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CLINICAL STUDY

Effect of selected cardiovascular factors on mortality in patients with ST-segment elevation myocardial infarction treated with primary coronary intervention

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ABSTRACT

OBJECTIVE: This work was aimed at analyzing in-hospital, 30-day and 1-year mortality rates, impact of selected cardiovascular factors on mortality of patients with ST-segment elevation myocardial infarction (STEMI) manifested on electrocardiogram (ECG) and treated by the percutaneous coronary intervention (PCI) at our cardiac center, comparing the subgroup of non-shock (survivors and deceased) patients after STEMI and evaluating how these patients differ from each other.

METHODS: In total, 270 patients with STEMI manifested on ECG and treated by PCI were enrolled between April 1, 2018, and March 31, 2019, at our cardiologic center. Our study sought to determine the risk of death after acute myocardial infarction with carefully selected factors and parameters such as the presence of cardiogenic shock, ischemic time, left ventricular ejection fraction (LVEF), post-PCI TIMI (thrombolysis in myocardial infarction) flow and serum levels of cardio-specific markers, namely troponin T, creatine kinase and N-terminal pro-brain natriuretic peptide (NT-proBNP). Further evaluation included in-hospital, 30-day and 1-year mortality rates in shock and non-shock patients and determination of factors that influence the survival separately in each subgroup. The follow-up was carried out for 12 months after the myocardial infarction in form of outpatient examinations. After 12 months of follow-up, the collected data were statistically evaluated.

RESULTS: Shock and non-shock patients differed in mortality and several other parameters including NT-proBNP values, ischemic time, TIMI flow defect and LVEF. In all outcomes (in-hospital, 30-day and 1-year mortality rates) the shock patients did worse than non-shock patients ($p < 0.001$). In addition, age, gender, LVEF, NT-proBNP and post-PCI TIMI flow less than 3 were found to be important factors influencing the overall survival. In shock patients, the survival was associated with age, LVEF and TIMI flow, while in non-shock patients, the factors predicting survival were age, LVEF, level of NT-proBNP and troponin levels.

CONCLUSION: Shock patients differed in terms of mortality in post-PCI TIMI flow, while non-shock patients varied in troponin and NT-proBNP levels. Despite early intervention, certain risk factors might affect the clinical outcome and prognosis of patients with STEMI treated by PCI *(Tab. 5, Fig. 1, Ref. 30)*. Text in PDF *www.elis.sk*

KEY WORDS: myocardial infarction, primary coronary intervention, shock, mortality, cardio-specific markers.

Introduction

Cardiovascular events are among the leading causes of death worldwide (1–3).

Our cardiology department treats approximately 250–300 patients with ST-segment elevation myocardial infarction (STEMI) every year. Despite early intervention, there are certain risk factors such as age, sex, family history, body mass index (BMI), tobacco smoke, high cholesterol level, high levels of uric acid, comorbidities (diabetes mellitus, hypertension, etc.) that may affect the clinical outcome and prognosis of patients with STEMI treated by percutaneous coronary intervention (PCI) (1). Among the factors, many of them increase the risk of the atherosclerotic disease per se. Out of lipid spectrum disorders, LDL (low-density lipoprotein) cholesterol has been specifically identified as a factor contributing to the atherosclerosis development, while statin treatment aimed

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at reducing the level of LDL-cholesterol contributes to the reduction in the risk of cardiovascular events (3–7).

Other potential risk markers linked to atherosclerosis include inflammatory molecules, given that inflammation also plays an important role in the pathogenesis of atherosclerosis and cardiovascular disease (8, 9). According to the latest data, atherosclerosis is considered a chronic inflammation. The CANTOS (Canakinumab Anti-Inflammatory Thrombosis Outcome Study) study investigating the interleukin IL-1 inhibitor canakinumab (anti-inflammatory therapy), demonstrated a reduction in cardiovascular events in patients who had already experienced an acute myocardial infarction (10–12). Drugs with an anti-inflammatory effect could become part of the treatment of atherosclerosis (13). In all cases, further factors influencing STEMI patient survival should be sought and investigated in the light of their potential influence on future treatment options (14, 15). In our study, we focused on parameters linked to the clinical course, results of imaging methods and laboratory markers at the time of STEMI manifestation.

