LabTech International, United Kingdom). RNA was reverse transcribed into cDNA with the High-Capacity RNA-to-cDNA Kit (Applied Biosystems, Spain). RIPK1 and RIPK3 gene expression was determined by real time Polymerase Chain Reaction using a QUANTSTUDIO 5 Applied Biosystems (Applied Biosystems, Thermo Fisher Scientific, Waltham, United States). The values of the threshold cycle (Ct) were calculated and normalized to the housekeeping gene 18S ribosomal ribonucleic acid.[4] We used specific primers pre-designed by Applied Biosystems (Thermo Fisher Scientific, Waltham, United Sttes) for analysis of human RIPK1 (Hs01041869_m1), RIPK3 (Hs00179132_m1) and the endogenous ribosomal 18S (4319413E).

TUNEL assay: This technique is employed to detect DNA fragmentation, which detects not only apoptosis activation but also non-apoptotic cells, including necrotic degenerating cells.[5,6] Terminal deoxynucleotidyl transferase catalyzes the incorporation of labelled deoxynucleotides to the free 3'-hydroxyl terminus of damaged DNA. Briefly, cells were harvested, dehydrated, and 200 000 cells were mounted on double gelatin-coated glass slides. Afterwards, HCAEC were stained using an immunohistochemical assay kit following the manufacturer's instructions (#ab206386, Abcam, Cambridge, United Kingdom). For each sample, five photographs at 20x magnification were taken in independent fields using an optical microscope, Leica DM3000

(Leica Microsystems, Wetzlar, Germany). The number of TUNEL-positive cells was morphometrically determined using Image ProPlus 7.0 software (Media Cybernetics Inc, Rockville, United States) performed blind on coded slides.

Permeability of endothelial monolayer assay: An *in vitro* vascular permeability assay (96-well) was employed (#ECM642, Merck, Burlington, United States) following the manufacturer's instructions. Briefly, HCAEC cells were seeded onto collagen-coated inserts with 1µm pores. After treatment, 75µl of FITC-dextran solution was added on the upper part for 20 min at room temperature. The fluorescent molecules passed through the endothelial barrier proportionally to the monolayer's permeability. Next, the insert with the HCAEC cells was removed and the amount of FITC-dextran in the bottom receiver was quantified using a microtiter plate reader at 485 nm and 535 nm excitation and emission, respectively. The relative permeability of the endothelial monolayer was expressed as the percentage of permeability in samples treated with control serum (100% relative permeability).

Once the inserts were removed, cells were stained with 50 µl toluidine blue staining for 20 min at room temperature. Afterwards, they were washed twice with phosphate buffer solution and ten photographs at 20x magnification were taken in independent fields using optical microscope Leica DMIL LED (Leica Microsystems, Germany). Pictures were digitized and then analyzed with Image ProPlus 7.0 software (Media Cybernetics Inc, Rockville, United States). Scoring was performed blind on coded slides.

Immunofluorescence: After 24 hours treatment, HCAEC were fixed carefully in 4% (w/v) paraformaldehyde at room temperature for 15 minutes and washed carefully with PBS three times. The blocking and permeating solution [10% fetal bovine serum; 0,1% Triton x-100] was added and allowed to act for 30 minutes at room temperature. Vascular endothelial-cadherin antibody (1:250, #MABT129, Merck, Burlington, United States) was diluted in blocking and

permeating solution and then incubated at 4 °C within a wet chamber in darkness overnight. Cultures were washed three times with PBS. Anti-mouse secondary antibody (Alexa Fluor 488 Conjugate, Thermo Fisher Scientific, Spain) was diluted in blocking and permeating solution and then incubated at room temperature within a wet chamber in darkness for 1 hour. Cover glasses were mounted with DAPI-Fluoromount-G (Thermo Fisher Scientific, Spain).[7] Samples were imaged with the Leica TCS SP2 spectral confocal and multiphoton system (Leica Microsystems, Germany).

