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The Use of Hounsfield Unit as a Quantitative Indicator for Detecting Stroke Phases on Non-Contrast Head CT-Scan

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ABSTRACT

Introduction: Ischemic stroke is a major cause of morbidity and mortality worldwide. Early determination of the stroke phase is essential for guiding appropriate management and predicting prognosis. Non-contrast head computed tomography (CT) remains the first-line imaging modality in acute stroke evaluation.

Material and Methods: This analytic observational study with a cross-sectional approach was conducted at Dr. Saiful Anwar General Hospital, Malang, Indonesia. A total of 45 patients diagnosed with ischemic stroke were included and divided equally into three groups: acute, subacute, and chronic (15 patients each). HU values were measured using three regions of interest (ROIs) of 1 cm² placed within the infarcted area and averaged for analysis.

Results: Mean HU values decreased progressively with stroke phase progression: acute 25.84 ± 1.07, subacute 18.11 ± 0.96, and chronic 9.24 ± 0.96. The Kruskal–Wallis test revealed a significant difference in HU values among the three groups ($p < 0.001$). Post hoc analysis using Dunn's test confirmed significant differences between all phases (acute–subacute = 7.73 HU; subacute–chronic = 8.87 HU; acute–chronic = 16.60 HU; all $p < 0.001$). Clinically, decreased consciousness predominated in the acute phase (66.7%), while hemiparesis was more common in subacute (66.7%) and chronic (60%) phases, reflecting underlying tissue pathophysiology.

Conclusion: Quantitative HU measurement on non-contrast CT provides a reliable and objective tool for differentiating ischemic stroke phases. The progressive decline in HU values corresponds with the temporal evolution of infarction, making HU analysis a useful adjunct to conventional qualitative CT interpretation.

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INTRODUCTION

Stroke is a neurological deficit resulting from a disturbance in cerebral blood flow, classified into ischemic and haemorrhagic types. Globally, stroke ranks as the second leading cause of death, accounting for approximately 11.6% of all fatalities in 2019 [1]. The lifetime risk of experiencing any stroke after the age of 25 is 24.9%, with ischemic stroke specifically accounting for 18.3% [2]. In Indonesia, stroke remains the number one cause of mortality, contributing to 15.9% of all deaths, with a steadily increasing prevalence linked to hypertension, diabetes mellitus, smoking, and obesity [3].

Non-contrast CT (NCCT) remains the most widely available and rapid imaging modality for evaluating suspected acute stroke, capable of distinguishing ischemic from haemorrhagic stroke [4]. However, during the early phase of ischemic stroke, visual detection may be challenging due to minimal density changes [4]. This limitation increases reliance on radiologist experience, leading to potential inter-observer interpretation variability. Quantitative CT-based parameters such as Hounsfield Unit (HU) measurement have therefore been explored to provide a more objective evaluation of parenchymal density alterations during stroke evolution [5].

HU reduction reflects increased tissue water content associated with cytotoxic and vasogenic oedema, making it a promising biomarker for ischemic phase characterization [5]. Quantitative CT analysis using Hounsfield Units (HU) offers an objective approach to detect subtle density variations, thereby improving diagnostic reliability and inter-observer agreement, also shows the cut off for each phases [5].

HU values represent tissue radiodensity measured on CT scans, reflecting variations in water content and tissue composition. During ischemia, cytotoxic oedema leads to decreased HU values due to increased intracellular water. As ischemia progresses, vasogenic oedema and necrosis alter tissue attenuation further, producing characteristic HU value changes across different stroke phases [5]. Prior studies have suggested that HU measurement can assist in phase determination and prognosis prediction, but its application in clinical settings, particularly in Indonesia, remains limited.

This study aims to investigate the use of HU values as quantitative, standardized indicators for identifying stroke phases on non-contrast CT scans. Establishing specific HU cut-off values for each phase is expected to enhance diagnostic accuracy, support timely therapeutic decisions, and promote objective imaging assessment in stroke management.

MATERIAL AND METHODS

This study employed an analytical observational design with a cross-sectional approach. The research was conducted in the Department of Radiology, Dr. Saiful Anwar General Hospital, Malang, East Java, Indonesia, over a period of three months (April-June 2025). Patient's demographic and stroke phase categories were collected from the medical record, while CT scan images were taken from PACS. The study population consisted of all patients clinically diagnosed with ischemic stroke who underwent non-contrast head CT as part of their diagnostic evaluation. Research ethics approval was obtained for this research from the Ethics Committee for Health Research of Dr. Saiful Anwar General Hospital, number board 400/311/K.3/102.7/2025.

