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Initial experience of a telemetry EEG amplifier (Headset™) in the emergent diagnosis of nonconvulsive status epilepticus

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ABSTRACT

Objectives: In the diagnosis of nonconvulsive status epilepticus (NCSE), the use of ongoing ictal electroencephalographic (EEG) findings is the gold standard. However, this is difficult without the availability of continuous EEG monitoring facilities. Given this, a small telemetry EEG amplifier (Headset-EEG; AE-120A EEG Headset™) was introduced for use in simple EEG monitoring facilities. In this study, we examined the diagnostic performance of the Headset-EEG for NCSE in a neuroemergency setting.

Methods: We retrospectively selected five patients who were diagnosed with NCSE using Headset-EEG by attending physicians between October 2017 and July 2018. Reevaluation with Headset-EEG was performed, and the findings were compared with the initial diagnosis. Results: In Case 1, Headset-EEG clearly revealed repeated ictal discharges associated with acute subdural hematoma. However, in Cases 2 and 3, the initial EEG diagnosis was incorrectly made because of an EEG interpretation error. In Cases 4 and 5, various artifacts, caused by unstable electrode placement, resulted in an initial diagnosis of ongoing seizure activity. Nevertheless, the initial diagnoses and subsequent treatment of Cases 2–5 did not directly affect the patients' outcomes.

Conclusion: The present study indicates that Headset-EEG is a possible tool for use in NCSE screening. However, considerable knowledge of EEG interpretation is needed to establish a correct NCSE diagnosis. When NCSE is suspected per Headset-EEG, a subsequent routine EEG with a full montage of 10–20 EEG electrodes should be performed in collaboration with an epileptologist or electroencephalographer.

1. Introduction

Nonconvulsive status epilepticus (NCSE) has recently been recognized as a critical cause of unexplained impaired consciousness in the field of neuroemergency medicine [1]. While convulsive status epilepticus is easily recognized, capturing ongoing seizure activity or continuous epileptiform discharge on electroencephalography (EEG) is the gold standard for diagnosing NCSE [1–6]. However, it is difficult to record these ictal discharges without continuous EEG monitoring facilities, which are available only in epilepsy centers or neuro-intensive care units [4,6]. In many hospitals in Japan, even a routine EEG examination is often unavailable outside of working hours or on weekends [2,4], and a delay in performing EEG recordings may cause a diagnosis of NCSE to be overlooked [2,4,6,7]. Another difficulty in recording/monitoring EEGs in emergency rooms (ERs) or intensive care units (ICUs) is that very few physicians can apply International 10–20 EEG electrodes to the correct positions on a patient's head.

To overcome these problems, a telemetry EEG amplifier (AE-120A EEG Headset™: Headset) was first developed by Nihon Kohden (Tokyo, Japan) (Fig. 1a). One of the advantages of this device is that anyone can use it by following several simple steps. Curved lines follow the contours of the patient's skull and the Headset's small dimensions allow for less interference with other medical equipment that might be attached to the patient's head (Fig. 1b). Disposable electrodes enable quick application without having to first prepare attachment sites. The Headset's flexible design also allows electrodes (recording electrodes F3, F4, C3, C4, T3, and T4 and Cz for common reference) to be positioned correctly simply by placing the pad on the patient's forehead (Fig. 1c). Cz is indicated by '0 V' in this system. A second advantage compared with traditional EEG equipment is that measurement data can be transmitted wirelessly via Bluetooth™, enabling EEGs to be measured in an ER/ICU without adding to the number of medical devices interacting with the

