ORIGINAL RESEARCH

Predictive Value of Subdural Effusion Thickness for the Transformation of Post-Traumatic Subdural Effusion into Chronic Subdural Hematoma

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ABSTRACT

Objective • The primary aim of this research is to investigate the predictive value of subdural effusion thickness in determining the progression of post-traumatic subdural effusion to chronic subdural hematoma. Studying this progression is crucial as it helps in early diagnosis and effective management of chronic subdural hematoma, which is a serious and life-threatening condition. This research is valuable and relevant for improving patient outcomes and reducing the associated risks and complications.

Methods • We conducted a retrospective examination of the clinical data obtained from 124 patients who were treated for post-traumatic subdural effusion at our neurosurgery department between March 2017 and March 2021. The data collection process involved reviewing the patients' medical records, radiographic images, and follow-up visits. We used strict criteria for patient selection, including a confirmed diagnosis of post-traumatic subdural effusion, availability of follow-up data, and no prior history of chronic subdural hematoma. Patients who experienced a progression of subdural effusion to chronic subdural hematoma were assigned to the hematoma group (26 cases). In comparison, those who did not show such progression were categorized into the effusion group (98 cases). We endeavored to identify potential risk factors contributing to the progression from subdural effusion to chronic subdural hematoma. The predictive strengths of these risk factors were evaluated using receiver operating characteristic (ROC) curves.

Results • There were no statistically significant disparities between the two groups in terms of gender, hypertension, COPD, and GCS scores ($P > .05$). However, significant differences were noted in the variables of age, tSAH, the location of subdural effusion, and subdural effusion thickness ($P < .05$). Multivariate logistic regression analysis disclosed age (1.213), tSAH (12.542), and subdural effusion thickness (1.786) as independent risk factors for the conversion of TSE to CSDH ($P < .05$). The ROC curve showed the AUC values of age, tSAH, and subdural effusion thickness for predicting CSDH to be 0.739, 0.670, and 0.820, respectively, with a combined AUC value of 0.942, thereby outperforming the individual tests.

Conclusion • In patients suffering from post-traumatic subdural effusion, the thickness of the subdural effusion emerges as a strong predictor for its progression into a chronic subdural hematoma. Clinicians should be particularly cautious when the effusion thickness exceeds 10.7 mm, as the likelihood of transformation increases significantly. These findings have important implications for clinical practice and patient management, highlighting the need for prompt and effective treatment to prevent chronic complications. (*Altern Ther Health Med.* [E-pub ahead of print.])

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INTRODUCTION

Traumatic Subdural Effusion (TSE) is a common consequence of traumatic brain injury. It occurs when brain tissue is displaced within the skull following a traumatic event, resulting in a breach in the arachnoid membrane. This breach allows for the leakage of blood and the flow of cerebrospinal fluid into the subdural cavity, driven by intracranial pressure gradients. However, due to the operation of the living valve in the arachnoid, the accumulated cerebrospinal fluid cannot return to the subarachnoid space. As a result, it accumulates in the subdural cavity.[1]

MAYO first described TSE in 1894,[2] and it was originally thought to be a relatively rare condition. However, recent studies have suggested that a certain subset of patients subsequently evolve into Chronic Subdural Hematoma (CSDH). Clinical observations also suggest that numerous

patients with Traumatic Brain Injury (TBI) simultaneously develop TSE, which later progresses into CSDH.[2,3] As TBIs are often mild and require brief hospitalization periods, TSE may often be overlooked by healthcare providers and patients alike, which can even result in cases being lost to follow-up.[4,5] Chronic Subdural Hematoma (CSDH) is a severe health event that can have significant consequences for patients. Potential symptoms include headaches, confusion, seizures, plications can arise from the pressure of the hematoma on the brain, leading to neurological deficits and potentially life-threatening conditions. The impact on patients' lives can be substantial, with long-term disability and even death in severe cases. Studying the progression of CSDH is important to better understand its development, improve early detection, and develop effective treatments to prevent complications and improve patient outcomes.

