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Prognostication of Incidence and Severity of Ischemic Stroke in Hot Dry Climate From Environmental and Non-Environmental Predictors

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ABSTRACT **Background**: Recently, rapid fluctuations of ambient temperature were found to be associated with hospital admission for cardiovascular diseases in general and the ischemic stroke in particular. **Objective**: to test if climatic factors predict the incidence of stroke reliably and to study the predictive potential of risk factors for a stroke. **Materials and methods**: In a retrospective design, we studied 566 patients admitted to the stroke unit in 2016-2019. A distributed lag nonlinear model was used to explore immediate and delayed effects of weather and clinicodemographic risk factors on the stroke incidence. Supervised machine learning was used to build models predictive of the mRS score. We assessed model performance by calculating R^2, mean absolute error and root-mean-square error. **Results and conclusions:** We found a non-correlation between the weather parameters and statistics on stroke. The disparities in their trends lead us to investigate behind time effects of the environment with distributed lag models and a concordant impact of all the settings - with machine learning models. If categorized into two classes by severity and functional outcomes, the cases have few disparities in the weather parameters within a week before the stroke onset. In contrast to the groups classified by severity, the ones grouped by outcomes have a significant difference in age, nationality, the presence of background diseases and smoking status. We ranked environmental and individual risk factors by the information gain that they provide to the models. Inclusion of the weather parameters into the machine learning model predicting the mRS score provides a slight boost in performance.

INDEX TERMS Ethnicity, ischemic stroke, machine learning classification model, middle east, risk, sex, weather.

I. INTRODUCTION

Stroke is one of the top 20 diseases contributing to life expectancy with disabilities. Over 7 million people die annually from cerebrovascular diseases worldwide. Half of them die of the ischemic stroke (IS) [1]. Arterial hypertension, atherosclerosis, atrial fibrillation, diabetes mellitus and smoking are commonly studied risk factors for IS [2]–[7]. The time of the stroke onset and the meteorological factors

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refer to additional risk factors which have not been studied well. In recent years, there has been a rise in interest to the impact of meteorological factors on public health because of the climate changes, observed and projected [8]–[10].

A. AMBIENT TEMPERATURE AS A RISK FACTOR OF IS

The effect of temperature on the population has received a particular attention of researchers [10]. Studies on its delayed effect (up to 7 days) have evidenced an impact of extreme cold weather on the risk of IS [11]. Relevant findings for hot dry climate are missing. Studies have provided discrepant findings onto whether an increase [12]–[14] or decrease [15]–[17] in the air temperature triggers IS.

It is challenging to elucidate pathogenic mechanisms of extreme heat or cold because of their cumulative effect and a lag of response to them [18], [19]. Furthermore, the perception of temperature changes according to relative humidity and wind power. Socioeconomic factors may also interact with the weather to affect the prevalence of IS. It is also necessary to consider the financial availability of air conditioners or heaters. Today the information on this issue is missing [20].

B. TEMPERATURE VARIANCE

Large-scale shifts in the weather patterns may put a burden on society as individuals are maladjusted to them. This increases the occurrence of cardiovascular disasters [21] and risk of mortality [22]. The temperature variance also has attracted particular attention of epidemiologists. They found that rapid fluctuations of ambient temperature are positively associated with hospital admission for cardiovascular diseases in general [23] and for IS in particular [24].

C. RELATIVE HUMIDITY (RH) AS A RISK FACTOR OF IS

RH changes the thermal conductivity of the air and thus contributes to the impact of ambient temperature [25]. An association between IS and ambient temperature is shown to be stronger on the days with high RH levels [16]. There is little evidence on the relationship of low partial pressure of oxygen and high RH levels with IS risk in summer [26]. Some authors examined the effects of changes in RH without considering other weather parameters. A meta-analysis of such studies done in Switzerland, Portugal, Russia, South Korea and Taiwan showed no association of the occurrence of IS with air humidity [27]. In support of these findings, a study conducted on different age groups in Slovenia did not show significant correlation [28]. So, the effects of RH, considered either apart or in combination with other meteorological findings, remain disputable for different geographic locations.

D. ATMOSPHERIC PRESSURE (AP) AS A RISK FACTOR OF IS

A supposed effect of AP on IS is that it exerts some stress on an atherosclerotic plaque and may contribute to its rupture [29]. Researchers have reported conflicting findings: while some studies did not find a significant relationship between AP changes and the IS occurrence [27], [28], [30] the others showed that a drop in AP increased the IS incidence within the next 24 hours [31]. This IS rate was consistent with the maximal and minimal values of AP [32]. Unfortunately, these data are not fully reliable. *First*, the authors carried out single center-based studies limited to a specific climatic zone. *Second*, the number of patients was also limited. *Third*, they did not take into consideration that the time of the IS onset may not coincide with hospitalization.

E. WIND SPEED (WS) AS A RISK FACTOR OF IS

The impact of wind speed on IS has not been studied well. Some authors collected facts that the number of IS cases increases when the maximum WS is low 3 days before the stroke [31]. An exploration found an association between the increased WS and a high prevalence of IS in the elderly [33]. Some authors did not consider confounding risk factors. In another observation, IS count was associated with the WS range and wind chill [34]. A later study justified an effect of age, sex, and smoking status on the IS morbidity [34]. Some scientists analysed changes in the air masses due to the wind and justified that dry polar air was associated with a lower IS risk [35]. Further studies are required to get an insight if the weather parameters interact with individual risk factors to influence the IS incidence in distinct geographic locations.

II. OBJECTIVES

We wanted to test if climatic factors predict the incidence of IS reliably and to study the predictive potential of IS risk factors. The hypothesis of the study was that harsh desert climate of the UAE is an additional environmental IS risk factor, worsening the disease course and outcomes. *The primary objective* was to estimate the associations of the IS incidence with weather parameters and clinicodemographic risk factors in the desert climate. We explored if climatic factors contribute to IS severity *(second objective)*. We also built a regression model to predict the outcome of IS at discharge *(third objective)*.

III. MATERIALS AND METHODS

A. DATASET DESCRIPTION

The data on de-identified IS cases was collected from the hospital information system in the city of Al Ain, Abu Dhabi Emirate. The dataset was labeled the PRAS_IS dataset after the project title ''Prognostication of Recovery from Acute Stroke''. The following weather parameters were obtained from the National Oceanic and Atmospheric Administration: daily ambient temperature (TEMP), relative humidity (RH), wind speed (WDSP) and AP. The number of days between the stroke onset and a given weather event was measured and expressed by a number after the acronym, e.g., WDSP7 is the wind speed 7 days before the stroke onset. We also calculated the humidity index (humidex) from ambient temperature and RH. From these data, we calculated the derivative parameters characterizing the weather. The daily change was calculated

TABLE 1. Incidence of ischemic stroke in Al Ain stratified by sex and age group.

Variable		Total number	Mean number per annum	Mean per 100,000 people	City population
IS cases	female gender	125	31.25	25.76	249'940
	male gender	441	110.25	139.86	315'310
	totally	**566**	**141.5**	**100.13**	**565'250**
	in 2016	135	-	25.76	524'000
	in 2017	113	-	20.40	554'000
	in 2018	146	-	24.96	585'000
	in 2019	172	-	28.76	598'000
Age groups	0-34 years	21	5.25	5.40	389'057
	35-44 years	86	21.50	82.22	104'595
	45-54 years	160	40.00	330.29	48'443
	55-64 years	139	34.75	827.09	16'806
	65-74 years	95	23.75	2001.69	4'746
	$\geq$75 years	65	16.25	4054.90	1'603

for air temperature (TDIF), pressure (PDIF), wind speed (WDIF), relative humidity (RHDIF) and humidex (HDIF).

We also analyzed the following possible clinicodemographic predictors of the IS incidence and outcomes: age (DEMOGRAPHY_age), sex (DEMOGRAPHY_sex), ethnicity (DEMOGRAPHY_ethnicity), body mass index (BMI), history of a stroke (History_OldStroke), history of smoking (History_Smoking), current diabetes mellitus (History_DM), arterial hypertension (History_HyperTension), ischemic heart disease (History_IschemicHeartDisease), arterial hypertension (History_ArterFibrillation), and hyperlipidemia (History_HyperLipidaemia), year of the stroke onset (year), day of the onset (ONSET_Date), time of day at the onset (ONSET_LKW_time), National Institutes of Health Stroke Scale (NIHSS) score at hospital admission (Screening_tools_NIHSS), final diagnosis (Diagnosis_Final), and mRS at discharge (Discharge_Plan_Modified_Rankin_Score). To classify the cases, we added DEMOGRAPHY_agerange as a categorical variable (age groups 18–44, 45–59, 60–74, 75–89 and >90 years).

B. STUDY DESIGN AND SUBJECTS

The primary outcome was the number of daily emergency hospital admissions for IS. We analyzed the records of the IS patients admitted to the stroke unit of Al Ain Hospital from January 1, 2016 to December 31, 2019 (tertiary level of care). The final study cohort included 566 patients (see Table 1). In accordance with the national healthcare standards, all the patients were examined by a neurologist; underwent brain CT, a complete etiologic review and other tests to fulfill the IS diagnostic criteria. The inclusion criteria were absence of intracranial bleeding on CT on admission and conformation of IS with diffusion restriction found on MRI [I63 in International Classification of Diseases (ICD-10) and 434.91 in ICD-9].

