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D-dimer/high-sensitivity troponin I ratio in the diagnosis of acute pulmonary embolism and/or non-ST-elevation myocardial infarction

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Abstract

This study aimed to determine whether the D-dimer/high-sensitivity troponin I (hs-TnI) ratio is useful in the differential diagnosis of acute pulmonary embolism (APE) and/or non-ST-elevation myocardial infarction (NSTEMI) in patients who presented to the emergency department with chest pain. The study included 219 patients with APE and 385 patients with NSTEMI over the age of 18 who presented to the emergency department with chest pain and were diagnosed with either APE or NSTEMI. Using statistical analysis, D-dimer, hs-TnI, creatine kinase myocardial band (CK-MB) levels, D-dimer/CK-MB, and D-dimer/hs-TnI ratios were compared in patients with APE and NSTEMI. The D-dimer/hs-TnI ratio in patients with APE was found to be considerably greater than in patients with NSTEMI. Similarly, the D-dimer/CK-MB levels in patients with APE were significantly higher than in individuals with NSTEMI. Patients with APE had higher D-dimer levels, while those with NSTEMI had higher hs-TnI levels. The D-dimer/hs-TnI ratio can be useful for emergency clinicians because it is affordable, quickly calculated, and easily accessible.

Abbreviations: ACS = acute coronary syndrome, APE = acute pulmonary embolism, AUC = area under the curve, CK-MB = creatine kinase myocardial band, DBP = diastolic blood pressure, ED = emergency department, hs-TnI = high-sensitivity troponin I, MAP = mean arterial blood pressure, NSTEMI = non-ST-elevation myocardial infarction, ROC = receiver operating characteristic, SaO_2 = Saturation percentage, SBP = systolic blood pressure.

Keywords: acute pulmonary embolism, D-dimer, high-sensitivity troponin I, non-ST-elevation myocardial infarction

1. Introduction

Two commonly encountered clinical symptoms in the emergency room are shortness of breath and sudden chest discomfort.[1] Because of the high risk of mortality and morbidity in acute coronary syndrome (ACS) and acute pulmonary embolism (APE), both should both be diagnosed promptly in these patients and adequate follow-up should be provided after establishing a differential diagnosis. Therefore, selecting the correct therapeutic approach in a timely manner is crucial in these situations.

Medical history, physical examination, and cardiac risk factors might not be enough to differentiate between similar conditions that present with chest pain.[2] One of the biomarkers used to exclude the diagnosis of APE is the D-dimer protein fragment. Although normal or low levels of D-dimer is useful for excluding venous thromboembolic events, high levels have low specificity and sensitivity for confirming pulmonary embolism diagnosis.[3] This low specificity is because several other thrombotic diseases, including ACS, increase serum D-dimer levels, which is a fibrin breakdown product.[4,5] Given the pathophysiology of APE, right ventricular load contributes to increased serum levels of high-sensitivity troponin I (hs-TnI), just as in ACS.

The D-dimer/hs-TnI ratio is easy to use and can be more effective than using each biomarker alone. Studies in the literature show that the ratio is a suitable parameter to use in the differential diagnosis of chest pain, such as APE, ACS, or thoracic acute aortic syndrome.[6-8]

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The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

The ethics committee of the Adana City Training and Research Hospital approved the study.

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This study aimed to investigate whether the D-dimer/hs-TnI ratio is effective in the differential diagnosis of APE and non-ST-elevation myocardial infarction (NSTEMI). We also wanted to establish whether this practical ratio could be helpful for the differential diagnosis of these 2 important diseases in the emergency department (ED) setting.

2. Materials and methods

This retrospective, cross-sectional case study conducted at a single institution. The study began following approval and conducted in accordance with the Helsinki declaration.

2.1. Participant selection

Patients who presented to the ED with a complaint of chest pain and were diagnosed with APE and/or NSTEMI between January 1, 2020 and January 1, 2022 were included in the study. The APE diagnosis was based on file records and/or computed tomography pulmonary angiogram, whereas NSTEMI diagnosis was based on hs-TnI levels. The pediatric age group (age <18 years), patients whose records could not be completely accessed, and patients with diagnoses other than APE and NSTEMI due to chest pain were all excluded from the study.

