A Dynamic Machine Learning Model to Predict Angiographic Vasospasm After Aneurysmal Subarachnoid Hemorrhage

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BACKGROUND AND OBJECTIVES: The goal of this study was to develop a highly precise, dynamic machine learning model centered on daily transcranial Doppler ultrasound (TCD) data to predict angiographic vasospasm (AV) in the context of aneurysmal subarachnoid hemorrhage (aSAH).

METHODS: A retrospective review of patients with aSAH treated at a single institution was performed. The primary outcome was AV, defined as angiographic narrowing of any intracranial artery at any time point during admission from risk assessment. Standard demographic, clinical, and radiographic data were collected. Quantitative data including mean arterial pressure, cerebral perfusion pressure, daily serum sodium, and hourly ventriculostomy output were collected. Detailed daily TCD data of intracranial arteries including maximum velocities, pulsatility indices, and Lindegaard ratios were collected. Three predictive machine learning models were created and compared: A static multivariate logistics regression model based on data collected on the date of admission (Baseline Model; BM), a standard TCD model using middle cerebral artery flow velocity and Lindegaard ratio measurements (SM), and a machine learning long short term memory (LSTM) model using all data trended through the hospitalization.

RESULTS: A total of 424 patients with aSAH were reviewed, 78 of whom developed AV. In predicting AV at any time point in the future, the LSTM model had the highest precision (0.571) and accuracy (0.776), whereas the SM model had the highest overall performance with an F1 score of 0.566. In predicting AV within 5 days, the LSTM continued to have the highest precision (0.488) and accuracy (0.803). After an ablation test removing all non-TCD elements, the LSTM model improved to a precision of 0.824.

CONCLUSION: Longitudinal TCD data can be used to create a dynamic machine learning model with higher precision than static TCD measurements for predicting AV after aSAH.

KEY WORDS: Machine learning, Cerebral vasospasm, Aneurysm, Subarachnoid hemorrhage, Transcranial Doppler, Prediction model

Cerebral angiographic vasospasm (AV) occurs in up to one-third of patients who suffer from aneurysmal subarachnoid hemorrhage (aSAH) and can result in a significant morbidity and mortality.[1-3] However, anticipation and treatment of AV remains complex and challenging due to its rapid onset, variable clinical presentation, and suboptimal diagnostic tests.[4-6] Screening includes serial neurological examination often with ancillary testing. Transcranial Doppler ultrasound (TCD) is noninvasive, inexpensive bedside imaging study that has been shown to be highly sensitive for AV and is supported by guideline recommendations.[7-11]

Despite the awareness of AV and the various monitoring methods available, it may not be identified until patients are symptomatic from hypoperfusion or completed ischemic stroke, resulting in a combined morbidity and mortality rates of up to 33.7%.[12,13] In

ABBREVIATIONS: AV, angiographic vasospasm; **BM,** Baseline Model; **CPP,** cerebral perfusion pressure; **DCI,** delayed cerebral ischemia; **LR,** Lindegaard ratios; **LSTM,** learning long short term memory; **MAP,** mean arterial pressure; **TCD,** transcranial Doppler ultrasound.

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response, significant effort has gone into developing predictive models.[14-18] Ultimately, none of these models have achieved high enough performance scores to be reliably used in clinical practice.

Given the effectiveness of TCDs and the rapid advancements in artificial intelligence, we aim to use a longitudinal time series data set centered around TCD measurements to create a machine learning model that can accurately stratify aSAH patients according to their risk of developing AV. The primary hypothesis is that a combination of clinical measurements and longitudinal TCD data are more predictive of AV than daily static TCD values.

METHODS

Patient Population

The following work was completed with the approval of our Institutional Review Board and submitted in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) reporting guidelines. Documented consent was waived by Institutional Review Board, given minimal risk to patients. A retrospective review of consecutive patients with aSAH admitted to the neurological intensive care unit (ICU) at our tertiary cerebrovascular referral center from 2015 to 2019 was conducted. Given the novel method of machine learning analytics used, we did not perform any detailed analysis to calculate the number needed to power the study. Adult patients (18 years or older) who underwent endovascular or microsurgical treatment of a ruptured intracranial aneurysm within 24 hours of admission and survived at least 7 days after admission were included. During admission in the neurological ICU, all patients underwent daily TCD, as well as standard of care critical care management according to American Heart Association guidelines,[19] including ventriculostomy for hydrocephalus if clinically indicated. Patients were excluded if there were inadequate data in the electronic medical record or the patient died within 14 days of admission.