C. METHODOLOGY

To examine associations between the IS incidence and the weather parameters, we constructed plots (Fig. 1) representing the regional circannual weather changes and IS morbidity rates. We also examined the immediate and delayed effects of the weather and its daily changes on the IS incidence with distributed lag nonlinear model (DLNM) analysis (Fig. 2).

Working on the second objective, we aimed to find out how clinicodemographic and the weather factors impact IS severity. We conducted a comparative analysis of the groups classified by NIHSS score with a threshold of 4 (362 minor vs 204 moderate and severe cases) and by mRS with a threshold of 2 (371 absent or slight disability vs 195 cases of moderate to severe disability and deaths). As the variables had non-normal distribution, we utilized non-parametric tests. The relationships between the continuous features were assessed with Kruskal-Wallis test.

The idea of the third objective was to build a machine learning model prognosticating the IS early outcomes (mRS score). By selecting the top 20 informative features to train the regression model in the 10-fold cross-validation technique, we highlighted the importance of the weather conditions in the ethyopathogenesis. We compared the strength of associations between the input variables and the values of NIHSS and mRS with Pearson's correlation. Fig. 3A-B show the ranked variables. With MAE, RMSE and R^2, we compared the performance of the prediction from different sets of predictors (see Table 5). For details on the models see Supplemental Material at https://www.dropbox.com/s/qx56wqh94u0p3bj/SupplemtalMaterial.pdf?dl=0.

D. STATISTICAL ANALYSIS

We calculated descriptive statistics for the groups with different levels of severity on admission and outcomes at discharge in a standard way. The discontinuous variables were presented as Mean±SD, while for the categorical ones the percentage was calculated. We resorted to Pearson's correlation to show the associations between the factors. This allowed us to reduce the "noise" from useless variables, thereby improving the model outcome metrics with the top valuable features: "NIHSS_score", "DEMOGRAPHY_age", "Lab_Investigation_C-reactive

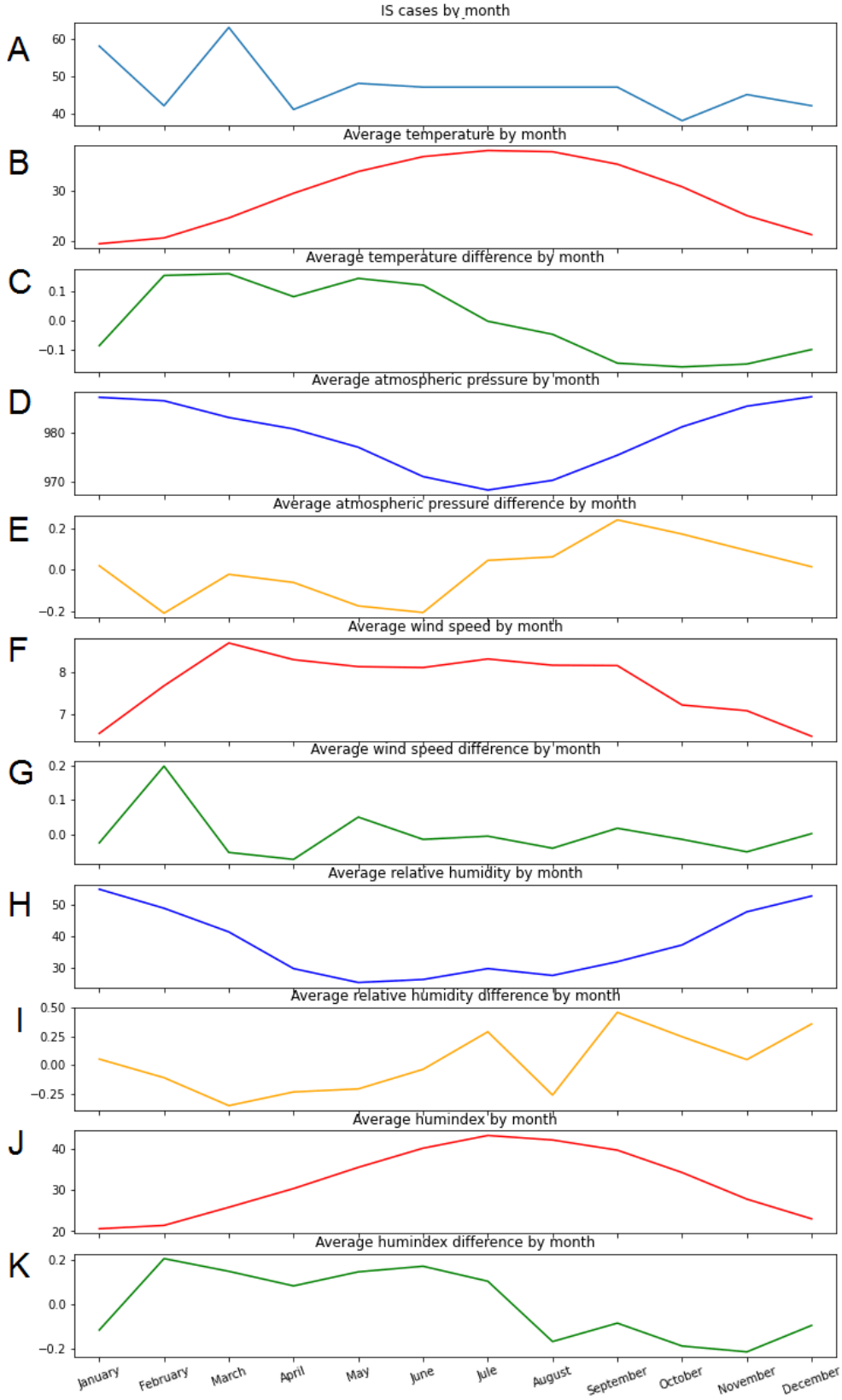

FIGURE 1. Plots of average monthly air temperature, average daily changes, mean relative humidity, and incidence of ischemic stroke by month.

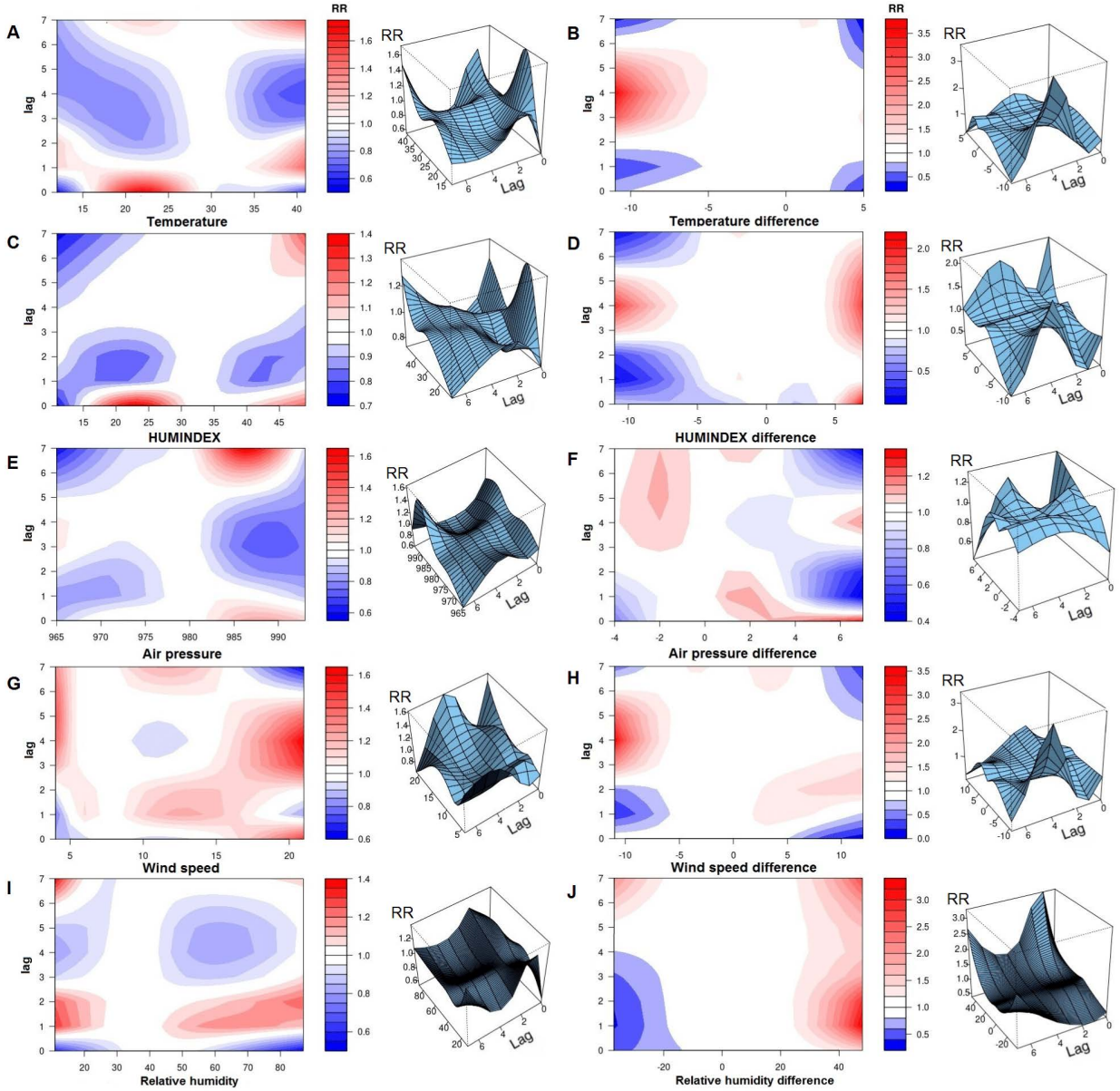

FIGURE 2. Contour exposure–lag-response plots and three-dimensional exposure–lag-response plots of ischemic stroke risk versus ambient temperature (a, b), perceived temperature (c, d), atmosphere pressure (e, f), wind speed (g, h), relative humidity (i, j) and daily changes in them (lag = 7 days).

protein'', ''History_OldStroke'', ''History_DM'', ''History_IschemicHeart Disease'', ''History_HyperTension'', ''DEMOGRAPHY_nati-onality'', ''History_ArterFibrillation'', ''WDIF3'', ''RH'', ''Lab_Investigation_Creatinine'', ''TDIF4'', ''RH1'', ''WDSP2'', ''TDIF5'', ''isMultiple'', ''History_HyperLypidAemia'', ''WDSP1'', ''Hemorrhagic transformation''.

