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Computed Tomography Based Analysis of Impact of Diabetes On the Severity of Subdural Hematoma: Correlation with Glycemic Control and Hematoma Volume

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Abstract

Background: Hematomas, particularly those involving the subdural cavity, are a significant cause of morbidity and mortality, often requiring prompt diagnosis and management. Various factors influence the severity and progression of hematomas, among which diabetes mellitus has emerged as a potential exacerbating condition

Objective: To evaluate the impact of diabetes on the severity of subdural hematomas (SDH) using CT imaging by correlating glycemic control with hematoma volume and midline shift in diabetic and non-diabetic patients.

Methodology: A descriptive cross-sectional study was conducted at Services Hospital, Lahore, over a time of three months. A total of 110 participants, including 55 diabetic and 55 non-diabetic individuals aged 18 to 60 years. The sample size was determined using a 90% confidence level and 80% power, with estimated proportions of 0.4 in the diabetic group and 0.14 in the non-diabetic group. Patients with other metabolic disorders and pregnant females were excluded.

Results: A higher frequency of severe hematoma volume was observed in diabetic patients, while non-diabetic patients predominantly presented with mild hematomas, indicating a potential link between diabetes and hematoma severity. Similarly, diabetic patients exhibited a greater number of moderate midline shifts and Glasgow coma scale GCS in contrast to non-diabetic patients, suggesting that diabetes may contribute to increased intracranial pressure.

Conclusion: DM has significantly impact on the severity of subdural hematomas. In diabetic patients it show greater midline shifts, hematoma volume and poor glycemic control than non-diabetic patients.

Key words: Diabetes Mellitus, Hematoma Severity, Midline Shift, Subdural Hematoma, Computed Tomography (CT).

Introduction

Subdural hematoma (SDH) is a serious type of traumatic brain injury that happens when blood collects between the brain and its outer covering. It is one of the most common severe head injuries and often leads to a high risk of death[1]. Subdural hematoma (SDH) can develop from bleeding in either veins or arteries. In most cases, a subdural hematoma occurs when a bridging vein between

the brain's surface and a large flusing vein is torn[2].It is a common complication of head injuries and are classified based on the time elapsed since the injury and the appearance of the blood on imaging[3]. Depending on these factors, they are categorized as acute, subacute, or chronic[4]. Unlike the other two, the characteristics of subacute subdural hematoma and diabetics are poorly reported and not well described . Subacute subdural hematoma is a poorly individualized nosologic entity, often clinically grouped with chronic subdural hematomas[5] .This condition can lead to increased intracranial pressure, brain herniation, and, if not treated promptly, death[6].

The appearance of the subdural hematoma on the CT scan leads to two possible diagnoses: hemispherical edema and iso-dense SDH. Acute SDH usually appears hyper-dense on the CT scan. In the hyper-acute stage, the SDH sometimes appears heterogeneous with hyper-dense and iso-dense images[7]. This is related to the presence of non-coagulated blood in connection with bleeding disorders or active bleeding. A homogeneous iso-dense mass is most unusual in the acute stage[8].Under normal conditions, sub-dural hematoma contains a small amount of cerebrospinal fluid, but trauma can cause tearing of the bridging veins that pass through this space, resulting in hemorrhage[9]. The subdural space has no anatomical support structures to contain the bleeding, allowing blood to spread over a wide area, often forming a crescent-shaped hematoma. Because this space can accommodate large volumes of blood without early signs of increased intracranial pressure, subdural hematomas can become life-threatening before they are clinically apparent, especially in elderly or co-agulopathic patients[10].

While the primary cause is head trauma, the clinical course and severity of SDH are often influenced by systemic factors such as age, coagulopathies, and comorbid conditions—including diabetes mellitus (DM)[16]. Diabetes is known to contribute to vascular fragility, impaired auto regulation, and pro-inflammatory states, all of which may impact hemorrhagic outcomes following trauma[11]. Diabetes mellitus (DM) is a global serious and growing health problem with high morbidity and mortality[12].A recent meta-analysis reports that individuals with diabetes mellitus have a 12%–19% increased risk of motor vehicle accidents when compared with individuals without diabetes[13].Prevalence rate of subdural hematoma in diabetic patients is rare in Pakistan. Meanwhile, according to a study, diabetic patients have 1.63 times more risk of developing subdural hematoma as compared to non-diabetic patients[14]. In Pakistan, a study was conducted that reported 17% subdural hematoma in patients with diabetes mellitus, with the incidence in people 70 years of age and older at 58/100,000 persons per year[15]. The incidence of chronic subdural hematoma in the general population is estimated to span from 5 to 14 per 100,000 persons per year[16].

