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ORIGINAL COMMUNICATION

Transient neurological deficit in patients with chronic subdural hematoma: a retrospective cohort analysis

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Abstract

Rationale Symptoms of chronic subdural hematoma (CSDH) vary widely, including transient neurological deficit(s) (TND). The precise prevalence and the clinical aspects of TND are yet to be determined. Most TNDs are regarded and treated as symptomatic seizures, but the rationale for this decision is not always clear.

Methods Patients with temporary symptoms were selected from a retrospective cohort of CSDH patients. We analyzed the association of TND characteristics with patients being classified as having a symptomatic seizure and with functional outcome using logistic regression analysis.

Results Of the included 1307 CSDH patients, 113 (8.6%) had at least one episode of TND. Most common TNDs were aphasia/dysphasia, impaired awareness or clonic movements. Of these 113 patients, 50 (44%) were diagnosed with symptomatic seizure(s) by their treating physician. Impaired awareness, clonic movements and the presence of 'positive symptoms' showed the strongest association with the diagnosis symptomatic seizure (OR 36, 95% CI 7.8–163; OR 24, 95% CI 6.4–85; and OR 3.1, 95% CI 1.3–7.2). Aphasia/dysphasia lowered the chance of TND being classified as symptomatic seizure together with a longer TND duration (OR 0.2, 95% CI 0.1–0.6; and OR 0.91, 95% CI 0.84–0.99). Treatment with anti-epileptic drugs was related to unfavorable functional outcome (aOR 5.4, 95% CI 1.4–20.7).

Conclusion TND was not a rare phenomenon in our cohort of CSDH patients. A TND episode of 5 min, aphasia/dysphasia and/or absence of 'positive' symptoms are suggestive of a different TND pathophysiology than symptomatic seizures. Our results further suggest that treatment of TND in CSDH deserves careful consideration as management choices might influence patient outcome.

Keywords Anti-seizure medication · Chronic subdural hematoma · Epilepsy · Functional outcome · Seizures · Transient neurological deficits

Abbreviations

ASM	Anti-seizure medication(s)
BHC	Burr hole craniostomy
CSD	Cortical spreading depression/depolarization
CSDH	Chronic subdural hematoma

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TIA Transient ischemic attack
TND Transient neurological deficit

Introduction

Patients with a chronic subdural hematoma (CSDH) commonly have symptoms, such as headache, hemiparesis, cognitive deficit and gait disturbances [1]. Another presentation of CSDH concerns temporary neurological symptoms [2]. These episodes have been described as transient cerebral ischemic attack(s), transient neurological symptom(s) and most commonly as transient neurological deficit(s) (TND) [3–6]. The reported prevalence of TND in CSDH varies strongly between 1 and 24% of CSDH patients, depending on the definition [4, 7, 8]. Clinically, aphasia/dysphasia is the most frequently observed TND, followed by one-sided weakness and hemisensory deficit [2, 4, 8]. Classic symptomatic seizure-like symptoms, such as transient impaired/changes in awareness and clonic movements, are as a rule not considered as TND in the literature. This may be considered surprising given that (focal) epileptic activity is also suggested to be one of the pathophysiological mechanisms that causes TND in CSDH [3, 4, 6, 9]. Multiple other pathophysiological mechanisms for TNDs have been proposed as well: cortical spreading depression, focal ischemia, recurrent micro-hemorrhages, temporary cerebral edema, and mechanical pressure caused by the hematoma. [3–6, 9]. Even though TNDs may be caused by any of the aforementioned mechanisms, TNDs in CSDH patients are most often regarded by clinicians as being epileptic in origin and treated as such [10].

TND is also seen postoperatively in patients with CSDH, as described in a recent article. [11]. The authors proposed an experimental grading scale, the 'NonEpileptic, Stereotypical and Intermittent Symptoms (NESIS) in patients with subdural hematoma', to aid clinicians in differentiating between symptomatic seizures and other alternative etiologies of *post-operative* TND. The correct diagnosis of TND is important, especially when considering that symptomatic seizures as a result of CSDH have been associated with unfavorable outcome [12, 13].