IV. RESULTS

A. EFFECTS OF WEATHER PARAMETERS ON STROKE INCIDENCE BY AGE AND SEX

Table 1 summarizes the IS incidence as estimated on admission. According to the recent census, the population of Al Ain was 560,250 with a greater proportion of males (56%). The mean number of IS cases per annum was 141.5 (100.13 per 100,000 people). It was three and a half times as common in males as in females (110.25, 39.01 vs 31.25, 11.057 per 100,000). Notably, the occurrence of IS rises dramatically across the lifespan.

Table 1 presents the average number of cases treated in the stroke unit in 2016-2019. Table 2 illustrates the basic descriptive statistics for the meteorological factors and their association with the IS incidence. There is no correlation between the average and daily changes of the meteorological factors and the incidence by month. The plots in Fig. 1A-1K depict disparities in the trends of the weather parameters and statistics on IS. Therefore, we studied the behind-time effects of the environment with DLNM. Machine learning models were used to describe a concordant impact of all the settings.

TABLE 2. Associations between meteorological factors and ischemic stroke incidence.

Variable		Mean ± SD	Median [IQR]	Min - Max		Correlation with IS incidence	
						r-coefficient	p-value
Temperature, $^\circ C$	absolute value	29.363 ± 7.146	30.389 [22.778 ÷ 35.889]	11.667	41.889	-0.03	0.255
	daily change	-0.003 ± 1.59	0.056 [-0.722 ÷ 0.889]	-11.611	5.5	0.004	0.889
Air pressure, *mbar*	absolute value	979.45 ± 7.007	980.5 [973.3 ÷ 980.6]	964.1	993.5	0.014	0.586
	daily change	-0.002 ± 1.406	0 [-0.8 ÷ 0.7]	-4.7	7.5	0.018	0.503
Wind speed, *knot*	absolute value	7.745 ± 1.952	7.5 [6.5 ÷ 8.7]	3.7	21.1	0.009	0.719
	daily change	-0.001 ± 1.885	-0.1 [-1 ÷ 1]	-11.9	12.1	-0.023	0.373
Relative humidity, %	absolute value	37.869 ± 14.939	36.216 [25.236 ÷ 48.488]	10.86	87.73	0.042	0.112
	daily change	0.022 ± 9.137	-0.067 [-5.244 ÷ 5.018]	-37.827	48.939	-0.019	0.461
Humindex, $^\circ C$	absolute value	32.081 ± 8.476	32.544 [24.741 ÷ 39.591]	10.188	49.955	-0.019	0.458
	daily change	-0.002 ± 1.847	0.037 [-0.895 ÷ 1.093]	-11.074	7.574	-0.007	0.799

Fig. 2 displays the immediate and delayed outcomes of ambient temperature, perceived temperature (humidex), air pressure, wind speed and their daily changes in terms of the IS risk. Contour plot 2A shows that a decrease in the risk follows extremely low and especially high levels of ambient temperatures with a 3- to 6-day lag.

From Fig. 2B, the risk reduces for a day after considerable changes of the air temperature. There is a rise in risk with a 3- to 5-day lag after a temperature drop by -5°C and more. This may reflect maladjustment of the population living in the desert climate to a dramatic drop in ambient temperature. Both a rise in humindex above 35°C and its drop below 28°C leads to a decreased IS risk, and the effect lasts 3-4 days. Negative changes in humindex below -5°C/day reduce the incidence of IS up to 3 days. Conversely, the positive changes in humindex result in the opposite trend lasting a day (see Fig. 2C-D). A week after the day of exposure IS risk falls back to the baseline for the major weather events. A decline in RH fosters a decreased IS risk values and vice versa.

An increase in air pressure above 980 mbar is associated with a slight rise in IS risk on the day of exposure (see Fig. 2E-F). From day 1 to 7 after the event, we see the opposite tendency. Both high and low absolute values of WS have a positive impact on the number of IS cases. The DLNM diagram for WD looks similar to the one on TD (see Fig. 2G-H). Fig. 2I-J present data for relative humidity.

B. IMPACT OF WEATHER PARAMETERS AND INDIVIDUAL RISK FACTORS ON STROKE SEVERITY

Table 3 provides descriptive statistics on the cases grouped by the level of severity and early outcomes. There is a discrepancy between the distribution of cases over severity on admission and the early outcomes on discharge. The level of severity or outcomes does not vary with the IS occurrence daytime which is presented with four six-hour time intervals. To present the same variable in ML more accurately, we encoded daytime and month of the IS onset with Cat2Vec technique [36].

In contrast to the groups classified by NIHSS score, the ones grouped by mRS had a significant difference in age ($p = 2.14e-06$), nationality ($p = 0.0008$), background diseases and smoking ($p = 7.53e-07$). The separability with regard to laboratory findings (levels of troponine I and C-reactive protein) is also more pronounced in the cohorts classified by mRS.

Patients with lacunar infarctions are more likely to present with a mild than a severe IS form (23.2 vs 25%; $p = 0.001$). The variance of IS location across distinct vascular territories differs pronouncedly in the groups stratified by both NIHSS and mRS ($p = 0.0007$ and $p = 0.03$). A major part of infarctions with multiple lesions and the ones allocated in the middle cerebral artery vascular territory have NIHSS score under 4 and mRS below 3. Contrarily to this, a mild clinics and early outcomes are characteristic of IS in such vascular territories as the anterior and posterior cerebral, cerebellar, basilar, vertebral arteries and the watershed area.

If categorized into two classes by NIHSS and mRS values, the cases have few disparities in the weather parameters within a week before the stroke onset (see Table 4). Two classes of NIHSS values differ substantially in RH two days before IS ($p = 0.02$ - 0.04), and two classes of mRS values - in WS on the 4-th and 7-th day ($p = 0.01$ - 0.04), and in WD the day before ($p = 0.016$ - 0.013).

C. EARLY OUTCOMES OF STROKE

Fig. 3 shows environmental and individual risk factors ranked by the information gain they provide to the model predicting NIHSS value. The top valuable predictors are background diseases: arterial fibrillation, hypertension, diabetes mellitus and smoking. They are followed by the weather parameters and the Cat2Vec-encoded data on the onset of the stoke in the calendar year [cos(day of year), sin(month), sin(day of year)]. Hyperlipidemia is the top-rank biochemical predictor as it is a marker of atherosclerosis. A negative association of arterial hypertension with IS severity can be explained by the medications prescribed. The total number of background diseases is positively associated with the metric of early outcomes. However, some background diseases may remain misdiagnosed and, therefore, not treated.

To predict the early IS outcomes from different features, we employed six conventional ML regressors (SVM non-linear, Gradient Boosting, AdaBoost, Random Forest, Lasso, and K Nearest Neighbours). The models were trained until convergence. To generalize our solution to the true-rate

TABLE 3. Comparison of clinicodemographic parameters among ischemic stroke cases classified by severity and early outcomes.