Definitions and Management of Vasospasm

For clarity, the following definitions were used in this study: Delayed cerebral ischemia (DCI) is defined as a neurological decline of at least 2 points in the Glasgow Coma Scale or new focal neurological deficit that could not be explained by other causes such as intracranial hemorrhage, seizure, medication, metabolic, or other origin.[19,20] Most patients with suspected DCI underwent CT angiography, although in some patients with significant clinical concern, digital subtraction angiography was obtained without cross-sectional imaging. AV is the radiographic entity defined as focal arterial narrowing within the vessels comprising the Circle of Willis identified on cerebral angiography. All patients with suspected DCI were treated with induced hypertension and those with AV were treated with intra-arterial vasodilatory medications and/or mechanical balloon angioplasty at the discretion of the neurointerventionalist.

We elected to use AV as the primary outcome since AV represents an objective radiographic finding (compared with the clinical diagnosis of DCI). Of note, not all patients with suspected DCI showed evidence of AV. These patients were included in the study, but DCI without AV was not considered as a positive primary outcome regarding the developed prediction models.

Clinical and Physiological Data Collection

Standard demographic, clinical, and radiographic data were obtained. In total, 335 data elements were extracted for each patient, including daily physiological quantitative metrics trended throughout each patient's hospitalization including hourly vitals (among them intracranial pressure, mean arterial pressure [MAP], and cerebral perfusion pressure [CPP] when available), daily mean serum sodium concentration, hourly external ventricular drain output, and daily TCD blood flow velocities of the cerebral vasculature. TCD measurements were obtained by highly trained technicians with a minimum of 5 years of experience. Studies were conducted every morning for the first 14 days of admission or until discharge from the ICU. More detailed description of the TCD protocol can be found in the **Supplemental Digital Content 1** (http://links.lww.com/NEU/E915). Of note, no boundaries were used when collecting continuous, physiological data. Radiographic data obtained included intracranial aneurysm characteristics (maximum size, arterial location) and Fisher score. Clinical outcomes abstracted included modified Rankin Score at discharge, 6-month, and 1-year follow up. Intensive care and hospital length of stays were also recorded.

Statistical Analysis and Modeling

Extracted patient data were used to train predictive machine learning models. We experimented with using time series data as inputs and incorporating them into models that use both sparse (logistic regression) and dense feature representations (neural networks) for AV risk prediction. Each model used patient representations from prior time points to predict the likelihood of AV during subsequent time points by sampling the predicted likelihood of these future events.

Given the small data set, we constructed fixed-duration time series segments by sampling with a moving window across the available dates for each patient. For example, given a patient who is observed for 14 days in whom AV occurred on day 10, we first subsampled the patient data using a 5-day window: days 1-5, days 2-6, … days 5-9, and used each windowed segment to predict the risk of a future AV event. This method allows the creation of multiple training instances from the same patient, while addressing the challenge of training models to handle time series data of varying lengths (some participants have short hospital stays and earlier AV events, while others have much later events). All patient variables from the time series window were used as input into each model.

We constructed a model to predict the likelihood of AV occurring in the future (any day subsequent to the last day of the time series window). We divided all windowed time series into 80% train and 20% test splits while keeping all time series data associated with a particular patient in the same split.

We trained and evaluated logistic regression models and long short-term memory (LSTM) networks for AV prediction. As described above, the primary prediction outcome was angiographically confirmed vasospasm. For the multivariate logistics regression models, we trained one model using clinical/radiographic data from the day of admission (the "Baseline Model" [BM]) and one using bilateral middle cerebral artery (MCA) velocities and Lindegaard ratios (LR)[21] as measured by TCD (Standard TCD Model). We also trained an LSTM model using all clinical features as input. Precision, recall, accuracy, and F1 score were calculated for each model. To evaluate the feature influence of TCDs on the occurrence of AV, we trained LSTM models with and without TCDs and compared performance metrics to assess the influence of TCDs in the dense feature representation models.

RESULTS

Baseline Characteristics

The overall cohort included 424 patients, 78 of whom developed AV (18.4%). Of these 78 patients, 17 were asymptomatic

and taken to the angiography suite due to elevated velocities on TCD. There were several differences in baseline demographic, clinical, and radiographic characteristics in the AV group summarized in Tables 1 and 2. There were no differences in overall averages among any of the trended quantitative parameters monitored throughout the hospitalization including intracranial pressure, MAP, CPP, hourly venitriculostomy output, and TCD velocities (Table 3).