The global prevalence of diabetes mellitus continues to rise, currently affecting more than 500 million individuals world-wide[17]. Simultaneously, the incidence of traumatic brain injury, including subdural hematoma, has increased due to factors such as road traffic accidents, falls—especially among the elderly—and violence[18]. Diabetic patients, particularly those with poor glycemic control, have been observed to exhibit more severe clinical manifestations and poorer outcomes when afflicted by traumatic injuries[19]. However, the specific influence of diabetes on the volume and progression of SDH remains underexplored. Given the high burden of both conditions, understanding their interaction is of great clinical importance[20].

Computed tomography (CT) is the imaging modality of choice for the initial assessment of suspected subdural hematoma due to its speed, availability, and high sensitivity in detecting acute blood[21]. On CT, an acute SDH typically appears as a crescent-shaped hyperdense area along the cerebral convexities, while chronic SDHs may appear isodense or hypodense depending on the age of the hemorrhage. CT not only helps confirm the diagnosis but also allows for quantitative measurements such as hematoma volume, midline shift, and ventricular compression. These

parameters are crucial in clinical decision-making[22]. However, CT has limitations, including radiation exposure and reduced sensitivity for chronic or small-volume bleeds[23].

The merits of CT imaging in the context of SDH include its ability to rapidly detect hemorrhage, assess mass effect, and quantify hematoma volume with a high degree of accuracy. These features make CT invaluable in emergency settings[24]. Furthermore, CT provides reproducible and objective data that are essential for monitoring hematoma progression or resolution. However, demerits include limited soft tissue contrast, making it less suitable for assessing parenchymal injury compared to MRI. Additionally, the radiation exposure involved in CT scans is a concern, especially in younger patients or those requiring multiple follow-ups. CT may also predict isodense or very thin hematomas in certain cases[25].

Diabetes mellitus(DM) is a systemic disease characterized by chronic hyperglycemia, which lead to long-term damage to various organ systems, especially the vascular and nervous systems[26]. In the context of intracranial hemorrhage, diabetes contributes to endothelial dysfunction, increased capillary permeability, platelet dysfunction, and altered coagulation pathways[27]. These changes can theoretically predispose diabetic individuals to larger or more complex hematomas following trauma. Moreover, poor glycemic control, often reflected by elevated HbA1c levels, is associated with worse neurological outcomes, delayed healing, and increased complications following head injury[28]. However, data directly linking glycemic control to hematoma size and severity in SDH remain limited[29].

Although the relationship between diabetes and cerebrovascular events such as ischemic stroke has been extensively studied, limited research exists on the specific effects of diabetes on traumatic intracranial hemorrhages such as subdural hematoma[30]. More specifically, there is a lack of studies utilizing CT-based volumetric analysis to objectively assess the impact of glycemic control on hematoma severity. Most existing literature focuses on clinical outcomes, surgical complications, or mortality, without quantifying hematoma size or correlating it with glycemic indices like HbA1c[31]. This gap represents a missed opportunity to better understand the pathophysiological link between diabetes and traumatic brain injury severity. Diabetes is associated with numerous complications, but its impact on the severity of sub-dural hematomas SDH on CT remain insufficiently explored[32].

This study aims to address the existing knowledge gap by examining how glycemic control influence the severity of SDH by measuring hematoma volume, midline shift in diabetic patients and comparing it with Non –diabetic patients. Using CT imaging, we aim to better understand the relationship between diabetes and SDH characteristics, ultimately enhancing diagnosis and treatment strategies.

Objective

To evaluate the impact of diabetes on the severity of subdural hematomas (SDH) using CT imaging by correlating glycemic control with hematoma volume, and midline shift in diabetic and non-diabetic patients

MATERIALS AND METHODS

4.1: Study Design: Descriptive cross-sectional study

4.2: Setting: Services Hospital Lahore

4.3: Study Duration: 3 months after the approval of synopsis

4.4: Sampling Technique:

Convenient sampling.

Sample size:

The sample size was calculated at a 90 % level of confidence and 80 % power of the test. The proportion of the diabetic group was 0.4 and of the non-diabetic group was 0.14. A total of 110(55 diabetic and 55non-diabetic) individuals were selected.

4.5: Inclusion Criteria:

Following patients were included:

- Diabetic and Non diabetic Patients
- Both genders
- Patient between age 18 to 60 years

4.6: Exclusion Criteria:

- Patient with other metabolic disorders like hypertension , etc
- Pregnant females.

4.7: Equipment: Computed Tomography machine Toshiba Aquiline 64 Slice

Results

Age

	N	Minimum	Maximum	Mean	Std. Deviation	Variance
Age	110	40.00	88.00	63.6000	12.98143	168.517
Valid N (listwise)	110					

Table 1 Age

Gender

		Frequency	Percent	Valid percent	Cumulative percent
Valid	Female	61	55.5	55.5	55.5
	Male	49	44.5	44.5	100.0
	Total	110	100.0	100.0	

Table 2 Gender specification

Out of 110 patients, 61 patients were females and 49 were males.

