

Predicting the incidence of pressure ulcers in the intensive care unit using machine learning

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Intensive care units (ICUs) have a high incidence of hospital acquired pressure ulcers (PUs) due to the long-term immobilization and complex morbidity profile of the patient population. Limited work has been done to characterize the profile of PUs in the ICU using observational data from the electronic health record (EHR). Consequently, there are limited EHR-based prognostic tools for determining a patient's risk of PU development, with most institutions relying on nurse-calculated risk scores such as the Braden score to identify high-risk patients. Using EHR data from 50,851 admissions in a tertiary ICU, we show that the prevalence of PUs at stage 2 or above is 7.8%. For the 1,690 admissions where a PU was recored on day 2 or beyond, we evaluated the prognostic value of the Braden score measured within the first 24 hours. A high-risk Braden score (≤ 12) had precision 0.09 and recall 0.49 for the future development of a PU. We trained a range of classifiers using demographic parameters, diagnosis codes, laboratory values and vitals from the EHR within the first 24 hours. A weighted elastic net showed precision 0.09 and recall 0.74 for future PU development. Performance was not improved by integrating Braden score elements into the model. This demonstrates that an EHR-based model can outperform the Braden score as a screening tool for PUs. This may be useful tool for automatic risk stratification early in an ICU admission, helping to inform the allocation of prophylactic interventions such as pressure support mattresses.

Keywords: Pressure ulcers; Electronic health records; Predictive models; Machine learning

1. Introduction

Pressure ulcers (PUs) represent a significant public health issue, afflicting Intensive Care Units (ICUs) internationally.¹ PUs occur when the skin is exposed to pressure and shear, typically as a result of long term patient immobilization, causing injury to the epidermis and underlying tissues. The prevalence of PUs in ICUs has been estimated between 22-49%.^{2,3} An ulcer can significantly extend a patients length of stay in the hospital, and can cause long term disability, with muscles and other deep tissues often impaired for months after the resolution of the external wound.⁴ PUs cause chronic morbidity through pain and associated psychological impacts; however, they are also responsible for significant mortality, typically as a consequence of sepsis following bacterial inoculation to the bloodstream via the ulcer.

PU are, however, eminently preventable and treatable in their early stages. Prophylactic measures include regular patient rolling, specialized pressure mattresses (e.g. powered active air and hybrid air surfaces), and good patient nutrition.^{5,6} Management of established ulcers includes pressure dressings (hydrocolloid, foam, and film), wound cleansers, negative pressure therapy, ultrasound therapy, and surgical intervention.⁷ To reduce PU incidence, it is critical to identify at-risk patients and intervene early. As many of these therapies are labor-intensive or expensive (e.g. pressure mattresses), allocating resources according to patient risk is an important clinical challenge.

Several risk stratification methods have been developed, including the Braden (1987), Norton (1965), and Waterlow (1985) scales. These are nurse-reported measures that combine local skin factors (such as moisture, friction and shear) with patient-level factors (such as mobility, sensory perception and nutrition).⁸ The sensitivity and specificity of these risk scores in predicting the later development of PUs is highly variable.⁹ For example, a survey of 7,800 ICU patients found that the best performance of the Braden score yielded an area-under-the-curve (AUC) of only 0.67.¹⁰ One Brazilian study found that although the Braden score had poor prognostic accuracy, its performance could be increased by including a broader range of patient-level factors.¹¹ These variables included: age, gender, comorbidities (specifically diabetes, hematological malignancies, and peripheral artery disease), hypotension, renal replacement therapy and mechanical ventilation within the first 24h of admission. All of these data are recorded in the EHR, raising the possibility to build more powerful predictive models using a broader range of variables than the traditional manual risk scores. This aligns with a recent meta-analysis of 17 studies evaluating these scores, which called for the development of more personalized risk algorithms.¹²

There have been several early attempts to model the incidence of PUs with statistical methods. Kaewprag *et al.* used Bayesian nets to classify patients based on the presence of an ulcer, achieving good specificity but poor sensitivity, consistently below 0.5.¹³ Park *et al.* performed multivariate linear regression on 61 clinical and laboratory variables to predict time-to-ulcer onset in 202 patients with PUs.¹⁴ Additionally, Cho *et al.* developed a Bayesian net to predict PUs based on 37 structured elements derived from the EHR.¹⁵ Most of these algorithms do not deal well with time-dependence - focusing more on classifying PU versus non-PU patients for the purpose of identifying risk factors, rather than predicting ulcer development after an index time. The exception is Cho *et al.* - the only study to date which has developed a risk model framed as a decision support tool. It is also the only algorithm deployed in practice - during a trial in 2010 in South Korea - where investigators observed a significant reduction in PU incidence from 21% to 4%.