		Total n=566	NIHSS Score			modified Ranking Score		
			NIHSS$\leq$ 4 n_1=362(63.96%)	NIHSS > 4 n_2=204(36.04%)	p_{1-2}	mRS $\leq$ 2 n_3=371(65.55%)	mRS > 2 n_4=195(34.45%)	p_{3-4}
EPIDEMIOLOGY AND SEVERITY OF CASES								
Year	2016	135(23.85%)	75(20.72%)	60(29.41%)		82(22.1%)	53(27.18%)	
	2017	113(19.96%)	78(21.55%)	35(17.16%)	0.120496	65(17.52%)	48(24.62%)	**0.046502**
	2018	146(25.8%)	96(26.52%)	50(24.51%)		103(27.76%)	43(22.05%)	
	2019	172(30.39%)	113(31.22%)	59(28.92%)		121(32.61%)	51(26.15%)	
NIHSS score		4.52[1.0-6.0]	1.62±1.36	9.68±4.94	**1.73e-88**	3.11±3.68	7.21±5.98	**1.26e-20**
mRS score		1.93[1.0-4.0]	1.41±1.34	2.85±1.55	**3.89e-24**	0.9±0.72	3.89±0.62	**2.63e-90**
NIHSS group	low	362(63.96%)	–	–	–	291(78.44%)*	71(36.41%)*	**8.66e-23**
	high	204(36.04%)	–	–		80(21.56%)*	124(63.59%)*	
MRS group	low	371(65.55%)	291(80.39%)*	80(39.22%)*	**8.66e-23**	–	–	–
	high	195(34.45%)	71(19.61%)*	124(60.78%)*		–	–	
Onset day time	morning	94(16.61%)	53(14.64%)	41(20.1%)		59(15.9%)	35(17.95%)	
	afternoon	168(29.68%)	110(30.39%)	58(28.43%)	0.410074	109(29.38%)	59(30.26%)	0.858217
	evening	180(31.8%)	119(32.87%)	61(29.9%)		122(32.88%)	58(29.74%)	
	night	121(21.38%)	78(21.55%)	43(21.08%)		80(21.56%)	41(21.03%)	
	month	6.22[3.0-9.0]	6.2±3.35	6.27±3.67	0.424045	6.22±3.48	6.23±3.45	0.491879
	day of week	2.85[1.0-4.0]	2.8±1.93	2.93±2.03	0.249068	2.85±1.98	2.85±1.94	0.485391
	day of year	173.63[78.25-263.0]	172.22±102.61	176.13±112.37	0.346884	173.33±106.51	174.19±105.74	0.447395
DEMOGRAPHICS, PERSONAL RISK FACTORS								
Age, $years$		56.81[47.0-66.0]	56.91±13.8	56.63±13.67	0.411235	54.85±13.22	60.55±13.99	**2.14e-06**
BMI		28.26[24.02-30.74]	28.53±8.15	27.77±6.34	0.164550	28.21±7.53	28.35±7.6	0.489967
Sex	males	441(77.92%)	278(76.8%)	163(79.9%)	0.460164	292(78.71%)	149(76.41%)	0.524708
	females	125(22.08%)	84(23.2%)	41(20.1%)		79(21.29%)	46(23.59%)	
Nationality	Asian	281(49.65%)	172(47.51%)	109(53.43%)		199(53.64%)*	82(42.05%)*	
	Arab	180(31.8%)	122(33.7%)	58(28.43%)	0.228032	120(32.35%)	60(30.77%)	**0.000818**
	UAE	101(17.84%)	64(17.68%)	37(18.14%)		49(13.21%)*	52(26.67%)*	
	Others	4(0.71%)	4(1.1%)	0(0.0%)		3(0.81%)	1(0.51%)	
BACKGROUND DISEASES								
OldStroke		85(15.01%)	54(14.91%)	31(15.19%)	0.455280	41(11.05%)	44(22.56%)	**0.000144**
Diabetes mellitus		305(53.88%)	206(56.91%)	99(48.53%)	**0.029450**	183(49.33%)	122(62.56%)	**0.001228**
Hypertension		435(76.86%)	289(79.83%)	146(71.57%)	**0.029238**	272(73.32%)	163(83.59%)	**0.006306**
Ischemic heart disease		67(11.84%)	40(11.05%)	27(13.24%)	0.498263	32(8.63%)	35(17.95%)	**0.001547**
Arterial fibrillation		46(8.13%)	23(6.35%)	23(11.27%)	*0.053477*	24(6.47%)	22(11.28%)	*0.052641*
Hyperlypidaemia		63(11.13%)	38(10.5%)	25(12.25%)	0.578075	36(9.7%)	27(13.85%)	0.15945
Smoking		141(24.91%)	98(27.07%)	43(21.08%)	0.129051	116(31.27%)	25(12.82%)	**7.53e-07**
LABORATORY FINDINGS DISEASES								
Troponine I		0.21[0.0-0.02]	0.26±3.71	0.1±0.53	**0.009486**	0.26±3.73	0.11±0.53	**4.33e-05**
Inversed normalized ratio		1.27[0.94-1.03]	1.39±2.43	1.03±0.23	**0.040077**	1.38±2.35	1.0±0.15	0.184509
C-reactive protein		12.43[1.43-9.73]	11.64±29.53	13.76±28.66	**0.000597**	8.95±20.29	18.07±38.97	**4.06e-07**
Total choleserol		13.02[3.74-5.39]	17.88±239.04	4.5±1.26	0.086549	17.46±235.15	4.49±1.25	0.104429
Low density lipoprotein		9.21[2.27-3.84]	12.72±173.62	3.03±1.18	0.226266	12.45±171.04	2.99±1.19	0.114936
Atherogenic index		2.17[1.5-2.6]	2.14±1.41	2.21±1.03	0.437810	2.2±0.99	2.1±1.72	0.140666
POC_Random blood sugar		8.25[5.6-9.7]	8.35±3.86	8.07±3.75	0.142876	8.16±3.89	8.42±3.7	0.083498
Creatinine		87.78[66.0-95.0]	87.81±49.61	87.72±55.96	0.347588	84.61±39.07	93.86±70.04	0.064143
MRI FINDINGS								
Hemorrhagic transformation		53(9.36%)	31(8.56%)	22(10.78%)	0.452583	34(9.16%)	19(9.74%)	0.879541
Lacunar stoke		109(19.26%)	84(23.2%)	25(12.25%)	**0.001293**	75(20.22%)	34(17.44%)	0.501068
Multiple lesions		153(27.03%)	96(26.52%)	57(27.94%)	0.767564	94(25.34%)	59(30.26%)	0.232276
Side of lesion	right	243(42.93%)	154(42.54%)	89(43.63%)		165(44.47%)	78(40.0%)	
	left	264(46.64%)	170(46.96%)	94(46.08%)	0.969082	170(45.82%)	94(48.21%)	0.525197
	bilateral	59(10.42%)	38(10.5%)	21(10.29%)		36(9.7%)	23(11.79%)	
Vascular territory	middle cerebral	299(52.83%)	175(48.34%)*	124(60.78%)*		184(49.6%)*	115(58.97%)*	
	vertebral and basilar	71(12.54%)	51(14.09%)	20(9.8%)		46(12.4%)	25(12.82%)	
	posterior cerebral	69(12.19%)	54(14.92%)*	15(7.35%)*		56(15.09%)*	13(6.67%)*	
	multiple	59(10.42%)	31(8.56%)	28(13.73%)		34(9.16%)	25(12.82%)	
	cerebellar	37(6.54%)	31(8.56%)*	6(2.94%)*		29(7.82%)	8(4.1%)	
	anterior cerebral	12(2.12%)	9(2.49%)	3(1.47%)	**0.000689**	7(1.89%)	5(2.56%)	**0.030604**
	watershed	9(1.59%)	7(1.93%)	2(0.98%)		7(1.89%)	2(1.03%)	
	internal carotid	5(0.88%)	3(0.83%)	2(0.98%)		5(1.35%)	0(0.0%)	
	anterior choroidal	3(0.53%)	0(0.0%)*	3(1.47%)*		1(0.27%)	2(1.03%)	
	anterior cerebral	1(0.18%)	1(0.28%)	0(0.0%)		1(0.27%)	0(0.0%)	
	venous infarction	1(0.18%)	0(0.0%)	1(0.49%)		1(0.27%)	0(0.0%)	

*If the variance of a variable differs significantly ($p < 0.05$) from other cases taken together, its $Mean \pm SD$ is marked with an asterisk.
The significant differences between cohorts are marked in bold.

TABLE 4. Comparison of weather parameters on days preceding ischemic stroke of distinct severity and early outcomes.