2.2. Study methods

During the data collection phase of the study, the vital signs (i.e., mean of pulse rate, systolic blood pressure [SBP], diastolic blood pressure [DBP], mean arterial blood pressure [MAP], fever, and oxygen saturation percentage), laboratory results at the time of admission, electrocardiography results, time spent in the ED, hospitalization time, and discharge details were also documented in addition to the patients' demographic details. Deaths that occurred during hospitalization were also recorded.

Blood samples to measure biochemical and complete blood count parameters were taken. The complete blood count results included hemoglobin, hematocrit, and platelets readings. The following biochemical parameters were also evaluated: glucose, albumin, sodium, potassium, calcium, urea, creatinine, alanine transaminase, hs-TnI, creatine kinase myocardial band (CK-MB), and D-dimer levels.

2.3. Statistical analysis

All data were calculated and analyzed using SPSS v25.0 software. Data were expressed as mean ± standard deviation for continuous variables and percentages for categorical variables. The Kolmogorov–Smirnov test was used to determine the normality of continuous variables. Normally distributed variables were reported as mean ± standard deviation, while nonnormally distributed parameters were expressed as median with interquartile range ($IQR_{25–75}$). The Student *t* test and Mann–Whitney *U* were used to compare continuous data. The Chi-squared test was used to compare categorical variables. Variables determined to be significant in univariance studies were used in the multivariance logistic regression analysis. The receiver operating characteristic (ROC) curve analysis was used to evaluate the predictive power of 4 inflammatory indices: hs-TnI, CK-MB, D-dimer, D-dimer/CK-MB ratio, and D-dimer/hs-TnI ratio. The area under the curve (AUC; <0.50 indicating no discrimination and >0.80 indicating good discrimination) was calculated for each index to measure diagnostic accuracy along with 95% confidence interval for precision. ROC curves were plotted to illustrate the sensitivity and specificity, offering a clear comparison of their performance. The optimal cutoff value was established using the maximum Youden index. Statistical comparisons of AUCs were conducted using the DeLong method to ensure robust evaluation of diagnostic accuracy. All statistical analyses used 2-sided tests with a significance level (α) of 0.05.

Table 1

The patients' vital signs and laboratory findings at the moment of admission to the emergency department.

Variables	APE (n = 219)	NSTEMI (n = 385)	*P*-value*
Age (yr)	66.4 ± 14.8	61.8 ± 12.0	<.001
Female gender, n (%)	116 (53.0)	135 (35.1)	<.001
Temperature (°C)	36.6 ± 0.5	36.5 ± 0.3	.078
Pulse (beat/min)	106 ± 19	90 ± 14	<.001
SBP (mm Hg)	118 ± 27	133 ± 25	<.001
DBP (mm Hg)	75 ± 15	82 ± 13	<.001
MAP (mm Hg)	89 ± 17	100 ± 17	<.001
SaO_2 (%)	90 ± 8	96 ± 5.5	<.001
Laboratory parameters	**Mean ± SD**	**Mean ± SD**	
Glucose (mg/dL)	172 ± 85	136 ± 83	<.001
Urea (mg/dL)	51 ± 30	40 ± 21	<.001
Creatinine (mg/dL)	1.0 ± 0.5	0.9 ± 0.5	.049
Sodium (mmol/L)	137 ± 5	136 ± 4	.079
Potassium (mEq/L)	4.4 ± 0.6	4.3 ± 0.5	.077
Calcium (mg/L)	9.0 ± 2.4	9.4 ± 2.7	.149
ALT	26 ± 14.8	24 ± 12	.239
White blood cell	13.3 ± 4.8	11.9 ± 4.1	.083
Hemoglobin (g/dL)	12.1 ± 2.1	13.2 ± 1.8	<.001
Hematocrit (%)	36.8 ± 6.9	39.5 ± 5.4	<.001
Platelet ($\times 10^3$/uL)	246 ± 105	255 ± 72	.218
Albumin (g/L)	34.0 ± 5.7	37.6 ± 3.9	<.001

ALT = alanine transaminase, APE = acute pulmonary embolism, DBP = diastolic blood pressure, dL = deciliter, g = gram, L = liter, MAP = mean arterial blood pressure, mEq = miliequalan, mg = milligram, min = minute, mm Hg = millimeters of mercury, mmol = millimoles, NSTEMI = non-ST-elevation myocardial infarction, SaO_2 (%) = oxygen saturation percentage, SBP = systolic blood pressure, SD = standard deviation, *U* = unit.