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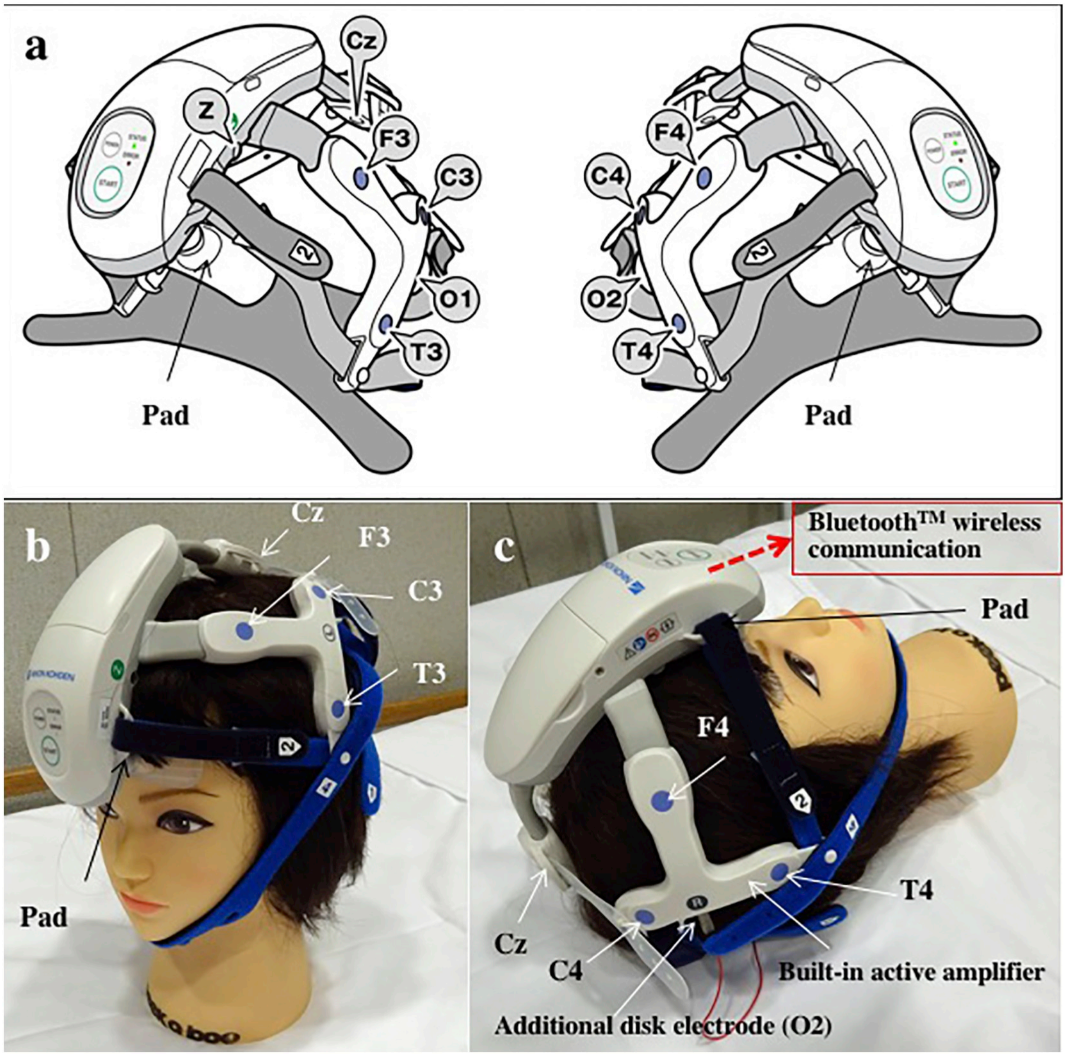

Fig. 1. (a) A telemetry electroencephalograpy (EEG) amplifier (AE-120A EEG Headset™: Headset). Seven electrode (F3, F4, C3, C4, T3, T3, Cz) positions are fixed on the Headset. There is no need to measure or mark the electrode positions. Apart from the Z electrode (ground), two more optional EEG disk electrodes such as O1 and O2 can be added. (b) The Headset is applied to a patient's head using disposable electrodes, which are attached beforehand. The disposable electrodes are prefilled with enough gel such that skin preparation is not required to obtain a low impedance. (c) The headset's flexible arms allow it to fit various head shapes. Also, the built-in active amplifier reduces mixed external noise. EEG measurement data can be transmitted wirelessly via Bluetooth™.

patient. Furthermore, the built-in active amplifier can reduce mixed external noise in the ER/ICU.