A diagnosis of seizure(s) may also result in the start of anti-seizure medication (ASM) treatment, which can have serious side effects and subsequently lower quality of life [14, 15]. Also, it has been shown that TND does not always improve after surgical treatment of CSDH. Therefore, a correct diagnosis of the cause of TND is relevant, as it might imply that surgery can be avoided.

In this study, we assessed the prevalence of TNDs in patients with CSDH. Also, we analyzed which clinical characteristics of TND are associated with the episodes being classified as symptomatic seizures in our cohort. Furthermore, we investigated the validity of the NESIS score for pre-operative TND. Finally, the association of several clinical characteristics and functional outcome in patients with TND was studied.

Methods

Patients

We retrospectively extracted data from the electronic patients' files of all consecutive adult CSDH patients in three hospitals with neurosurgical facilities in the Netherlands: the University Medical Center Groningen, Isala Hospitals and Medisch Spectrum Twente Enschede. Inclusion periods were from 2005 to 2019, but inclusion length varied between centers from 8 to 13 years. All three hospitals serve as a referral center for neurosurgical care in their region. Symptoms at presentation were registered and classified to be of transient or persistent nature. Other data that were collected included age, sex and functional outcome. Functional outcome was determined using the Glasgow Outcome Score Extended (GOS-E), an 8-point scale with scores varying from 1 (death) to 8 (full recovery) [16]. The GOS-E was dichotomized into unfavorable (GOS-E 1–4) and favorable (GOS-E 5–8) outcome for statistical purposes. GOS-E scores were based on the outpatients' notes at the first follow-up appointment after treatment. Also, 3-month recurrence rate was scored, defined as recurrence of symptoms/signs with re-accumulation of hematoma on head CT scan, and 3-month mortality.

The medical ethical committees/local ethical review boards of all three hospitals consented to this study (numbers: 2019/026, 190213 and KH19-13). Due to the retrospective nature of the study, the necessity for an informed consent was waived.

Definition of TND

All signs and/or symptoms with a transient nature were scored as TND, excluding the transient symptoms of headache, nausea and vomiting. In the literature on TND in CSDH, transient impaired awareness and/or clonic movements are regularly not included, probably because they are classical symptoms of a seizure and the TNDs are diagnosed as such. Nevertheless, we included these symptoms to be able to differentiate between TND and symptomatic seizures. Patients with a diagnosis of epilepsy prior to the CSDH were excluded, for the uncertainty of the TND being caused by CSDH or by the epilepsy. Also, patients with a more probable alternative explanation for the TND were excluded, for example if the treating physician deemed syncope, resulting in altered consciousness, more likely.

Clinical aspects of TND

If patients experienced a TND, we collected data on duration and frequency of the episodes together with their clinical presentation. All patients with transient altered awareness, changes in consciousness and/or loss of consciousness in our cohort were classified as 'impaired awareness'. Also, the treatment modality was recorded: burr hole craniostomy (BHC), dexamethasone, starting of ASM, 'watchful waiting' treatment or a combination of the aforementioned. The response of the TND to given treatment was classified as: (1) permanently resolved, (2) temporarily improved, (3) worsened or (4) no response.

NESIS score

To assess the validity of the NESIS score for our *pre-operative* TND patients, we determined and translated all components of the NESIS to the corresponding NESIS score. The NESIS score varies between -8 and 13 with a score of equal to or more than 4 in the absence of epileptic EEG activity indicating a different cause for TND than a symptomatic seizure [11] (Table 1). The sum score comprises the following characteristics which are all given a certain amount of points: migration of symptoms, a stereotype semiology, duration and frequency of symptoms, impaired awareness and clonic movements. Finally, in the NESIS score, points are awarded for a negative or positive nature of symptoms, meaning a loss of function (hemiparesis, hypesthesia) for negative symptoms and added sensations for positive symptoms (paresthesia). If available, the EEG diagnosis was registered too.

