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Neurogenic pulmonary edema following seizures: A retrospective computed tomography study

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ABSTRACT

Introduction: Data on the frequency and clinical relevance of neurogenic pulmonary edema (NPE) following epileptic seizures are limited. The aim of the present study was to analyze computed tomography (CT) examinations in patients with previous seizures.
Method: Incidence of NPE and related clinical factors were retrospectively assessed in patients admitted because of epileptic seizures who underwent thoracic CT imaging as part of emergency diagnostics.
Results: Between January 2010 and January 2016, we included all patients admitted with the International Classification of Diseases (ICD) diagnosis code of epileptic seizure or epilepsy and who underwent CT imaging, including visualization of the lungs, as part of emergency diagnostics.
Of the 47 included patients, 26 patients had suffered from generalized convulsive seizures (GCS), 17 patients had focal seizures with impaired and 4 without impaired consciousness. Signs of NPE were present in 5 out of 47 patients; all 5 patients had GCS prior to thoracic CT scan (i.e., 19% of patients with GCS). In four out of five cases, a single seizure was described; in one case, the seizure was only partially witnessed, but the indirect clinical signs strongly suggested a GCS.
Related factors such as the initial respiratory rate or the initial pCO2 value were not significantly different in patients with and without signs of NPE.
Conclusions: The highly selected and biased patient group warrants caution in the interpretation of the study results. Our data, however, confirm that signs of NPE appear to be rather frequent in patients with GCS. Its clinical significance as regards morbidity and sudden death in epilepsy is discussed.

1. Introduction

Generalized convulsive seizures (GCS) are frequently associated with transient alterations of various physiological functions such as respiratory failure, tachycardia, and metabolic acidosis. In most cases, the changes spontaneously resolve without permanent damage. In some cases, however, GCS are linked to sustained central apnea, secondary bradycardia, and asystole in the early postictal period, ultimately leading to sudden unexpected death in epilepsy (SUDEP) [1].

In postmortem studies of patients with SUDEP, signs of neurogenic pulmonary edema (NPE) were detected in 62% of the cases [2]. Neurogenic pulmonary edema is a type of acute pulmonary edema that occurs in association with a significant illness of the central nervous system (CNS) in the absence of primary pulmonary or cardiovascular injury. A variety of CNS disorders can trigger NPE including subarachnoid hemorrhage, traumatic brain injury, spinal cord injury, and intracranial hemorrhage.

Importantly, NPE has also been described following epileptic seizures [3]. Whether NPE is a surrogate marker of a previous GCS or involved in the pathophysiology of SUDEP remains unclear. Data on the frequency of NPE following seizures and its clinical relevance are scarce. Previous case reports [4,5] and case series studying postictal chest radiographs (CXR) [6–8] provided inconsistent results, maybe due to technical limitations of conventional X-ray diagnostics.

The aim of the present retrospective study was to assess occurrence and related clinical factors of NPE in patients with previous epileptic seizure who underwent computed tomography (CT) examinations.

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2. Materials and methods

2.1. Study design and setting

We conducted a retrospective analysis of patients who were admitted to a single tertiary care medical center (University Hospital RWTH Aachen, Aachen, Germany) with the International Classification of Diseases (ICD) diagnosis code of epileptic seizure or epilepsy and who underwent CT imaging, including visualization of the lungs, as part of emergency diagnostics. Data were collected from January 2010 to January 2016. Our study was approved by the ethics committee of University Hospital RWTH Aachen.

2.2. Study group

We enrolled all adult patients (≥18 years of age) who were (1) discharged from the Department of Neurology with the ICD diagnosis code of epileptic seizure or epilepsy and (2) who had received cranial CT imaging with CT angiography (CTA) of the supra-aortic vessels in the context of emergency diagnostics within the first 24 h after admission. These patients were initially suspected to have an ischemic stroke or CNS processes leading to acute neurological symptoms. However, diagnostics eventually revealed an epileptic seizure as the cause leading to emergency admission. The primary aim of CT imaging was the detection of cerebrovascular processes. Since our local CT protocol includes heart and thoracic imaging, we were able to screen for pulmonary pathologies. The final discharge diagnosis of included patients was GCS or focal seizure. The diagnosis was established on medical history and diagnostic tests, mainly neuroimaging or electroencephalography (EEG).