Clinical Outcomes

Although strokes rates were statistically similar between the 2 populations (30.8% for the AV group and 5.2% for the non-AV group; $P = .263$), the AV group had longer hospital stay (32.4 ± 20.5 days vs 23.1 ± 14.2 days; $P < .001$) and ICU stay (19.6 ± 8.63 vs 14.4 ± 6.97 days; $P < .001$), as well as worse modified Rankin Score at discharge (3.46 ± 1.19 vs 2.92 ± 1.32; $P = .003$) and 6-month follow-up (2.40 ± 1.64 vs 1.75 ± 1.75; $P = .025$). There were no differences in long-term outcomes (Table 4).

Quantitative Trends

Line graphs depicting trends of quantitative metrics throughout ICU hospitalization identify several patterns (Figure 1). TCD values including bilateral LR, maximum bilateral MCA, vertebral artery, and basilar artery velocities were all higher in the AV group compared with the non-AV group. Furthermore, all measurements showed notable elevation immediately before the occurrence of AV and all except for right vertebral artery velocities peaked immediately before the occurrence of AV. During the days leading up to an AV event, MAP and CPP noticeably increased relative to the non-AV group. Both groups showed similar numbers of red blood cells in the cerebrospinal fluid initially and both down trended during the days after presentation; however, the AV group tended to have a higher count through admission.

Model Performance

The LSTM model demonstrated the highest precision in predicting AV at any time point during the ICU stay (0.683) and was the most accurate in predicting AV at any time point (0.786). The BM had the highest F1 score in predicting AV at any time point (0.566) (Figure 2).

An ablation experiment was conducted to further understand the performance of the LSTM model in predicting AV at any time point by creating an LSTM model that used TCD values only, as opposed to all trended data. After removing all non-TCD features, precision increased to 0.824, recall decreased to 0.139, and F1 score decreased to 0.237 (Figure 3).

DISCUSSION

Prediction of AV and DCI after aSAH has been a topic of investigation since the inception of the Fisher score in 1980.[14] Advantages to accurately predict patients who will suffer from AV after aSAH include prophylactic treatment and prevention of ischemic stroke. In addition, identifying patients at low risk for AV may permit shorter ICU and hospitalization times, resulting in cost and resource savings. Previous attempts have been made to apply complex data analytics and machine learning to AV prediction.[22–25] However, these models have reached only a moderate level of predictive capacity due to limited sample sizes, heterogenous data, and the use of data collected at a single time point. Recently, other models have been developed using time series physiological parameters such as systolic blood pressure,

TABLE 1. Comparison of Baseline Clinical Characteristics of AV and No AV Patients

Clinical characteristic	AV (n = 78) [95% CI]	No AV (n = 346) [95% CI]	*P* value
Age, y, mean (SD)	52.9 [50.4, 55.4]	56.2 [54.8, 57.7]	.023
Sex, %			.055
Female	79.5 [68.8, 87.8]	69.4 [64.2, 74.2]	
Male	20.5 [12.2, 31.2]	30.6 [25.8, 35.8]	
BMI, kg/m^2, mean (SD)	27.3 [25.8, 28.8]	28.5 [27.7, 29.2]	.149
Tobacco use, %	35.9 [25.3, 47.6]	25.7 [21.2, 30.7]	.048
Hypertension, %	47.4 [36.0, 59.1]	46.5 [41.2, 51.9]	.689
Hunt-Hess, mean (SD)	3.51 [3.30, 3.73]	2.87 [2.76, 2.98]	<.001
Fisher, mean (SD)	3.51 [3.40, 3.63]	3.20 [3.12, 3.28]	<.001
GCS initial, mean (SD)	10.3 [9.47, 11.2]	11.9 [11.5, 12.3]	.002
GCS 24 h, mean (SD)	10.6 [9.91, 11.4]	11.9 [11.5, 12.2]	.002

AV, angiographic vasospasm; BMI, body mass index; GCS, Glasgow Coma Scale.