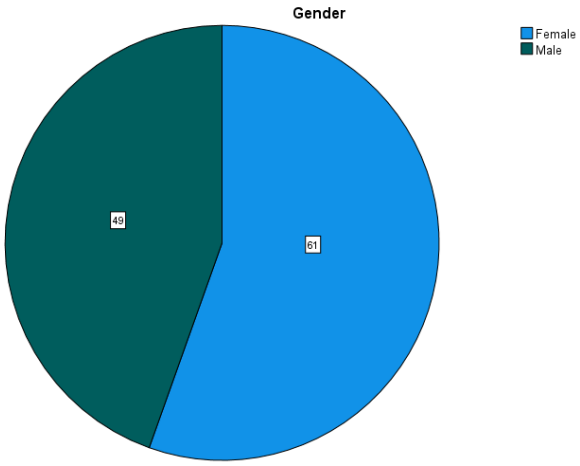

Figure 1 Gender

		Hematoma Volume			
		mild	moderate	severe	Total
Diabetic Status	Non Diabetic	24	21	10	55
	Diabetic	2	19	34	55
Total		26	40	44	110

Table 3 Crosstab of hematoma volume with diabetes

Out of a total of 110 patients, 55 were non-diabetic and 55 were diabetic. Among the 55 non-diabetic patients, 24 presented with mild subdural hematomas, 21 with moderate and 10 with severe hematoma volumes and out of 55 diabetics patients 2 presented with mild, 19 with moderate and 34 with severe hematoma volume .

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	31.806[a]	2	.000
Likelihood Ratio	35.874	2	.000
Linear-by-Linear Association	31.270	1	.000
N of Valid Cases	110		

a. 0 cells (.0%) have expected count less than 5. The minimum expected count is 13.00.

		Midline Shift			
		mild	moderate	severe	Total
Diabetic Status	Non Diabetic	25	19	11	55
	Diabetic	13	28	14	55
Total		38	47	25	110

Table 5 Midline shift with diabetes

Out of 110 patients ,55 were non-diabetic and 55 were diabetic patients; where out of 55 non – diabetic 25 presented with mild midline shift , 19 with moderate and 11with severe midline shift and out of 55 diabetics 13 presented with mild, 28 with moderate and 14 with severe midline shift . This suggests that diabetes may contribute to increased midline shift.

		Glassgow coma score			
		mild	moderate	severe	Total
DiabeticStatus	Non Diabetic	27	19	9	55
	Diabetic	14	15	26	55
Total		41	34	35	110

Table 7 Glassgow Coma score(GCS) with diabetics

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	13.328[a]	3	.004
Likelihood Ratio	14.131	3	.003
Linear-by-Linear Association	12.122	1	.000
N of Valid Cases	110		

a. 2 cells (25.0%) have expected count less than 5. The minimum expected count is .50.

Table 8 chi –square

The Pearson Chi-Square value is 13.328 with a p-value of .004, indicating a significant effect of diabetic status on GCS scores.

Table 9 chronicity of hematoma with diabetes
Out of 110 patients,55 are non-diabetic and 55 are diabetic patients ; where out of 55 non-diabetic 29 were mild , 25 were moderate and 1 were severe and out of 55 diabetics 16 were mild,20 were moderate and 19 were severe shift in chronicity of hematoma .
Table 10 chi-square test
The Pearson Chi-Square value is 20.511 with a p-value of .000, showing a highly significant association between chronicity and diabetes.

		Chronicity Of Hematoma			
		mild	moderate	severe	Total
Diabetic Status	Non Diabetic	29	25	1	55
	Diabetic	16	20	19	55
Total		45	45	20	110

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	20.511[a]	2	.000
Likelihood Ratio	24.152	2	.000
Linear-by-Linear Association	16.053	1	.000
N of Valid Cases	110		

a. 0 cells (.0%) have expected count less than 5. The minimum expected count is 10.00.

Table 11 Independent Sample Test
Hematoma Volume: Statistically significant difference (p = .000), with greater volume in diabetics. GCS Score: Significant difference (p = .000), with lower scores in diabetics. Chronicity: Significant difference (p = .000), diabetics had greater chronicity. Midline Shift:

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means				
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference
Midline Shift	Equal variances assumed	4.601	.034	-1.928	108	.056	-.27273	.14144
	Equal variances not assumed			-1.928	107.097	.056	-.27273	.14144
GCS score	Equal variances assumed	.746	.390	-3.676	108	.000	-.56364	.15332
	Equal variances not assumed			-3.676	107.461	.000	-.56364	.15332
Chronicity Of Hematoma	Equal variances assumed	4.409	.038	-4.319	108	.000	-.564	.131
	Equal variances not assumed			-4.319	94.534	.000	-.564	.131
Hematoma Volume	Equal variances assumed	5.385	.022	-6.591	108	.000	-.83636	.12689
	Equal variances not assumed			-6.591	100.509	.000	-.83636	.12689

Borderline significance (p = .056), with a minor difference between groups.