2.3. Procedures

2.3.1. Computed tomography (CT)

Computed tomography angiography examinations were performed on a 40-slice spiral CT scanner (Siemens SOMATOM® Definition AS, Siemens Healthcare, Forchheim, Germany). Technical parameters were as follows: Collimation 40 × 0.6 mm with reconstruction in 1 mm slices in an intermediate window as well as multiplanar reformations in the axial, coronal, and sagittal plane (3 mm slice thickness and 1.5 mm overlap). We used 80 ml Ultravist 300 and Saline 40 ml each with a flow of 5.0 ml/s as contrast medium.

The CTA was used to detect vascular stenosis or occlusions, vascular malformations, or cardiac sources of embolism and thus included partial imaging of the lungs. Consequently, a secondary assessment of the lungs was possible.

In the present study, the thoracic CT images of the included patients were retrospectively reexamined with the specific aim of detecting signs of pulmonary edema. In order to be included in the study group, patients' available imaging was required to include at least the beginning of the two lower pulmonary arteries, including the hili with the main pulmonary arteries. This specification was based on the fact that noncardiogenic pulmonary edema (including NPE) occurs centrally along the arteries and respects the subpleural space. In contrast, cardiogenic pulmonary edema develops along the pulmonary veins, towards the interlobular septa and distributes peripherally to the pleura [9]. There is no specific pattern for NPE in CT imaging. This entity belongs to the group of noncardiogenic pulmonary edemas, also known as increased-permeability pulmonary edemas, and is centrally located. Peripheral cardiogenic pulmonary edema, also known as hydrostatic or hemodynamic edema, presents a distribution from the central region towards the pleura and is usually associated with an enlarged heart and increased vascular pedicle width [9–12]. In addition, pulmonary edema can be classified as interstitial, alveolar, and atypical.

The radiological study investigator (Y.M.) had several years of experience in lung CT diagnostic.

2.4. Definition of outcome measures

The primary endpoint was the incidence of NPE in the study group. Neurogenic pulmonary edema is defined as an acute pulmonary edema occurring shortly after a central neurologic illness. In the present study, all pulmonary edema detected in CT, for which no other explanation could be found in the documentation and which corresponded CT-graphically to noncardiac pulmonary edema, were considered neurogenic.

As secondary endpoints, various clinical factors associated with the presence of NPE were considered. These included respiratory rate, oxygen saturation, and the need for oxygen supply or invasive ventilation therapy documented initially by the emergency physician as well as the measured respiratory rate in the emergency department (ED), the initial pCO2 value in blood gas analysis (BGA) in the ED, and the need for intensive care monitoring.

2.5. Statistical analysis

Statistical analysis was performed using SPSS (Statistic Package for Social Sciences) 24 Software (SPSS Inc. Chicago, IL, USA).

Continuous variables such as respiratory rate and oxygen saturation were compared using the t-test. We compared categorical variables (intensive care monitoring, oxygen supply, intubation) using Fisher's exact test.

3. Results

In total, 151 adult patients (≥18 years of age) were retrieved with an ICD diagnosis code of epileptic seizure or epilepsy and CT imaging in emergency diagnostics. Some patients received CT imaging twice or three times after epileptic seizures during the study period, resulting in a total of 174 analyzed instances of CT imaging. Altogether, 47 patients were included in the study. In 120 of the remaining instances of CT imaging, adequate imaging of the lungs in CT examination was not available. In 7 patients with sufficient CT imaging, the existing documentation provided insufficient certainty that an epileptic seizure had indeed occurred. These patients were also excluded from further evaluation (Fig. 1).