The majority of previous studies characterizing PUs in the ICU, which are predominantly found in the nursing literature, have used manual audits of clinical notes to profile relatively small patient cohorts. For example, the number of PU patients varied between 16 and 140 in the audits conducted by numerous studies.¹⁶⁻¹⁸ Very limited work has been done to auto-

matically extract PU information using structured EHR data. This is despite the increasingly widespread use of template-based reporting systems for nurse skin examinations, and the fact that EHR-derived PU data has consistently been found to more accurately represent the prevalence of ulcers than clinical progress notes.^{19,20} ICUs have the highest rates of hospital acquired ulcers; however, only one study to date has utilized ICU-specific EHR data at scale to characterize the disease burden of pressure injury.¹³

Our first aim is a large-scale descriptive study of PUs in an observational ICU dataset. This forms the basis for the second objective - developing a machine learning model to predict PU development. This is the first predictive model for PUs built on EHR data from an ICU in the US, and will make use of the largest training dataset to date (for comparison, Kaewprag *et al.* had 590 cases and 7,127 controls).¹³ Specifically, we aim to predict future PU development using data from the first 24h of admission, as a means to risk stratify patients early in their care.

2. Materials and Methods

Our study consisted of four key components: (1) identifying a cohort of patients who develop PUs in the ICU; (2) descriptive statistics comparing the PU versus non-PU populations; (3) producing feature vectors using EHR-derived data from the first 24h of admission; and (4) training and evaluating a range of classifiers to predict future PU development. We outline each of these steps below.

2.1. Data

MIMIC-III (v1.4) is a publicly-accessible database of de-identified EHR data for approximately 54,000 patients admitted to the ICU of Beth Israel Deaconess Medical Center between 2001 and 2012.²¹ The database includes tables for chart events (such as vital signs), medications, diagnosis codes, laboratory measurements, observations and notes recorded by care providers. EHR data are linked to 12-month out-of-hospital survival data obtained from social security records. MIMIC-III spans two EHR recording systems: in 2008 the Beth Israel Deaconess Medical Center switched from the Carevue (Philips Healthcare, Cambridge MA) to the Metavision system (iMDSoft, Wakefield MA). Data were extracted from both systems to create three separate datasets: a collective set of all data, and respective Carevue and Metavision data sets. Patients aged under 18 years were excluded (Figure 1).

2.2. Cohort identification & descriptive statistics

PUs were identified using the following codes for PU staging: 551, 552, 553 for Carevue; 224631, 224965, 224966 for Metavision. PUs recorded at stage 2 or above, representing an injury to the dermis, were counted as ulcers. Stage 1 PUs, characterized by redness of the skin with no epidermal breach, were ignored. In total, there were 4,174 ICU admissions where a stage 2 or greater PU was recorded, accounting for 7.8% of all admissions. For our prediction task, patients with a PU recorded within the first 24h of ICU admission were excluded (2,001

patients, 2484 ICU stays). Patients who developed a PU after the initial 24h window were classified as cases. This yielded 1,606 cases (1690 ICU stays) and 36,561 controls (49,161 ICU stays). Descriptive statistics were calculated for these populations and compared using two sample t-tests.