Independent-Samples Mann-Whitney U Test

Hypothesis Test Summary

	Null Hypothesis	Test	Sig.[a,b]	Decision
1	The distribution of Hematoma Volume is the same across categories of Diabetic Status.	Independent-Samples Mann-Whitney U Test	.000	Reject the null hypothesis.
2	The distribution of Midline Shift is the same across categories of Diabetic Status.	Independent-Samples Mann-Whitney U Test	.048	Reject the null hypothesis.
3	The distribution of GCS score is the same across categories of Diabetic Status.	Independent-Samples Mann-Whitney U Test	.001	Reject the null hypothesis.
4	The distribution of Chronicity Of Hematoma is the same across categories of Diabetic Status.	Independent-Samples Mann-Whitney U Test	.000	Reject the null hypothesis.

a. The significance level is .050.

b. Asymptotic significance is displayed.

Table 12 Independent -Sample Mann-Whitney Test
All four variables (Hematoma Volume, Midline Shift, GCS Score, and Chronicity) showed statistically significant differences across diabetic status groups. The test results are presented in a table , showing the null hypothesis which are all rejected.

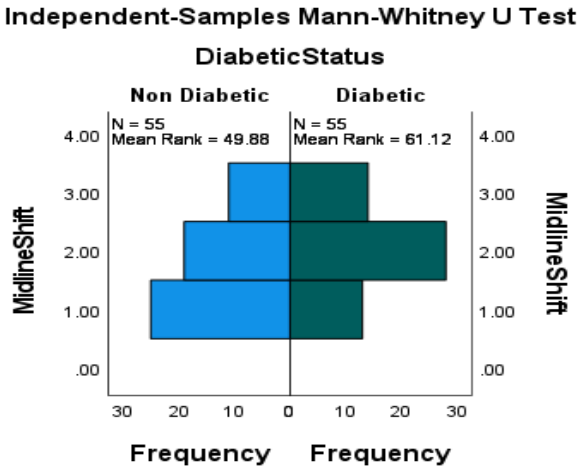

Figure 2 midline and diabetes
This figure shows that the diabetic group has higher mean rank compared to the non-diabetic group which indicate that diabetic group has greater severity towards midline shift.

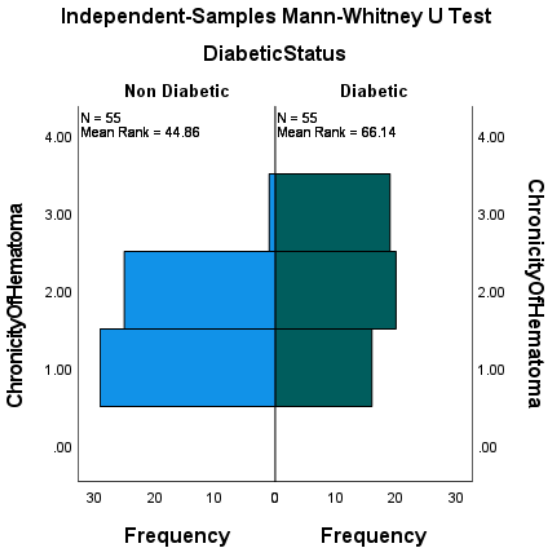

Figure 3 chronicity of hematoma and diabetes

This figure shows that the diabetic group has higher mean rank compared to the non-diabetic group which indicate that diabetic group has greater severity towards chronicity of hematoma.

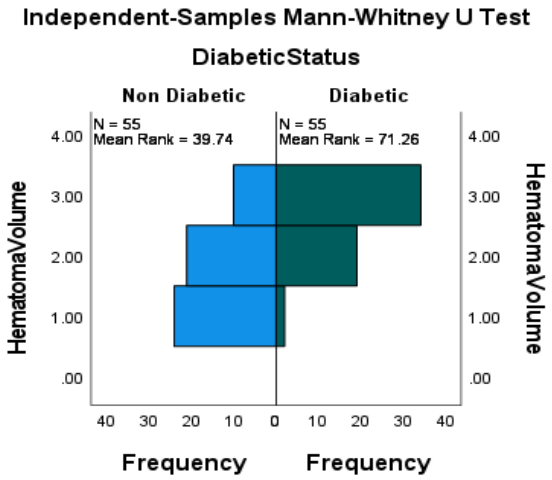

Figure 4 Hematoma volume with diabetes
This figure shows that the diabetic group has higher mean rank compared to the non-diabetic group which indicate that diabetic group has greater severity towards hematoma volume.