The inclusion criteria were patients diagnosed with ischemic stroke with a clearly documented onset time of symptoms who underwent a non-contrast CT examination at Dr. Saiful Anwar General Hospital. Exclusion criteria comprised patients diagnosed with haemorrhagic stroke and brain neoplasms. The sample size was determined using the formula for a cross-sectional study design, resulting in a minimum of 11 respondents for each stroke phase group (acute, subacute, and chronic), ensuring adequate statistical representation. The independent variable in this study was the ischemic stroke phase categories based on the time from symptom onset, while the dependent variable was the quantitative Hounsfield Unit (HU) value measured on non-contrast CT scans.

Accordingly, the acute phase was defined as within 1–7 days after symptom onset, representing cytotoxic oedema and early parenchymal hypodensity; the subacute phase spanned 8–21 days, indicating progressive vasogenic oedema, necrosis, and partial disruption of gray–white matter differentiation; and the chronic phase was defined as beyond 21 days after onset, characterized by gliosis, encephalomalacia, and

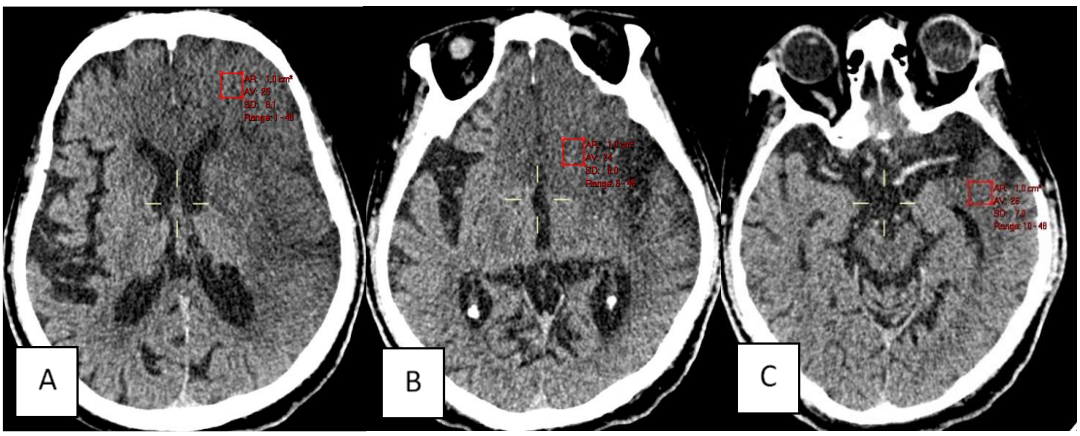


Fig.1. Placement of Square Regions of Interest (ROI) on Non-Contrast Head CT

A 71-year-old patient with a clinical onset of 48 hours. Three square ROIs, each measuring 1.0 cm², were manually positioned within the infarcted brain parenchyma at different representative locations to account for spatial variation in attenuation. The HU values obtained from the three regions were 25 HU in image A, 24 HU in image B, and 26 HU in image C. The mean HU value for the lesion was then calculated as the average of these three measurements, resulting in a representative quantitative value of 25 HU for that infarcted region.