Availability of data and materials: The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

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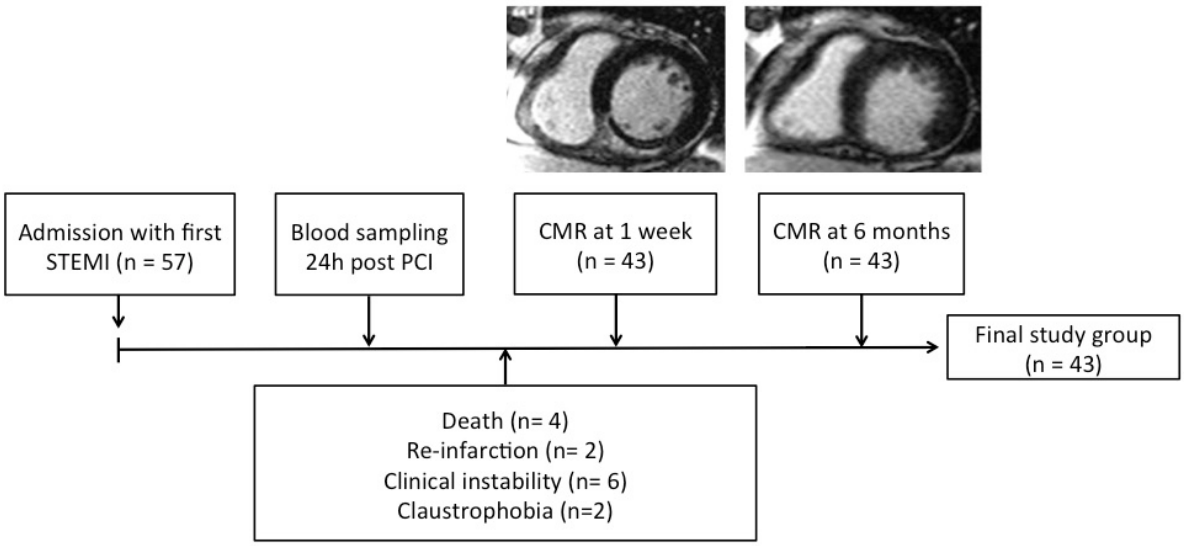
SUPPLEMENTARY FIGURE

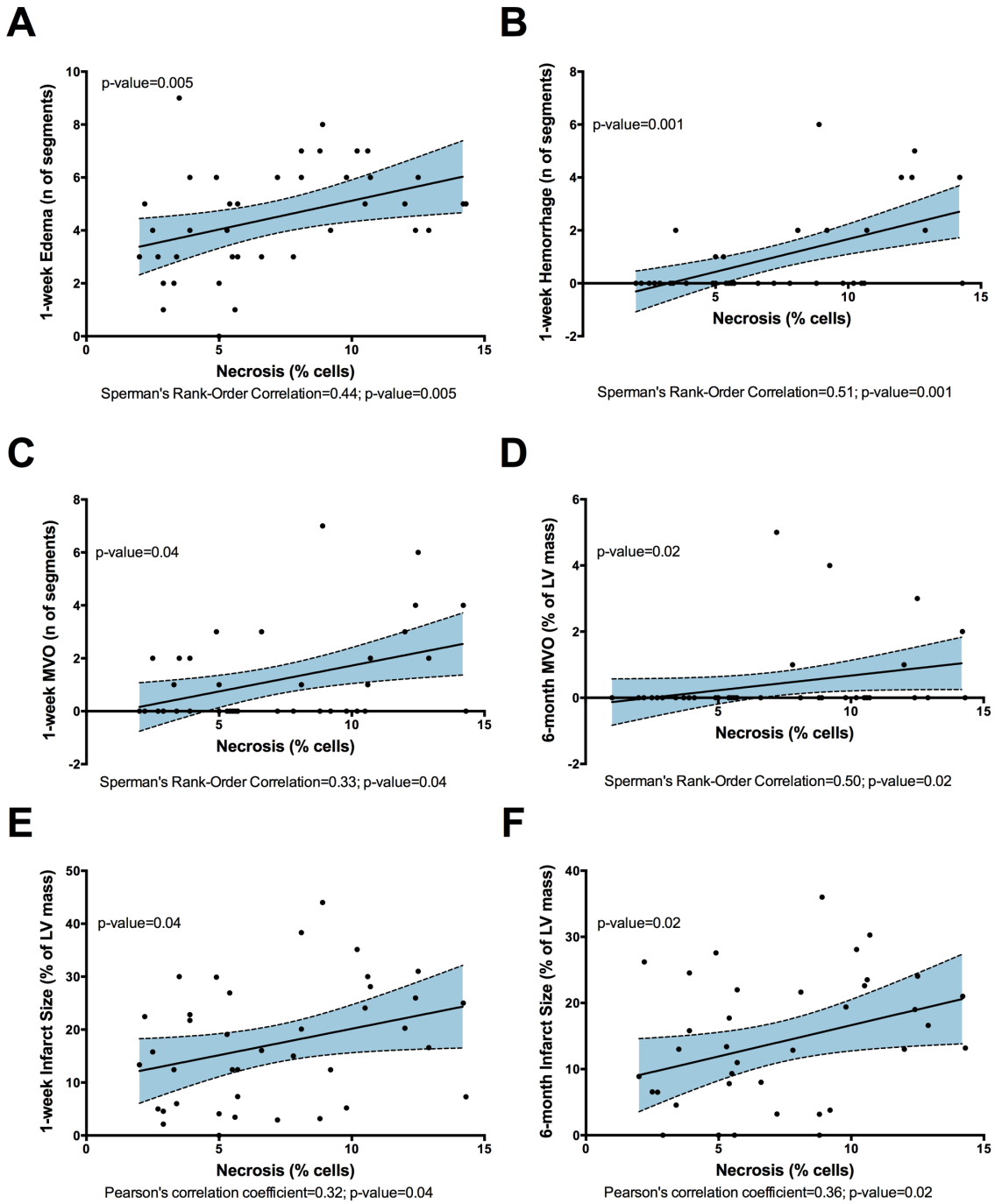
Figure 1 of the supplementary data. Flow chart showing STEMI patient enrolment protocol.