Table 1 Items of the Nonepileptic, Stereotypical, and Intermittent Symptom (NESIS) score [11]. Other diagnosis than synptomatic seizures probable if total score ≥ 4 and no positive EEG

Features supporting other etiology than synptomatic seizure	Point values
Negative symptoms	+4
Duration ≥ 5 min	+3
Dysphasia	+3
Migration	+1
≥ 5 Episodes	+1
Stereotypy	+1
Features supporting synptomatic seizures	
Impaired awareness	− 4
Clonic movements	− 4

Clinical diagnosis of epilepsy

The TND episodes were classified as symptomatic seizures based on the diagnosis made by the treating physician. This resulted in a binary outcome: seizure 'yes' or 'no'. In some cases, the treating physician did not specifically mention this diagnosis, but ASM were started. If ASM was started, regardless of TND symptomatology, we classified the TND in these patients as seizure(s). For instance, in a patient with transient aphasia that received ASM, but without a chart note stating seizure as cause of the TND, this TND was classified as a seizure.

The association of all TND characteristics with the clinical diagnosis symptomatic seizure was assessed using binary logistic regression analysis.

Statistical analysis

TND vs. non-TND patients

We compared baseline demographic and hematoma characteristics of TND and non-TND using Chi-square or Mann–Whitney *U* tests when appropriate. Furthermore, the 3-month mortality, 3-month recurrence and functional outcome were compared between both groups using univariable analyses.

Association with outcome

Within the TND group, the association between all clinical characteristics of TND episodes and functional outcome was assessed using binary logistic regression analyses, adjusted for age and sex (aOR). Also, the relationship between functional outcome and the (clinical) diagnosis of symptomatic seizure(s) and the starting of ASM treatment was assessed. For the analyses, duration and frequency of TND were subdivided per 5 min or episodes to facilitate interpretation.

All associations are presented in odds ratio (OR) with 95% confidence interval (CI). If data were unknown, for instance, the duration of TND, it was analyzed as 'missing data' to prevent bias. All statistical analyses were performed with IBM SPSS statistics version 23. Figures were made using the "ggplot" package in RStudio. Reporting was done following the STROBE guidelines for observational studies.

Results

Prevalence

We identified 1307 patients diagnosed with a CSDH, of which 113 patients had at least one episode of TND, resulting in a prevalence of 8.6%. In 50 of these patients

Table 2 Clinical characteristics of 113 CSDH patients with Transient Neurological Deficit

Variable	*n* (%) (Total = 113)
Sex (male)	82 (73)
Age (years ± SD)	73 (± 12.5 SD)
Type of transient neurological deficit (TND)	
Aphasia/ dysphasia	45 (40)
Impaired awareness	30 (27)
Motor: clonic movements	29 (26)
Motor: weakness/hemiparesis	21 (19)
Dysarthria	15 (13)
Sensory: hypesthesia	17 (15)
Sensory: paresthesia	13 (12)
Confusion	6 (5)
Amnesia	1 (1)
Location of CSDH	
Unilateral	93 (82)
Bilateral	20 (18)
Duration in minutes median (range)	10 (0.5–1440)
Amount of episodes median (range)	2 (1–15)
Treatment modality	
Surgical evacuation	40 (35)
Anti-seizure medication + surgical evacuation	21 (19)
Close observation	17 (15)
Anti-seizure medication	11 (10)
Dexamethasone	10 (9)
Dexamethasone + surgical evacuation	6 (5)
Anti-seizure medication + dexamethasone	6 (5)
Anti-seizure medication + surgical evacuation + dexamethasone	1 (1)
Antiplatelet medication started	1 (1)
Effect of treatment:	
Permanently gone	90 (80)
Temporarily improved	13 (11)
Worsened	3 (3)
No reaction	6 (5)
Unknown	1 (1)

(3.8%), the diagnosis of a symptomatic seizure was made by the treating physician, leaving 63 patients (4.8%) with a TND diagnosis other than seizure.