* A *P*-value of <.05 was considered statistically significant. As appropriate, *P*-value was calculated using an independent samples *t* test or the Mann–Whitney *U*-test for continuous variables and a Chi-squared test or the Fishers exact test for categorical variables.

3. Results

Six hundred four patients were included of which 219 were patients diagnosed with APE and 385 with NSTEMI. The mean age of patients with APE (66.4 ± 14.8 years) was statistically higher than patients with NSTEMI (61.8 ± 12.0 years; $P < .001$). Of the patients with APE, 53% (n = 116) were female and 47% (n = 103) were male. Of the patients with NSTEMI, 64.9% (n = 250) were male, compared to 35.1% (n = 135) who

Table 2

Cardiac evaluation and mortality rates.

Variables	APE (n = 219)	NSTEMI (n = 385)	*P*-value*
Ejection fraction, (%)	50 ± 10	48 ± 11	.178
hs-TnI (pg/mL)	32 (12–153)	2099 (845–5250)	<.001
CK-MB (mcg/L)	2.4 (1.4–3.9)	16.5 (5.0–55.9)	<.001
D-dimer (mcg/L)	7620 (3810–15,390)	520 (260–1250)	<.001
D-dimer/hs-TnI ratio	214 (58–642)	0.28 (0.10–0.85)	<.001
D-dimer/ CK-MB ratio	3130 (1375–6115)	34 (10–110)	<.001

Values are presented as numbers (n) and percentages (%) or median (interquartile range).

P-value was calculated using the Mann–Whitney *U*-test for continuous variables and the Chi-squared test or the Fishers exact test for categorical variables, as appropriate.

APE = acute pulmonary embolism, CK-MB = creatine kinase-myocardial band, hs-TnI = high-sensitivity troponin I, mcg/L = microgram/liter, NSTEMI = non-ST-elevation myocardial infarction, pg/mL = picogram/milliliter.

* A *P*-value of <.05 was considered statistically significant.

were female. Compared to those with NSTEMI, the female sex ratio in patients diagnosed with APE was significantly higher ($P < .001$).

Patient vital signs at ED admission are summarized in Table 1. There were statistically significant differences in the mean pulse rate, SBP, DBP, MAP, and oxygen saturation between the APE and the NSTEMI patient groups ($P < .001$); however, no significantly statistical difference in the temperature measured. Laboratory values obtained at the time of ED admission are given in Table 1. Hemoglobin, hematocrit, urea, and albumin values were statistically significantly different between both groups (all $P < .001$).

Patient cardiac evaluations and mortality rates are summarized in Table 2 and Figure 1. The ejection fractions of the 2 patient groups did not differ significantly. The hs-TnI, CK-MB, D-dimer level, and D-dimer/CK-MB and D-dimer/hs-TnI ratios were statistically significantly different between both groups (all $P < .001$). After admission to the hospital, patients with APE had an in-hospital mortality rate of 21.1% (n = 46), whereas patients with NSTEMI had a rate of 2.9% (n = 11). The difference in mortality rates was significantly higher in patients with APE ($P < .001$).

The ROC analysis comparison of the performances of hs-TnI, D-dimer, CK-MB, D-dimer/CK-MB, and D-dimer/hs-TnI parameters in discriminating APE from NSTEMI are shown in Table 3 and Figure 2. According to the ROC analysis, all 5 variables were statistically significant and are shown in Table 4 (all $P < .001$). The ratio of D-dimer/hs-TnI (>4.4) had 99.5% sensitivity and 94.0% specificity (AUC 0.995) among the predictive values determined by the AUC, which suggests that this ratio is the most significant factor for differentiating APE from NSTEMI.

4. Discussion

Literature research reveals some reports of the usefulness of using the D-dimer/Troponin I ratio ratio to distinguish between APE and NSTEMI, both of which cause chest pain.[6] No studies have investigated the use D-dimer/hs-TnI ratio in this context. In our study, we found that D-dimer/hs-TnI (>4.4) had higher sensitivity and specificity than hs-TnI and D-dimer alone in differentiating APE from NSTEMI. The main conclusion of our study was that D-dimer/hs-TnI ratio could be useful as an independent and effective parameter to distinguish between APE and NSTEMI in patients presenting to the ED with chest pain.