TABLE 2. Comparison of Aneurysm and Treatment Characteristics Between AV and No AV Patients

Aneurysm/treatment characteristics	AV (n = 78) [95% CI]	No AV (n = 346) [95% CI]	*P* value
Aneurysm location, anatomic, %			.011
Anterior	91.0 [82.4, 96.3]	81.2 [76.7, 85.2]	
Posterior	9.0 [3.68, 17.6]	18.5 [14.5, 23.0]	
None	0 [0.00, 4.62]	0.3 [0.01, 1.60]	
Aneurysm location, vessel, %			.155
Middle cerebral artery	21.8 [13.2, 32.6]	14.7 [11.2, 18.9]	
Internal carotid artery	7.7 [2.88, 16.0]	9.2 [6.41, 12.8]	
Anterior communicating artery	34.6 [24.2, 46.2]	38.4 [33.3, 43.8]	
Anterior cerebral artery/pericollosal	5.1 [1.41, 12.6]	3.2 [1.60, 5.62]	
Posterior communicating artery	19.2 [11.2, 29.7]	15.3 [11.7, 19.6]	
Posterior inferior cerebellar artery	2.6 [0.31, 8.96]	5.8 [3.57, 8.79]	
Anterior inferior cerebellar artery	0 [0.00, 4.62]	0.6 [0.07, 2.07]	
Basilar artery	6.4 [2.11, 14.3]	5.5 [3.34, 8.44]	
Other	2.6 [0.31, 8.96]	7.2 [4.73, 10.5]	
Aneurysm size, mm, mean (SD)	6.38 [3.45, 10.41]	6.95 [4.12, 12.67]	.354
Intervention, %			.025
Clip	57.7 [46.0, 68.8]	45.4 [40.0, 50.8]	
Coil	41.0 [30.0, 52.7]	49.1 [43.7, 54.5]	
Bypass	1.3 [0.03, 6.94]	3.2 [1.60, 5.62]	
Parent vessel sac	0 [0.00, 4.62]	1.2 [0.32, 2.93]	
Flow-diverting stent	0 [0.00, 4.62]	0.3 [0.01, 1.60]	
None	0 [0.00, 4.62]	0.9 [0.18, 2.51]	

AV, angiographic vasospasm.

heart rate, and respiratory rate trended throughout the patient's ICU hospitalization to predict AV risk.[20] This concept is intriguing given the dynamic nature of AV and its variable time of onset. Furthermore, a dynamic model would provide continuous risk stratification throughout the patient's hospitalization aiding in day-to-day prophylactic management. However, these previous models have all relied on physiological data such as vital signs which can be nonspecific and confounded by multiple factors,[26–28] limiting their power.

TCD has been shown to be a safe, noninvasive, and effective method for screening for AV after aSAH. Several studies have validated its high sensitivity and negative predictive value[7-10]; thus, it has been recommended by the American Heart Association/American Stroke Association (Class IIA/Level B evidence)[19] and Neurocritical Care Society.[29] At our institution, TCDs are performed daily on all patients with aSAH throughout their 14-day ICU admission. Thus, we hypothesized that incorporating TCD data in a dynamic fashion may yield a predictive dynamic model that could be used in practice.

Long Short-Term Memory is a form of recurrent neural network that is able to recall past inputs in a data series that may influence future outcomes or events.[30] LSTM models have been successfully used in a wide range of healthcare fields including prediction of surgical site infections,[31] delirium onset,[32] and emergency department wait times.[33] In this study, we created an LSTM model using TCD data along with other relevant clinical and radiographic data to predict AV and compared it with 2 conventional multivariate logistics regression models: One using clinical/radiographic data from the day of admission (BM) and one using baseline MCA velocities and LRs measured by TCD (Standard TCD Model). The rationale for the Standard TCD Model was current clinical practice, in which static MCA