Recognized risk factors for the progression of TSE to CSDH include age, bilateral TSE, and CT values of subdural effusion. However, the possibility of other contributing factors in the progression from TSE to CSDH in TBI patients remains uncertain.[6-9] CSDH is considered a severe health event, particularly in the elderly population. International data suggests an alarming 5-year mortality rate of up to 34.8%, which questions its label as a benign condition.[10]

Our study aimed to address the knowledge deficit regarding the progression from traumatic subdural effusion (TSE) to chronic subdural hematoma (CSDH) following traumatic brain injury (TBI). The existing literature lacks a comprehensive understanding of the risk factors involved in this progression and their predictive power for such a transition. This research gap drove our investigation, as we sought to identify specific risk factors implicated in the development of CSDH among TSE patients. By filling this knowledge gap, we aimed to provide valuable insights for the establishment of clinical prevention and treatment strategies targeting this critical transition. In this investigation, we evaluated the clinical data of 124 patients suffering from TSE following TBI, 26 of whom developed CSDH. We aimed to identify risk factors implicated in the progression from TSE to CSDH and to determine their predictive power for such a transition. The ultimate goal was to provide a reference point for establishing clinical prevention and treatment strategies. In summary, our study contributes to the field of neurosurgery by providing valuable insights into the risk factors and predictive power associated with the progression from TSE to CSDH. The clinical implications of our findings include the development of targeted prevention and treatment strategies, risk stratification, and resource allocation optimization.

PATIENTS AND METHODS

Research Participants

We performed a retrospective analysis of clinical records from 124 TBI patients with concurrent TSE who were treated in our neurosurgery department between March 2017 and March 2021. The patient cohort comprised 85 males (68.55%) and 39 females (31.45%) ranging in age from 42 to 82 years, with an average age of 66.36±7.14 years. Hypertension was identified in 21 cases (16.94%), and Chronic Obstructive Pulmonary Disease (COPD) was noted in 13 cases (10.48%). The average Glasgow Coma Scale (GCS) score was 14.14±1.65, and traumatic subarachnoid hemorrhage (tSAH) co-occurred in 22 cases (17.74%). The mean thickness of the subdural effusion was 9.43±2.52 mm, with subdural effusion encompassing ≤2 cerebral lobes in 67 cases (54.03%) and ≥3 cerebral lobes in 57 cases (45.97%).

Inclusion Criteria: (1)Patients who sought treatment within 24 hours of sustaining a head injury and underwent cranial CT scanning, (2)Patients who demonstrated subdural effusion on cranial CT or MRI within 10 days post-injury, with an increase in subdural effusion thickness of >3 mm compared to the first 24 hours post-injury, (3)Patients without any further head trauma post-TSE diagnosis, (4) Patients with at least a 3-month follow-up after TSE diagnosis, (5)Patients who were 18 years of age or older.

Exclusion Criteria: (1)Patients whose initial cranial CT scan post-injury showed subdural effusion but who displayed no subsequent increase in subdural effusion thickness after a minimum 10-day follow-up, indicating a lack of significant correlation between the effusion and trauma, (2)Patients with an effusion thickness >1.0 cm and significant mass effect requiring immediate surgical intervention, (3)Patients with a history of long-term oral antiplatelet or anticoagulation medication usage, (4)Patients with evident coagulation disorders, (5)Patients with concurrent trauma to vital organs such as the heart, liver, or kidneys, (6)Those with a medical history of hepatic or renal failure or malignant tumors.

Prognostic Criteria

(1) Diagnostic criteria for TSE[4]: TSE is recognized within 10 days post-traumatic brain injury; the CT scan displays a homogenous subdural low-density region similar in density to cerebrospinal fluid, exceeding 3 mm in thickness and compressing the adjacent brain tissue; the CT Hounsfield unit value of the effusion is less than 20 Hu; and there is no significant enhancement of the encapsulating layer.

(2) Criteria for predicting the progression of TSE into CSDH[4]: Clear evidence of post-traumatic subdural effusion is noted; subdural hematoma appears three weeks post-traumatic brain injury; serial CT or MRI scans demonstrate the transformation of subdural effusion into chronic subdural hematoma; cranial CT presents as low or slightly low-density areas, while MRI exhibits hyperintense changes on both T1- and T2-weighted images, with an enhanced encapsulating layer after contrast administration.

Observational Parameters

Relevant clinical data was collected from patients, including variables such as age, gender, hypertension, COPD, co-existing tSAH, GCS score upon admission, and the thickness and size of the subdural effusion. The thickness of the subdural effusion for each patient was independently

Table 1. Univariate Analysis of Clinical Characteristics and Potential Risk Factors in Two Patient Groups

Items	Case count	Hematoma Group (n = 26)	Effusion Group (n = 98)	t/χ^2 value	*P* value
Age (years, $\bar{x} \pm s$)		71.65±7.14	63.27±7.94	4.881	.000
sex [case (%)]					
Male	85	21	64	2.279	.1311
Female	39	5	34		
Hypertension [case (%)]					
Yes	21	6	15	0.882	.348
No	103	20	83		
COPD [case (%)]					
Yes	13	3	10	0.039	.844
No	111	23	88		
tSAH [case (%)]					
Yes	22	9	13	6.418	.011
No	102	17	85		
Location of Subdural Effusion [case (%)]					
≤2 lobes	67	6	61	12.691	.000
≥3 lobes	57	20	37		
GCS Score (score, $\bar{x} \pm s$)		13.75±1.55	14.43±1.80	1.760	.081
Thickness of Subdural Effusion (mm, $\bar{x} \pm s$)		12.14±2.68	9.15±2.47	5.390	.000

Abbreviations: COPD, Chronic Obstructive Pulmonary Disease; tSAH, Traumatic Subarachnoid Hemorrhage.

measured by an attending physician and an associate chief physician from the Department of Neurosurgery, with the derived average value being recorded.