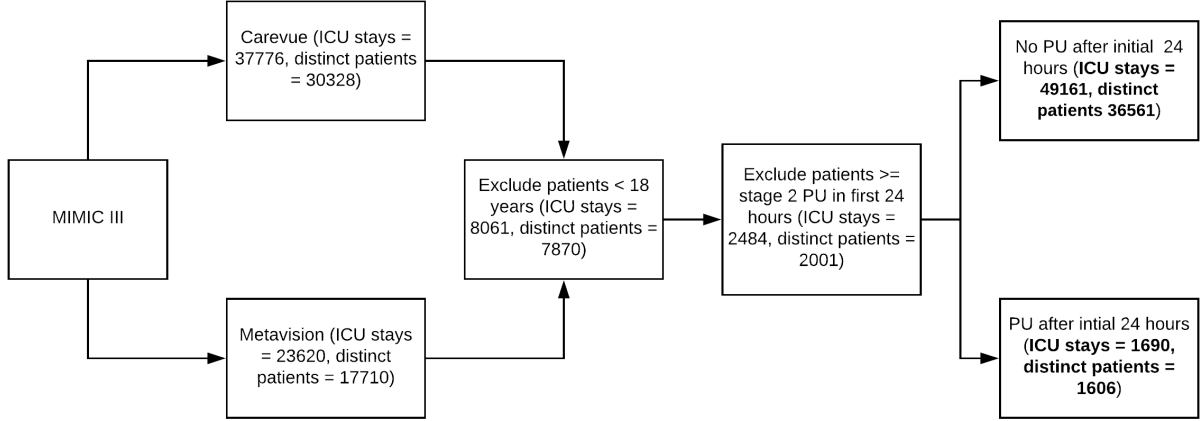


Fig. 1. Flow diagram of cohort selection with exclusion criteria.

2.3. Braden score prognostic evaluation

For the Carevue patients, where 93.5% of ICU admissions had Braden score documented within 24h, we evaluated the performance of standard Braden score thresholds for high and severe risk (≤ 12 and ≤ 9 respectively) in predicting future PU development.

2.4. Feature engineering

The design of our prediction task is illustrated in Figure 2. Data from the first 24h post ICU admission were used to generate a feature vector in order to predict, as a binary outcome, the occurrence of a PU in the remainder of that admission. A wide range of demographic, clinical, laboratory and environmental features were used to populate the feature vector. The rationale behind feature engineering was to use only common variables that could be readily extracted from the EHR at the 24h timepoint, taking into account known risk factors for PU.²² No Braden score data was used in the original feature vectors, with the intention to determine if EHR-derived model could match or outperform nurse-calculated Braden scores.

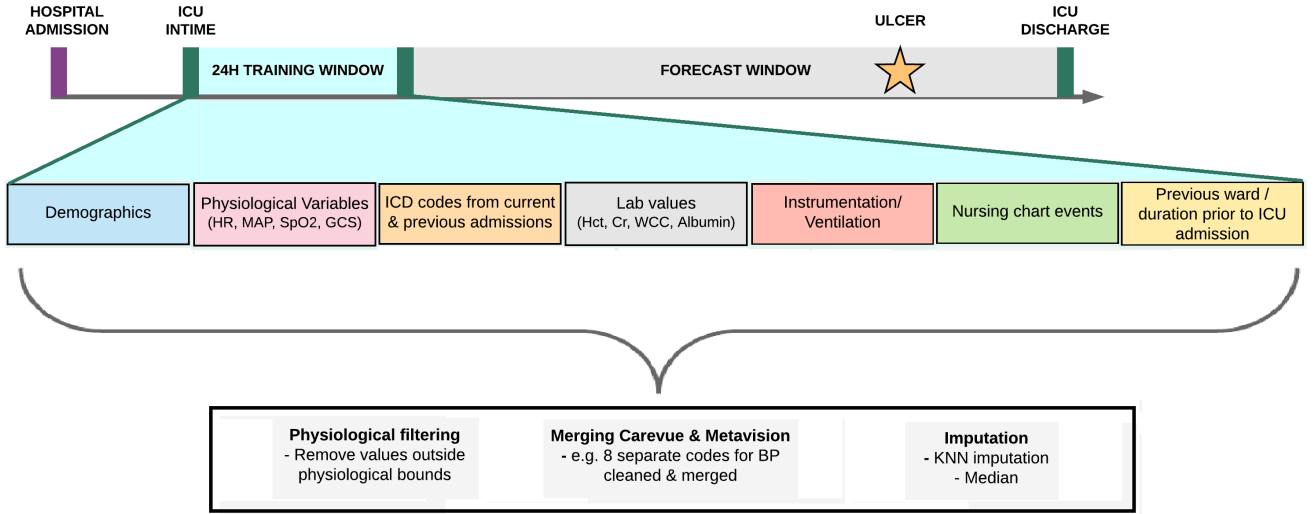


Fig. 2. Feature engineering using multiple data sources from the first 24h of ICU admission.