TABLE 3. Comparison of Trended Physiological Data Between AV and No AV Patients

Variable	AV (n = 78) [95% CI]	No AV (n = 346) [95% CI]	*P* value
MAP, mm Hg, mean (SD)	101 [98.4, 103]	99.7 [98.6, 101]	.442
CPP, mm Hg, mean (SD)	94.2 [91.6, 96.8]	93.4 [92.2, 94.6]	.586
EVD output, cc/hour, mean (SD)	8.38 [7.72, 9.04]	8.72 [8.38, 9.05]	.365
Lindegaard ratio, right, mean (SD)	3.08 [2.46, 3.71]	2.59 [2.44, 2.73]	.126
Lindegaard ratio, left, mean (SD)	2.67 [2.42, 2.92]	2.58 [2.42, 2.73]	.533
Max MCA velocity, right, cm/s, mean (SD)	98.9 [90.9, 107]	91.9 [88.2, 95.6]	.118
Max MCA velocity, left, cm/s, mean (SD)	97.7 [91.2, 104]	93.9 [90.2, 97.6]	.314
Max ICA velocity, right, cm/s, mean (SD)	68.7 [62.7, 74.8]	65.3 [62.6, 68.0]	.303
Max ICA velocity, left, cm/s, mean (SD)	66.5 [61.5, 71.4]	64.4 [61.9, 66.9]	.462
Max ACA velocity, right, cm/s, mean (SD)	75.7 [70.8, 80.6]	73.9 [71.2, 77.2]	.522
Max ACA velocity, left, cm/s, mean (SD)	75.5 [69.8, 81.1]	74.5 [71.9, 77.2]	.77
Max PCA velocity, right, cm/s, mean (SD)	49.0 [46.0, 51.9]	48.8 [47.0, 50.6]	.925
Max PCA velocity, left, cm/s, mean (SD)	48.1 [45.5, 50.7]	48.3 [46.6, 50.0]	.893
Max VA velocity, right, cm/s, mean (SD)	46.4 [43.2, 49.7]	44.4 [42.8, 46.0]	.257
Max VA velocity, left, cm/s, mean (SD)	46.6 [43.1, 50.0]	43.5 [42.0, 45.0]	.108
Max BA velocity, cm/s, mean (SD)	67.1 [62.4, 71.8]	62.9 [60.4, 65.4]	.121
Na, average, mean (SD)	138 [138, 139]	138 [138, 138]	.052
CSF, red blood cells, cells/μL, mean (SD)	70 581 [55 460, 85 703]	72 896 [64 998, 80 795]	.788
CSF, nucleated, cells/μL, mean (SD)	511 [324, 698]	644 [528, 759]	.231
CSF, glucose, mg/dL, mean (SD)	82.2 [78.8, 85.7]	80.6 [78.8, 82.5]	.42
CSF, protein, mg/dL, mean (SD)	122 [90.7, 153]	113 [103, 123]	.593

ACA, anterior cerebral artery; AV, angiographic vasospasm; BA, basilar artery; CPP, cerebral perfusion pressure; CSF, cerebrospinal fluid; EVD, external ventricular drain; ICA, internal carotid artery; MAP, mean arterial pressure; MCA, middle cerebral artery; Na, daily mean serum sodium concentration; PCA, posterior cerebral artery; VA, vertebral artery.

TABLE 4. Comparison of Clinical Outcomes Between AV and No AV Patients

Outcome	AV (n = 78) [95% CI]	No AV (n = 346) [95% CI]	*P* value
Stroke, %	30.8 [20.8, 42.2]	5.2 [3.11, 8.10]	.263
Hospital LOS, days, mean (SD)	32.4 [27.8, 37.0]	23.1 [21.5, 24.6]	<.001
ICU LOS, days, mean (SD)	19.6 [17.7, 21.6]	14.4 [13.7, 15.2]	<.001
mRS, discharge, mean (SD)	3.46 [3.16, 3.76]	2.92 [2.72, 3.11]	.003
mRS, 6 months, mean (SD)	2.40 [1.89, 2.90]	1.75 [1.49, 2.01]	.025
mRS, 1 year, mean (SD)	2.08 [1.47, 2.70]	1.50 [1.23, 1.78]	.089
Mortality, %	7.7 [2.88, 16.0]	6.1 [3.80, 9.13]	.624

AV, angiographic vasospasm; ICU, intensive care unit; LOS, length of stay; mRS, modified Rankin Scale.