In October 2017, we introduced the Headset to EEG monitoring facilities in our ER/ICU. In this retrospective study, we present our initial experience with Headset-EEG monitoring and its diagnostic performance, as well as some limitations of the device.

2. Material and methods

Between October 2017 and July 2018, 484 patients were admitted to our department as neuroemergency cases. Most patients underwent computed tomographic (CT) scanning as an initial emergent neuroradiological examination. Nine patients underwent further EEG monitoring with the Headset (Headset-EEG) device. Among these patients, we retrospectively selected five (three men, two women, mean age of 66 years, range 25–91) who were initially diagnosed with NCSE using Headset-EEG by attending physicians.

Reevaluation of Headset-EEG was performed by two board certified electroencephalographers (T.M. and A.S.) who were blinded to all clinical data. No differences in the electroencephalographers' interpretations were noted on independent assessments (*kappa* = 1) [8]. Diagnoses based on reevaluation of Headset-EEG were compared to initial Headset-EEG diagnoses. Subsequent treatment and functional outcomes were also reviewed. Functional outcomes were assessed at discharge using a modified Rankin Scale (mRS) and compared with mRS values from before the onset of each patients' neuroemergent event. Informed consent was obtained from the patients (or for those unable to provide it, from their families).

3. Results

Detailed clinical courses and the initial and reevaluated Headset-EEG diagnoses of five patients are summarized in Table 1. Four patients (Cases 1, 2, 4, and 5) experienced acute traumatic intracranial lesions. Case 3 also had partial symptomatic epilepsy. In three patients (Cases 3, 4, and 5), generalized convulsive seizures preceding unconsciousness were observed and an initial diagnosis of subtle status epilepticus (SE), which is a persistent NCSE after withdrawal of prolonged convulsive seizures [3,4,9,10], was made.

Case 1 featured a left-lateralized acute subdural hematoma (Fig. 2a); Headset-EEG data revealed repeated ictal discharges, which originated from the left anterior quadrant (Fig. 2b, black lines) and

Table 1
Clinical profile and EEG findings of 5 patients of this study.

Case No	Age/ gender	Clinical findings	Clinical diagnosis	Initial Headset-EEG diagnosis	Performed treatment	Reevaluated Headset-EEG diagnosis	Outcome(mRS)
1	73/F	Unconsciousness (JCS200)	ASH, Lt	Repeated ictal discharges, Lt anterior quadrant → NCSE	DZP&fPTH iv, Burr-hole surgery, CBZ po	Repeated ictal discharges, Lt anterior quadrant→NCSE	Unchanged(4 → 4)
2	83/M	Restless confusion	Contusion Rt frontal, Traumatic SAH, CSHs, Bil	Frequent paroxysmal discharges, Lt frontal → NCSE	DZP&fPTH iv, Burr-hole surgery, LEV iv	Triphasic wave→Metabolic encephalopathy	Worse(renal failure)(4 → 5)
3	25/M	Impaired consciousness (JCS20) following generalized seizure	Symptomatic epilepsy with operated AVM, Rt frontal	Repeated ictal discharges, Lt temporal → Subtle SE	LCM po	Muscle artifact	Good(1 → 1)
4	91/F	Unconsciousness (JCS200) following generalized tonic seizure, predominantly on Rt	ASH, Lt, Old contusion Lt frontal	Repeated ictal discharges, Lt frontal → Subtle SE	DZP & fPTH iv	Artifact due to unstable electrode attachment	Worse(head trauma)(3 → 5)
5	58/M	Impaired consciousness (JCS20) following generalized tonic seizure	ASH, Rt	Frequent paroxysmal discharges, C3&C4 → Subtle SE	LEV iv & po	Artifact due to unstable electrode attachment	Good(1 → 1)