A total of 29 features were used to populate feature vectors. Demographic features included age, gender and ethnicity. Clinical features included specific vitals (mean arterial pressure (MAP), peripheral oxygen saturation (SpO₂), Glasgow Coma Scale (GCS)), which were averaged across the first 24h. Results outside of physiological bounds were censored. Laboratory features included complete blood counts, electrolytes, albumin, arterial blood gases, blood urea nitrogen, bilirubin, blood glucose and international normalized ratio (INR). The patient's ventilation status was encoded into no ventilation, non-invasive ventilation and mechanical ventilation, with the highest level during the first 24h used.

Comorbidities were evaluated by mapping International Classification of Diseases (ICD) codes from the current and previous admissions to 10 high-level diagnoses: anemia, coronary artery disease, amputation, heart failure, diabetes, leukemia, neuropathy, peripheral vascular disease, spinal cord injury and stroke. These comorbidities were chosen because of known associations with PU development, either due to immobility or impaired wound healing. Our reasoning for using ICD codes from the present admission was that these conditions are all chronic. Even when an ICD is assigned at discharge, it would likely have been present in the problem list of a patient even within the first 24h. If a patient had a previous ICU admission, recording of a PU at stage 2 or above during that admission was encoded as a specific feature. Additional logistical features included the patient's previous ward location, current ICU ward, and the duration from hospital admission to ICU admission.

Features were harmonized across the Metavision and Carevue datasets. The range and missingness of features was evaluated in consultation with clinical mentors, with an iterative process for refining the scope of features selected.

2.5. Classifier training

Figure 3 shows the model development pipeline. Models were built using both the entire dataset, and the Carevue data only, which was not only more complete than Metavision but had Braden scores evaluated for most patients. We trialled both median imputation and k-nearest neighbors imputation (with 5 nearest neighbors) to populate missing values. Categorical variables were dummy coded using full rank encoding, except for ICD codes which were encoded with one-hot encoding. Continuous variables were standardized.

Data was split into training and test sets with an 80:20 ratio. The following classifiers were trained using 5-fold cross-validation on the training set over a standard search grid of hyperparameters: logistic regression, elastic net, support vector machines (with linear and radial kernels), random forest, gradient boosting machines, and a feed-forward neural network with a single hidden layer. During cross-validation, classifiers were optimized on Cohen’s kappa - a measure of variability between the expected and observed accuracy that better accounts for class imbalance (3.3% PUs).²³

We cross-validated each classifier with a range of minority class sampling techniques including class weightings, up-sampling the minority class, down-sampling the majority class, and Synthetic Minority Oversampling Technique (SMOTE). Each classifier was evaluated during cross validation, and the final models were chosen on that basis.

Finally, the optimal type of classifier was re-trained using the original predictor variables as well as Braden score information, including the overall Braden score as well as scores for the six subcomponents: mobility, activity, moisture, shear, nutrition and sensory.