Aim of the study

In our study, we assessed mortality (in-hospital, 30-day, and 1-year mortality rates) among patients treated for STEMI who met or did not meet the criteria for cardiogenic shock. We also looked for other factors that could affect mortality in patients after STEMI, specifically, the presence of shock at the onset of myocardial infarction, laboratory parameters, namely troponin T, creatine kinase, N-terminal pro-brain natriuretic peptide (NT-proBNP), as well as ischemic time, post-PCI TIMI flow (thrombolysis in myocardial infarction) and LVEF (left ventricular ejection fraction). The association of these factors with survival was further evaluated separately in the patients with or without cardiogenic shock.

Materials and methods

The authors describe a one-year prospective study involving 270 consecutive patients with STEMI as diagnosed by electrocardiogram (ECG) record, who were treated at our department (First Department of Internal Medicine – Cardioangiology, St. Anne´s University Hospital Brno, Czech Republic) with primary PCI in the period from April 1, 2018, to March 31, 2019. Most patients presented typical clinical symptoms accompanying a heart attack and a STEMI myocardial infarction plotted on ECG. Immediately upon arrival at the catheterization laboratory, the infarcted artery was treated with PCI using drug-eluting stent (DES) implantation. Another important part of acute coronary syndrome management was the pharmacological treatment inclusive of antiplatelet and anticoagulant drugs. After the intervention, patients were hospitalized in a coronary unit to monitor vital signs and facilitate their further care. Following their discharge from the hospital, the patients remained in our ambulatory care with follow-ups scheduled at 1, 3, 6 and 12 months after intervention/ heart attack. Upon completion of the follow-up, we statistically evaluated the in-hospital, 30-day and annual mortality rates and the effect of examined factors on the survival of patients after STEMI. Patients with stimulated rhythm captured on ECG and subacute Q-STEMI were excluded from the study.

Statistical analysis

For a cross-sectional comparison of continuous variables, Mann–Whitney U-test was chosen due to the highly asymmetric distribution of most of them. In these cases, the data are shown as median with interquartile range (IQR); in the variables with normal distribution such as age and LVEF, the data are expressed as mean ± standard deviation. Two-tailed Fisher exact test was used to compare binary variables including the in-hospital survival and 1-month survival, where the survival time was not regarded as a parameter to be assessed by the analysis. For 1-year survival, Kaplan–Meier curves, log-rank test and univariate Cox regression models were employed. In the latter case, the absence of disease/ complication and male sex (more frequent in the study group) were used as reference categories. In the case of continuous parameters

Tab. 1. Baseline characteristics.

Parameter		Value
Age [years]		66.7±13.1
Sex [male/female; n (%)]		189 (70.0 %)/81 (30.0 %)
Coronary occlusion		
Culprit lesion [n (%)]	LM	7 (2.6 %)
	LAD	124 (45.9 %)
	LCx	32 (11.9 %)
	LIM	5 (1.9 %)
	RCA	101 (37.4 %)
	CABG	1 (0.4 %)
TIMI 3 (complete perfusion) [n (%)]	Pre-PCI	46 (17.0 %)
	Post-PCI	257 (95.1 %)
Time to revascularization (ischemic time)* [min]		203 (126–368)
Cardiogenic shock [n (%)]		49 (18.1 %)
LVEF at admission [%]		46.4±11.5
Personal history		
Hypertension [n (%)]		169 (62.6 %)
Diabetes [n (%)]		101 (37.4 %)
BMI [kg.m-2]		27.9±5.1
LDL-cholesterol [mmol/l]		3.09±1.06
Smoking [current/past; n (%)]		100 (45.7 %)/44 (16.6 %)
Known ischemic heart disease [n (%)]		44 (16.3 %)
Stroke [n (%)]		17 (6.3 %)
Family history of MI [n (%)]		136 (53.3 %)
Laboratory values		
CK (peak) [μkat/l]*		14.9 (7.6–27.7)
Troponin-T (peak) [ng/l]*		2.444 (895–4834)
NT-proBNP [ng/l]*		538 (141–1802)
C-reactive protein (peak) [mg/l]*		7.5 (2.0–48.1)

The values of continuous parameters with normal distribution are expressed as mean ± standard deviation, those with asymmetric distribution are marked by asterisk and shown as median (lower quartile–upper quartile). Categorical parameters are shown as number (percentage of total).
LM = left main; LAD = left anterior descendent; LCx = left circumflex; LIM = left intermediate; TIMI = thrombolysis in myocardial infarction; PCI = percutaneous coronary intervention; LVEF = left ventricle ejection fraction; BMI = body mass index; LDL = low density lipoprotein; MI = myocardial infarction; CK = creatine kinase; NT-proBNP = N-terminal pro-brain natriuretic peptide. RCA = right coronaryartery, CABG = coronary artery bypass graft

Tab. 2. Comparison between shock and non-shock patients.