Twenty-six of the 47 included patients had suffered GCS (1 generalized onset tonic–clonic seizure (GTCS), 19 focal to bilateral tonic–clonic seizures, and 7 GCS with unknown onset). Of the 7 generalized tonic–clonic seizures with unknown onset, three patients had acute symptomatic seizures, and in four patients, the etiology was unclear. The patient with GTCS had a genetic cause. Nineteen focal to bilateral tonic–clonic seizures all had a structural etiology. Of the patients with GCS, 4 patients suffered status epilepticus, and 5 patients had recurrent GCS. Focal seizures with impaired consciousness were reported in 17 patients, including 6 patients with status epilepticus. Four patients had focal seizures without impaired consciousness, one of which developed into status epilepticus. Overall, the proportion of patients with status epilepticus was 23.4%. The etiologies of focal seizures were structural in 19 patients and unclear in one patient. In addition, another patient had an acute symptomatic seizure.

The cardiopulmonary comorbidities present in our study population were heart failure in 7 patients, atrial fibrillation in 14 patients, coronary heart disease (CHD) in 10 patients, and chronic obstructive pulmonary disease (COPD) in 6 patients. Two patients had previous pulmonary artery emboli, and one patient had pulmonary fibrosis.

Forty patients were discharged from hospital after acute treatment. Of the patients with GCS, 22 of 26 were discharged from the hospital, including all 5 patients with NPE. In 6 patients, long-term intensive care was required. Four of the patients had a GCS without an NPE. The remaining two patients had complex partial seizures.

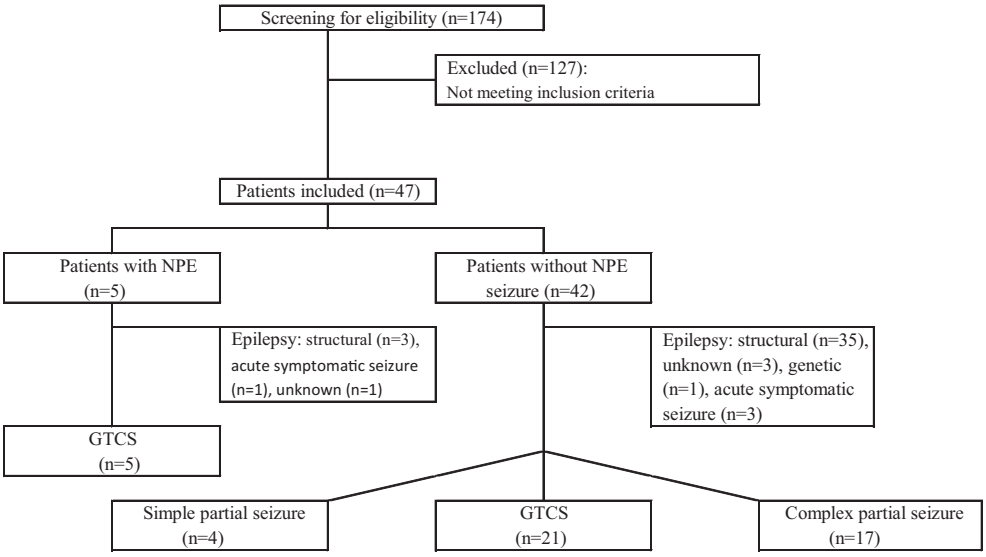

Fig. 1. Study flow chart of patient selection.

Inpatient treatment with rehabilitation was necessary in 6 patients. One patient was in need of further inpatient psychiatric treatment because of drug abuse. None of the patients died during the hospital stay.

The cranial CT imaging with CTA was performed because patients were suspected of having a cerebral stroke or CNS process. With regard to seizure-associated focal neurological deficits, 16 patients showed clinical signs of a right hemispheric process, compatible with middle cerebral artery syndrome in 2 cases; and 18 patients presented signs of left hemispheric process, of which 10 displayed symptoms consistent with middle cerebral artery syndrome. The remaining 13 patients manifested clinical symptoms that were not confined to a specific brain region.