Clinical aspects of TND

The most common TND was aphasia/dysphasia, found in 45 patients (40%) (Table 2). Impaired awareness was present in 30 patients (27%) and clonic movements were observed in 29 patients (26%), 21 patients (19%) had both impaired awareness and clonic movements. Median duration of the TND was 10 min (IQR 0–30) and median number of episodes was 2 (range: 1–15). Median time period from first TND to diagnosis of the CSDH was 1 day (IQR 0–8), and median time from first TND to treatment was 3 days (IQR 0–10).

Treatment modality and effect of treatment

BHC was performed in 68 (60%) of TND patients, in 40 patients (35%) as monotherapy and in 28 patients (25%) in combination with other treatment modalities (Table 2). In total, 39 patients (35%) were treated with ASM of which in 11 patients the ASM was the only treatment. Most often used ASM were levetiracetam ($n = 23$) and valproic acid ($n = 13$).

In 90 out of the 113 patients (80%), the TNDs dissolved after receiving treatment and 13 (11%) had a temporarily improvement after which the TNDs recurred. Of these 13 patients, 11 (85%) received the clinical diagnosis epilepsy. In the other 10 patients, complaints worsened in three patients, were unchanged in three patients and the effect of treatment was unknown in one case.

Table 3 Comparison between patients with and without Transient Neurological Deficit (TND) using chi square or Mann–Whitney *U* test

Variable	Patients with TND *n* (%) Total = 113	Patients without TND *n* (%) Total = 1194	*p*-value
Age	72.7 (± 12.5)	73.8 (± 11.4)	*0.520*
Sex (male)	82 (73)	876 (74)	*0.825*
3-month Recurrence	9 (8)	143 (12)	*0.281*
3-month Mortality	5 (4)	68 (6)	*0.827*
Unfavorable functional outcome (GOS-E 1–4)	13 (13)[a]	116 (12)[a]	*0.627*

[a]Percentage of the known cases, for TND patients $n = 99$, for non-TND $n = 992$

Significant value = $p < 0.05$

TND vs. non-TND patients

We found no differences in age, sex, recurrence rate, mortality or functional outcome when comparing TND to non-TND patients (Table 3).

Clinical diagnosis of epilepsy

The treating physicians classified the episodes of TND as symptomatic seizure in 50 patients (44%). In 53 patients, the TND was considered a result of CSDH, without further specification and 8 patients were suspected to suffer from a vascular ischemic event. This suspicion was based on clinical symptoms/signs, without confirmation by diffusion weighted MRI or perfusion CT in the majority of patients. In one patient that was suspected to have suffered from a vascular ischemic event, an MRI was made, showing both a subdural hematoma and an area of recent ischemia. In two patients, the TND was reported to result from cortical stimulation, without further explanation.

In seven of the patients that received the diagnosis of a symptomatic seizure, an EEG was made, of which two EEGs showed lateralized epileptic activity corresponding with the clinical features. The clinical variables significantly associated with the diagnosis symptomatic seizure were clonic movements and impaired awareness (OR 36, 95% CI 7.8–163 and OR 24, 95% CI 6.4–85, respectively) (Fig. 1). When combining patients with both impaired awareness and clonic movements, all 21 received the diagnosis symptomatic seizure from their treating physician. The occurrence of 'positive symptoms' was also associated with the diagnosis symptomatic seizure (OR 3.1, 95% CI 1.3–7.2). Longer duration of the TND (per 5 min) (OR 0.91, 95% CI 0.84–0.99) and the presence of an aphasia/dysphasia disorder (OR 0.2, 95% CI 0.1–0.6) lowered the possibility of the diagnosis of a seizure.