Parameter	Cardiogenic shock (n=49)	No shock (n=221)	p
Age [years]	67.9±11.7	66.3±13.3	0.47
Sex [female; n (%)]	18 (36.7 %)	63 (28.5 %)	0.30
LVEF [%]	**40.8±14.6**	**47.6±10.3**	**0.005**
Ischemic time [min]*	**159 (111–247)**	**227 (127–380)**	**0.018**
TIMI flow defect [n (%)]	**6 (12.2 %)**	**7 (3.2 %)**	**0.016**
CK (peak) [μkat/l]*	14.9 (6.1–38.7)	14.8 (8.5–27.2)	0.72
Troponin-T (peak) [μmol/l]*	3080 (794–8300)	2331 (895–4447)	0.12
NT-proBNP [ng/l]*	**1,816 (349–4550)**	**500 (133–1338)**	**0.004**

LVEF = left ventricle ejection fraction; CK = creatine kinase; NT-proBNP = N-terminal pro-brain natriuretic peptide. Statistically significant results with $p < 0.05$ are marked in bold.

assessed by the Cox regression, a continuously increasing/decreasing effect on survival was assumed without introducing any artificial cut-offs – linear in the case of age and LVEF, while the logarithmic transformation was employed in laboratory values and ischemic time (hazard ratios are always per doubling of a given parameter in this case). Post-interventional TIMI flow score lower than 3 was grouped as "TIMI flow defect" due to the low number of patients with TIMI 0–2 (n = 13) and compared with TIMI 3 as binary data. The value of $\alpha = 0.05$ was used throughout the study and only the tests with p-values lower than this were considered to be statistically significant. All analyses were performed using Statistica software (version 14.0. TIBCO Software Inc., Palo Alto, California, USA, 2020).

Patients with cardiogenic shock induced by acute myocardial infarction were defined by a decrease in systolic blood pressure below 90 mmHg with the need for catecholamine support to maintain systemic blood pressure. They also showed signs of peripheral hypoperfusion.

Ischemic time was defined as the time from the symptom onset (angina pectoris) up to that of balloon inflation at the culprit lesion coronary artery site in the catheterization laboratory.

Results

Out of the total number of 270 patients, 70 % were male (n = 189) and 30 % female (n = 81), with the mean age of 66.7 ± 13.1 years. In regard to shock, 18 % (n = 49) of patients had a baseline cardiogenic shock, and the remaining 82 % (n = 221) were non-shock patients. More detailed parameters and characteristics of the cohort are shown in Table 1.

Of the evaluated parameters, the median troponin T concentration was 2444 ng/L (IQR: 895–4834) while cut-off troponin T concentration in our center is 14 ng/L, median NT-proBNP concentration was 538 ng/L (IQR: 141–1802). The median value of the laboratory parameter of creatine kinase was 14.9 μkat / L (IQR: 7.64–27.67). The median ischemic time was 203 minutes (IQR: 126–368). The average value of the left ventricular ejection fraction in the examined population was 46.4 ± 11.5 %.

As shown in Table 2, the differences between patients with and without shock in natriuretic peptide NT-proBNP ($p = 0.004$), ischemic time ($p = 0.018$), TIMI flow defect ($p = 0.016$), and left ventricular ejection fraction ($p = 0.005$) were statistically significant.