When comparing for age and sex, there were no significant differences between the groups of patients with pulmonary edema and those without pulmonary edema (Table 1).

3.1. Primary outcome

Neurogenic pulmonary edema was detected in CT imaging in 5 of 47 cases (10.6%). All 5 patients had a GCS before admission. Four patients had witnessed GCS. In the case of one patient, the seizure was only partially witnessed, but the indirect clinical signs (postictal phase, tongue biting, enuresis, hyperlacticemia) strongly suggested a GCS.

The underlying causes of the documented seizures with NPE varied. Symptomatic epilepsy was present in 3 cases. One patient had a hyperglycemic-induced seizure. In one case, the cause of the seizure remained unknown, because the diagnostic work-up was incomplete because of the patient's voluntary early discharge (Table 2).

Overall, 19.2% of patients with GCS (5/26) had an NPE whereas no patients in the groups with focal seizures showed signs of NPE.

Analysis of the CT examinations showed variable findings in the individual patients with NPE:

Patients 1 and 2 showed mild interstitial pulmonary edema in the form of a thickening of the bronchial walls with a reticular pattern. In patient 3, in addition to mild interstitial pulmonary edema, there was a mild interlobar effusion on the right. Computed tomography examination in patient 4 revealed pronounced alveolar pulmonary edema in the lower lobes with interstitial pulmonary edema in the upper lobes (Fig. 2). Patient 5 also had alveolar pulmonary edema. This was mainly located in the upper lobes and only slightly pronounced (Fig. 3).

3.2. Secondary outcome

As secondary endpoints, we compared clinical parameters of the two groups (Group 1: patients with NPE vs. Group 2: patients without NPE).

Table 1
Comparison of patients with NPE with all patients without NPE and the subgroup patients with GCS without NPE.

	Group 1: seizure patients with NPE (n = 5)	Group 2: all seizure patients without NPE (n = 42)	p-Value Group 1 vs. Group 2	Group 3: patients with GCS without NPE (n = 21)	p-Value Group 1 vs. Group 3
Male sex (%)	2/5 (40%)	16/42 (38.1%)	1.0	6/21 (28.57%)	0.628
Age (years) (mean ± SD, range)	68.8 ± 13.32, 52–88 (n = 5)	72.33 ± 14.95, 27–92 (n = 42)	0.617	70.76 ± 17.84, 27–90 (n = 21)	0.82
Initial respiratory rate per minute (mean ± SD, range)	11.25 ± 4.27, 5–14 (n = 4)	14.86 ± 4.53, 8–29 (n = 22)	0.152	13.58 ± 2.75, 8–18 (n = 12)	0.218
Initial oxygen saturation (%) (mean ± SD, range)	86.4 ± 15.11, 60–98 (n = 5)	93.53 ± 5.63, 75–100 (n = 32)	0.353	93.44 ± 4.43, 82–99 (n = 16)	0.359
Respiratory rate per minute in ED (mean ± SD, range)	21.0 ± 2.58, 18–24 (n = 4)	18.4 ± 3.56, 13–27 (n = 20)	0.182	18.30 ± 3.83, 13–27 (n = 10)	0.224
pCO2 in ED (mm HG) (mean ± SD, range)	48.53 ± 12.42, 32.2–58.4 (n = 4)	45.86 ± 8.63, 30.3–66.3 (n = 24)	0.59	43.58 ± 8.36, 30.3–59.7 (n = 9)	0.411
Need for oxygen supply	5/5 (100%)	21/34 (61.8%)	0.1488	12/17 (70.59%)	0.289
Intubation due to respiratory failure	1/5 (20%)	1/42 (2.4%)	0.2035	1/21 (4.76%)	0.354
Need for intensive care monitoring	2/5 (40%)	29/42 (69.1%)	0.302	15/21 (71.43%)	0.302

SD = Standard Deviation.