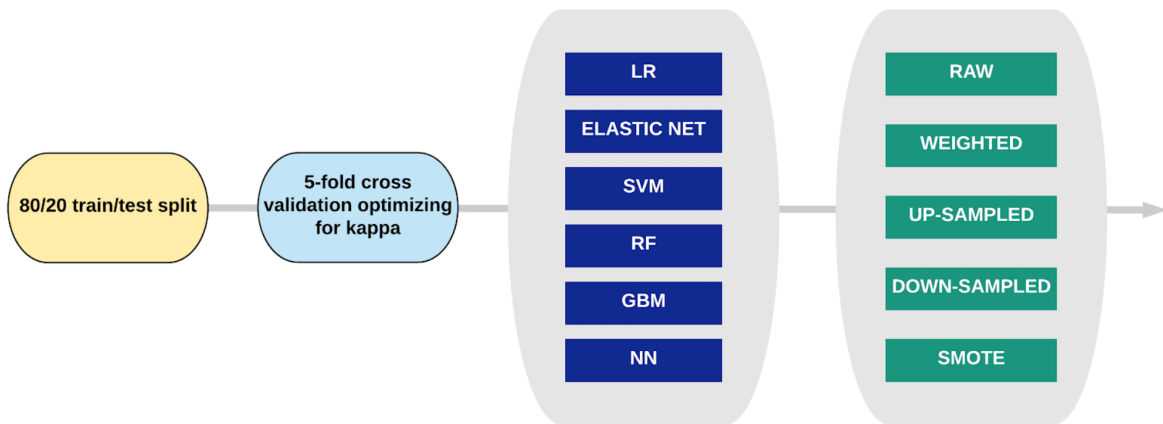


Fig. 3. Classifier training pipeline illustrating the range of models and sampling techniques used.

3. Results

3.1. Patient characteristics

Table 1 compares a range of demographic and clinical features between the PU (post 24h) and non-PU populations. The mean weight and age were higher among patients with PUs, as was the median length of stay. Additionally, PU patients had higher rates of ventilatory support (both non-invasive and mechanical ventilation), lower mean arterial pressure and lower arterial oxygen pressure. Of these 1690 PU patients, the median time to onset was 4.2 days post ICU admission and 13% demonstrated healing during the ICU, as evidenced by a reduction in stage between the final-recorded stage and maximum stage.

Table 1. Demographic and clinical features used in classifier training, compared between the PU and non-PU populations.

| | No pressure ulcers (n = 49161) | Pressure ulcers (n = 1690) | P value | % missingness |
|--|--------------------------------|----------------------------|---------|---------------|
| Demographics | | | | |
| Age (years, mean, SD) | 63.7 (17.2) | 66.5 (15.4) | <0.001 | 0 |
| Gender (% male) | 56 | 60 | 0.002 | 0 |
| Weight (kg, mean, SD) | 84 (25) | 87 (31) | <0.001 | 40 |
| Ethnicity (%) | | | <0.001 | 0 |
| Asian | 2.4 | 1.6 | | |
| Black | 9.5 | 7.5 | | |
| Hispanic | 3.4 | 2.7 | | |
| Other | 2.5 | 1.7 | | |
| Unknown | 10.5 | 12.7 | | |
| White | 71.6 | 73.8 | | |
| Length of stay (days, median, IQR) | 2.0 (1.2, 3.8) | 11.7 [6.1, 22.8] | <0.001 | 0.006 |
| Mortality within 12 months (%) | 42 | 56 | <0.001 | 0 |
| Glasgow Coma Scale (mean, SD) | 12.3 (3.3) | 9.6 (3.7) | <0.001 | 45 |
| Noninvasive ventilation (%) | 3.5 | 3.4 | <0.001 | 0 |
| Mechanical ventilation (%) | 39.4 | 67.9 | <0.001 | 0 |
| No prior admission with ulcer (%) | 25.4 | 33.8 | <0.001 | 0 |
| Braden score (mean, SD) | 15.0 (2.7) | 12.6 (2.2) | <0.001 | 47 |
| Time to ulcer onset (median, IQR) | NA | 4.2 (2.2, 9.3) | NA | 97 |
| Physiology (mean, SD) | | | | |
| Mean arterial pressure (MAP) | 79 (12) | 76 (12) | <0.001 | 44 |
| Cutaneous O2 | 97 (2) | 97 (2) | 0.3 | 44 |
| Arterial O2 | 163 (71) | 148 (57) | <0.001 | 67 |
| Arterial CO2 | 41 (9) | 41 (10) | 0.6 | 67 |
| Albumin | 3.1 (1.2) | 2.8 (0.6) | <0.001 | 77 |
| Blood glucose | 138 (50) | 148 (57) | <0.001 | 10 |
| Troponin (median, IQR) | 0.08 (0.01, 0.40) | 0.11 (0.04, 0.45) | NA | 74 |
| Hemoglobin | 10.8 (1.8) | 10.3 (1.6) | <0.001 | 6 |
| Creatinine | 1.4 (1.5) | 1.8 (1.6) | <0.001 | 1 |
| Sodium | 138 (4) | 138 (5) | 0.12 | 1 |
| Potassium | 4.1 (0.6) | 4.2 (0.6) | 0.3 | 1 |
| C-reactive protein (median, IQR) | 34 (7, 118) | 85 (30, 182) | NA | 99 |
| Erythrocyte Sedimentation Rate (median, IQR) | 35 (12, 74) | 38 (15, 76) | NA | 99 |
| Hemoglobin A1c (median, IQR) | 6.0 (5.6, 7.1) | 6.2 (5.8, 7.3) | NA | 96 |
| White blood cell count (median, IQR) | 11 (8, 14) | 12 (8, 17) | NA | 11 |
| Platelets (median, IQR) | 201 (148, 266) | 194 (132, 274) | NA | 11 |
| Chronic diseases (%) | | | | |
| Diabetes | 27 | 22 | <0.001 | 0 |
| Neuropathy | 5 | 4 | 0.16 | 0 |
| Peripheral vascular disease | 27 | 22 | <0.001 | 0 |
| Amputation | 0.1 | 0 | 0.7 | 0 |
| Spinal cord injury | 0.2 | 0.1 | 0.9 | 0 |
| Coronary artery disease | 4 | 4 | 0.9 | 0 |
| Leukemia | 1 | 1 | 0.5 | 0 |
| Stroke | 1.5 | 1.2 | 0.4 | 0 |
| Heart failure | 27 | 25 | 0.03 | 0 |
| Anemia | 18 | 14 | <0.001 | 0 |