Abbreviations of this table: EEG, electroencephalography; mRS, modified Rankin Scale; M, male; F, female; JCS, Japan coma scale; Lt, left; Rt, right; ASH, acute subdural hematoma; SAH, subarachnoid hemorrhage; CSH, chronic subdural hematoma; NCSE, Nonconvulsive status epilepticus; SE, status epilepticus; DZP, diazepam; fPHT, fosphenytoin; LEV, levetiracetam; LCM, lacosamide; iv, intravenous; po, per os.

lasted for approximately 3–5 min. On the basis of an initial diagnosis using Headset-EEG, it was determined that the patient's unconsciousness was caused by NCSE rather than by the mass effect of the hematoma. Following intravenous administration of diazepam and fosphenytoin, partial evacuation of subdural clots via a less-invasive burr-hole rather than via craniotomy was performed. The patient recovered consciousness and returned to the nursing facility.

In Cases 2 and 3, initial Headset-EEG diagnoses were incorrectly made because of inaccurate interpretations of the EEG findings. In Case 2, the patient also had various metabolic disorders including diabetes mellitus, renal dysfunction due to diabetic nephropathy, and chronic alcoholism, as well as intracranial traumatic lesions (Fig. 3a); additionally, the patient developed restless confusion six days after experiencing head trauma. An initial Headset-EEG diagnosis of frequent paroxysmal discharges in the bilateral anterior quadrants was made. However, reevaluation with a Headset-EEG revealed triphasic waves caused by metabolic encephalopathy (Fig. 3b). At this time, the patient's renal function was also noted to be moderately impaired. Blood urea nitrogen, creatinine, and an estimated glomerular filtration ratio of 47 mg/dl (normal 8–20), 1.70 mg/dl (normal 0.65–1.07), and 30.54 ml/min/1.73 m^2, respectively, were noted. Due to renal dysfunction, aggressive treatment for NCSE could not be performed. At discharge, the patient's functional outcomes worsened because of deterioration of his metabolic function, despite of intensive medical treatment. In Case 3, the patient had symptomatic epilepsy associated with a resected arteriovenous malformation in the right frontal lobe (Fig. 3c) and showed electromyogenic discharges in the left temporal muscle via the Headset-EEG. These were incorrectly diagnosed as repeated ictal discharges (Fig. 3d, black lines). However, he regained his full consciousness after Headset-EEG recording and hence intravenous administration of anticonvulsants was not performed.

In Cases 4 and 5, various artifacts, caused by unstable electrode placement, were incorrectly attributed to ongoing seizure activity in the initial Headset-EEG diagnosis. The patient in Case 4, a 91-year-old female with a prior resolved frontal lobe contusion, developed an acute, left-lateralized subdural hematoma (Fig. 4a). During unconsciousness and after the generalized seizure, repetitive waveforms without quasi-rhythmic spatio-temporal evolution were recorded by electrode F3 in addition to the myogenic responses by electrode T3 (Fig. 4b). Large motion artifacts were mixed on the T3 and C3 electrodes (Fig. 4b, black arrows). Additionally, there was no potential for C4-F4 derivation in this case (Fig. 4b, asterisk). The patient's condition ultimately worsened because of the head trauma. Case 5 featured an acute, right-lateralized subdural hematoma (Fig. 4c). Repetitive waveforms on F3, T3, F4, and T4 were initially diagnosed with ictal discharges (Fig. 4d). No potentials were recorded on C3–0V, C4–0V, or F3-T3 derivations (Fig. 4d, asterisks), and the patient showed good outcomes.

4. Discussion

Despite the attempts to define the EEG characteristics of NCSE, a universally accepted definition has yet to be established [5]. However, it is generally accepted that repeated ictal discharges with quasi-rhythmic, spacio-temporal evolution are truly ictal in nature [4,11]. In Case 1, repeated ictal discharges, which originated from the anterior quadrant of the ipsilateral side of the patient's hematoma, were clearly recorded. On the basis of this patient's prompt diagnosis of NCSE using the Headset-EEG apparatus, subsequent appropriate therapy was administered, and good outcomes were obtained.