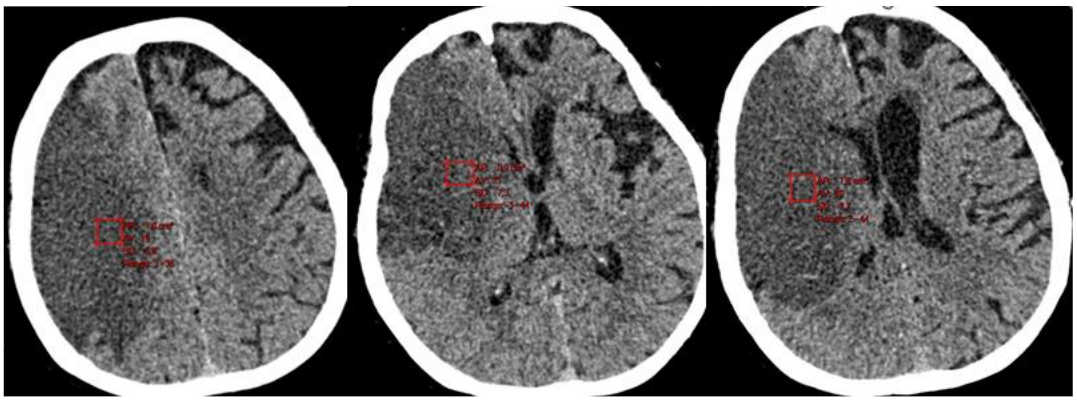


Fig.2. Placement of Square Regions of Interest (ROI) on Non-Contrast Head CT

A 67-year-old patient with a clinical onset of 12 days. Three square ROIs, each measuring 1.0 cm², were manually positioned within the infarcted brain parenchyma at different representative locations to account for spatial variation in attenuation. The HU values obtained from the three regions were 19 HU in image A, 21 HU in image B, and 20 HU in image C. The mean HU value for the lesion was then calculated as the average of these three measurements, resulting in a representative quantitative value of 20 HU for that infarcted region.

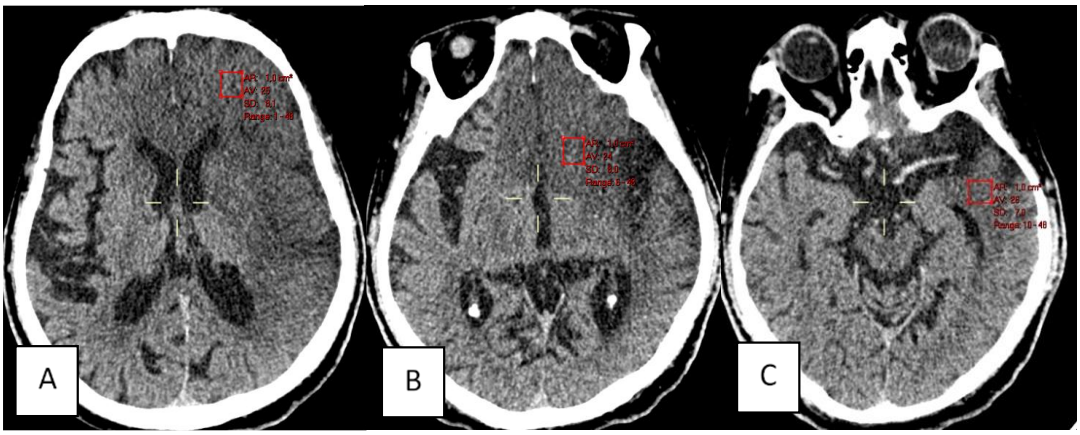


Fig.3. Placement of Square Regions of Interest (ROI) on Non-Contrast Head CT

A 57-year-old patient with hemiparesis for the last 2 months. Three square ROIs, each measuring 1.0 cm², were manually positioned within the infarcted brain parenchyma at different representative locations to account for spatial variation in attenuation. The HU values obtained from the three regions were 9 HU in image A, 10 HU in image B, and 10 HU in image C. The mean HU value for the lesion was then calculated as the average of these three measurements, resulting in a representative quantitative value of 9,67 HU for that infarcted region.

parenchymal volume loss with attenuation values approximating cerebrospinal fluid. This temporal classification is consistent with the radio pathological evolution of cerebral infarction as reported in previous HU-based analyses [5–8].

HU measurements were performed manually using the region of interest (ROI) technique, with each ROI standardized to an area of 1 cm² to maintain measurement consistency as shown in Fig.1 [6]. For each patient, the ROI was placed in three different representative locations within the infarcted area, and the mean HU value from these three regions was calculated. A corresponding ROI of equal size was also placed in the contralateral normal brain tissue for comparison. ROI placement avoided cerebrospinal fluid spaces, calcifications, bone margins, and large vascular structures to minimize measurement bias. All CT scans were performed using a multidetector CT scanner with standard non-contrast brain protocols and a slice thickness of 5 mm. HU values were measured using DICOM viewer software in Vue PACS.

RESULTS

A total of 45 patients diagnosed with ischemic stroke were included in this study. The subjects were divided into three groups based on the phase of infarction: acute,

subacute, and chronic, with 15 patients in each group. All patients underwent non-contrast head CT examination as part of their diagnostic evaluation. Quantitative assessment of the infarcted regions was performed using Hounsfield Unit (HU) measurements obtained from three distinct regions of interest (ROIs) within each lesion, and the mean HU value for each patient was calculated. The HU values were then analysed descriptively to identify characteristic attenuation patterns across different stroke phases.