Statistical analysis

All statistical analyses were performed using Statistic Package for Social Science (SPSS) software (version 21.0, IBM, Armonk, NY, USA). For experimental data following a normal distribution, values were expressed as $\bar{x} \pm s$. Comparisons between the two groups were conducted using a paired *t* test. Count data were reported as frequencies or proportions, and differences between the two groups were evaluated using a chi-square test. Factors that were statistically significant in the univariate analysis were included in the multivariate analysis. Multivariate analysis was executed using a logistic regression model. The predictive power of relevant factors for the progression from TSE to CSDH was evaluated using receiver operating characteristic (ROC) curves. These curves provide a visual representation of the trade-off between sensitivity and specificity for different cut-off points of a diagnostic test, in this case, the factors being evaluated in the study. A P-value less than 0.05 was considered indicative of statistical significance.

RESULTS

Clinical Characteristics of Patients and Univariate Analysis

Within this study cohort, 26 cases (20.97%) developed into hematoma, and 98 cases (79.03%) remained as effusion, indicating a 20.97% conversion rate of TSE into CSDH. Comparisons between the two groups uncovered no statistically significant differences in terms of gender, presence of hypertension, COPD, or GCS scores ($P > .05$). Nevertheless, significant differences were observed in variables including age, incidence of tSAH, location of subdural effusion, and thickness of subdural effusion ($P < .05$). In the univariate analysis, age, incidence of tSAH, location of subdural effusion, and thickness of subdural effusion were found to be significantly associated with the progression of TSE into CSDH ($P < .05$, Table1).

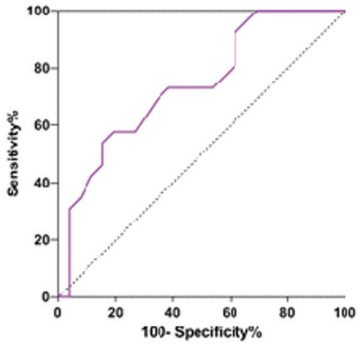
Table 2. Multivariate Regression Analysis of Risk Factors for Conversion of TSE to CSDH

Factors	*B* value	*SE* value	*Ward* value	*OR* value	95%*CI*	*P* value
Age	0.193	0.076	6.455	1.213	1.045~1.407	.012
tSAH	2.529	0.889	8.093	12.542	2.196~71.630	.000
Subdural effusion thickness	0.579	0.236	6.039	1.786	1.124~2.836	.006
Effusion location	1.092	0.914	1.429	2.983	0.497~17.892	.254

Table 3. Predictive Value of Age, tSAH, and Subdural Effusion Thickness for Conversion of TSE to CSDH

Indicator	Cutoff Value	AUC	95%CI	Specificity (%)	Sensitivity (%)	*P* value
Age	70	0.739	0.604 ~0.874	71.65	61.80	.003
tSAH	/	0.670	0.516~0.823	61.28	50.27	.035
Subdural effusion thickness	10.7	0.820	0.702~0.937	76.26	76.37	.000
Combined detection	/	0.942	0.882~0.999	93.28	81.45	.000

Figure 1. ROC Curve for Age Prediction of CSDH



Multivariate Analysis of Risk Factors for TSE Progressing into CSDH

A multivariate logistic regression analysis was conducted on factors with a $P < .05$ in the univariate analysis. The results suggested that age (1.213), co-occurrence of tSAH (12.542), and thickness of subdural effusion (1.786) were identified as independent risk factors for the progression of TSE into CSDH ($P < .05$). Multivariate logistic regression analysis revealed that age (OR 1.213), co-occurrence of tSAH (OR 12.542), and thickness of subdural effusion (OR 1.786) were independent risk factors for the progression of TSE into CSDH ($P < .05$, Table2).