Table 2
Individual patient characteristics of the group with NPE.

	Age	Sex	Epilepsy type	Seizure type	Epilepsy etiology	Pulmonary edema	Initial respiratory rate per minute	Initial oxygen saturation (%)	Respiratory rate per minute in ED	pCO2 (mm Hg) in ED	Need for oxygen supply	Intubation due to respiratory failure	Need for intensive care monitoring	Cardiopulmonary diseases
Patient 1	88	Female	Focal	GCS	Structural	Interstitial	12	92	24	58.0	Yes	No	Yes	CHD, COPD
Patient 2	52	Male	Focal	GCS	Structural	Interstitial	Unknown	89	20	58.4	Yes	No	No	No
Patient 3	62	Female	Focal	GCS	Structural	Interstitial	5	60	Intubated	Intubated	Yes	Yes	Yes	CHD
Patient 4	70	Female	Generalized	GCS	Acute symptomatic	Alveolar/ interstitial	14	93	18	57.9	Yes	No	No	No
Patient 5	72	Male	Unknown	GCS	Unknown	Alveolar	14	98	22	45.5	Yes	No	No	Unknown

Fig. 2. Computed tomography image of patient 4 showing mild interstitial (arrowheads) and alveolar (arrows) edema without clinical or laboratory signs of pneumonia.

Because NPE was only seen in patients with GCS, a further subgroup analysis including patients with GCS with NPE (Group 1) or without NPE (Group 3) was performed. In some cases, however, the documentation was incomplete (Table 1).

When comparing all patients with or without NPE, the initially documented respiratory rate was not significantly different in the t-test between the two groups ($t[24.0] = 1.487$, $p = 0.152$). In the comparison of the initially measured oxygen saturation ($t[4.175] = 1.044$, $p = 0.353$) and the frequency of an oxygen supply ($p = 0.1488$), there were also no significant differences. Altogether, 10 patients were endotracheally intubated, though this was due to respiratory failure in only two patients, one of which displayed NPE in CT imaging. We determined the N-terminal pro Brain Natriuretic Peptide (NT-pro-BNP) value only in this patient. Though it was initially normal, there was a significant increase during the hospitalization.

The cause of intubation in the remaining cases was either altered consciousness or a persistent status epilepticus. Nevertheless, when comparing the two groups, the difference was not significant ($p = 0.2035$). The t-test revealed no significant differences among groups in the documented respiratory rate in the emergency room ($t[22.0] = 1.379$, $p = 0.182$) as well as the pCO2 in the first venous BGA ($t[26.0] = 0.54$, $p = 0.594$).

Intubated patients were excluded from the analysis of respiratory rate and pCO2 values in the ED.

Intensive care monitoring was necessary in 29 patients without pulmonary edema and in 2 patients with pulmonary edema. There were no statistically significant differences between both groups ($p = 0.302$).

As a possible competing cause of cardiopulmonary decompensation, one patient had a previous diagnosis of COPD, and at least two patients were known to have CHD.

When comparing only the patients with GCS with or without NPE, we found similar results (Table 1): In the t-test, there were no significant differences between the initial respiratory rate ($t[14.0] = 1.289$, $p = 0.218$) and the initially measured oxygen saturation ($t[4.23] = 1.028$, $p = 0.359$). The respiratory rate in the emergency room ($t[12] = 1.282$, $p = 0.224$) as well as the pCO2 in the first venous BGA ($t[11] = 0.854$, $p = 0.411$) also did not differ relevantly. There were also no significant differences regarding the need for initial oxygen substitution supply ($p = 0.289$), intubation ($p = 0.354$), or intensive care monitoring ($p = 0.302$).