CMR: cardiovascular magnetic resonance, PCI: percutaneous coronary intervention, STEMI: ST-segment elevation myocardial infarction.

Figure 2 of the supplementary data. Correlation between serum-induced necrosis and CMR-derived indices of cardiac structure 1 week and 6 months after STEMI.

Patients whose serum caused a higher percentage of HCAEC to undergo necrosis presented more edema (A), hemorrhage (B), MVO (C), and infarct size (E) at acute phase (1 week post-revascularization), as well as higher MVO (D) and infarct size (F) at chronic phase (6 months).



CMR: cardiovascular magnetic resonance, HCAEC: human coronary artery endothelial cells, LV: left ventricle, MVO: microvascular obstruction, STEMI: ST-segment elevation myocardial infarction.

SUPPLEMENTARY TABLES

Table 1 of the supplementary data. Comparison of baseline characteristics between STEMI patients and controls.

	STEMI n = 43	**Controls** n = 14	***P***
Age (years)	58±13	62±12	.4
Male (%)	30 (70)	10 (71)	1
Diabetes mellitus (%)	6 (14)	4 (29)	.3
Hypertension (%)	20 (47)	7 (50)	1
Dyslipidemia (%)	16 (37)	6 (42)	.5
Smoker (%)	26 (60)	4 (29)	.2

STEMI: ST-segment elevation myocardial infarction.

Age is expressed as mean ± SD.

Table 2 of the supplementary data. Raw data from flow cytometry analysis to quantify the percentages of cells undergoing necrosis, apoptosis, early apoptosis and late apoptosis after human coronary artery endothelial cells treatment with serum from STEMI patients and controls.

Necrosis (% of EC)		**Apoptosis (% of EC)**		**Early apoptosis (% of EC)**		**Late apoptosis (% of EC)**	
STEMI	**Controls**	**STEMI**	**Controls**	**STEMI**	**Controls**	**STEMI**	**Controls**
4.9	4.0	12.3	9.4	8.3	11.6	4.0	7.8
2.7	2.6	7.1	13.8	5.0	7.6	2.1	6.2
3.9	5.1	18.6	15.4	13.2	11.4	5.4	4.0
7.2	2.8	17.3	14.4	12.2	8.3	5.1	6.1
6.6	1.8	18.4	14.2	13.4	9.6	5.0	4.6
5.3	1.4	27.1	14.7	19.3	9.7	7.8	5.0
3.7	1.7	32.7	14.4	23.7	10.3	9.0	4.1
8.1	11.0	26.0	8.4	17.4	4.8	8.6	3.6
2.9	5.6	35.6	16.8	28.0	8.6	7.6	8.2
5.0	1.4	27.7	14.6	19.3	13.0	8.4	11.6
2.9	2.1	11.4	18.0	6.0	7.3	5.4	10.7
3.9	1.5	10.0	10.0	7.0	3.8	3.0	6.2
3.4	1.1	11.9	20.8	8.4	8.0	3.5	12.8
5.4	3.4	8.7	11.9	5.2	5.7	3.5	6.2
5.0		13.1		8.6		4.5	
5.6		11.1		7.6		3.5	
3.5		17.6		12.6		5.0	
4.1		11.7		8.2		3.5	