The distribution of clinical symptoms differed among the stroke phases. In the chronic phase, the most frequent presenting complaint was hemiparesis, observed in 9 patients (60%), followed by gradual decrease of consciousness (DOC) in 3 patients (20%), and aphasia in 3 patients (20%). In the subacute phase, hemiparesis remained the predominant symptom, affecting 10 patients (66.7%), while decrease of consciousness was reported in 5 patients (33.3%). In contrast, in the acute phase, decrease of consciousness was the most common clinical manifestation, occurring in 10 patients (66.7%), followed by hemiparesis in 4 patients (26.7%) and aphasia in 1 patient (6.7%), as shown in Table 2.

Overall, these findings indicate that hemiparesis tends to dominate in the later phases of ischemic stroke (subacute and chronic), reflecting established motor deficits due to infarction and neuronal loss, whereas decreased consciousness is more frequently observed in the acute phase, likely associated with cerebral oedema and perfusion failure occurring in the early stage of ischemic injury.

Quantitative analysis of Hounsfield Unit (HU) measurements demonstrated a progressive decline in mean attenuation values corresponding to the chronological phase of ischemic stroke. The acute phase group exhibited the highest mean HU value of 25.84 ± 1.07, indicating relatively higher tissue density consistent with the early stage of cytotoxic oedema and minimal liquefactive necrosis. In the subacute phase, the mean HU value decreased to 18.11 ± 0.96, reflecting ongoing vasogenic oedema, tissue necrosis, and partial loss of parenchymal density. The chronic phase showed the lowest mean HU value of 9.24 ± 0.96, representing advanced infarct evolution characterized by gliosis and encephalomalacia, where the attenuation approaches that of cerebrospinal fluid, as shown in Table 3.

This descending trend of HU values from the acute to the chronic phase illustrates the expected radiological evolution of ischemic brain tissue on non-contrast CT imaging. The consistent reduction in HU values across the phases indicates that quantitative HU measurement can serve as a reliable and objective indicator for determining the temporal stage of ischemic stroke, complementing conventional qualitative CT assessment.

In the acute phase, HU values are tightly clustered with a narrow IQR, indicating relatively low variability. The subacute phase shows a slightly wider spread of HU values, while the chronic phase demonstrates both the lowest median HU and the smallest overall range. One mild outlier is observed in the chronic group, suggesting a few values slightly below the main cluster.

Overall, the boxplot confirms the trend that HU values decrease progressively from the acute to the chronic phase, consistent with the results of the Kruskal–Wallis analysis. This pattern reflects the expected reduction in tissue density as ischemic damage evolves over time, as shown in Fig.2.

The Kruskal–Wallis test was performed to compare Hounsfield Unit (HU) values among the acute, subacute, and chronic stroke phases. The results showed in Table 3 with a significant difference in HU values among the three groups ($p < 0.001$).

The acute phase had the highest mean and rank sum of HU values, followed by the subacute phase, while the chronic phase had the lowest. This indicates that HU values decrease progressively as the stroke phase advances from acute to chronic.

Post hoc analysis using Dunn's test revealed statistically significant pairwise differences in HU values among all stroke phases ($p < 0.001$). The mean rank difference between the acute and subacute phases was 7.73, between the subacute and chronic phases was 8.87, and between the acute and chronic phases was 16.60.

These results confirm that each stroke phase demonstrates distinct attenuation characteristics, with HU values progressively declining from acute to chronic stages. This finding aligns with the underlying pathophysiological sequence of ischemic tissue—transitioning from cytotoxic oedema in the acute phase, to vasogenic oedema and necrosis in the subacute phase, and finally to gliosis and encephalomalacia in the chronic stage.

All pairwise comparisons showed $p<0.001$, confirming that each stroke phase differed significantly in mean HU value. The progressive reduction in attenuation across phases can objectively distinguish between acute, subacute, and chronic infarctions.

DISCUSSION

Stroke remains a major global cause of mortality and disability, with ischemic stroke constituting nearly 85% of all cases [1,2]. In Indonesia, the national health survey also reports a rising incidence of stroke, emphasizing the need for accurate, accessible diagnostic tools [3]. Non-contrast computed tomography (NCCT) continues to serve as the first-line imaging modality for acute stroke assessment because of its speed,

availability, and capability to detect early parenchymal changes [4]. Beyond detecting haemorrhage, the use of Hounsfield Unit (HU) values provides a promising quantitative parameter for evaluating infarct evolution.