During the first 12 months after myocardial infarction, 38 patients died (14.1 %). Of these, 20 patients were initially in cardiogenic shock, 17 of whom died within 1 month of STEMI. The remaining 18 patients were free of cardiogenic shock (8 of them died within 1 month after STEMI, and the remaining 10 patients died between 2 and 12 months). The one-year mortality rates in patients with and without initial cardiogenic shock were 40.8 % and 8.1 %, respectively. The survival analysis of shock and non-shock patients using log-rank tests demonstrated that shock patients had a statistically significantly worse survival as compared to patients who were not in the shock state at the beginning ($p < 0.001$). The survival of both groups expressed as Kaplan-Meier curves are shown in Figure 1.

The percentages of patients who died in the hospital and those who deceased during the first 30 days were very similar, as there was a huge overlap between the two groups. Namely, the 30-day mortality of the whole group was 9.6 % and the total in-hospital mortality was 9.3 %. Both in-hospital and 30-day mortality rates in shock patients was 34.7 %, while in patients without shock at the beginning of STEMI, the in-hospital mortality was 3.6 % and 30-day mortality was also 3.6 %. In both cases, the risk of death was significantly higher in shock patients, $p < 0.001$.

Our results suggest that the presence of shock significantly affected mortality. In univariate Cox regression models, the association with survival was the strongest of all evaluated factors [$p < 0.001$, HR = 6.47 (95% CI 3.42–12.26)]. Apart from shock, other parameters also influenced the survival of patients after STEMI. These were LVEF [$p < 0.001$, HR per each 10 %: 0.47 (95% CI 0.36–0.62)]; NT-proBNP [$p < 0.001$, HR per doubling = 1.42

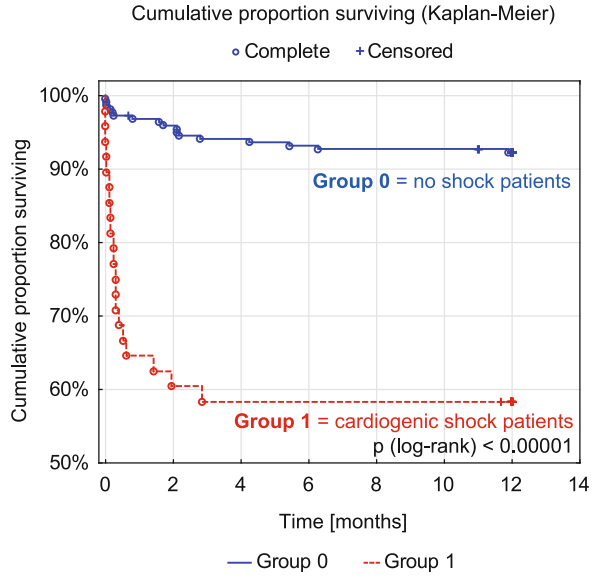

Fig. 1. Cumulative proportion survival (Kaplan-Meier).

Tab. 3. Univariate Cox regression models of survival, all groups.

Parameter	HR (95 % CI)	P-value
Age (per each 10 years)	**1.72 (1.32–2.24)**	**< 0.001**
Sex (female vs. male)	**1.95 (1.03–3.69)**	**0.041**
Cardiogenic shock	**6.47 (3.42–12.26)**	**< 0.001**
LVEF (per each 10 %)	**0.47 (0.36–0.62)**	**< 0.001**
Ischemic time (per doubling)	0.98 (0.71–1.35)	0.90
CK (peak) (per doubling)	1.20 (0.93–1.55)	0.15
Troponin-T (peak) (per doubling)	1.25 (1.00–1.57)	0.051
Post PCI TIMI flow defect vs. 3	**4.78 (1.99–11.45)**	**< 0.001**
NT-proBNP (per doubling)	**1.42 (1.20–1.68)**	**< 0.001**

HR = hazard ratio; CI = confidence interval; LVEF = left ventricle ejection fraction; CK = creatine kinase; NT-proBNP = N-terminal pro-brain natriuretic peptide; TIMI = thrombolysis in myocardial infarction.
Statistically significant results with p < 0.05 are marked in bold.

Tab. 4. Univariate Cox regression models of survival in non-shock patients who survived and died within 12 months from STEMI.