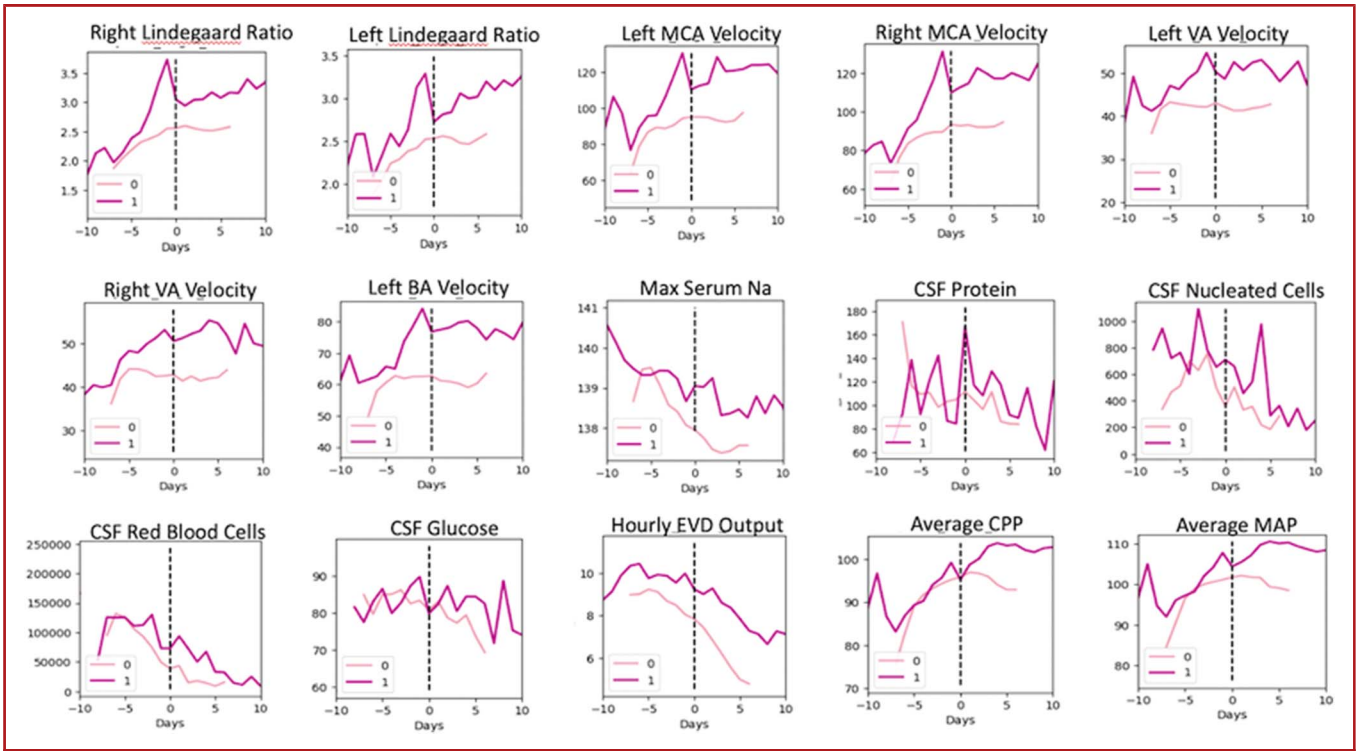
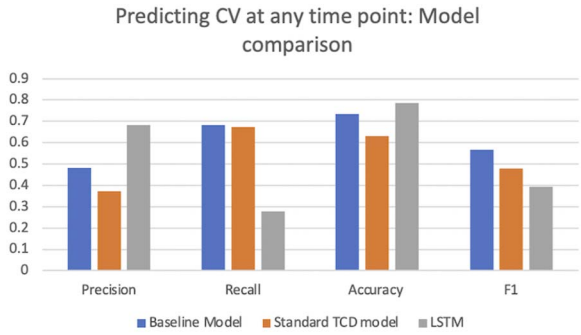


FIGURE 1. *Line charts depicting patterns of quantitative metrics comparing patients with vasospasm (dark pink, 1) and patients without vasospasm (light pink, 0). Day 0 (dotted line) represents the day of vasospasm. Lindegaard ratio is the maximum internal carotid artery velocity/maximum MCA velocity. CPP, cerebral perfusion pressure; CSF, cerebrospinal fluid; EVD, external ventricular drain; MAP, mean arterial pressure; VA, vertebral artery.*

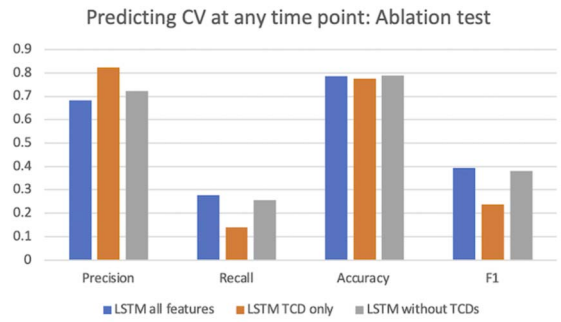


	Precision	Recall	Accuracy	F1
Baseline Model	0.483	0.683	0.736	0.566
Standard TCD model	0.372	0.673	0.632	0.479
LSTM	0.683	0.277	0.786	0.394