Chest pain is one of the commonest complaints of patients presenting to the ED.[10] In patients presenting with chest pain, APE and ACS are 2 life-threatening conditions that should be identified or ruled promptly. Large-scale multicenter studies have shown that compared to traditional tests, hs-TnI tests improve the diagnostic accuracy for identifying NSTEMI and allow quicker diagnosis and exclusion of ACS, particularly in patients who present to the ED immediately following the onset of chest pain.[11] Another critical diagnosis among patients with chest discomfort is APE. Clinical findings, electrocardiography, laboratory parameters, and chest radiology are used in the diagnosis of APE.[12–14] The primary biomarker that is used to rule out the diagnosis of APE is D-dimer, which is a fibrin breakdown product formed following fibrin production and destruction.[15–18] The emergency medicine physicians frequently use plasma D-dimer levels to differentiate between ACS and APE.[19,20] Thrombus in the coronary arteries of patients with NSTEMI can result in increased D-dimer levels.[15] Given the pathophysiological mechanisms involved, it is well known that pulmonary embolism (PE) might also result in increased cardiac marker levels and right ventricular involvement.[3,21] Elevated cardiac troponin in PE is related to the prognosis and severity of the disease rather than the diagnosis.[22,23] According to some studies, patients with massive PE had greater cardiac troponin levels than patients without.[24,25] In our study, we observed that patients with APE had lower hs-TnI levels than those with NSTEMI.

In a study conducted by Kim et al, patients with APE had a significantly higher D-dimer/Troponin-I ratio (50.6 ± 85.3

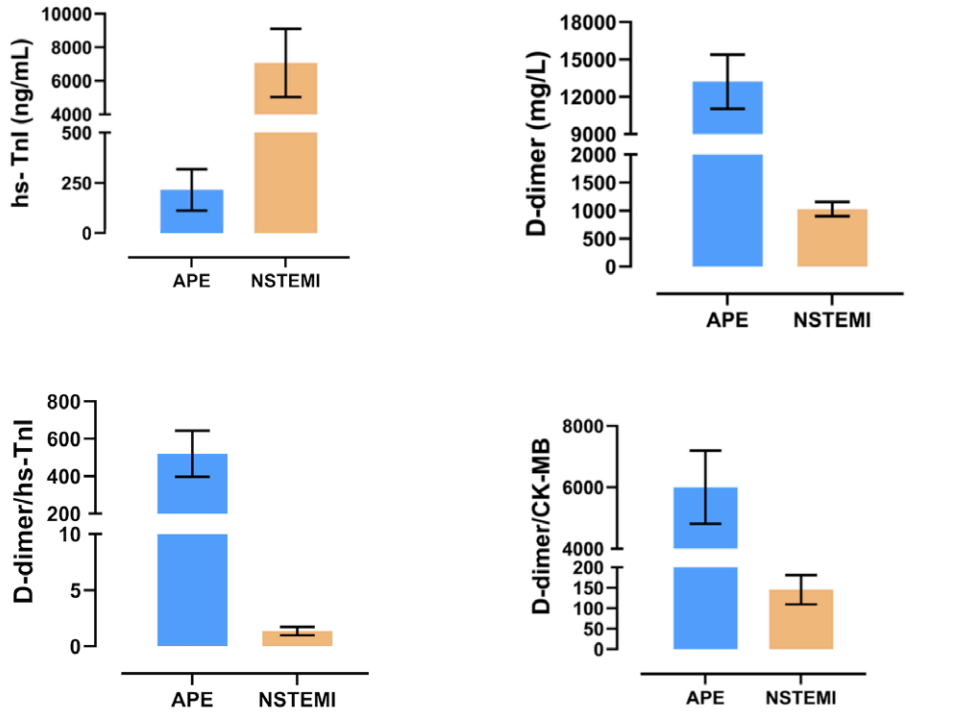

Figure 1. Analysis of hs-TnI, D-dimer, D-dimer/hs-TnI, D-dimer/CK-MB values in pulmonary embolism and NSTEMI patients. CK-MB = creatine kinase myocardial band, hs-TnI = high-sensitivity troponin I, NSTEMI = non-ST-elevation myocardial infarction.