	Days before stroke	Total n=566	NIHSS Score			modified Ranking Score		
			NIHSS $\leq$ 4 n_1=362(63.96%)	NIHSS > 4 n_2=204(36.04%)	p_{1-2}	mRS $\leq$ 2 n_3=371(65.55%)	mRS > 2 n_4=195(34.45%)	p_{3-4}
WEATHER PARAMETERS WITHIN A WEEK BEFORE STROKE								
Temperature, $^{\circ}C$	0	29.7[23.28-36.01]	30.02±7.05	29.12±6.89	0.0707123	29.58±7.08	29.92±6.86	0.304969
	1	29.65[22.99-36.12]	29.96±7.15	29.08±7.02	0.0841308	29.53±7.19	29.87±6.98	0.27604
	2	29.66[23.33-35.67]	29.91±7.1	29.21±6.92	0.13143	29.51±7.13	29.94±6.88	0.244159
	3	29.5[23.43-35.79]	29.77±7.19	29.01±6.99	0.0964896	29.28±7.35	29.92±6.67	0.202608
	4	29.34[22.65-35.74]	29.71±7.15	28.66±7.08	*0.05728*	29.16±7.27	29.67±6.88	0.218522
	5	29.47[22.72-35.79]	29.72±7.13	29.02±6.97	0.154388	29.34±7.24	29.72±6.77	0.354178
	6	29.34[22.82-35.67]	29.59±7.21	28.89±6.89	0.105537	29.17±7.32	29.66±6.67	0.28464
	7	29.29[22.33-35.89]	29.55±7.2	28.84±7.23	0.153111	29.07±7.35	29.72±6.92	0.167705
Atmospheric pressure, $mbar$	0	979.24[973.2-985.12]	978.93±7.01	979.79±6.64	0.0884093	979.38±6.99	978.97±6.69	0.208791
	1	979.17[973.4-984.82]	978.95±6.94	979.56±6.81	0.157933	979.3±6.97	978.92±6.76	0.233129
	2	979.07[973.13-984.9]	978.82±6.98	979.51±7.13	0.126161	979.13±7.02	978.95±7.08	0.361886
	3	979.28[973.4-984.8]	979.03±7.01	979.74±6.65	0.145652	979.37±7.03	979.11±6.63	0.247751
	4	979.16[972.77-985.05]	978.88±7.0	979.65±6.81	0.102131	979.22±7.03	979.03±6.76	0.351149
	5	979.23[973.2-985.3]	978.89±7.1	979.82±7.25	*0.0673457*	979.21±7.29	979.26±6.93	0.474549
	6	979.35[973.15-985.6]	979.08±7.03	979.82±7.25	0.111861	979.33±7.17	979.38±7.03	0.499892
	7	979.36[973.27-985.32]	979.09±7.1	979.84±7.32	0.0843787	979.42±7.23	979.25±7.11	0.370472
Wind speed, $knot$	0	7.84[6.5-8.8]	7.8±1.93	7.91±2.09	0.403019	7.84±1.99	7.84±1.99	0.484828
	1	7.95[6.4-8.93]	7.84±1.93	8.16±2.34	0.149447	7.94±2.03	7.98±2.2	0.42905
	2	7.73[6.5-8.6]	7.69±1.83	7.8±2.02	0.243733	7.69±1.98	7.8±1.75	*0.0576691*
	3	7.9[6.5-8.8]	7.9±2.03	7.9±2.4	0.132535	7.85±1.96	7.99±2.51	0.386254
	4	7.98[6.5-8.9]	8.03±2.09	7.88±2.19	0.0938021	8.12±2.16	7.71±2.05	**0.0135324**
	5	7.74[6.5-8.6]	7.75±2.04	7.72±2.05	0.381578	7.83±2.24	7.55±1.58	0.345392
	6	7.72[6.4-8.7]	7.7±1.78	7.75±1.86	0.437463	7.75±1.84	7.67±1.77	0.367167
	7	7.82[6.5-8.7]	7.8±2.1	7.85±2.24	0.434702	7.73±2.14	7.99±2.17	**0.0409716**
Relative humidity, %	0	37.02[25.7-46.73]	36.13±13.61	38.61±14.55	**0.0249914**	36.54±13.59	37.92±14.72	0.15573
	1	37.25[25.5-47.84]	36.36±14.04	38.84±15.38	**0.0425925**	36.89±14.2	37.92±15.26	0.280876
	2	37.19[24.98-48.44]	36.73±14.74	38.01±14.24	0.0974633	37.27±14.49	37.05±14.75	0.47455
	3	37.51[24.92-47.97]	37.06±15.11	38.34±15.25	0.169178	37.79±15.4	36.99±14.72	0.322599
	4	38.08[25.43-48.81]	37.33±14.41	39.42±14.97	0.06224	38.39±14.95	37.49±14.04	0.327978
	5	37.82[25.49-48.75]	37.01±14.78	39.26±14.83	0.0253716	37.98±14.71	37.51±15.06	0.324942
	6	37.57[24.93-48.42]	37.19±14.37	38.25±14.72	0.211685	37.83±14.85	37.07±13.8	0.331519
	7	37.32[24.67-48.91]	36.63±14.87	38.55±15.33	0.0812183	37.65±14.99	36.69±15.18	0.20872
Humindex, $^{\circ}C$	0	32.45[25.02-39.89]	32.74±8.5	31.92±8.26	0.128188	32.2±8.56	32.92±8.14	0.187192
	1	32.37[25.08-40.17]	32.64±8.45	31.87±8.35	0.138988	32.14±8.55	32.8±8.16	0.219407
	2	32.41[25.79-40.07]	32.67±8.53	31.96±8.42	0.168974	32.24±8.69	32.74±8.11	0.286489
	3	32.23[24.84-39.71]	32.52±8.61	31.7±8.44	0.121592	31.94±8.78	32.77±8.09	0.171897
	4	32.12[24.84-39.59]	32.52±8.58	31.4±8.47	0.071007	31.9±8.68	32.53±8.31	0.198801
	5	32.25[24.91-40.08]	32.45±8.55	31.91±8.35	0.235094	32.11±8.69	32.53±8.06	0.324942
	6	32.03[24.69-39.25]	32.32±8.69	31.5±8.17	0.11687	31.82±8.77	32.42±8.01	0.245785
	7	31.85[24.23-39.5]	32.08±8.56	31.43±8.52	0.19441	31.61±8.8	32.29±8.05	0.195408
DAILY CHANGES OF WEATHER WITHIN THE SAME WEEK								
Temperature difference, $^{\circ}C$	1	0.05[-0.79-0.89]	0.06±1.5	0.03±1.64	0.371769	0.05±1.54	0.05±1.57	0.323945
	2	-0.16[-1.11-0.96]	-0.13±2.03	-0.22±2.26	0.392118	-0.19±2.17	-0.1±2.0	0.370362
	3	-0.11[-2.01-1.61]	-0.11±3.24	-0.1±4.3	0.397725	-0.03±3.55	-0.25±3.84	0.226366
	4	-0.13[-3.11-3.06]	-0.4±5.85	0.35±5.73	0.117874	-0.2±5.8	-0.01±5.84	0.382021
	5	0.6[-4.9-6.78]	0.59±11.25	0.6±10.85	0.432173	0.37±11.21	1.02±10.9	0.228834
	6	0.22[-10.9-10.83]	0.11±20.3	0.42±18.66	0.45612	0.19±19.94	0.28±19.32	0.374277
	7	0.36[-20.83-23.07]	2.1±41.03	-2.76±40.05	0.150202	1.86±41.79	-2.47±38.53	0.166687
Pressure difference, $mbar$	1	0.07[-0.7-0.82]	-0.01±1.42	0.23±1.43	*0.0605182*	0.09±1.42	0.05±1.45	0.499024
	2	0.14[-0.9-1.4]	0.13±1.74	0.17±1.88	0.374822	0.16±1.8	0.1±1.77	0.352639
	3	-0.19[-1.7-1.5]	-0.12±2.89	-0.33±2.94	0.195724	-0.17±2.79	-0.24±3.13	0.383048
	4	0.05[-3.0-3.3]	0.08±4.86	-0.01±5.23	0.281164	0.02±4.83	0.09±5.29	0.146179
	5	-0.06[-4.8-5.43]	-0.16±9.33	0.12±9.12	0.389229	0.04±9.38	-0.25±9.01	0.229984
	6	-0.74[-11.4-8.4]	0.03±16.57	-2.11±17.88	0.0929461	-0.66±16.86	-0.88±17.5	0.435798
	7	1.06[-16.2-19.6]	0.29±30.6	2.44±32.7	0.271173	0.07±32.08	2.93±29.93	0.132516
Wind speed difference, $knot$	1	-0.11[-1.12-1.0]	-0.04±1.77	-0.25±1.93	0.295659	-0.1±1.84	-0.14±1.81	0.42841
	2	0.06[-1.6-1.6]	-0.0±2.9	0.16±2.95	0.221702	0.04±2.84	0.09±3.06	0.40763
	3	0.03[-2.6-2.42]	-0.22±4.94	0.49±5.29	0.074995	-0.32±5.16	0.71±4.84	**0.0163922**
	4	-0.34[-6.1-4.93]	-0.14±10.24	-0.7±10.25	0.165662	-0.4±10.3	-0.23±10.15	0.474658
	5	1.86[-7.6-11.72]	1.68±19.34	2.17±15.84	0.228191	1.64±18.47	2.28±17.58	0.0833779
	6	0.3[-18.22-18.0]	0.36±31.59	0.19±29.12	0.473252	-1.64±30.21	3.98±31.35	**0.0139776**
	7	-1.66[-39.4-33.45]	-1.88±58.18	-1.26±59.54	0.39223	-1.17±61.14	-2.57±53.66	0.416522
Relative humidity difference, %	1	-0.23[-4.94-4.44]	-0.23±8.95	-0.23±8.93	0.4155	-0.35±9.02	0.0±8.79	0.363315
	2	-0.55[-7.48-6.37]	-0.63±12.87	-0.42±14.13	0.285553	-0.16±13.57	-1.3±12.84	0.101278
	3	0.86[-11.38-12.79]	0.69±21.58	1.17±24.25	0.497205	0.3±22.66	1.92±22.37	0.297873
	4	-1.21[-23.01-21.54]	0.41±40.21	-4.11±38.49	0.192046	0.55±41.01	-4.54±36.74	0.151931
	5	-2.29[-38.96-32.96]	-2.51±73.54	-1.9±70.73	0.276287	0.21±75.69	-7.02±65.94	0.0745491
	6	-2.02[-83.69-79.86]	-2.52±141.62	-1.13±129.04	0.486672	0.01±141.88	-5.87±127.95	0.360261
	7	0.13[-152.87-164.91]	-8.53±293.99	15.65±284.96	*0.067456*	-8.04±296.4	15.58±279.91	0.0982327
Humindex difference, $^{\circ}C$	1	0.08[-0.85-1.12]	0.1±1.77	0.05±1.82	0.455159	0.06±1.74	0.13±1.87	0.355899
	2	-0.29[-1.76-1.09]	-0.24±2.32	-0.39±2.51	0.315162	-0.18±2.29	-0.51±2.55	*0.0669074*
	3	0.01[-2.15-2.08]	-0.0±4.22	0.02±3.97	0.442366	-0.02±4.16	0.05±4.09	0.48494
	4	-0.26[-4.27-4.02]	-0.23±7.56	-0.31±7.36	0.396483	-0.2±7.55	-0.37±7.36	0.372118
	5	0.28[-6.63-7.1]	0.62±12.76	-0.31±13.24	0.232122	0.84±12.82	-0.77±13.12	0.206543
	6	-0.44[-14.81-13.81]	-0.78±23.01	0.18±23.98	0.307543	0.29±23.64	-1.81±22.78	0.17721
	7	0.43[-27.44-26.67]	1.1±47.02	-0.78±46.54	0.4957	0.81±49.52	-0.29±41.34	0.442653