7.8		15.4		9.2		6.2	
5.7		11.6		7.6		4.0	
2.5		18.9		13.0		5.9	
3.3		22.8		18.4		4.4	
1.0		27.7		22.8		4.9	
2.0		15.4		10.6		4.8	
5.7		12.0		8.6		3.4	
8.1		15.3		10.9		4.4	
5.5		14.9		10.6		4.3	
10.2		16.6		11.7		4.9	
8.9		17.1		11.0		6.1	
10.7		19.6		13.2		6.4	
10.6		23.6		16.1		7.5	
12.9		19.8		12.6		7.2	
9.2		21.7		15.1		6.6	
14.2		21.3		12.9		8.4	
9.8		26.5		11.2		15.3	
12.5		12.7		6.0		6.7	
12.0		15.5		5.5		10.0	
12.4		23.9		10.8		13.1	
8.8		22.0		11.0		11.0	
10.5		28.1		12.9		15.2	
14.3		29.2		16.6		12.6	
2.2		28.6		23.1		5.5	
5.4		27.7		22.2		5.5	

EC: endothelial cells, STEMI: ST-segment elevation myocardial infarction.

Table 3 of the supplementary data. Raw data from MTT assay, LDH release and gene expression of RIPK1 and RIPK3 obtained from human coronary artery endothelial cells treated with serum from STEMI patients and controls.

MTT assay (% of control)		**LDH release (cytotoxicity)**		**RIPK1 (mRNA fold change)**		**RIPK3 (mRNA fold change)**	
STEMI	**Controls**	**STEMI**	**Controls**	**STEMI**	**Controls**	**STEMI**	**Controls**
42.01	77.74	5.42	0.37	2.42	1.82	2.24	0.06
62.27	100.62	6.53	1.70	3.58	1.93	5.42	0.74
61.79	88.82	5.13	1.39	3.00	2.36	5.88	0.33
42.47	93.29	6.02	1.63	3.37	2.88	2.60	0.08
70.45	102.45	5.33	0.68	2.40	2.28	1.20	2.11
75.32	149.91	2.96	0.03	1.77	1.98	4.96	1.33
57.03	107.26	2.00	2.00	1.57	2.03	0.76	0.14
55.57	105.59	4.42	0.19	3.15	0.19	0.80	1.43
42.24	120.15	4.93	0.65	1.56	0.22	3.85	0.17
38.54	86.09	10.40	0.90	2.91	1.46	1.06	1.13
15.83	127.61	18.83	1.00	3.04	1.12	2.12	0.75
37.94	79.58	6.96	1.02	2.58	1.66	0.55	0.85
46.02	127.51	5.37	1.20	2.03	0.29	0.73	0.65
58.70	79.30	4.54	1.17	2.61	0.35	2.10	0.77
38.96		3.24		3.52		3.50	
27.66		19.17		2.40		1.22	
52.09		8.42		2.20		2.03	
72.27		8.13		2.72		2.79	
71.79		8.33		2.14		4.22	

52.47		9.96		3.59		6.18	
85.32		3.00		1.49		4.10	
67.03		7.42		3.57		0.64	
65.57		8.43		0.86		0.30	
52.24		12.90		1.76		1.70	
48.54		9.46		2.00		0.31	
25.83		7.87		1.50		2.55	
47.94		7.04		2.31		2.55	
56.02		5.74		4.09		0.22	
68.70		21.67		1.64		0.24	
48.96		2.42		3.32		1.01	
37.66		3.53		3.54		0.18	
32.09		2.13		1.89		5.04	
52.27		3.02		2.92		0.19	
51.79		2.33		0.96		0.27	
32.47		1.00		1.60		2.62	
47.03		1.42		1.39		0.53	
45.57		2.43		0.50		4.69	
32.24		7.90		0.61		0.38	
28.54		16.33		0.78		2.15	
27.94		4.46		1.73		2.00	
36.02		2.87		1.05		2.30	
48.70		2.04		1.72		2.25	
28.96		16.67		1.86		2.92	

LDH: lactate dehydrogenase, RIPK: receptor interacting protein kinases, STEMI: ST-segment elevation myocardial infarction.