3.2. Prognostic performance of Braden score

Figure 4 shows the distribution of Braden scores at 24h across the Carevue population. Figure 5 shows the precision and recall of various thresholds of the Braden score at 24h in predicting future PU development. High risk is defined as a score of 12 or below and severe risk as 9 or below.^{24,25} The high-risk threshold had precision 0.09 and recall 0.49; while the severe-risk threshold had precision 0.15 and recall 0.08.

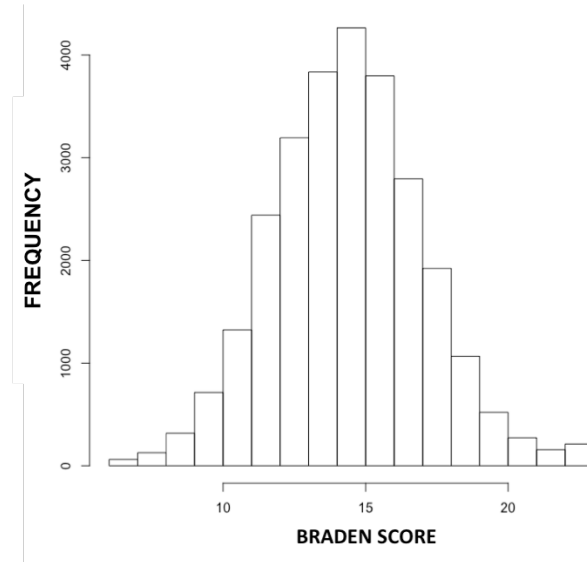


Fig. 4. Histogram of Braden scores measured within the first 24h of admission for Carevue patients (range 6-23).

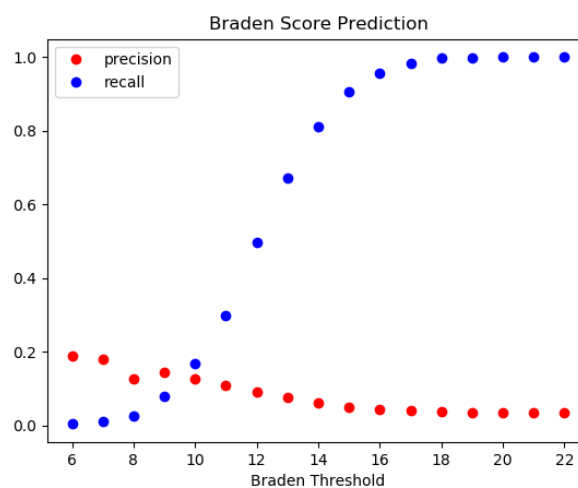


Fig. 5. Precision and recall at various thresholds of Braden score (measured within 24h) in predicting future PU development.