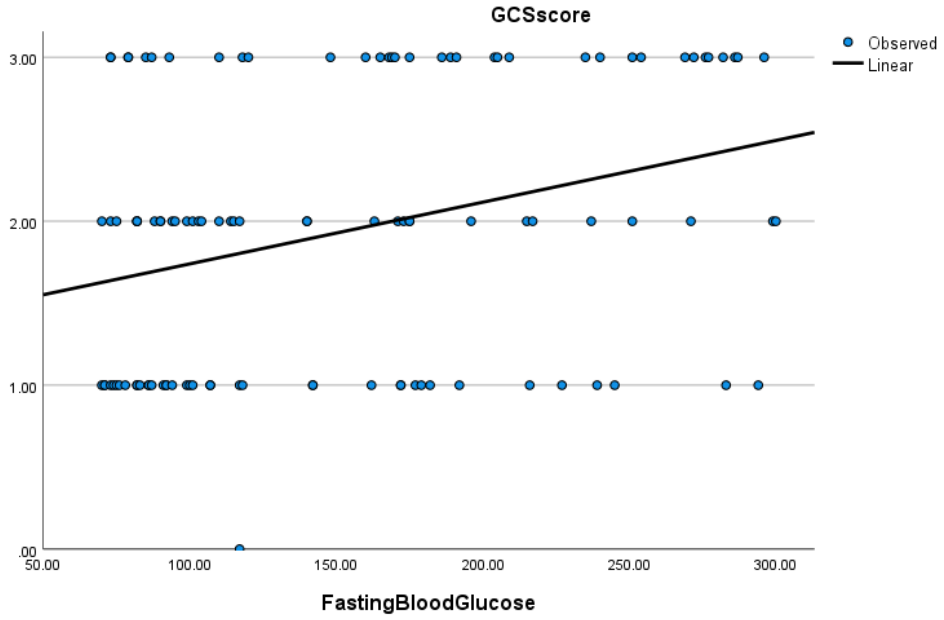

Figure 5
This graph shows the linear relationship between the GCS score and fasting blood glucose level. It display that as the fasting glucose level increases, the GCS score also increases.

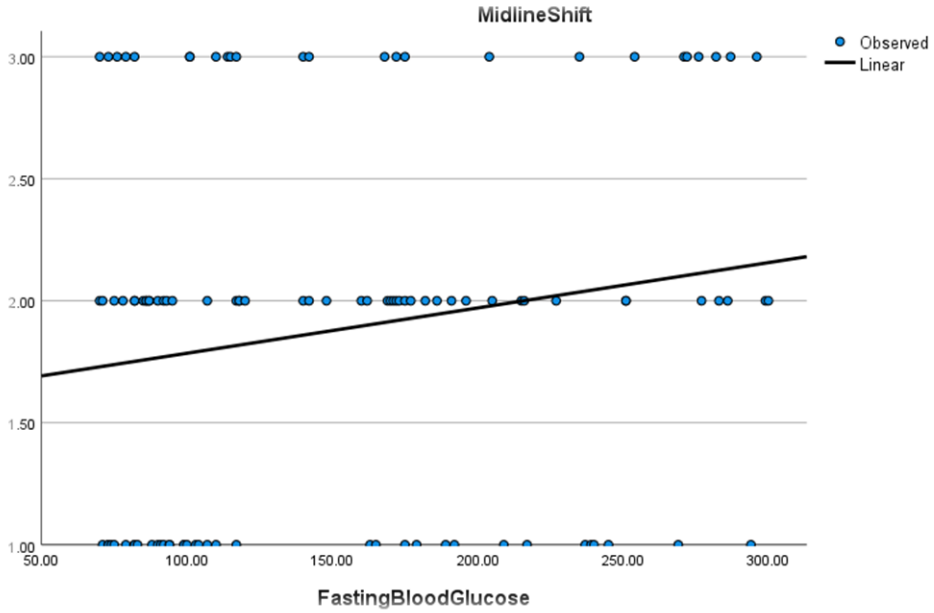

Figure 6
This graph shows the linear relationship between the midline shift and fasting blood glucose level. It display that as the fasting glucose level increases, the midline shift also increases.