Association with outcome

Median time from diagnosis to follow-up and determining of GOS-E was 47 days (IQR 35–74).

Of the analyzed characteristics of TND, only the presence of clonic movements was associated with unfavorable functional outcome (aOR 5.8, 95% CI 1.4–23.2). The combination of impaired awareness and clonic movements was also related with unfavorable outcome (aOR 5.0, 95% CI 1.1–22.3) (see Fig. 2).

Classification of the TND as a symptomatic seizure by the treating physician was not associated with functional outcome (aOR: 0.48, 95% CI 0.14–1.7). Treatment with ASM, however, was related with unfavorable functional outcome (aOR 5.4, 95%CI 1.4–20.7). Adjusting both clonic movements and the combination of impaired awareness and clonic movements for ASM treatment, resulted in a non-significant relationship with functional outcome (OR 1.7 95% CI 0.82–17.2 and OR 1.0, 95% CI 0.2–4.6, respectively).

NESIS score

We found that 32 of 50 patients (64%) with the clinical diagnosis of a symptomatic seizure had a corresponding NESIS score of less than 4 (indicating seizures as a probable cause of the TND). Of the patients with a NESIS score of 4 or more (suggesting another etiology than seizure), 19% were still diagnosed with a symptomatic seizure.

Discussion

Key results

In this study, we found a prevalence of 8.6% of transient neurological deficit (TND) in patients with a CSDH, of which 3.8% received the clinical diagnosis of a symptomatic seizure. The most common transient sign was aphasia/dysphasia. We could not establish significant differences between TND and non-TND patients concerning age, sex, recurrence rate, mortality or functional outcome. Treatment of TND with ASM showed a negative association with outcome.

Interpretation

The prevalence of TND in our cohort (8.6%; 4.8% when excluding impaired awareness and clonic movements as TND symptoms) is comparable with a previous study that found TND in 9.2% of CSDH patients [4]. In their analysis of 130 CSDH patients, recruited in a single center, it is unclear whether this concerned consecutive patients. The authors reported that aphasia/dysphasia and hemiparesis were the most common forms of TND, similar to our study [4]. Our prevalence is far less than the 24% of another study describing symptoms of 114 consecutive CSDH patients. Unfortunately that study did not provide the specific details of the 'fluctuating symptoms' they described, making exact comparisons difficult [7]. The phenomenon of TND is rarely discussed in CSDH literature. This could be caused by a reporting bias as most studies do report on symptomatic seizures, but do not classify this as TND. Therefore, the true prevalence seems highly dependent on the definition of TND.

The fact that only seven patients underwent EEG examination (out of 50 patients diagnosed with a symptomatic seizure) suggests that clinicians predominantly diagnose the cause of TND in CSDH using solely clinical