Prognostic Utility of Age, tSAH, and Thickness of Subdural Effusion in Predicting TSE Conversion into CSDH

With age, co-occurrence of tSAH, and subdural effusion thickness as predictive variables and the development of CSDH as the actual outcome, a receiver operating characteristic (ROC) curve was constructed. The resultant areas under the curve (AUCs) for predicting CSDH based on age, tSAH, and subdural effusion thickness were 0.739, 0.670, and 0.820, respectively. When these predictors were combined, the AUC increased to 0.942, which surpassed those of the individual predictors. For more detailed information, please refer to Table 3 and Figures 1-4.

Figure 2. ROC Curve for tSAH Prediction of CSDH

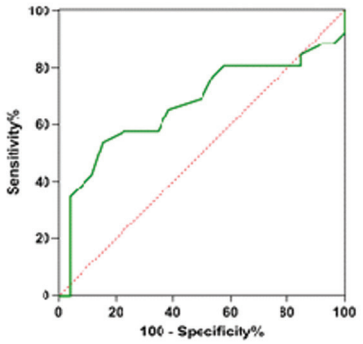


Figure 3. ROC Curve for Subdural Effusion Thickness Prediction of CSDH

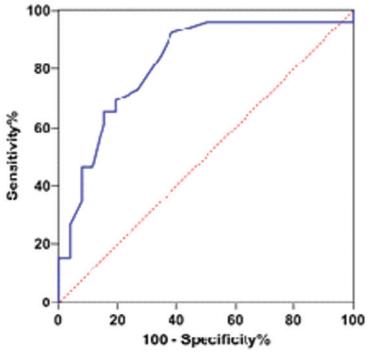
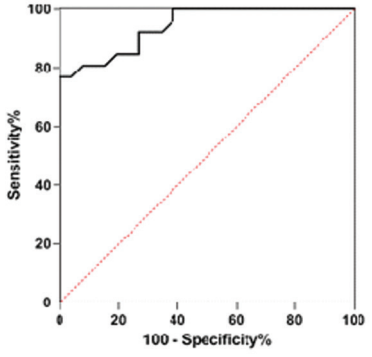


Figure 4. ROC Curve for Combined Detection Prediction of CSDH



DISCUSSION

Typically, Traumatic Subdural Effusion (TSE) may follow one of three trajectories: absorption, stagnation, or enlargement, progressing to Chronic Subdural Hematoma (CSDH). The possibility of TSE transitioning into CSDH was first proposed by Yamada in 1979, leading some researchers to view TSE as a preliminary stage of CSDH. In 2008, Professor Jiang Jiyao and his team suggested that TSE and CSDH might represent different stages of the same inflammatory response.[11] While various theories and hypotheses have been presented to explain this transformative tendency, such as the rupture of bridging veins and neutrophil oxidative bursts induced by interleukin-8, these mechanisms are not fully understood.[4] Although there is a consensus that TSE can evolve into CSDH, an essential task for clinicians is to identify which TBI patients are at a heightened risk for this transformation. Our study revealed that, in TBI patients with TSE, the thickness of the subdural effusion serves as a reliable indicator of TSE's potential progression to CSDH. The probability of this transition increases when the effusion thickness exceeds 10.7 mm.

Our study establishes a link between the thickness of subdural effusion in TSE patients and the progression of TSE to CSDH. An increased thickness of subdural effusion signifies a larger subdural space, which in turn heightens the risk of bridging vein rupture and subsequent bleeding. This raises the likelihood of CSDH development. Additionally, an increased effusion thickness may prolong the absorption time and lead to encapsulation of the effusion. This encapsulation can develop into a hematoma, with incomplete capillary walls potentially increasing permeability and exudation, thereby contributing to the formation of CSDH. The persistence of TSE may also result in CSDH due to recurring microbleeds within the neomembrane.[12] Existing literature suggests that early surgical intervention for subdural effusion showing a mass effect can reduce the risk of this transformation, potentially by interrupting the process of the effusion transforming into a cystic tumor and the formation of encapsulation.[5,13,14] Several studies have identified the thickness of subdural effusion as an independent risk factor for the transition of TSE to CSDH,[15,16] although the direct relationship between effusion thickness and this transition has yet to be fully elucidated. We conducted a Receiver Operating Characteristic (ROC) analysis on this risk factor and obtained an Area Under the Curve (AUC) of 0.820, with a cutoff value of 10.7 mm. This showcases its predictive value; the risk of transition in TBI patients heightens when the thickness of the subdural effusion exceeds 10.7 mm. These findings are consistent with those reported by Fan et al.[17], who suggested a cutoff value for effusion thickness of 11.37 mm.