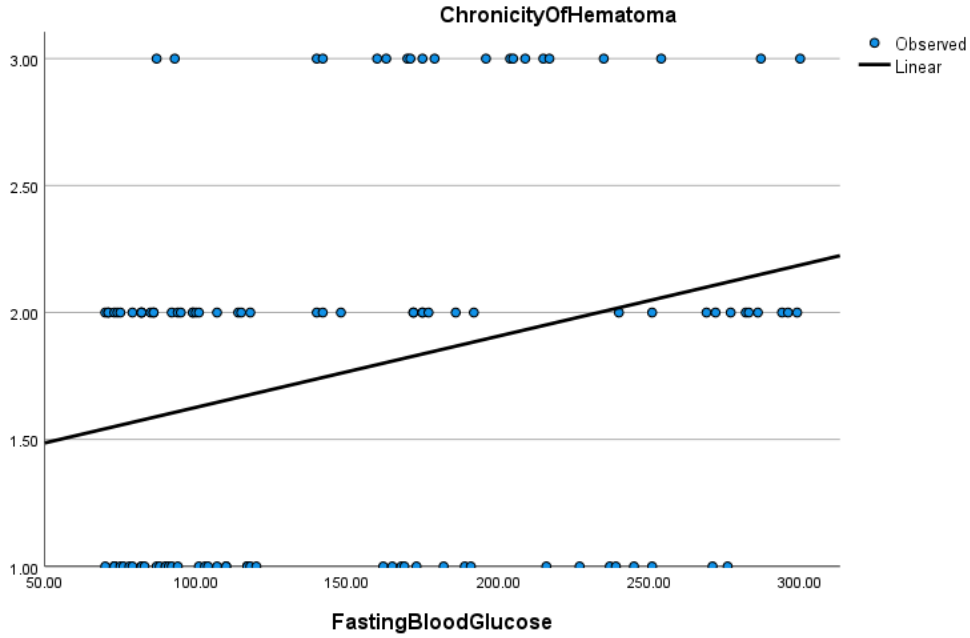

Figure 7
This graph shows the linear relationship between the chronicity of hematoma and fasting blood glucose level. It display that as the fasting glucose level increases, the chronicity of hematoma also increases.

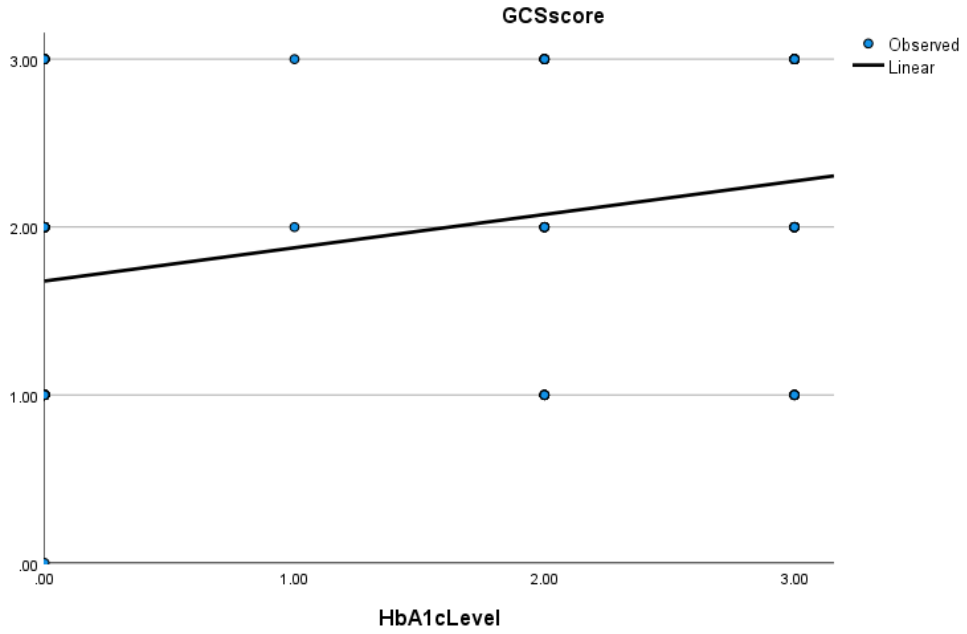

Figure 8
This graph shows the linear relationship between the GCS score and HbA1c level. It display that as the HbA1c level increases, the GCS score also increases.