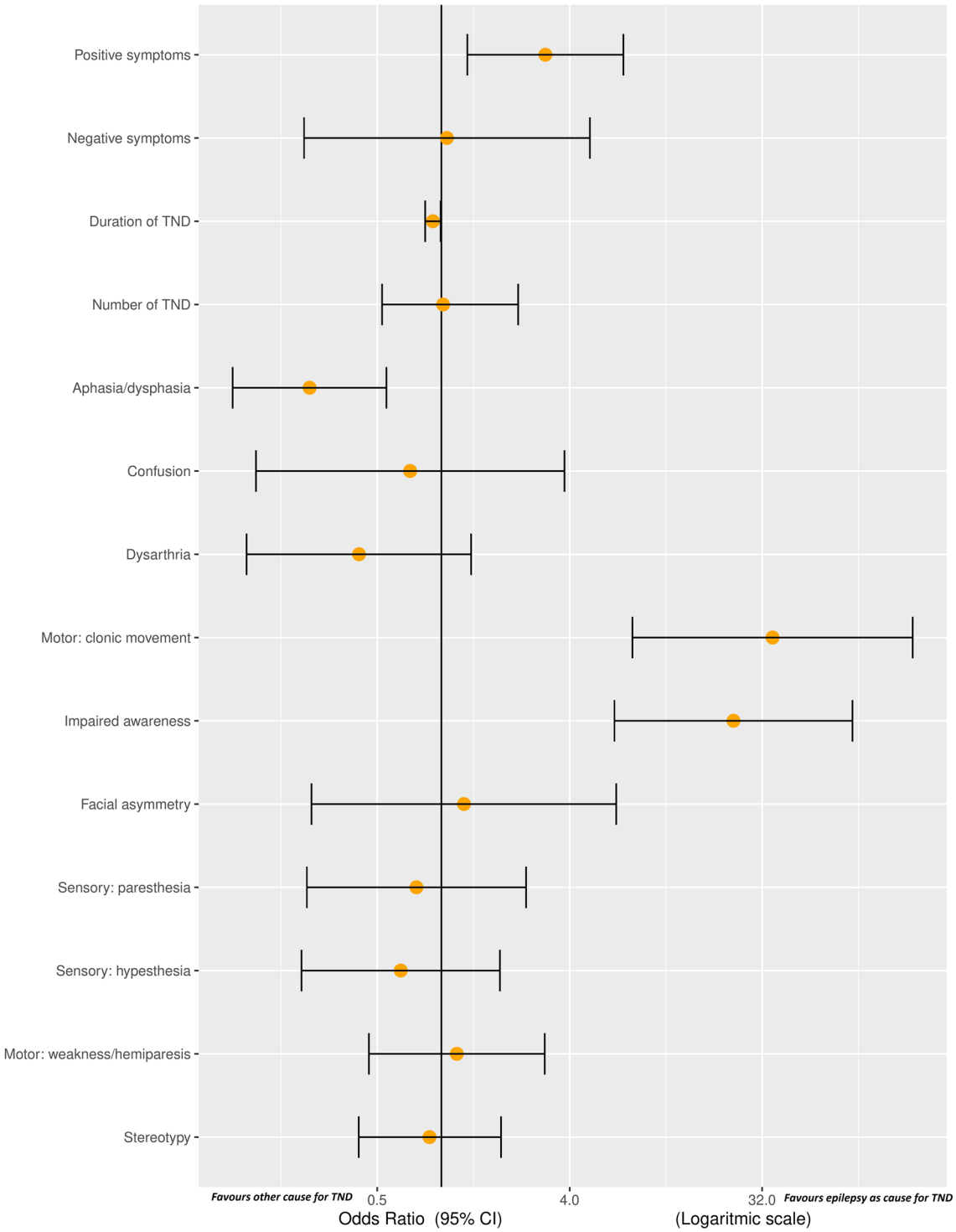

Fig. 1 Forest plot for Odds Ratio and 95% Confidence Intervals for different clinical characteristics of Transient Neurological Deficit (TND) and clinical diagnosis of symptomatic seizures

characteristics. One of these characteristics is the duration of episodes: A longer duration was negatively associated with the clinical diagnosis of a seizure. In general, seizure duration is dependent of the type of seizure with a median time of 30–120 s [17]. Because the median duration of the TNDs in our cohort was 10 min, this might have triggered clinicians to consider a different diagnosis. This assumption also corresponds with the NESIS score, in which an