A limitation of this Headset apparatus, however, is that epileptic spread to the ipsilateral posterior quadrant is not identifiable, as the seven Headset electrode positions are located only on the anterior half of the head. Thus, when ictal discharges originate from, and are confined to, the posterior half of the head, the Headset-EEG is unable to detect NCSE. Critically, however, two optional EEG disk electrodes can be added to the Headset apparatus to address this problem (Fig. 1a). We

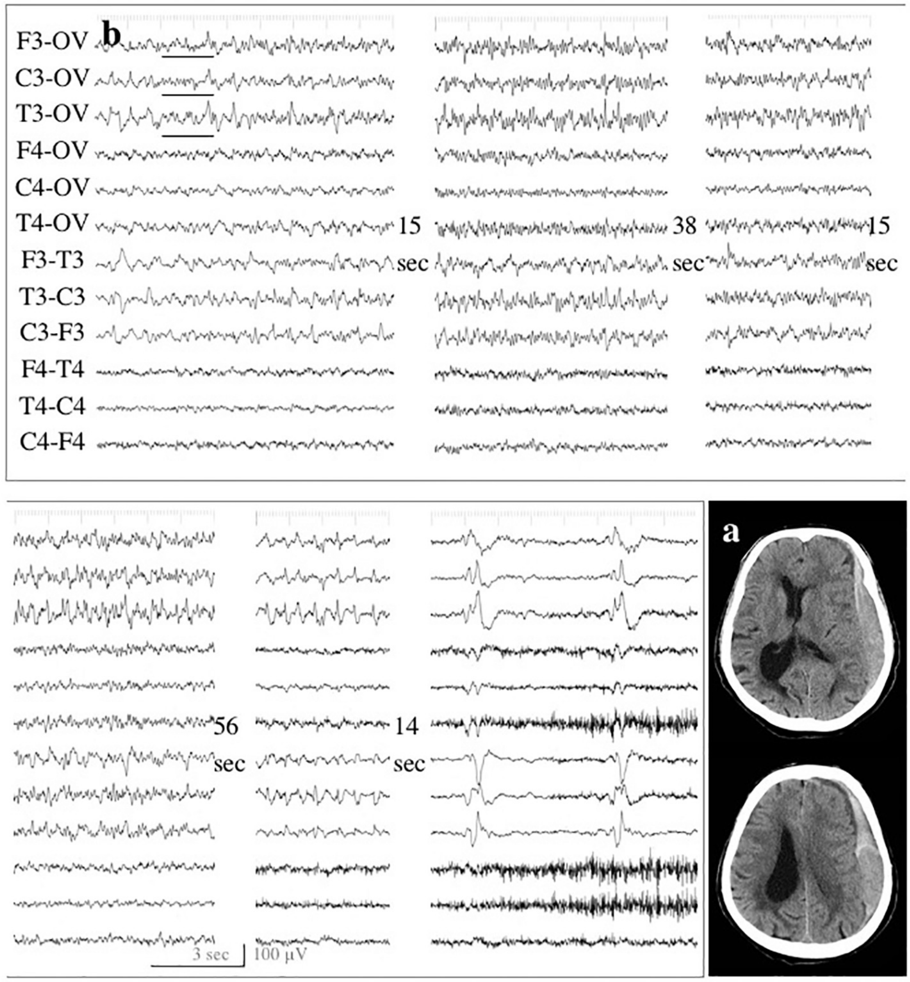

Fig. 2. (Case 1) (a) A computed tomographic (CT) scan revealed an acute subdural hematoma in the left hemisphere with a midline shift. (b) Headset-EEG results revealed ictal discharges that originated from the left anterior quadrant (black lines) and lasted for approximately 3 min. Spread of the ictal discharges to the left posterior quadrant was not demonstrated.

recommend placing these additional disk electrodes at positions O1 and O2 to monitor patient vigilance and detect ictal discharges in the posterior half of the head. A further structural limitation of the Headset-EEG is that Cz, the common EEG reference point for conventional monopolar recordings with A1 and A2 reference electrodes, is not recorded with this apparatus.