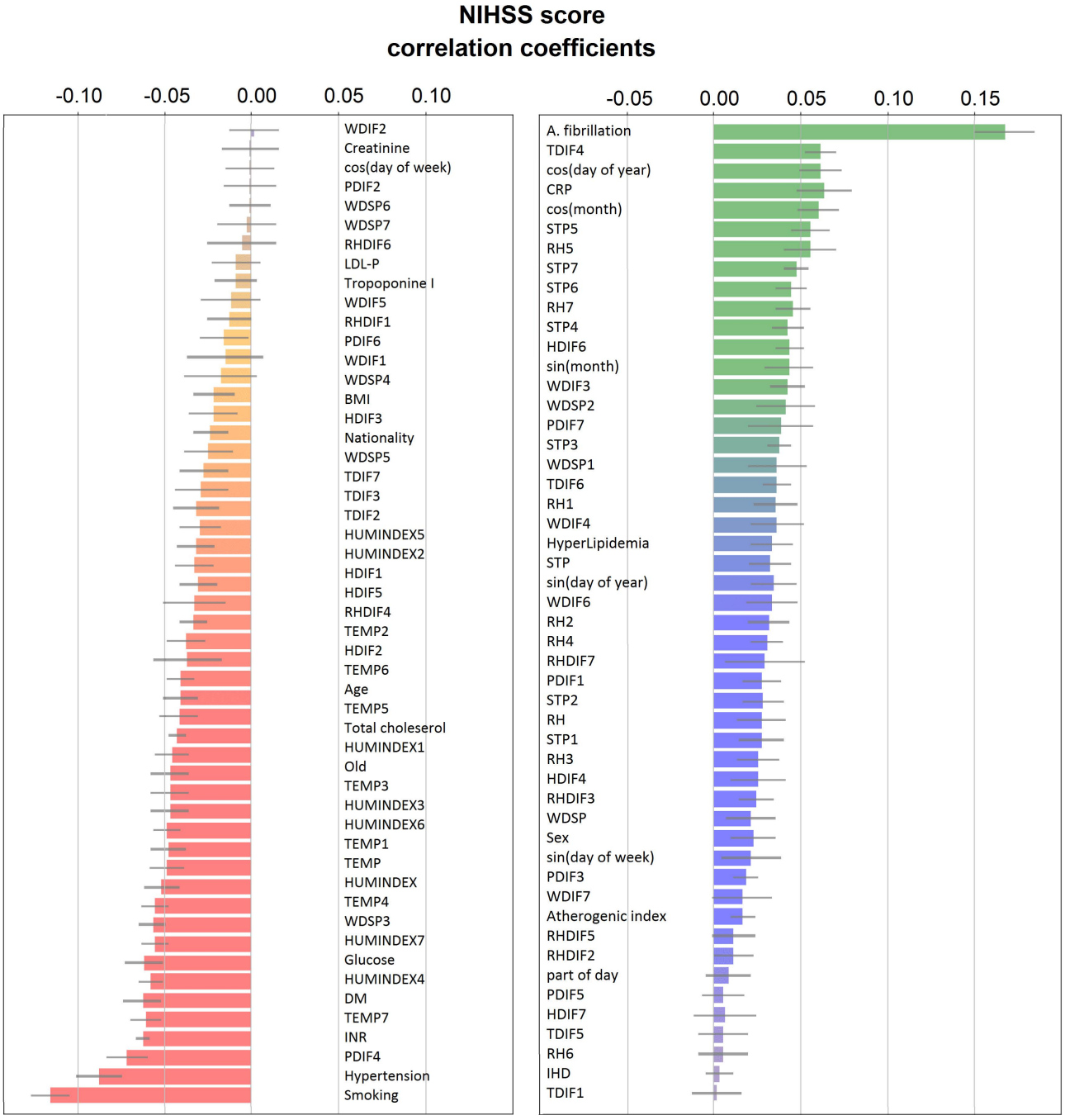

FIGURE 3. NIHSS score predictors ranked with correlation feature selection method in 10-fold cross-validation technique.

error, the models were trained in the ten-fold cross-validation technique. The performance of the regression models was expressed in the mean absolute error (MAE), root mean square error (RMSE) and coefficient of determination (R2). We reported the performance metrics averaged over 10 folds. The Python scikit-learn library v. 0.24.2 was used to implement ML models.

Fig. 4 shows correlation feature selection for the regression model trained to forecast mRS value. The model for calculating mRS incorporates the aforementioned predictors of NIHSS and the data received on admission. The top valuable individual risk factors are severity on admission, age, the history of an old stroke. The weather parameters also have a high-performance value in the model. To judge on the additive value of the parameters for the total performance of ML, see Table 5. For all regressors, except AdaBoost, the inclusion of the weather estimates improved the accuracy of the final model. After performing feature selection,

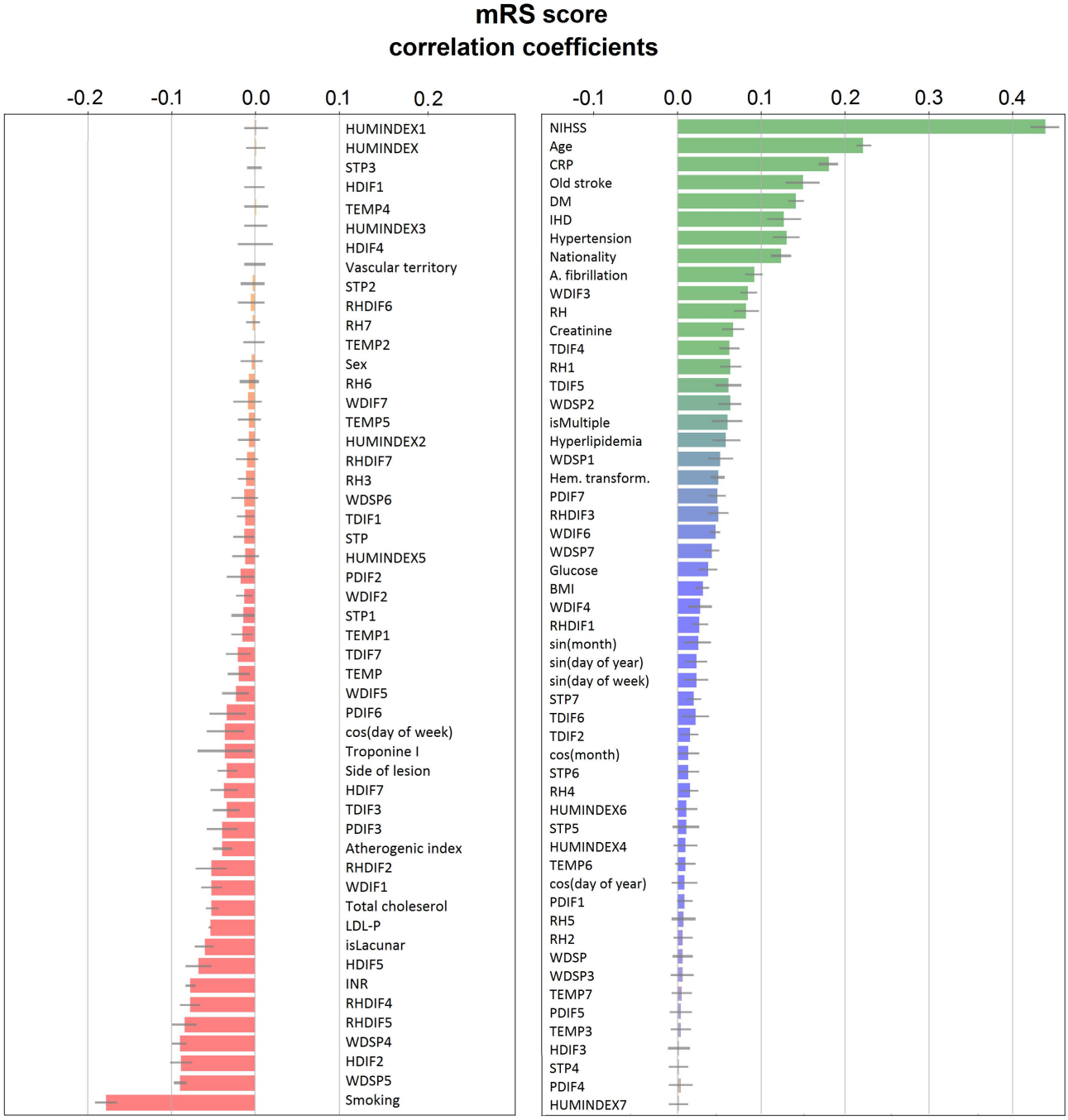

FIGURE 4. mRS score predictors ranked with correlation feature selection method in 10-fold cross-validation technique.

we received the most accurate prediction with Random Forest algorithm.