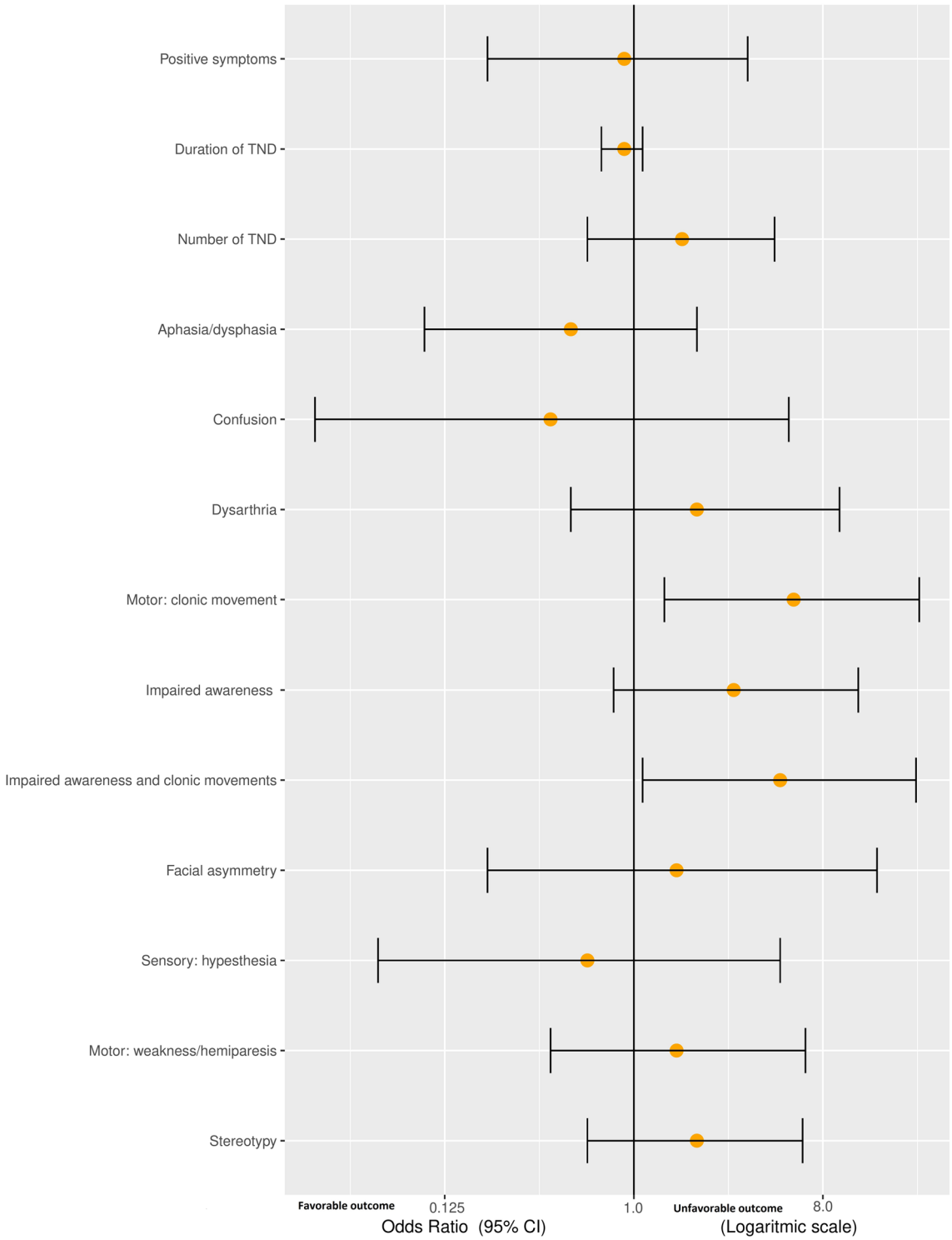

Fig. 2 Forest plot for Odds Ratio and 95% Confidence Intervals for different clinical characteristics of Transient Neurological Deficit (TND) and functional outcome as measured with Glasgow Outcome Scale-Extended. Adjusted for age and sex. The TNDs 'negative symptoms' and 'sensory: paresthesia' are not shown as the calculated OR was not applicable

episode of more than 5 min decreases the chance of seizure as the cause of TND.