3.3. Classifier performance

The optimal classifier on the entire dataset during cross-validation was the weighted elastic net with alpha 0.1, lambda 0.002. This had precision 0.09, recall 0.74 on the test set. The optimal classifier on the Carevue-only dataset was a logistic regression model, which had precision 0.09, recall 0.73.

The performance of all tuned models on the test set is shown in Table 2. Many of the models operated as majority classifiers in the unweighted scenario, labeling every case as a non-ulcer. Kappa score, and performance of the test set, tended to increase when trained with model weights or SMOTE sampling. There was no significant performance boost observed after training on the entire dataset.

Table 2. Classifier performance on Carevue-only and all data, based on raw training data (unweighted), model class weights (weighted) or synthetic minority oversampling (SMOTE).

| CAREVUE (n=28,886) | | | | | | |
|----------------------------|------------------|---------------|------------------|---------------|------------------|---------------|
| | UNWEIGHTED | | WEIGHTED | | SMOTE | |
| | <i>Precision</i> | <i>Recall</i> | <i>Precision</i> | <i>Recall</i> | <i>Precision</i> | <i>Recall</i> |
| BRADEN SCORE | 0.09 | 0.50 | | | | |
| LR | 0.60 | 0.03 | 0.09 | 0.73 | 0.11 | 0.49 |
| Elastic Net | NA | 0 | 0.09 | 0.74 | 0.13 | 0.57 |
| RF | NA | 0 | NA | 0 | 0.14 | 0.29 |
| GBM | 0.11 | 0.33 | 0.05 | 0.95 | 0.07 | 0.61 |
| SVM | NA | 0 | NA | 0 | 0.10 | 0.49 |
| Neural net | NA | 0 | 0.09 | 0.75 | 0.11 | 0.45 |
| ALL DATA (n=50,851) | | | | | | |
| LR | 0.29 | 0.006 | 0.09 | 0.72 | 0.12 | 0.49 |
| Elastic Net | 0.25 | 0.006 | 0.09 | 0.74 | 0.12 | 0.51 |
| RF | NA | 0 | NA | 0 | 0.16 | 0.31 |
| GBM | 0.15 | 0.13 | 0.33 | 0.005 | 0.07 | 0.66 |
| SVM | NA | 0 | NA | 0 | 0.1 | 0.41 |
| Neural net | NA | 0 | 0.08 | 0.69 | 0.13 | 0.43 |

3.4. Feature importances

Table 3 shows the standardized regression coefficients of the top 10 predictors in two of the highest performing models. Multiple features are represented in both, including GCS, BUN, paO₂, albumin and previous ulcer-free admission. Notably, both amputation and previous ulcer-free admission were negatively correlated with PU development.

Table 3. Top 10 features for the weighted logistic regression model trained on Carevue data (left) and elastic net trained on all data (right) based on relative variable importance.

| Weighted logistic regression (Carevue) | | Weighted Elastic Net (all data) | |
|--|----------------------------|---------------------------------|----------------------------|
| <i>Variable</i> | <i>Standardized coeff.</i> | <i>Variable</i> | <i>Standardized coeff.</i> |
| GCS | -0.63 | Amputation | -1.07 |
| BUN | 0.36 | Mechanical ventilation | 1.03 |
| paO ₂ | -0.33 | Spinal Cord Injury | 0.92 |
| Previous ulcer-free admission | 0.51 | Albumin | -0.61 |
| Cardiac Surg. Recovery Unit | -0.74 | Cardiac Surg. Recovery Unit | -0.57 |
| Albumin | -0.45 | Non Invasive Ventilation | 0.36 |
| Potassium | -0.20 | BUN | 0.32 |
| Male gender | 0.33 | Previous ulcer-free admission | 0.30 |
| Medical ICU | -0.49 | paO ₂ | -0.28 |
| Time to ICU admission | 0.15 | GCS | -0.26 |

3.5. Classifier with Braden

When Braden features were integrated into the feature matrix, the optimal classifier on all data was again the weighted elastic net, which showed essentially unchanged performance relative to the classifier without Braden data - precision 0.09, recall 0.75.