V. DISCUSSION

A. ASSOCIATION OF STROKE INCIDENCE WITH WEATHER AND INDIVIDUAL RISK FACTORS

1) IMPACT OF WEATHER ON PREVALENCE OF IS

The relation between the weather changes and IS has been studied in many papers, but inconsistency in the findings leads to further debate. This inconsistency might rise out of discrepancies among studies in design, methodology, population susceptibility, climate and geographical regions. Similarly to other papers [30], [37], [38] we failed to find a straight-forward correlation between the meteorological factors and the incidence of IS. On the other hand, unlike other papers we did not find either a non-correlation with the relative humidity [15], [27] or a positive association of the IS incidence with temperature variations [19], [39] and barometric pressure [40], [41].

TABLE 5. Performance of regression models for predicting early outcomes of ischemic stroke from different features.

Regressor	Non-weather features			All features			Selected features*		
	MAE	RMSE	R^2	MAE	RMSE	R^2	MAE	RMSE	R^2
Gradient Boosting	1.132	1.4110	0.181	1.099	1.3857	0.213	1.046	1.3258	0.281
AdaBoost	1.139	1.3879	0.193	1.177	1.3901	0.195	1.248	1.4358	0.149
K nearest neighbours	1.369	1.6295	-0.082	1.250	1.5171	0.056	1.156	1.4417	0.141
Lasso	1.159	1.3775	0.226	1.132	1.3580	0.246	1.075	1.3216	0.280
Random Forest	1.106	1.3666	0.232	1.071	1.3407	0.258	**1.046**	**1.3166**	**0.285**
SVR non-linear	1.311	1.5421	0.036	1.237	1.5271	0.052	1.207	1.6367	-0.101
Mean $\pm$ std	1.2 ± 0.1	1.45 ± 0.1	0.13 ± 0.12	1.16 ± 0.07	1.42 ± 0.07	0.17 ± 0.08	1.13 ± 0.08	1.41 ± 0.11	0.17 ± 0.14

*Top 20 listed features were identified from correlation feature selection method

Pathophysiological links between the weather and IS have been discussed recently. Hot weather may initiate IS by increasing blood viscosity, the platelets count and the level of serum cholesterol [42], [43], while cold seasons induce IS because of peripheral vasospasm and an increased blood pressure [44]. Heat stroke is a special case of brain ischemia which typically happens due to exercising in hot environment [45].

A high level of humidity in a hot climate may cause dehydration, which increases the risk of thrombosis [46]. Analogously, in these conditions an association between cold temperature and IS morbidity is also strong [16].

Changes in AP may exert stress on atherosclerotic plaques up to rupturing them [29]. A study found an association between the incidence of non-lacunar IS and the daily decrease in AP [31], [47]. We observed the opposite tendency and found that the IS occurrence increased after the moderate changes of AP within the previous day.

Extreme weather conditions may initiate stress that induces heart arrhythmia and contributes to the formation of thrombi. Additional research is required to confirm and get a new insight into the relationship between IS and the meteorological factors at diverse geographic locations [32].

2) INDIVIDUAL RISK FACTORS FOR IS

Age, sex, obesity, smoking, atherosclerosis, hypertension, low physical activity, atrial fibrillation and diabetes mellitus are the known risk factors for IS [48], [49]. Many studies show that a sedentary lifestyle, a low physical activity, smoking and obesity [50] lead to atherosclerosis, arterial hypertension [51] and diabetes mellitus [52] which are the main risk factors for IS, especially in the young generation [53], [54]. IS morbidity is higher in men [55]–[57]. Many authors believe that the risk of IS doubles each decade after the pivotal age of 55 [58], [59]. An alternative opinion is that the tendency starts at an early age [60], [61]. Particularly, our data suggest that from 45 to 54 the IS morbidity is four times higher than from 35 to 44 (330.29 vs 82.22 per 1000). Starting from 55 years of age, the risk of IS doubles compared to the preceding 10-year interval. The factors that account for the rising IS incidence in young adults - smoking, alcohol and drug abuse, a non-balanced diet promoting atherosclerosis, diabetes mellitus (DM) and obesity - are identical to the risks of developing cardiovascular complications of the disease [6], [62]–[64]. Genetic disorders contributing to cardiovascular pathologies improve the identification of IS at all ages including young adulthood [65]. A high number of pregnancies and childbirths may make young women vulnerable to IS [66], [67]. It is debatable if estrogen in birth-control pills account for IS in young women [68], [69]. Recent facts tell us that the dosage of estrogen is too small to promote IS [70]–[72].

3) INTERACTION BETWEEN WEATHER AND INDIVIDUAL RISK FACTORS FOR IS

Little is known on the interaction between the environmental and individual risk factors. For example, obesity may account for the elevated noradrenaline level which is a risk factor for IS [73]. Researchers observe a pick of noradrenaline and blood pressure in obese men in low temperature conditions [74]. An exposure to low temperatures may increase the blood and pulse pressure [75]. Patients with hypertension and diabetes mellitus are more vulnerable to the pressure changes [76], [77]. This raises the IS risk for them.

The tendency described for the low ambient temperature may also be relevant to the desert climate because of considerable day and night gaps. From our data, the occurrence of IS is maximal in March when both the temperature gap and the wind speed difference is maximal (see Fig. 1A-C, 1F).

B. ASSOCIATION OF STROKE SEVERITY WITH INDIVIDUAL AND ENVIRONMENTAL RISK FACTORS

Table 3) and Fig. 3A shows a strong association between individual risk factors and disease severity. From our data, the levels of C-reactive protein, troponin I, glucose, hyperlipidemia and INR are the top valuable laboratory factors reflective of the level of IS severity. These results correlate with the findings of other authors (see Section V-B2).

There was no evident difference in ethnicity of the patients stratified with regard to severity (p=0.23). However, the outcomes of IS among the UAE-Arabs were much worse, and in Asians - notably more favourable than in other

nations ($p<0.001$). Presumably, economy-related lifestyle habits rather than genetic factors account for the difference.

Some studies have shown that low temperature may contribute to high IS severity [37], [78]. But in our work, change in RH two consequent days before IS was a predictor of severe IS. Maybe this factor exerts a cumulative effect.

1) STROKE SEVERITY AND BACKGROUND DISEASES

Arterial hypertension may cause IS [79], [80], and affect its severity. Hypertension may influence endothelial dysfunction and large artery stiffness that transmits the pulsatile flow to the cerebral microcirculation [81], [82]. Because of vasospasm, hypertension may initiate lacunar IS [83]–[86].

Atrial fibrillation is a strong predictor of IS severity as seen from our data. This matches the results of other studies [87]–[89].

2) LABORATORY MARKERS OF STROKE SEVERITY

C-reactive protein (CRP) is a known marker of an early diagnosis of stroke, its recurrence and severity [90], [91]. A permanent release of the inflammatory mediator occurs because of a persistent irritation of the blood vessel wall by an atherosclerotic plaque. Since the level of CRP reflects atherosclerotic changes of the vessel wall, its high values may be indicative of an atherosclerotic embolus in a brain vessel, development of IS and stroke severity [92].

Troponin I is also a biomarker of IS severity, the poor outcomes and a coincidence of IS and the myocardial infarction (MI). Many studies have found a rising level of troponin I in the patients with IS who had neither heart nor kidney failure [93]–[97]. Commonly, the elevation of troponin I in IS does not exceed the level of 2 ng/mL [95]. As an explanation, some authors suggest an association of the elevated level of circulating epinephrine with the increased concentration of serum troponin I in IS [98]. Other studies failed to confirm a link between IS and troponin I [98], [99].

International normalized ratio (INR) is an important indicator for monitoring blood clotting in the patients taking anticoagulants for the secondary prevention of IS after an old stroke and transitory ischemic attack or atrial fibrillation. We check the level of INR to control thrombolytic therapy and prevent secondary brain hemorrhage or hemorrhagic transformation. A study of preadmission use of warfarin demonstrated an inverse relation between admission INR and volumes of acute IS lesion [100]. In our study the analysis of INR is challenging as the information on the preadmission use of blood-thinning medication was missing.

The blood glucose level may reflect severity of macro and micro-vascular IS lesions in the patients with DM [101]. As the continuous variables are more accurate than the categorical ones, hyperglycemia on admission indicates IS severity more reliably than the history of DM. Stress increases the level of serum glucose and the area of cerebral ischemia thus worsening the functional outcome [102], [103]. Furthermore, hyperglycemia can activate oxidative stress and damage neurons [102], [103].

3) VASCULAR TERRITORY OCCLUSION AND STROKE SEVERITY

Typically, IS severity depends on the location of brain damage caused by cardioembolic occlusion. Cerebral herniation and concomitant occlusion of several vessels also increase IS severity [104]. Evidently, the larger brain vessel is obstructed the more severe the stroke will be, as small collateral vessels may fail to substitute the blood supply. Cases with MCA stenosis are severe with a high likelihood of stroke-in-evolution and severe disability. At the same time, stenosis of MCA and extracranial internal carotid artery (ICA) has a worse functional outcome and a graver risk of stroke recurrence or death [105].