In Case 2, Headset-EEG results demonstrated triphasic waves caused by metabolic encephalopathy. The word "triphasic" has been used to describe a waveform characterized by an initial small negative component, followed by a large positive wave, and culminating in a slow negative wave. Because these findings were originally limited to patients with hepatic failure, triphasic wave encephalopathy was synonymous with hepatic encephalopathy [12–14]. Subsequently, triphasic wave encephalopathy has become associated with a wide range of metabolic abnormalities, such as renal dysfunction [12]. Furthermore, it may become generally accepted that triphasic waves are critical EEG features in patients with NCSE, although it is often difficult to distinguish epileptogenic triphasic waves from metabolic ones [2,15,16].

The presence of metabolic abnormalities is the most distinct, differentiating feature of NCSE triphasic waves and encephalopathy [2]. Renal function in Case 2 was moderately impaired, while liver dysfunction, including serum ammonia level dysregulation, was not evident. In NCSE, a triphasic wave assumes a particular epileptiform morphology with prominent and distinctive first phases exhibiting "spike and slow wave" complexes [2,17]. In Case 2, triphasic waves did not exhibit these complex morphologies. Also, the distinct asymmetrical and predominant focal location of triphasic waves associated with epileptogenic lesions is a characteristic EEG feature of NCSE. However, triphasic encephalopathy waves usually tend to be symmetric, anterior, and bilateral [2,15,16]. Additionally, to detect the distribution of the triphasic waves via Headset-EEG, additional disk electrodes may be placed on the bilateral occipital region.

In Case 3, electromyogenic discharges from the temporal muscle detected by the Headset-EEG were initially diagnosed as repeated ictal discharges. Frequencies of electromyogenic discharges of the temporal muscle are often higher than those of ictal discharges. In this case, electromyogenic discharges localized to the temporal region of the side contralateral to the epileptogenic lesion. To detect a myogenic response, it is critical to observe the patient's behaviors, such as swallowing.

In Cases 4 and 5, various artifacts, caused by unstable electrode placement, were initially diagnosed as ongoing seizure activity. In Case