Discussion about the role of tSAH in the progression from TSE to CSDH is scant in existing literature. When a TBI occurs, the formation of arachnoid fissures can be anticipated, especially in the presence of tSAH. The rupture in the arachnoid membrane functions similarly to a one-way active valve, allowing cerebrospinal fluid (CSF) mixed with blood from the subarachnoid space to accumulate in the subdural cavity through the fissures, thus leading to TSE.[18] It is widely acknowledged that the source of effusion in TSE patients is the CSF from the subarachnoid space.[2,19] When tSAH accompanies TBI, the resultant subdural effusion may contain blood elements. These blood components break down to form inflammatory mediators, which in turn stimulate the production of cytokines such as Interleukin-6 (IL-6) and Interleukin-8 (IL-8). These cytokines can foster the formation of an encapsulating membrane. Simultaneously,

IL-8 facilitates the infiltration of inflammatory cells and enhances the permeability of neovasculature. Continual bleeding into the effusion encapsulation gradually evolves into CSDH.[17,20] Our study posits that tSAH can accelerate this progression, potentially due to an increased presence of blood components in the subdural effusion during tSAH, as suggested by a rise in the CT value of the effusion. Prior literature has identified an independent association between a higher CT value of the subdural effusion and its progression from TSE to CSDH.[15,21] An increased CT value in TSE patients following tSAH, combined with the proven association between tSAH and effusion progression, indirectly confirms the link between elevated effusion CT values and effusion progression.

There is currently no consensus on whether age serves as a risk factor for the transition from TSE to CSDH. Some researchers posit that extreme ages represent a risk factor for this transition.[5] Within the context of surgical interventions for unruptured aneurysms via craniotomy clipping, it is suggested that the risk of transition escalates when the patient's age exceeds 60 years.[2] On the contrary, some reports refute age as a risk factor for the transformation from TSE to CSDH.[15,21] This study revealed a statistically significant age difference between the hematoma and effusion groups ($P<0.05$). It appears that TSE patients older than 70 years exhibit an increased propensity for progression to CSDH. This can be ascribed to the fact that older patients tend to exhibit some degree of cerebral atrophy, which consequentially establishes a larger subdural space. In these situations, the arachnoid granulation veins and bridging veins are prone to tension, potentially leading to hemorrhage—a recognized risk factor for the transition to CSDH.[8,22,23]

In conclusion, a plethora of risk factors contribute to the transition from TSE to CSDH. The evolving trajectory from TSE to CSDH merits close scrutiny. Particularly when patients are of advanced age, present with concurrent tSAH, and exhibit significant subdural effusion thickness, such transition tends to occur more readily. The collective predictive power of these risk factors, demonstrated by an area under the curve (AUC) of 0.942, bears significant predictive value. Therefore, for TSE patients, especially those manifesting the aforementioned risk factors, timely CT or MRI examinations should be initiated to diagnose CSDH and enable swift treatment definitively. Timely CT or MRI examination is important for the definitive diagnosis of chronic subdural hematoma (CSDH) in patients with traumatic subdural effusion (TSE), especially in older patients with traumatic subarachnoid hemorrhage (tSAH) and showing significant subdural effusion thickness. By identifying these risk factors, healthcare providers can proactively initiate appropriate interventions that improve patient outcomes and promote rapid treatment.

The clinical implications of our findings highlight the importance of timely CT or MRI examinations for patients with TSE, particularly those who exhibit the aforementioned risk factors. Early detection and diagnosis of CSDH enable prompt treatment initiation. Healthcare providers should be aware of these risk factors and proactively initiate appropriate interventions to improve patient outcomes. However, it is important to acknowledge the limitations of our study. Potential sources of bias or confounding factors should be considered, and the generalizability of our findings to other populations or settings should be examined. Future research in this area should focus on addressing unanswered questions and exploring areas that require further investigation. This will provide a roadmap for researchers interested in expanding upon our work and advancing the understanding of TSE and CSDH. The identification of risk factors for the transition from TSE to CSDH has significant implications for clinical decision-making. Healthcare providers should consider specific interventions or preventive measures for at-risk patients. By recognizing these risk factors, clinicians can make informed decisions and implement appropriate strategies to mitigate the progression to CSDH. In summary, our study emphasizes the importance of early detection, timely intervention, and patient management in the context of TSE and CSDH. By understanding the key findings and their broader implications, healthcare providers can enhance patient care and improve outcomes in this population.

ETHICAL COMPLIANCE

This study was approved by the ethics committee of Xinyi People's Hospital.

CONFLICT OF INTEREST

The authors have no potential conflicts of interest to report relevant to this article.

AUTHOR CONTRIBUTIONS

JL and ZX designed the study and performed the experiments, XC and FW collected the data, XY and MZ analyzed the data, JL and ZX prepared the manuscript. All authors read and approved the final manuscript.

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