C. COMBINED EFFECT OF CLINICODEMOGRAPHIC FACTORS AND WEATHER PARAMETERS ON EARLY OUTCOME OF STROKE

Interestingly, age and an old stroke as non-modifiable risk factors and the atherogenic index as modifiable risk are much stronger associated with the IS early outcomes than with its severity. In part, this matches the results obtained by other authors (see Sections V-C1-V-C3)

1) INDIVIDUAL RISK FACTORS FOR UNFAVOURABLE STROKE OUTCOMES

A recent study justified smoking, age and NIHSS score on admission as reliable predictors of mRS score at discharge [106]. Another study showed no link between median mRS scores before the onset and at discharge in smoking patients [107]. The high NIHSS at discharge may indicate a poor prognosis for the patient [19]. In our study we found NIHSS to be a strong predictor of mRs.

Thrombolized patients with IS and AF have worse outcome at discharge, a higher recurrence rate compared to non-AF patients [87], [108]. This assumption requires further study.

Physicians should also consider the impact of DM and hyperglycemia on IS outcomes: patients with diabetes have a higher mortality rate, more severe disability and slower recovery [109], [110]. Perhaps, this is due to the fact that acute IS and stress stimulate the hypothalamus–pituitary–adrenal axis as well as the sympathetic nervous system leading to release of stress hormones, including cortisol and catecholamines, which increase glucose levels [111].

From our data, the strongest predictors of IS prognosis are the value of NIHSS on admission, age, the history of an old stroke and DM as well as the smoking status. The weather parameters also contribute to the IS outcomes.

2) BIOCHEMICAL MARKERS OF UNFAVOURABLE STROKE OUTCOMES

Our study justifies the level of CPR, troponin I, creatinine and atherogenic index (AG) as predictors of the early IS outcomes.

CRP. Our findings are compliant with a study that stated the CRP threshold level for predicting survival after IS to

be 10.1 mg/L [112]. Other studies found a link between an increased CRP level and the unfavourable long-term outcomes [113].

Troponin I is another marker of the IS outcomes. Its high level is related to increased stroke scale scores at discharge [114], [115].

The end products of the protein metabolism, such as the level of creatinine and the blood urea nitrogen to creatinine ratio, are also reflective of the poor IS outcomes [116]. Authors suggest that their ratio rises as a consequence of dehydration [117], [118] which could impact the delivery of oxygen to the brain thus worsening the outcomes. There is a positive association of the creatinine level with NIHSS and mRS scores in our study.

Atherogenic index (AI), when elevated, is a marker of early atherosclerosis [119], [120]. Patients with IS may suffer from intracranial atherosclerosis that worsens IS severity and outcomes [121], [122]. In our study, hyperlipidemia and AI are positively correlated with NIHSS on admission. Contrarily, there is a negative association of the total cholesterol and AI with the mRS at discharge. The association of lipids and lipid ratios with stroke is an issue of ongoing research and discussion [123]. Instead of using serum AI, recent studies suggest AI of plasma as a new predictive biomarker for cardiovascular illnesses [124]. Some researchers found an association between atherogenic dyslipidemia and recurrent stroke risk in patients with different IS subtypes [125]. A recent study justifies the low-density-lipoprotein particle size as a biomarker for the prognosis of atherothrombotic stroke [126]. Another study suggests predicting IS mortality from the cholesterol level [127]. In contrast, another study shows that low triglyceride concentration precisely predicts higher mortality whereas serum cholesterol level is not an independent predictor of IS outcomes [128].

3) VASCULAR TERRITORIES OCCLUSION AND STROKE OUTCOMES

An acute internal carotid artery occlusion (ICAO) is associated with poor clinical outcomes and mortality [129], [130].

A posterior inferior cerebellar artery (PICA) infarction is the most frequent IS type, it is usually atherothrombotic by aetiology. An occlusion in this vascular territory is frequently associated with early (within the first week) severe complications [131].

Posterior circulation strokes have better outcomes than the strokes in the anterior circulation of the brain. Among the posterior circulation strokes, the poorest outcome is observed in the non-treated cases of *basilar artery occlusion* [102], [132] and *middle cerebral artery* (MCA) occlusion [105], [133]. In the vascular territory of the posterior cerebral artery (PCA), cardioembolism is a less common reason for IS than in the middle (MCA) and anterior cerebral artery (ACA) [134]. For this reason, lacunar infarction is the most common IS subtype in the PCA territory [134] and the short-term IS prognosis in the PCA is more favourable than that in MCA territory as shown by a higher frequency of symptom-free cases at discharge and shorter hospital stay [135]. Another reason for the disproportion in the outcomes is that a posterior circulation stroke is associated with a lower risk of intracranial hemorrhage after intravenous thrombolysis than an anterior circulation stroke [136].

The acute cerebral infarction in more than one arterial territory (MACI) occurs in patients with cardioembolism [137]. It can be associated with AF or atherosclerosis of brain vessels [138]. A damage to ICA may cause MACI in both the anterior and posterior circulation simultaneously [139]. Another supposed reason for MACI is a lesion of azygos artery which is an unpaired ACA. As it supplies both hemispheres with blood, its occlusion may result in bilateral infarct [140]. Patients with MACI stroke have higher rates of short-term in-hospital complications and worse functional status at discharge compared to patients with single arterial territory stroke [137].

A watershed infarct involves a junction of distal fields of two nonanastomosing arterial systems. For the cases of mildly symptomatic ICA occlusion, the forecast of neurological deterioration after hospitalization depends on whether the watershed infarction is internal (IWI) or cortical (CWI) [141]. IWI is associated with hemodynamic impairment and critical stenosis of ICA, it leads to worse hospital courses, early [142] and midterm [143] prognosis compared to CWI. Supposedly, severity of IWI is related to reduced perfusion altering blood flow currents and prompting microembolism to reach the blood vessels with the least efficient blood flow [144].

A collateral flow through the circle of Willis and anastomotic connections between distal segments of cerebral arteries may facilitate partial reperfusion of ischemic territories after a focal stroke thus sustaining brain tissue for hours after an occlusion of major arteries [145]. Collateral vessels in the brain can weaken the effects of arterial occlusion but the outcomes depend on angiogenesis, age and concomitant diseases. There are leptomeningeal anastomoses and perforating arteries between MCA and accessory MCA, which are observed in over 3% of cases. The collateral flow can provide a sufficient supply to the MCA area [146], [147].

Analyzing the vascular territory of the occlusion, we should also look at the other radiomics data: the size, shape of the lesion. Progressive motor deficits and IS severity are closer associated with tubular than oval lacunae [148].

VI. CONCLUSION

- We found a non-correlation between the weather parameters and statistics on IS. The disparities in their trends encouraged us to investigate behind-time effects of the environment with DLNM and determine a concordant impact of all the settings with machine learning models.
- If categorized into two classes by NIHSS and mRS values, the cases have few disparities in the parameters of the weather within a week before the IS onset. The classes of NIHSS values differ markedly in RH on two consequent days before IS. The classes of mRS values

vary in WS on the fourth and seventh day, and in WD - a day before IS.

- We ranked environmental and individual risk factors by the information gain that they provided to the model predicting NIHSS value. The top valuable predictors were the background diseases, the weather parameters, the Cat2Vec-encoded data on the IS onset in the calendar year, and hyperlipidemia.
- In contrast to the classes by NIHSS score, the ones grouped by mRS have a pronounced difference in age ($p = 2.14e-06$), nationality ($p = 0.0008$), presence of background diseases and smoking ($p = 7.53e-07$). The separability with regard to the laboratory findings (levels of troponine I and C-reactive protein) is also more pronounced in the cohorts classified by mRS than NIHSS.
- The model for predicting mRS incorporates the aforementioned predictors of NIHSS and the data received on admission (e.g., the stroke location as per MRI). The top valuable individual risk factors are severity on admission, age, history of an old stroke. Inclusion of the weather parameters into the machine learning model predicting mRS score provides a slight boost in performance.

STRENGTH AND LIMITATIONS

The present study has several limitations. *First*, for natural reasons we could not study the effects of extreme cold temperatures in Al Ain City. *Second*, we did not consider a possible effect of the air pollution on IS though it may affect the morbidity in conjunction with other environmental factors [149]–[151]. A research reported the absence of association between air pollutant exposure and a short-term IS risk in Lyon (France) [152]. Because of the economic aspect and controversy of the previous findings, this question should be an issue of a separate study.

Third, the desert climate of the city is not representative of the entire country, the biggest cities of which are located along the Gulf with the prevalence of humid air masses over the dry ones. Thus, the study we did is not reflective of the risks for the entire population of the country, but it covers a large multinational cohort exposed daily to the harsh environment of the desert. Similar studies should be conducted in other regions to evidence the impact of the weather on the IS risk.

On the plus side we have a big study cohort of patients admitted to the IS unit within 4 consecutive years. Each case was verified with MRI diffusion-weighted imaging, which is considered to be the golden standard of the IS diagnostics. This study addressed the limitations of previous studies by analyzing a full set of the climate parameters - ambient temperature, RH, humidex, AP, WS - and the changes in these parameters at various times preceding the IS onset, both individually and in combination. We provided physicians a reliable machine learning model for assessing the risk of severe IS forms and outcomes. To gain an advanced accuracy, the models were trained on both internal (clinicodemographic) and external (climatic) risk factors.

CONTRIBUTIONS

YS, EF and JAK contributed to the conceptual idea of the paper; YS and JAK formulated the objectives; YS and EF wrote the manuscript; VL and TH performed the statistical analysis, prepared the figures and tables for data presentation and illustration; TMA, FAZ, KNVG, ML, MS and AP contributed to the literature review and data analysis.

COMPETING INTERESTS

The authors declare no competing interests.

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