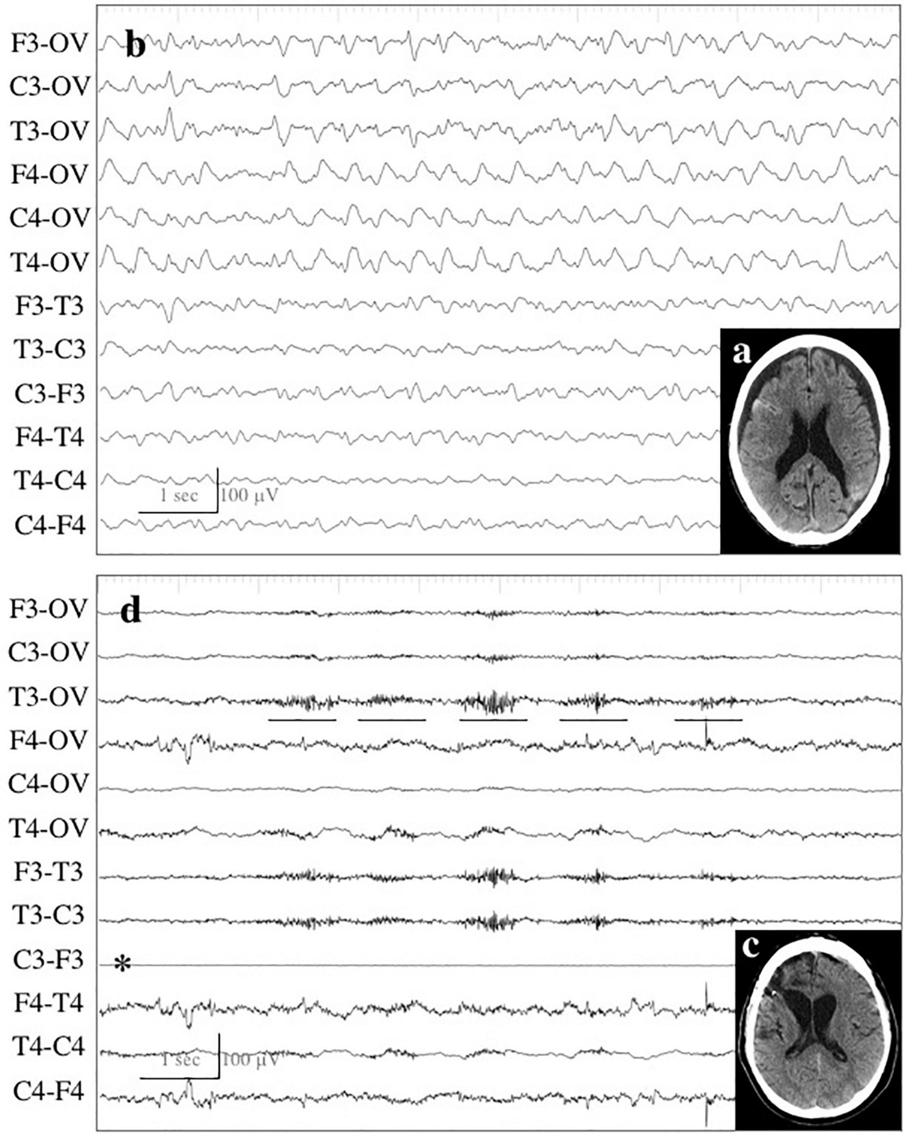

Fig. 3. (Case 2) (a) A CT scan revealed multiple contusional and subarachnoid hemorrhages with bilateral subdural effusions. (b) Headset-EEG results revealed triphasic waves in the bilateral anterior quadrants. (Case 3) (c) A surgical defect was observed in the right frontal lobe with a CT scan. (d) Electromyogenic discharges in the left temporal muscle were noted on T3 (black lines). There was no potential for C3-F3 derivation (asterisk).

4, two different types of high-frequency waveforms without spacio-temporal evolution were recorded only on F3 and T3. Critically, these waveforms did not affect the surrounding electrodes. Furthermore, with patient motion, large electrode artifacts were found to be mixed on these electrodes, as is typically observed on poorly or unstably placed electrodes. In Cases 3–5, there were no potentials on some of the derivations. This was attributed to an electrical short circuit between the two electrodes, likely caused by excessive hair wetness because of blood, sweat, and/or the conduction gel.

The present study indicated that the Headset-EEG is a possible new tool for NCSE screening in the ER/ICU, as was clearly demonstrated in Case 1. However, considerable knowledge on the "art" of EEG interpretation is required for the correct diagnosis of NCSE [5], as was demonstrated by Cases 2–5. When NCSE is suspected via Headset-EEG data, a routine EEG with a full montage of 10–20 EEG electrodes should be performed; additionally, results should be examined by an epileptologist and/or electroencephalographer, even following initial treatment. According to recent reports on magnetic resonance perfusion imaging with arterial spin labeling (ASL) reveals ictal hyperperfusion in the epileptically activated cortex during NCSE [4,18,19], and ASL and Headset-EEG in combination may be used for the precise diagnosis of NCSE.

5. Conclusion

In conclusion, Headset-EEG is a possible NCSE screening tool. However, to establish an accurate diagnosis of NCSE, considerable knowledge of EEG interpretation is needed. If NCSE is suspected after using Headset-EEG, a subsequent routine EEG with 10–20 EEG electrodes should be performed in consultation with an epileptologist or electroencephalographer.