4. Discussion

We demonstrated that a weighted elastic net using 29 EHR-derived features from the first 24h of an ICU admission is able to outperform the industry standard (nurse-calculated Braden score) in recall and match its precision. Given the high recall, this classifier may have utility as an automated screening tool for PUs early in a patient’s admission. Preventing a PU in a high-risk patient is more effective and less costly than treating an evolving PU. Moreover, interventions such as frequent rolling and foam padding are relatively low-cost, increasing our tolerance for false positives.

This elastic net model is interpretable and allows clinicians to better understand the impact of clinical and demographic factors on PU development. Important features were broadly consistent with domain knowledge and clinical intuition surrounding PUs. For example, it is intuitive that mechanical ventilation, low GCS and low albumin would be positively associated with PU development as they are proxies for immobility and impaired nutritional status. The time between hospital admission and ICU admission was also positively correlated, supporting the hypothesis that longer patient stays on the wards prior to ICU admission are associated with PU incidence. Although amputation was the leading feature in the elastic net model, the extremely low prevalence of this condition and the known association between amputation and PUs casts doubt over this feature as a key driver. Iterative feature elimination of low variance and low prevalence features could improve our confidence around these regression coefficients.

Including Braden features in the model did not improve performance, suggesting that the

Braden scoring system does not add additional information to the EHR-based model. As Kaewperg *et al.* describe, Braden subscales for activity, nutrition, mobility, sensory perception, and moisture are often not useful predictors because they tend to have similar values among ICU patients.¹³ By using a broader range of EHR-derived features, our model can be used to capture risk factors that meaningfully differ between patients with critical illness, and thereby achieve better discrimination of risk status. Additionally, an EHR-based model removes the need for nurses to manually calculate Braden risk scores, which is time consuming, subjective and can easily be overlooked.

The descriptive statistics provide a unique insight into the burden of PUs in a tertiary ICU, using EHR data alone. The prevalence of PUs has been quoted as high as 49%, however we find 7.8% of admissions have a PU of stage 2 or higher recorded. Interestingly, of the 1690 PU cases that developed after 24h (which removes chronic PUs), only 13% demonstrate healing during the patient’s admission, which is a metric not previously investigated in EHR data.

The key limitation of this study is that the underlying pathophysiology of PUs may simply not be reflected in EHR data. The main driver of PUs is consistent skin pressure over bony prominences,²² and although we can find proxies in the EHR for immobility and impaired healing, these are second-order features that do not directly capture the pressure waveform on, for example, a patient’s sacrum. This makes it very challenging to predict future onset of pressure injury. Furthermore, the EHR features that are available are affected by missingness. For example, a significant proportion of the population are missing height and weight data, preventing the calculation of Body Mass Index (BMI), an important risk factor in PU development. Finally, there was a significant class imbalance in our study design, with only 3.3% of cases developing a PU. Many of the classifiers defaulted to predicting the majority class. This was addressed with selection of kappa score as the optimization metric, along with several class re-balancing techniques; however we only observed incremental performance improvements.

Future work will involve an iterative process of feature engineering and model tuning in an effort to increase the precision of our classifier. A higher precision model could help to inform the allocation of expensive prophylactic resources such as pressure support mattresses. Additionally, the model must be deployed as a clinical decision support tool, as in Cho *et al.*, in order to fully evaluate its impact on PU incidence. This is the practical benchmark for utility, beyond precision and recall scores.¹⁵

5. Conclusion

In this paper we develop a predictive model which outperforms the Braden scale for predicting future PU development at 24h. This model uses EHR-derived data elements and could be a means to automatically screen patients for PU risk early in an admission without the need manual Braden scoring by nurses. The optimal models show an association between PU

development and EHR-derived proxies for immobility (e.g., spinal cord injury and low GCS), nutritional status (e.g., low albumin) and impaired skin healing, (e.g., low paO_2). While additional refinement of our model is warranted, implementation in a clinical setting as a decision support tool may decrease the incidence of PUs, as has been demonstrated in other literature.

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