vs 1.6 ± 5.7, $P < .001$) than those with NSTEMI. An D-dimer/Troponin-I ratio >1.82 has been reported to have higher sensitivity and specificity for discriminating APE with increased troponin from acute NSTEMI.[6] However hs-TnI was not the cardiac biomarker used in this study. Among cardiac troponins, hs-TnI has the highest sensitivity and specificity for the diagnosis of NSTEMI, hence why hs-TnI was used as the primary cardiac biomarker in our study.

Our study showed that the D-dimer/CK-MB ratio could be a useable marker for the differential diagnosis of APE and NSTEMI. We were unable to find any studies that looked into this ratio in our literature review. Nevertheless, the study by He et al showed that CK-MB alone can be used for the differential diagnosis of NSTEMI and APE.[26]

In our study, the female sex ratio was higher in the APE group, whereas the male sex ratio was higher in the NSTEMI group ($P < .001$), which is corroborated in the literature.[6,8]

Our research showed that patients with APE had significantly higher in-hospital mortality rates than those with NSTEMI. For patients with APE, hemodynamic stability is vital. According to a study, patients with APE had a higher 30-day mortality rate when their SBP levels decreased.[27] According to a large population-based study conducted in patients with acute myocardial infarction, low SBP is an independent and strong predictor of 1-year cardiovascular mortality.[28] Hemodynamic instability and high D-dimer levels in patients with APE are associated with the severity of APE. In our research, patients with APE had much lower SBP and DBP (including MAP, which is related to these 2 blood pressures),

Table 3

ROC analysis comparing performance of D-dimer, hs-TnI, CK-MB, D-dimer/CK-MB, and D-dimer/hs-TnI parameters in identifying non-ST-elevation myocardial infarction from pulmonary embolism.

Variable	AUC	Cutoff value†	Sensitivity (%)	Specifity (%)	SE	95% CI‡	*P*-value*
hs-TnI	0.942	≤544	93.2	80.3	0.010	0.92–0.96	<.001
CK-MB	0.855	≤4.4	79.9	77.5	0.002	0.82–0.88	<.001
D-dimer	0.952	>1565	94.5	83.6	0.009	0.94–0.97	<.001
D-dimer/hs-TnI ratio	0.995	>4.4	99.5	94.0	0.002	0.99–0.99	<.001
D-dimer/CK-MB ratio	0.980	>428.6	95.4	92.1	0.005	0.97–0.99	<.001

AUC = area under the curve, CI = confidence Interval, CK-MB = creatine kinase myocardial band, hs-TnI = high-sensitivity troponin I, SE = standard error.

† Calculated a maximal Youden index.

‡ Binomial exact.

* A *P*-value of <.05 was considered statistically significant.