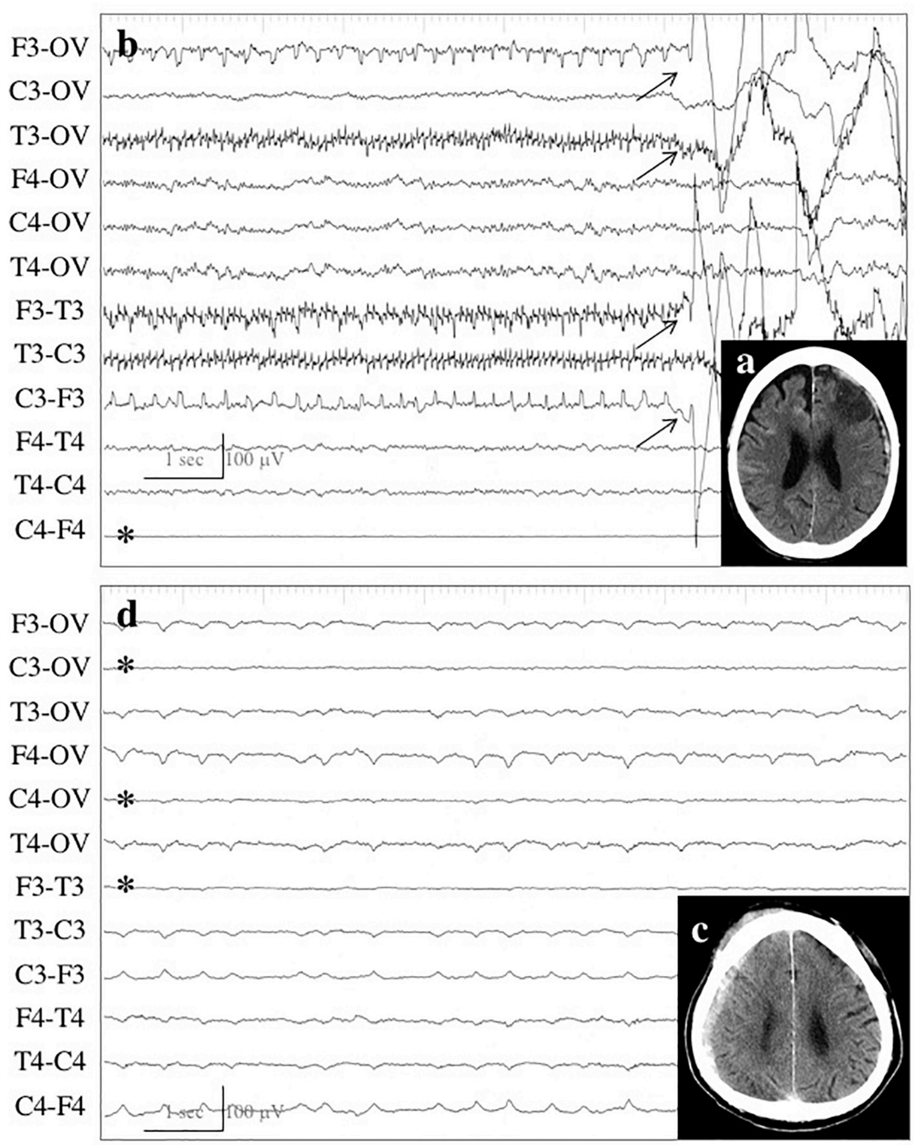

Fig. 4. (Case 4) (a) A CT scan revealed an old contusion in the left frontal lobe and an acute subdural hematoma also on the left. (b) Headset-EEG demonstrated two types of artifacts, caused by unstable electrode placement on F3 and T3: repetitive waveforms on F3-0 V and C3-F3 derivations and on T3-0 V, F3-T3, and T3-C3. With motion, large artifacts were mixed (back arrows). No potential was noted for C4-F4 derivation (asterisk). (Case 5) (c) A CT scan revealed an acute subdural hematoma on the right. (d) Headset-EEG revealed repetitive waveforms on F3, T3, F4, and T4, with no potential for C3–0V, C4–0V, and F3-T3 derivations (asterisks).

Declarations of Competing Interest

None.

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