Parameter	HR (95 % CI)	p
Age (per each 10 years)	**2.05 (1.37–3.05)**	**< 0.001**
Sex (female vs. male)	2.03 (0.80–5.13)	0.14
LVEF (per each 10 %)	**0.44 (0.28–0.68)**	**< 0.001**
Ischemic time (per doubling)	0.92 (0.56–1.49)	0.72
CK (peak) (per doubling)	1.40 (0.94–2.10)	0.10
Troponin-T (peak) (per doubling)	**1.84 (1.20–2.83)**	**< 0.001**
Post PCI TIMI flow defect vs. 3	1.93 (0.26–14.53)	0.52
NT-proBNP (per doubling)	**1.42 (1.14–1.76)**	**< 0.001**

HR = hazard ratio; CI = confidence interval; LVEF = left ventricle ejection fraction; CK = creatine kinase; NT-proBNP = N-terminal pro-brain natriuretic peptide; TIMI = thrombolysis in myocardial infarction.
Statistically significant results with p < 0.05 are marked in bold.
Male sex and absence of cardiogenic shock were used as reference categories in categorical risk factors. Continuous increase/decrease of risk was assumed in continuous risk factors with no artificial cut-offs.

Tab. 5. Univariate Cox regression models of survival in shock patients who survived and died within 12 months from STEMI.

Parameter	HR (95 % CI)	P-value
Age (per each 10 years)	**1.48 (1.02–2.14)**	**0.041**
Sex (female vs. male)	0.66 (0.27–1.58)	0.35
LVEF (per each 10 %)	**0.69 (0.50–0.95)**	**0.024**
Ischemic time (per doubling)	1.24 (0.85–1.79)	0.26
CK (peak) (per doubling)	0.96 (0.72–1.28)	0.77
Troponin-T (peak) (per doubling)	0.86 (0.67–1.11)	0.24
Post PCI TIMI flow defect vs. 3	**3.22 (1.15–8.99)**	**0.026**
NT-proBNP (per doubling)	1.26 (0.93–1.70)	0.13

HR = hazard ratio; CI = confidence interval; LVEF = left ventricle ejection fraction; CK = creatine kinase; NT-proBNP = N-terminal pro-brain natriuretic peptide; TIMI = thrombolysis in myocardial infarction.
Statistically significant results with p < 0.05 are marked in bold.

(95% CI 1.20–1.68)]; age [p < 0.001, HR per every 10 years: 1.72 (95% CI 1.32–2.24)]; post-PCI TIMI flow defect [p = 0.0005, HR = 4.78 (95% CI 1.99–11.45)]; and female gender [p = 0.041; HR = 1.95 (95% CI 1.03–3.69)]. The cardio-specific marker, troponin T, came out with borderline significance [p = 0.051, HR per doubling = 1.25 (1.00–1.57)]. Ischemic time was not associated with survival (p = 0.90). The detailed outcomes of univariate Cox regression including the 95% confidence intervals are in Table 3.

We then assessed the association of the above/mentioned factors with survival separately in non-shock (Tab. 4) and shock (Tab. 5) patients with STEMI using univariate Cox regression models. In both subgroups, age and LVEF were associated with survival. Further, in the non-shock patients, laboratory markers, troponin T [p = 0.003; HR per doubling = 1.84 (95% CI 1.20–2.83)] and NT-proBNP [p = 0.001; HR per doubling = 1.42 (1.14–1.76)] were significant predictors of survival. In shock patients, laboratory markers did not predict survival, but the post-PCI TIMI flow defect [p = 0.026; HR = 3.22 (95% CI 1.15–8.99)] was a significant factor. There was no difference in gender, CK or ischemic time in either of the two subgroups were assessed separately.

Regarding the causes of death in the study population within 12 months of STEMI, 71 % (27 causes) were cardiovascular causes. Of these, cardiogenic shock was the most common, in up to 30 % of patients (8 patients), while 22 % of patients died of heart failure (6 patients), 18.5 % (5 patients) had post hypoxic central nervous system (CNS) involvement, in 11 % (3 patients), the infarction resulted in sudden cardiac death (SCD, 7.4 % (2 patients) died of ischemic stroke and 3.7 % of patients died of other cardiovascular causes such as ruptured abdominal aortic aneurysm (1 patient), ischemic lower limb disease and its associated complications (1 patient), and mechanical complication of myocardial infarction (1 patient). Other non-cardiovascular causes of death (11 patients) were renal failure, urosepsis, pneumonia and malignant mesothelioma.

