

Automated Diagnosis of Parkinson's Disease Using Machine Learning

Kajal Kumari Yadav, Shyam Shankar Dwivedi

Computer science and Engineering Department,
Rameshwaram Institute of Technology & Management, Lucknow
kajalyadav707146@gmail.com

Abstract: The prediction of Parkinson's disease has been a focal point for researchers for over a decade. The Parkinson's disease dataset is readily available on machine learning repository websites. Researchers have employed various techniques including data mining, machine learning, and deep learning to develop prediction systems aimed at early detection of Parkinson's disease, crucial for managing a disease whose healthcare costs are often prohibitive in developing and underdeveloped countries. In our proposed study, we will preprocess the dataset through exploratory data analysis and build a prediction system using optimized machine learning models such as k-nearest neighbors, logistic regression, decision tree classifier, and random forest classifier. Each algorithm will be applied to predict outcomes, followed by a comprehensive multidimensional analysis to identify the most suitable model. The dataset comprises data from a study involving 31 individuals, among whom 23 are diagnosed with Parkinson's disease.

Keywords: Parkinson's Disease, Classification, Random Forest, Support Vector Machine, Machine Learning.

1. Introduction:

Parkinson's disease (PD) is a chronic and progressive neurodegenerative disorder characterized by the loss of nigrostriatal dopaminergic pathways [1]. Its prevalence in the general population ranges from 0.1% to 0.3%, with a notable increase among individuals aged over 65 years [2]. Key clinical features of PD include tremor, rigidity, akinesia (such as bradykinesia and hypokinesia), and postural instability. Alongside these motor symptoms, PD commonly presents with a spectrum of non-motor signs and symptoms encompassing autonomic dysfunctions (such as hyperhidrosis, orthostatic hypotension, and sexual-urinary dysfunction), sleep disturbances, neuropsychiatric issues (including apathy, fatigue, depression, anxiety, dementia, and psychosis), sensory problems (like internal tremor, restless leg syndrome, numbness, paresthesia, visual disturbances), and pain [4-7]. Pain affects approximately 30% to 50% of PD patients and can increase to 68% to 85% when all types of pain are considered [8]. It can manifest at any stage of the disease, sometimes preceding formal diagnosis [9]. Despite ongoing research, consensus on the classification and underlying mechanisms of pain in PD remains elusive. This review aims to explore existing data on potential mechanisms, classification,

assessment, and risk factors associated with pain in individuals with PD.

Parkinson's disease is increasingly recognized as a significant degenerative disorder of the central nervous system, profoundly impacting the quality of life for millions of older adults globally [1]. The progression of PD symptoms varies widely among individuals due to the disease's heterogeneity. Patients may exhibit resting tremors, limb rigidity, and difficulties with gait and balance. PD symptoms are broadly categorized into motor symptoms, which are movement-related, and non-motor symptoms that include depression, sleep disorders, anosmia (loss of sense of smell), and cognitive impairment. Non-motor symptoms often impose greater burden than motor symptoms. According to the Centers for Disease Control and Prevention (CDC), PD complications rank as the 14th leading cause of death in the United States. Despite extensive research, the precise cause of PD remains largely unknown. The economic impact of PD, encompassing direct medical costs, social security expenditures, and lost productivity, is estimated to exceed \$52 billion annually in the United States alone. Globally, more than 10 million people are affected by PD. Early detection is crucial as it enables prompt treatment initiation and significant symptom alleviation [2]. Detecting PD early is pivotal in slowing disease progression and potentially enabling access to disease-modifying therapies when available.

2. Literature Review:

Pain, characterized as a profound and tangible experience linked to actual or potential injury, is defined by the International Association for the Study of Pain (IASP) [10]. Nociception, the process initiating pain, involves several anatomical structures. Nociceptors, including thinly myelinated Ad fibers responsive to mechanical and thermal stimuli, and unmyelinated C fibers (polymodal) sensitive to mechanical, thermal, or chemical stimuli, transmit signals to dorsal horn neurons of the spinal cord. Lamina II, known as the substantia gelatinosa, plays a crucial role in regulating these pain signals [10,11]. This region acts as a transitional gateway, modulating transmission to lamina V neurons, which further relay sensory inputs to the brain. The substantia gelatinosa inhibits these neurons, reducing their inhibitory effect on ascending sensory pathways. Consequently, sensory transmission is enhanced, aligning with the gate control theory at the spinal level [12].