Table 4 of the supplementary data. Raw data from caspase-3 activity, caspase-8 activity, and TUNEL-positive cells flow obtained from human coronary artery endothelial cells treatment with serum from STEMI patients and controls.

Caspase-3 activity (% of control)		**Caspase-8 activity (% of control)**		**TUNEL-positive EC (cells/field)**	
STEMI	**Controls**	**STEMI**	**Controls**	**STEMI**	**Controls**
1.62	0.99	1.29	0.99	9.00	4.00
1.19	1.00	1.21	1.20	8.00	2.00
1.28	1.04	1.07	0.89	9.00	0.50
0.73	0.88	1.49	1.09	2.00	0.50
0.98	0.95	1.14	1.08	3.00	2.00
1.13	1.18	1.53	0.95	2.00	1.00
1.20	0.97	0.92	1.05	8.00	0.00
2.17	1.02	1.10	1.01	12.00	3.00
1.76	0.99	0.63	1.01	5.00	3.00
1.73	0.86	1.26	0.96	10.00	5.00
2.17	1.25	0.97	0.94	11.00	1.00
1.46	1.16	1.02	0.91	12.00	1.00
2.07	0.90	1.03	0.92	11.00	0.00
1.82	1.02	0.96	1.04	11.00	0.00
1.72		0.92		0.00	
2.11		0.93		5.00	
1.69		1.14		7.00	
1.78		1.26		8.00	
1.23		1.11		14.00	

1.48		1.08		11.00	
1.63		0.86		12.00	
1.70		1.60		9.00	
2.67		1.53		6.00	
2.26		0.98		6.00	
2.23		1.03		8.00	
2.67		1.02		2.00	
1.96		0.98		0.00	
2.10		0.93		0.00	
2.32		0.93		2.00	
2.22		1.13		4.00	
1.11		1.27		3.00	
0.69		1.11		4.00	
0.60		1.35		5.00	
0.23		0.99		2.00	
0.48		1.61		3.00	
0.63		1.54		4.00	
0.70		1.20		2.00	
1.67		1.11		0.00	
1.26		1.03		3.00	
1.23		1.09		5.00	
1.67		1.04		6.00	
0.96		1.33		6.00	
1.57		1.13		7.00	

EC: endothelial cells, STEMI: ST-segment elevation myocardial infarction.

Table 5 of the supplementary data. Raw data from relative permeability, area free of cells and roundness obtained from human coronary artery endothelial cells treatment with serum from STEMI patients and controls.

Relative permeability (% of control)		**Area free of cells (mm^2)**		**Roundness (a.u.)**	
STEMI	**Controls**	**STEMI**	**Controls**	**STEMI**	**Controls**
309	247	0.336	0.230	33.58	17.34
523	201	0.316	0.310	54.03	16.82
170	210	0.335	0.300	53.41	29.58
320	100	0.304	0.250	42.87	32.65
168	17	0.351	0.270	41.47	24.57
498	60	0.348	0.270	49.67	13.90
266	249	0.336	0.310	52.55	19.33
155	84	0.302	0.300	44.56	22.45
172	163	0.330	0.350	33.82	36.60
150	46	0.310	0.244	25.76	39.27
198	58	0.330	0.297	37.84	22.60
203	21	0.300	0.357	37.50	17.03
165	60	0.350	0.276	34.68	29.42
48	247	0.340	0.297	34.46	11.65
85		0.330		53.24	
558		0.300		58.98	
461		0.320		49.87	
169		0.315		53.56	
109		0.340		50.54	

81		0.310		46.95	
12		0.340		55.47	
75		0.330		46.15	
86		0.330		68.27	
262		0.300		52.97	
270		0.337		36.75	
407		0.317		26.52	
207		0.334		54.86	
738		0.303		36.16	
243		0.354		60.30	
250		0.342		46.63	
240		0.339		29.58	
200		0.306		52.22	
300		0.334		61.39	
350		0.302		61.96	
150		0.349		31.46	
100		0.348		42.20	
324		0.332		38.10	
345		0.301		73.19	
124		0.322		33.73	
201		0.311		48.29	
187		0.343		48.72	
311		0.312		44.43	
128		0.344		52.55	

STEMI: ST-segment elevation myocardial infarction.