It is not surprising that we found an association of myoclonic movements and/or impaired awareness with the diagnosis of a symptomatic seizure. These are probably the

strongest clinical manifestations suggesting a seizure [18, 19], and these symptoms combined with the presence of a CSDH will most likely cause clinicians to make this diagnosis. In general, 'positive symptoms', such as paresthesia, auras and automatisms (repeated movements, smacking), are also commonly found in patients with seizures [19–21]. This might explain why 'positive symptoms' were associated with the diagnosis of a symptomatic seizure in our cohort as well. However, these 'positive symptoms' could not be confirmed in our CSDH patients. This is probably due to a reporting bias, as symptoms had to be retrieved from the notes in the electronic patient files. In the NESIS score, 'positive symptoms' are also associated with seizures. When applying the NESIS score to our cohort, keeping in mind that it is developed for *post-operative* TND, we found that 64% of patients with the diagnosis of a seizure had a corresponding NESIS score of < 4. This might suggest that the NESIS score can also aid clinicians in the differentiation between seizures and other causes for TND in pre-operative CSDH patients.

Another aim of our study was to assess the relationship of symptomatic seizures and outcome after CSDH, a finding that has been reported before [13]. We were able to establish this relationship for clonic movements and outcome, but this association was not found for the clinical diagnosis of seizure, as made by the treating physician, and outcome. The explanation possibly lies within the association of ASM treatment and outcome, since the relationship between clonic movements and unfavorable outcome disappeared after adjusting for ASM treatment. The unfavorable influence of ASM on outcome has been described before and it is suggested to be related to poor cognitive status as a result of ASM treatment [22, 23]. Therefore, we emphasize the importance of a correct diagnosis of TNDs in CSDH before starting treatment with ASM. The association of unfavorable outcome and ASM treatment could also be caused by more severely affected patients (with multiple seizures) receiving ASM therapy sooner. This poorer outcome could also be an effect of the CSDH itself, with seizures being amongst the more severe signs of CSDH, reflecting more advanced disease and therefore poorer outcomes.

Limitations and generalizability

Strong points of our study are the large number of patients with TND that are included with multicenter recruitment in three hospitals and with an extensive collection of clinical variables. A limitation lies in the retrospective nature of our study making it susceptible to reporting and allocation bias as well as confounding by indication, as for example the exact considerations regarding the diagnosis of TND by physicians could not be retrieved. A reporting bias might have resulted in an underestimation of the true prevalence of TND in CSDH. The lack of EEG (seven in total) and MRI examination is another important limitation of our study. As a result, we could not make more definite statements about the presence or absence seizures or other explanatory causes for TNDs in our patients. Another limitation is that the duration of ASM treatment was not known in our cohort, which might have influenced the association with outcome. Outcome could also have been biased as the GOS-E was determined based on out-patient notes. Interpretation of the outcome and association with outcome should therefore be done with caution. Finally, whether our results are valid for all CSDH patients with transient symptoms requires further prospective study with EEG and MRI examination to rule out presence of for instance (temporary) ischemia as a cause.

Conclusion

In conclusion, TND is not a rare phenomenon in CSDH patients, with a prevalence of 8.6% (or 4.8% without patients diagnosed with seizures) in our cohort. Aphasia/dysphasia is the most common presentation form of TND. Clonic movements as a TND are related to functional outcome, which might be due to subsequent starting of ASM treatment. A TND episode that lasts more than 5 min with aphasia/dysphasia and absence of 'positive symptoms' suggests another pathophysiology of the TND than a symptomatic seizure. Management of TND in CSDH requires careful consideration of the responsible mechanism, as certain management choices can influence functional outcome of patients. The NESIS score might aid clinicians in management of TND, but further prospective study on this topic is required.

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Data availability statement Anonymised data are available upon reasonable request from the corresponding author and after clearance by the competent ethics committee.

Declarations

Conflicts of interest The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Ethical approval The medical ethical committees/local ethical review boards of all three hospitals consented to this study (numbers: 2019/026, 190213 and KH19-13). Due to the retrospective nature of the study, the necessity for an informed consent was waived.

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