Two phylogenetically distinct systems, the medial and lateral pain pathways, relay pain signals to higher brain centers. The medial system comprises fibers such as paleospinothalamic, spinomesencephalic, spinoreticular, spinoparabrachial, hypothalamic, and spinothalamic tracts. These fibers ascend and descend to various brain structures, including the parabrachial nucleus, locus coeruleus (reticular formation), periaqueductal gray (mesencephalon), thalamic nuclei (intralaminar and medial), insula, parietal operculum, secondary somatosensory cortex, amygdala, and hippocampus.

3. Methodology:

This section focuses on detailing the dataset utilized and the methodologies employed for early prediction of Parkinson’s disease in patients. The chosen approaches aim to differentiate Parkinson’s disease patients from healthy individuals. The strategy involves conducting a comparative analysis of various machine learning techniques by applying different models to the dataset and determining the optimal technique based on performance metrics such as accuracy, ROC (Receiver Operating Characteristic), AAE (Average Absolute Error), and ARE (Average Relative Error). Additionally, the study extends its scope by integrating the Boruta feature selection technique.

Algorithm:

- Step1: Data Gathering
- Step 2: Data preparation
- Step 3: Model Selection
- Step 4: Training
- Step 5: Evaluation
- Step 6: Prediction

Random Forest

Random Forest is a versatile machine learning method utilized for both classification and regression tasks. It belongs to the ensemble methods category, where a collection of weak models collectively forms a robust predictive model. In a random forest, multiple trees are generated, and each tree provides a classification. The final classification is determined by aggregating the votes from all trees in the forest, selecting the class with the highest number of votes.

Random Forest Prediction Pseudo code:

Here’s a revised version:

1. It uses the features of the test set to build decision trees for predicting outcomes, saving these predictions.
2. Votes are computed for each predicted outcome.
3. The final prediction is based on the highest-voted outcome among all predictions.

4. Result and discussion:

The data correlation is determined using the Pearson coefficient technique. Each feature in the feature set is correlated with the status label. In the correlation table depicted in Figure 4.5, it is evident that features such as sex, test time, and those related to Shimmer do not exhibit strong correlations with the status column. Utilizing these columns could result in an undertrained

machine learning model. Figure 2 displays a heatmap illustrating these correlations.

status	1.000000	Shimmer	-0.092105
age	0.258102	Shimmer(dB)	-0.079906
sex	0.005190	Shimmer:APQ3	-0.092579
test_time	0.045205	Shimmer:APQ5	-0.100546
motor_UPDRS	0.428504	Shimmer:APQ11	-0.051410
total_UPDRS	0.412621	Shimmer:DDA	-0.092577
Jitter(%)	-0.045653	NHR	-0.118059
Jitter(Abs)	-0.057245	HNR	0.130205
Jitter:RAP	-0.052936	RPDE	-0.143498
Jitter:PPQ5	-0.073836	DFA	-0.156187
Jitter:DDP	-0.052922	PPE	0.014561

Fig. 1: Correlation table.

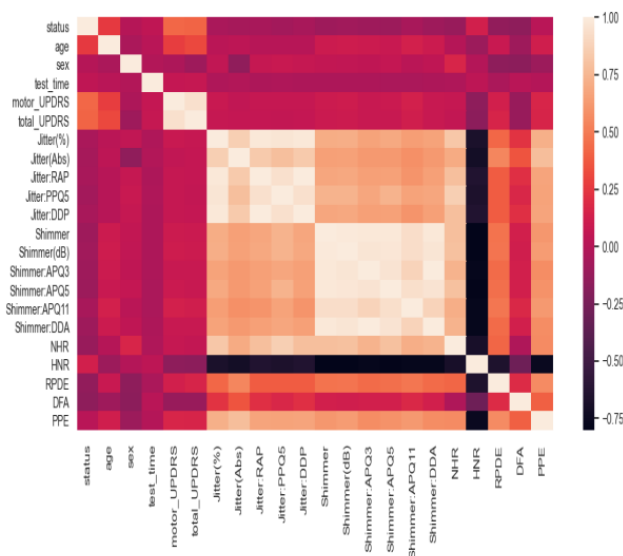


Fig. 2: heat map of the Correlation.