In the present study, a significant and progressive decline in mean Hounsfield Unit (HU) values was observed across the acute, subacute, and chronic phases of ischemic stroke. The Kruskal–Wallis test showed a statistically significant difference among the three groups ($p < 0.001$). Post hoc analysis using Dunn's test confirmed significant differences between the acute–subacute and subacute–chronic phases ($p < 0.001$), indicating that each stage of infarction exhibits distinct attenuation characteristics on non-contrast CT. These findings indicate that attenuation differences between each stage are not random but correspond closely to the biological progression of ischemic injury. The largest HU gap was observed between the acute and chronic groups, consistent with the complete transition from cytotoxic to vasogenic oedema and finally to encephalomalacia tissue degeneration [5–8].

This trend mirrors the results of Govind (2015), who reported mean HU values of 27 HU for acute, 19 HU for subacute, and 9 HU for chronic infarction, confirming a consistent pattern of attenuation reduction with infarct maturation [5]. In line with Mair *et al.* (2021) and Alzahrani *et al.* (2023), the current findings further validate that changes in tissue attenuation can serve as an indirect marker of infarct timing and viability [6,8]. Similarly, Ali *et al*. (2025) demonstrated significant inter-phase differences using HU quantification, supporting its diagnostic validity for stroke staging [9].

The physiological explanation for this progressive attenuation loss lies in the evolution of tissue water dynamics [5]. During the acute phase, cytotoxic oedema predominates due to ionic pump failure, resulting in intracellular water influx without major extracellular accumulation, hence relatively preserved HU values [5,8]. As the infarct progresses to the subacute stage, disruption of the blood–brain barrier allows extracellular fluid entry, increasing total water content and decreasing attenuation. In the chronic phase, tissue necrosis, gliosis, and cystic changes lead to further HU decline [7,8]. This pattern is supported by quantitative water uptake models, where HU values inversely correlate with infarct age and edema severity [4,11].

This study identified a progressive reduction in HU values across acute, subacute, and chronic infarction stages, consistent with established temporal changes in ischemic brain tissue [11]. Early in the acute phase, cytotoxic oedema produces intracellular water accumulation, resulting in modest attenuation reduction on NCCT [12]. As ischemia progresses, blood-brain barrier disruption and vasogenic oedema further decrease HU values in the subacute phase [13]. Chronic infarction then demonstrates marked attenuation loss due to gliosis and encephalomalacia, yielding HU values approaching cerebrospinal fluid levels [14]. These findings align with previous studies confirming a strong relationship between HU decline and infarct maturity, supporting HU measurement as a surrogate biomarker for tissue evolution in ischemic stroke [15]. Quantitative densitometry, including net water uptake (NWU) derived from HU differences, has also been shown to hold prognostic value by predicting malignant oedema and long-term outcomes [16]. Recent evidence further supports this concept, as HU-based NWU measured in subcortical regions has been associated with functional outcome in acute ischemic stroke patients [17]. Despite these advantages, HU values may vary based on scanner calibration and reconstruction settings, emphasizing the need for standardized protocols and multicentre validation [18]. Present findings showed, semi-automated HU-based analysis has been shown to enhance early infarct detection on NCCT through measurement of relative density differences and automated hypodensity mapping, reducing reliance on subjective reader interpretation [19]. Such methods further support the clinical value of HU quantification and provide a foundation for future integration of automated decision-support tools in acute stroke imaging workflows. Nonetheless, HU analysis remains a practical, accessible adjunct to conventional NCCT interpretation, especially in resource-limited environments where rapid, objective stroke assessment is needed [20].

Clinically, this study found that decreased consciousness was most common in the acute phase, while hemiparesis predominated in subacute and chronic stages, and aphasia occurred mainly in chronic infarction. These findings are consistent with earlier research indicating that acute global ischemia often manifests with altered consciousness, while chronic infarcts result in persistent motor and cortical deficits.

Despite these promising results, several limitations should be acknowledged. The sample size was relatively small, and measurements were manually performed, which may introduce bias in observing. Furthermore, the cross-sectional design precluded longitudinal observation of HU evolution within the same individual. Future multicentric studies employing larger samples, automated region-of-interest segmentation, and standardized calibration protocols are warranted to confirm these findings and establish reproducible HU reference thresholds.

CONCLUSION

Quantitative assessment of brain parenchymal attenuation using Hounsfield Unit (HU) values on non-

contrast CT provides a reliable and objective method for distinguishing ischemic stroke phases. The consistent decline in HU from acute to chronic stages reflects the underlying pathophysiological changes in tissue water content and cellular integrity. HU measurement can serve as a practical, cost-effective biomarker for infarct grading and may enhance diagnostic confidence, particularly in settings where advanced imaging modalities are unavailable.

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CONFLICT OF INTEREST

The authors declare there is no conflict of interest.

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