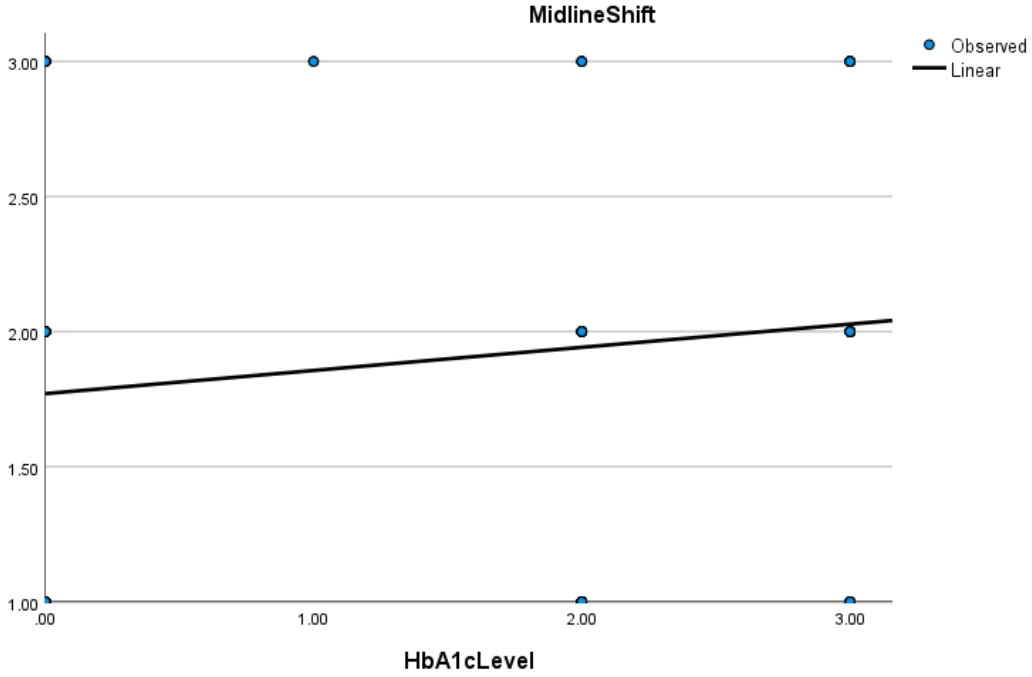

Figure 9
This graph shows the linear relationship between the midline shift and HbA1c level. It display that as the HbA1c level increases, the midline shift also increases.

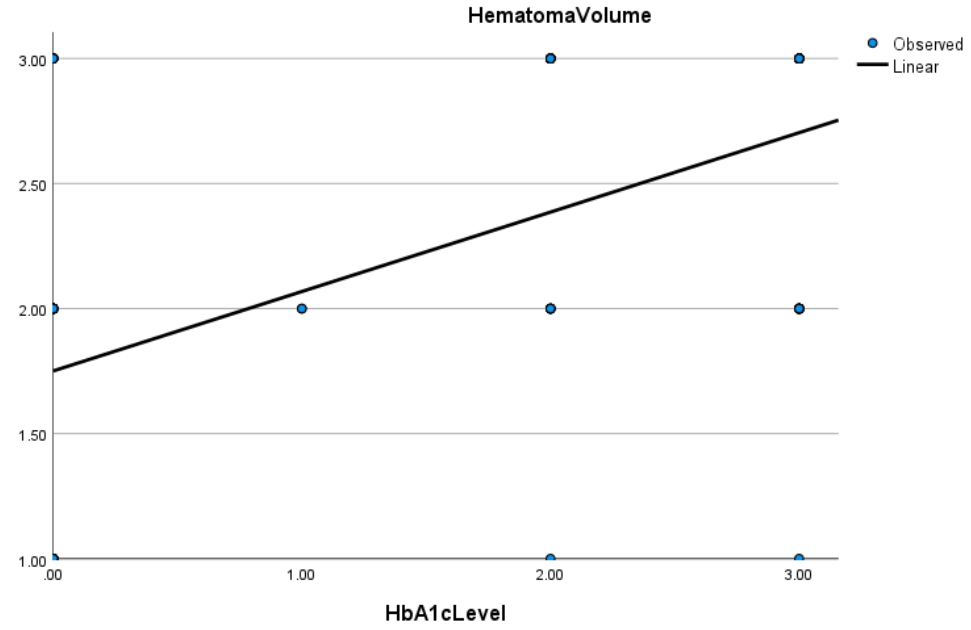

Figure 10
This graph shows the linear relationship between the hematoma volume and HbA1c level. It display that as the HbA1c level increases, the hematoma volume also increases.