The dataset is divided into a feature set and labels, followed by splitting into an 80:20 ratio for training and testing. Standard scaling is applied using standard scalers, which normalizes data based on its standard deviation. Training data is utilized to train multiple machine learning models such as K-neighbors, Logistic Regression, Decision Tree Classifier, and Random Forest Classifier. These models undergo hyperparameter tuning using GridSearchCV before training. For instance, the KNN classifier is tuned for the 'n_neighbors' parameter, typically set to 5 by default, achieving maximum accuracy with a value of 7. Figure 3 illustrates the resulting prediction accuracies of both tuned and non-tuned models.

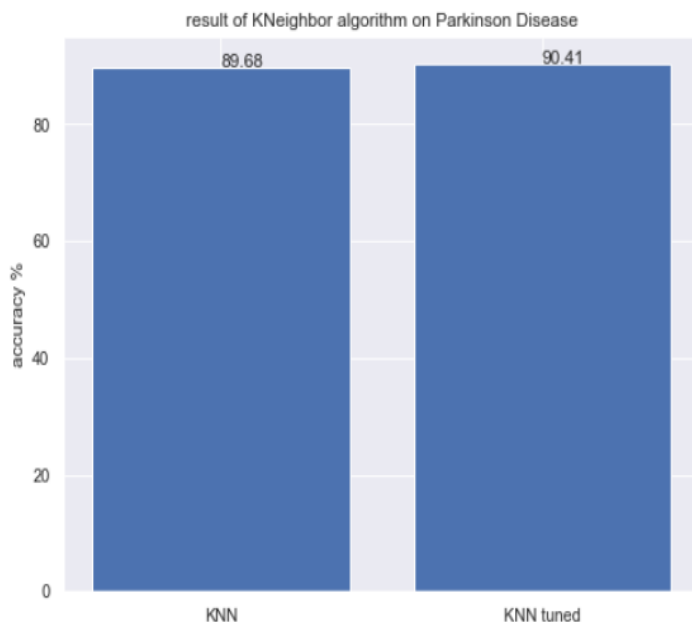


Fig 3: the prediction accuracy of KNN.

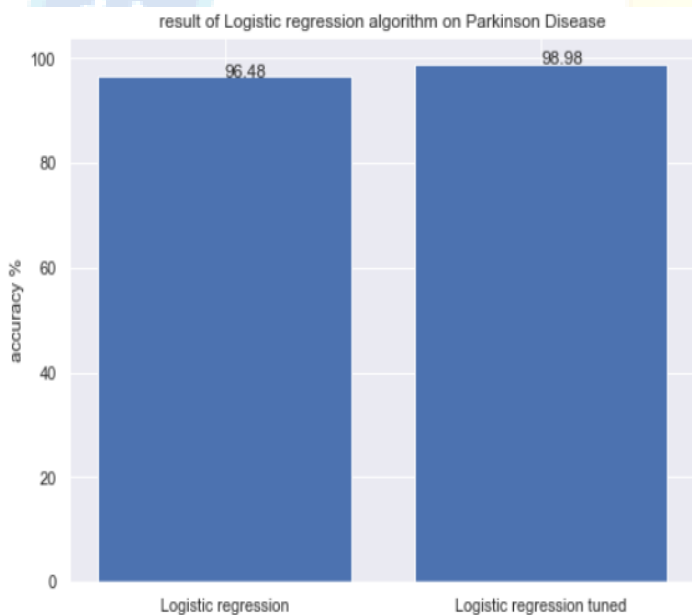


Fig. 4: the prediction accuracy of Logistic regression.

The logistic regression model undergoes hyperparameter tuning for the learning rate (C), penalty, and maximum iterations to optimize the theta parameters crucial for building effective logistic regression models. After hyperparameter tuning, the values are set to C = 1000, penalty = 12, and maximum iterations = 120000.