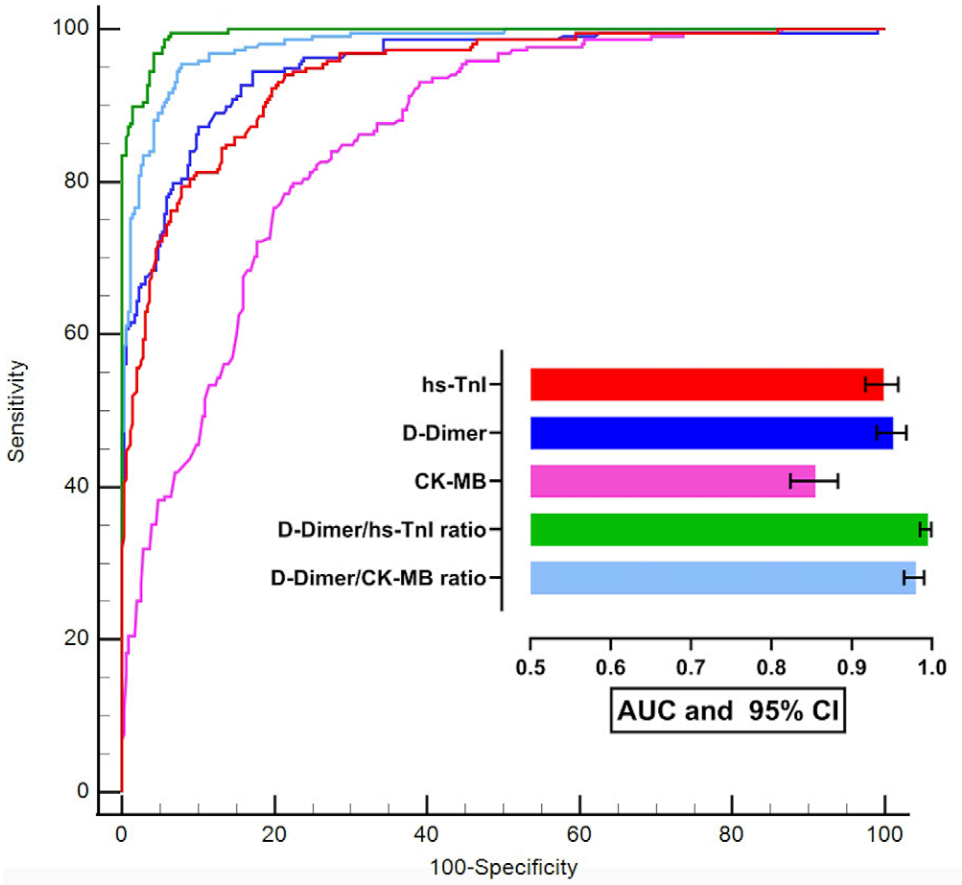

Figure 2. ROC analysis comparing performance of D-dimer, hs-TnI, D-dimer/CK-MB, and D-dimer/hs-TnI parameters in identifying NSTEMI from pulmonary embolism. CK-MB = creatine kinase myocardial band, hs-TnI = high-sensitivity troponin I, NSTEMI = non-ST-elevation myocardial infarction, ROC = receiver operating characteristic.

Table 4

Receiver operating characteristic curve analysis comparing performance of D-dimer, hs-TnI, D-dimer/CK-MB, and D-dimer/hs-TnI parameters in identifying non-ST-elevation myocardial infarction from pulmonary embolism.

Variables	Difference between AUC†	95% CI	*P*-value*
hs-TnI–D-dimer	0.0126	–0.02 to 0.04	0.363
hs-TnI–D-dimer/hs-TnI ratio	0.0553	0.04–0.07	<.001
hs-TnI–D-dimer/CK-MB ratio	0.0406	0.02–0.06	<.001
D-dimer–D-dimer/hs-TnI ratio	0.0427	0.03–0.06	<.001
D-dimer–D-dimer/CK-MB ratio	0.0281	0.01–0.04	<.001
D-dimer/hs-TnI ratio–D-dimer/CK-MB ratio	0.0146	0.01–0.02	<.001

AUC = area under the curve, CI = confidence interval, CK-MB = creatine kinase myocardial band, hs-TnI = high-sensitivity troponin I.

† DeLong et al[9].

* A $P < .05$ was considered statistically significant.

than those with NSTEMI. In addition to these findings, hemoglobin, hematocrit, urea, albumin, and glucose abnormalities were statistically significant in the APE group. These results might explain the significantly higher mortality rate in the APE group.[29–31] Early interventional revascularization could be the reason for less cardiac damage and lower mortality in patients with NSTEMI.

5. Limitations

This was a single-center, retrospective study. There was bias in the patient selection due to the nature of the retrospective design and the number of patients was relatively small. There was also a difference between the time onset of patients' symptoms and the time of blood tests. We could not obtain information about the patients' comorbidities and the medications they used. Since computed tomography pulmonary angiogram was not performed on all patients, APE that might have accompanied patients with NSTEMI could not be recognized.

6. Conclusion

Our research showed that the D-dimer/hs-TnI ratio is a useful and independent indicator in the differential diagnosis of APE and NSTEMI in patients who present to the ED with chest pain. We believe that emergency medicine physicians will benefit from this parameter because it is cheap, easily available, and quick to determine. Clearer results regarding the D-dimer/hs-TnI ratio's dependability can be obtained from large series, multicenter studies, and randomized controlled trials. Our work could serve as a foundation for further research.

Author contributions

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