Discussion

There is a strong consensus that early revascularization of the ischemic myocardium in STEMI patients by percutaneous coronary intervention improves patient survival (1, 16). The possibility of early PCI (within 90–120 minutes from the onset of pain) depends on the availability of the catheterization laboratory and arrival time. In consideration of the fact that some regions (e.g. North America) lack catheterization laboratories or have relatively longer arrival times, special protocols are employed using thrombolysis as the first-line treatment (17, 18). In the Czech Republic due to the dense network of catheterization laboratories with a very good access, thrombolysis is hardly used at all.

The risk of death after STEMI is still relatively high, although it has declined over several decades (19). Several previous studies have shown that one-year mortality is around 7–10 % and in-hospital mortality less than 7 %, which is slightly better than shown in our data (14 % and 8 %, respectively) (20–22). Laboratory levels of troponin and creatine kinase are strongly associated with the extent of the heart attack (23, 24).

Our study is focused on the evaluation and description of in-hospital, 30-day and 1-year mortality rates after acute STEMI. In the analyzed group, we assessed in-hospital, 30-day and 1- year mortality rates in both shock and non-shock patients, as well as in the whole cohort. As expected, the results in shock and non-shock patients are shown to be significantly different from each other, namely in the presence of a severe clinical condition in shock patients.

Aspartate aminotransferase (AST) and lactate dehydrogenase (LDH) were among the first biomarkers used in the diagnosis of acute myocardial infarction (25–27). Highly sensitive troponins T and I are currently used to diagnose myocardial infarction (25, 28).

A Chinese study as of 2021 observed that a higher entry level of troponin I is a significant predictor of 30-day and 1-year events (29).

In our study, we evaluated parameters as follows: cardio-specific laboratory markers (troponin T, CK, natriuretic peptides), clinical data (ischemic time, presence of shock), results of imaging methods (post-PCI TIMI flow, LVEF), age and gender. The association of these factors with survival was further evaluated separately in the patients with and without cardiogenic shock. In the subgroup of non-shock patients (deceased vs. survivors) the laboratory parameters that showed to have a significant effect on mortality were troponin T and NT-pro BNP whereas in shock patients, it was the post- PCI TIMI flow value. On the other hand, according to our results, ischemic time is not associated with mortality. This can be explained by the fact that shock patients generally had shorter ischemic times which is associated with their severe clinical condition at the beginning.

A 2017 Japanese study, which evaluated cardiac and non-cardiac causes of death after STEMI, revealed that cardiac causes of death predominate in the first 6 months, while non-cardiac causes prevail in the last 6 months (30). In our study, within the first year after STEMI, 71 % of the causes of death were of cardiovascular origin, while only the remaining 29 % were non-cardiovascular and almost all deaths in our study were in the first half of the year, and only two deaths occurred in the second half when non-cardiac causes of death should dominate.

Conclusion

Shock and non-shock patients in our study differed in mortality and other parameters including NT-proBNP, ischemic time, TIMI flow defect and LVEF. NT-proBNP is associated with survival when higher values indicate a worse survival. As to other factors, age, gender, post-PCI TIMI flow less than 3 and LVEF are associated with survival (for all groups). Cardio-specific marker, troponin T, is slightly below the limit of statistical significance (for all groups). Ischemic time is not a useful predictor of survival in the non-stratified patient population because it is not an independent factor as it depends on the patient's condition (shock patients had shorter ischemic time). In the subgroup of shock patients, the survival was associated with age, LVEF and TIMI flow, while in non-shock patients, the factors predicting survival were age, LVEF, NT-proBNP and troponin T.

Detailed description, identification and analysis of risk factors might aid clinicians in better understanding the nuances associated with increased morbidity and mortality in these patients and help to develop guidelines for preventing and eliminating these factors using population-wide strategies.

Limitations

The data of study patients come from one center. It is a small sample when considering the study period is limited to one year. In the Czech Republic, we have a relatively dense network of catheterization laboratories (22 per 10.7 million inhabitants). The study reflects the population of the Czech Republic (all patients were Caucasian), which makes the sample less comparable to the global population. Our data were collected before the COVID-19 pandemic; in the following years, the annual numbers of patients with STEMI were slightly lower, the delay tended to be higher, and patients with myocardial infarction used to arrive at the hospital later. Considering these factors, their mortality rate could have been worse.

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