5. Conclusion:

Parkinson's disease directly impacts the central nervous system and primarily affects patients' motor functions. The disease is primarily caused by a reduction in dopamine levels due to damage to nerve cells. Key symptoms include stiffness, impaired movement, and tremors. It is a progressive disease with no cure, managed through ongoing medical care. Machine learning models were applied to a dataset after performing exploratory data analysis and preprocessing steps such as scaling and feature extraction. The models underwent hyperparameter tuning using the gridsearchcv module. Based on the results, the random forest model emerged as the most effective for predicting Parkinson's disease.

References:

- [1] McNamara P, Stavitsky K, Harris E, Szent-Imrey O, Durso R. Mood, side of motor symptom onset and pain complaints in Parkinson's disease. *Int J Geriatr Psychiatry* 2010;25:519e24.
- [2] Weintraub D, Comella CL, Horn S. Parkinson's disease-Part 1: pathophysiology, symptoms, burden, diagnosis, and assessment. *Am J Manag Care* 2008; 14:40e8.
- [3] Alves G, Forsaa EB, Pedersen KF, Dreetz-Gjerstad M, Larsen JP. Epidemiology of Parkinson's disease. *J Neurol* 2008;255:18e32.
- [4] Park A, Stacy M. Non-motor symptoms in Parkinson's disease. *J Neurol* 2009; 256:293e8.
- [5] Simuni T, Sethi K. Nonmotor manifestations of Parkinson's disease. *Ann Neurol* 2008;64:S65e80.
- [6] Bayulkem K, Lopez G. Non-motor fluctuations in Parkinson's disease: clinical spectrum and classification. *J Neurol Sci* 2010;289:89e92.
- [7] Wolters ECH. Non-motor extranigral signs and symptoms in Parkinson's disease. *Parkinsonism Relat Disord* 2009;3:S6e12.
- [8] Beiske AG, Loge JH, Rønningen A, Svensson E. Pain in Parkinson's disease: prevalence and characteristics. *Pain* 2009;141:173e7.
- [9] Tolosa E, Compta Y, Gaig C. The premotor phase of Parkinson's disease. *Parkinsonism Relat Disord* 2007;13:S2e7.
- [10] Almeida TF, Roizenblatt S, Tufik S. Afferent pain pathways: a neuroanatomical review. *Brain Res* 2004;1000:40e56.
- [11] Millan MJ. The induction of pain: an integrative review. *Prog Neurobiol* 1999; 57:1e164.
- [12] Wall PD, Melzack R. *Textbook of pain*. Churchill London: Livingstone; 1994.
- [13] Scherder E, Wolters E, Polman C, Sergeant J, Swaab D. Pain in Parkinson's disease and multiple sclerosis: its relation to the medial and lateral pain systems. *Neurosci Biobehav Rev* 2005;29:1047e56.
- [14] Willis WD, Westlund KN. Neuroanatomy of the pain system and of the pathways that modulate pain. *J Clin Neurophysiol* 1997;14:2e31.
- [15] Nolano M, Provitera V, Estraneo A, Selim MM, Caporaso G, Stancanelli A, et al. Sensory deficit in Parkinson's disease: evidence of a cutaneous denervation. *Brain* 2008;131:1903e11.



- [16] Braak H, Sastre M, Bohl JR, de Vos RA, Del Tredici K. Parkinson's disease: lesions in dorsal horn layer I, involvement of parasympathetic and sympathetic pre- and postganglionic neurons. *Acta Neuropathol* 2007;113: 421e9.
- [17] Braak H, Del Tredici K, Rub U, de Vos RA, Jansen Steur EN, Braak E. Staging of brain pathology related to sporadic Parkinson's disease. *Neurobiol Aging* 2003;24:197e211.
- [18] Braak H, Rub U, Jansen-Steur EN, Del Tredici K, de Vos RA. Cognitive status correlates with neuropathologic stage in Parkinson disease. *Neurology* 2005; 64:1404e10.
- [19] Chudler EH, Dong WK. The role of the basal ganglia in nociception and pain. *Pain* 1995;60:3e38.