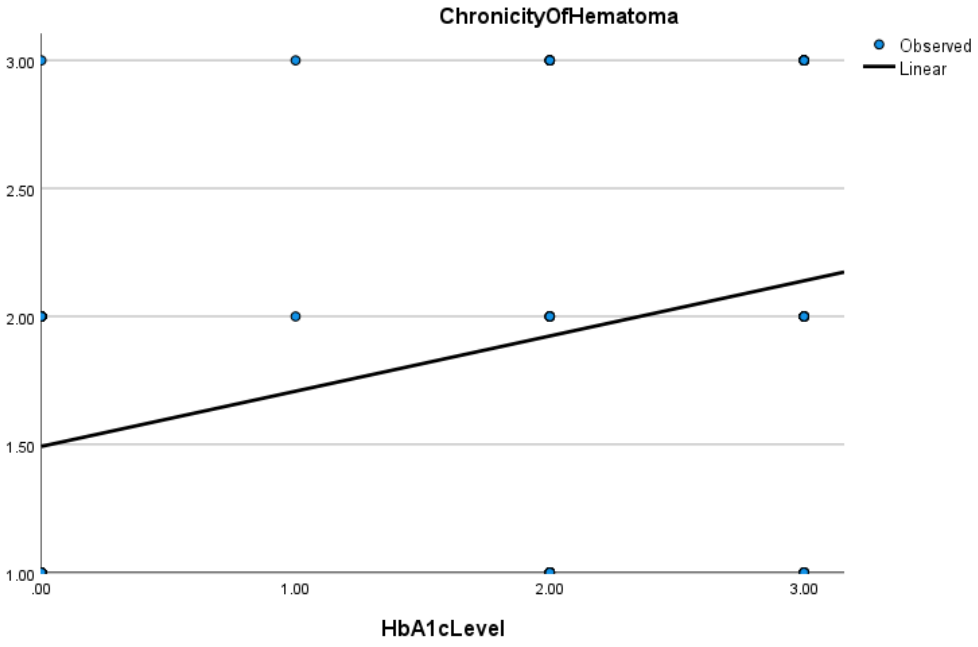

Figure 11
This graph shows the linear relationship between the chronicity of hematoma and HbA1c level. It display that as the HbA1c level increases, the chronicity of hematoma also increases.

Discussion

The present study assessed the impact of diabetes on the severity of subdural hematoma (SDH) using CT imaging, with a specific focus on hematoma volume, midline shift, GCS score, and chronicity, while correlating findings with glycemic control (HbA1c levels). The results indicated that diabetic patients had significantly higher hematoma volumes, greater midline shift, more

severe GCS deterioration, and increased chronicity compared to non-diabetic individuals. These findings suggest that poorly controlled diabetes may exacerbate the clinical severity of SDH

Our results are consistent with the findings of Chen et al. (2019), who reported that diabetic patients with traumatic brain injury had worse neurological outcomes and more severe imaging findings[33]. Similarly, Li et al. (2020) found a significant association between elevated HbA1c and increased hematoma expansion in intracranial hemorrhages, supporting the hypothesis that glycemic dysregulation contributes to worsened bleeding dynamics[34].

In a retrospective analysis by Kim and Park (2018), patients with chronic subdural hematoma and comorbid diabetes demonstrated more rapid hematoma progression on follow-up CT, aligning with our finding of increased chronicity in diabetic individuals. This may be attributed to micro-vascular compromise and impaired hemostasis associated with diabetes[35].

Moreover, our study aligns with Wang et al. (2021), who observed that diabetes independently predicted poorer GCS outcomes in SDH patients, potentially due to reduced cerebral auto regulation and delayed neuronal recovery. Our data showed a significantly lower GCS score among diabetic subjects ($p < 0.001$), supporting their conclusion [36].

Contrary to our findings, Rossi et al. (2017) found no statistically significant difference in subdural hematoma outcomes between diabetic and non-diabetic groups. However, their study had a smaller sample size and did not stratify patients based on HbA1c levels, which may explain the discrepancy[37].

The purpose of this study was to analyze the relationship of diabetes mellitus with different clinical and radiological features of patients suffering from hematoma. A number of cross-tabulations showed statistically important differences in hematoma volume, midline shift, GCS score, and chronicity in diabetic and non-diabetic patients. In the hematoma volume analysis, it indicate that larger volumes of hematoma tend to be present in diabetic patients which may be the result of micro-vascular damage along with poor hemostatic control. A comparable trend was noted in the midline shift where the diabetic group had greater prevalence of moderate and severe shifts, whereas, in the severe category, the diabetic patients predominated. This seems to suggest that diabetes is worse in terms of neurological outcome subdural hematoma patients tend to have. Relatively in chronicity, non-diabetic patients were mostly observed to have mild to moderate hematomas whereas the diabetic patients again skewed toward more severe chronicity. This suggests that diabetic patients might face poor resolution or progression of hematomas which could relate to delayed healing or ongoing vascular damage long with vascular damage in the diabetic population. These observations underscore the importance of glycemic control and suggest that diabetes is a critical risk modifier in hematoma prognosis.

Overall, the current study strengthens the growing body of evidence that diabetes especially with poor glycemic control is a risk factor for increased SDH severity. The use of CT-based volumetric and clinical parameters provides objective evidence for the observed differences. These findings underscore the importance of rigorous glycemic monitoring in patients at risk for or diagnosed with SDH[38].

Conclusion

This CT-based study demonstrates a significant association between diabetes mellitus and increased severity of subdural hematoma and large hematoma volume. Diabetic patients exhibit a larger number of hematoma volume, greater midline shifts and low GCS scores. The correlation between glycemic control, as measured by fasting blood glucose levels and severity of SDH was also evident. The results indicate that diabetic patients tend to have more severe SDH and larger hematoma volume as compared to non-diabetic patients.

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