Table 6 of the supplementary data. Baseline characteristics of patients with serum-induced apoptosis above or below median.

	Serum-induced apoptosis below median (18.4%)	**Serum-induced apoptosis above median (18.4%)**	***P***
Number of patients	22	21	
Age (years)	55 ± 11	62 ± 13	.08
Male sex (%)	19 (86)	11 (52)	.1
Diabetes mellitus (%)	3 (14)	3 (14)	.8
Hypertension (%)	9 (41)	11 (52)	.2
Dyslipidemia (%)	10 (45)	6 (29)	.5
Smoker (%)	13 (59)	13 (62)	.8
GRACE risk score	123 ± 30	130 ± 38	.5
TIMI risk score	2.0 ± 1.8	3.0 ± 1.8	.07
Time to reperfusion (min)	321 ± 259	296 ± 182	.7
Anterior infarction (%)	13 (59)	12 (57)	.5
Multivessel disease (%)	6 (27)	6 (29)	.6
Culprit artery (%)			.5
LAD	12 (55)	14 (67)	
RCA	7 (32)	3 (14)	
Cx	3 (13)	4 (19)	
Killip class (%)			.5
1	21 (95)	16 (76)	
2	1 (5)	4 (19)	
3	0 (0)	0 (0)	
4	0 (0)	1 (5)	

DAPT (%)	18 (82)	15 (71)	.8
Statins (%)	19 (86)	16 (76)	.7
ACEi/ARA-II/ARNi (%)	16 (73)	13 (62)	.4
Beta-blockers (%)	17 (77)	14 (67)	.8
Diuretics (%)	0 (0)	2 (10)	.1
Anticoagulants (%)	4 (18)	1 (5)	.2
Mineralocorticoid receptor antagonists (%)	3 (14)	3 (14)	.8

ACEi: angiotensin-converting enzyme inhibitors, ARA-II: angiotensin II receptor antagonists, ARNi: angiotensin receptor neprilysin inhibitors, Cx: circumflex coronary artery, DAPT: dual antiplatelet therapy, GRACE: Global Registry of Acute Coronary Events, LAD: left anterior descending coronary artery, PCI: percutaneous coronary intervention, RCA: right coronary artery, TIMI: Thrombolysis in Myocardial Infarction.

All variables are expressed as mean ± SD.

Table 7 supplementary data. CMR characteristics of ST-segment elevation myocardial infarction patients with serum-induced apoptosis above or below median.

	Serum-induced apoptosis below median (18.4%)	**Serum-induced apoptosis above median (18.4%)**	***P***
Number of patients	21	22	
1-week CMR			
LVEF (%)	53 ± 10	55 ± 11	.7
LV end-diastolic volume index (ml/m²)	78 ± 16	77 ± 19	.9
LV end-systolic volume index (ml/m²)	37 ± 13	35 ± 11	.6
LV mass (g/m²)	75 ± 16	75 ± 13	1
Infarct size (% of LV mass)	18 ± 12	16 ± 10	.6
Edema (% of LV mass)	27 ± 14	26 ± 14	.9
MVO (n of segments)	0 [0-2]	0 [0-2]	.3
Hemorrhage (n of segments)	0 [0-0]	0 [0-2]	.9
6-month CMR			
LVEF (%)	57 ± 11	61 ± 10	.3
LV end-diastolic volume index (ml/m²)	76 ± 22	85 ± 20	.2
LV end-systolic volume index (ml/m²)	34 ± 18	34 ± 13	1
LV mass (g/m²)	69 ± 17	69 ± 14	1
Infarct size (% of LV mass)	14 ± 10	15 ± 10	.7
MVO (n of segments)	0 [0-0]	0 [0-0]	.8

CMR: cardiovascular magnetic resonance; LV: left ventricular, LVEF: left ventricular ejection fraction, MVO: microvascular obstruction.

MVO and hemorrhage are expressed as median [percentile 25–percentile 75].

All other variables are expressed as mean ± SD.