FIGURE 2. *Model performance for predicting vasospasm at any time point in the future. CV, cerebral vasospasm; LSTM, long short-term memory; TCD, transcranial Doppler ultrasound.*

velocities and LR measurements have been shown to be most sensitive for AV.[34–36]

We focused on one primary outcome in this study: The occurrence of AV at any time point during the patient's admission. The metrics we used to evaluate the models were precision, recall, F1 score, and accuracy. Precision measures how a model correctly predicts the positive class, more commonly known as positive predictive value. Recall measures how often positive classes are correctly identified, more commonly known as sensitivity. F1 score is a balance of a model's precision and recall, therefore measuring its overall performance. Accuracy measures the how often the model is correct in identifying both positive and negative classes. In predicting AV at any time point, the BM had the highest overall performance with an F1 score of 0.566. However, the LSTM model had the highest precision at 0.571, meaning that it was relatively more successful at identifying patients who would develop AV. For context, an F1 score of greater than 0.9 is considered to be "very good," whereas 0.5-0.7 is considered "average."[37] By contrast, the LSTM model had the lowest recall score giving it a low sensitivity when compared with the other models. Although all 3 models had high accuracy score among the various metrics, accuracy can have a paradoxical meaning when working with highly imbalanced data sets where one outcome



	Precision	Recall	Accuracy	F1
LSTM all features	0.683	0.277	0.786	0.394
LSTM TCD only	0.824	0.139	0.776	0.237
LSTM without TCDs	0.722	0.257	0.789	0.38

FIGURE 3. *LSTM performance comparing using all features vs longitudinal TCDs only. LSTM, long short-term memory; TCD, transcranial Doppler ultrasound.*

greatly outnumbers the other. In this case, there were significantly more patients who did not have AV vs those who did. Overall, these results suggest that the LSTM model is best for early identification of patients who may have AV with fewer misses than the other models and can be used for triaging patients who may benefit from closer monitoring or prophylactic treatment. On the other hand, given the lower recall score, this LSTM model would not be suited for ruling out AV in the future and, thus, would be suboptimal for identifying patients at low risk for developing AV.

To further understand the potential of our LSTM model, we performed an ablation study whereby the clinical/radiographic elements of the input were removed to isolate the TCD data. The LSTM model's performance regarding precision was enhanced by using TCD data alone, which increased the precision score from 0.683 to 0.824 when predicting AV at any time point. This dramatic improvement in the LSTM model performance highlights the positive predictive value of trended TCD measurements in correctly identifying patients who may develop AV. However, the concomitant decrease in F1 score may suggest using TCDs alone results in an over-fitted model that yields more false positives. The clinical implication of false positives is unnecessary preventative tests and treatments for AV, which adds additional risk to aSAH patient care. Thus, any final predictive model must be interpreted in the overall clinical context.

Although none of the models developed in this study achieved an F1 score greater than 0.8, the precision score of 0.824 of the LSTM model using only TCD data implies that there may be future value in using longitudinal (rather than static daily) TCD data to identify patients who are at high risk for AV, which can have significant impact on resource utilization and clinical outcomes.

Limitations

There are several shortcomings to this study that, if optimized, may eventually yield a clinically applicable model. Data quality is a critical factor in creating a highly predictive machine learning algorithm. Owing to the retrospective nature of this study, suboptimal data quality could have contributed negatively to the performance in all aspects of the models. This would pertain more to the clinical and radiographic data, as opposed to the TCD data which is reported and collected in a more uniform fashion. For example, physiological data including MAP, CPPs, and laboratory values can be skewed by medication or other environmental factors, which are not controlled for due to the limitations of the chart review process, as evidenced by the improvement of the LSTM model when such elements were removed. Furthermore, although we attempted to amplify our data by segmenting patient data into multiple time windows, the relatively small sample size of this study hindered our ability to thoroughly train the algorithm. To address these issues, we plan to perform a prospective study focusing on more precise collection of the clinical and radiographic data while enhancing the TCD data by including more granular waveform data. These adjustments will improve the precision of our models, potentially leading to application in clinical practice.

CONCLUSION

A dynamic machine learning model using time series data including daily TCD measurements may identify patients with aSAH who are at high risk for developing AV.

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