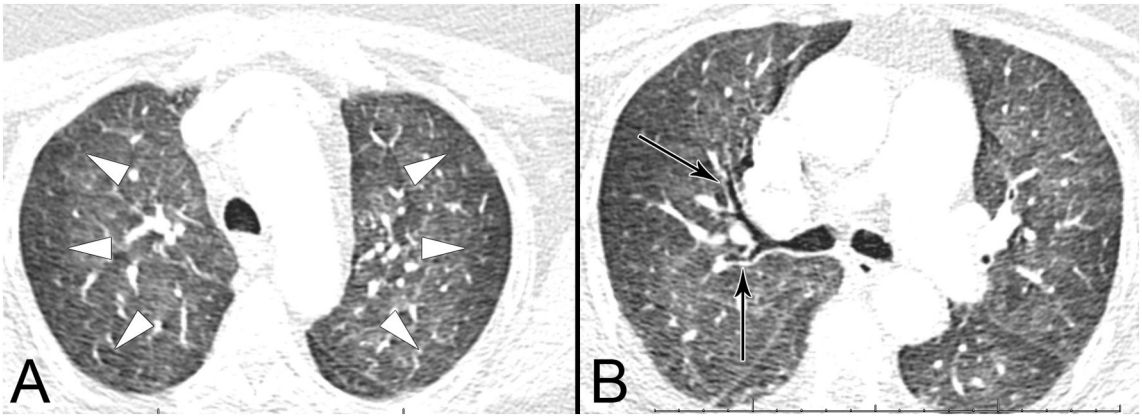

Fig. 3. Computed tomography images of patient 5 showing areas of ground glass opacity (pulmonary alveolar edema), which respect the subpleural space (A: arrowheads). There is also thickening of bronchial walls (B: arrows).

4. Discussion

In the present study, signs of NPE were detected in approximately 19% of patients admitted to an ED due to GCS but not in patients with other seizure types. Neurogenic pulmonary edema is defined as an acute pulmonary edema occurring shortly after a central neurologic illness probably due to an increased vascular permeability in the pulmonary capillaries triggered by inflammatory processes with resulting edema [13] and/or an increased adrenaline release after brain damage, resulting in pulmonary vasoconstriction and increased vascular permeability [14].

Neurogenic pulmonary edema has been reported as a consequence of many acute neurological disorders including subarachnoid hemorrhage, intracranial hemorrhage, traumatic brain injury, or epileptic seizures [15]. Data on incidence and clinical relevance of NPE or radiological abnormalities following epileptic seizures are scarce. Darnell and Jay found NPE in only one of 45 patients in a retrospective analysis of patients who had received CXR in the ED following a GCS [6]. Similar results were reported in a study evaluating postconvulsive CXR in patients with electroconvulsive therapy [7]. Only one in 12 patients had subclinical radiographic signs of NPE. Kennedy et al. found abnormalities in CXR in 11 out of 24 patients in a study of patients who had a GCS in an epilepsy monitoring unit [8]. In 7 of the 11 cases, the finding was consistent with pulmonary edema, with no significant respiratory dysfunction compared with patients with no changes in CXR. Interestingly, however, the duration of seizures correlated with the onset of pulmonary edema. In the present study, the duration of seizures could not be determined because they were preclinical. However, there was an above-average proportion of status epilepticus, and thus prolonged seizure events, in the study group affecting 23.4% of patients. However, status epilepticus or recurrent seizures were not reported in any of the five cases, suggesting that the presence of pulmonary edema was unlikely to correlate with seizure duration in the present group. It should be noted that not only patients with GCS but also patients with simple and complex partial seizures were included in the present study.

Regarding the frequency of NPE after GCS, the results suggest that this is a frequent complication. By comparison, the incidence of NPE in partial seizures seems to be rare. The results of the present study are more informative because of better imaging method used (CT versus CXR).

The role of NPE in the pathophysiology of SUDEP is unclear. A postmortem study found signs of pulmonary edema in 52 of 74 of SUDEP cases [2]. In a study comparing the occurrence of NPE in baboons, pulmonary edema was detected in all cases of sudden death in epilepsy, and in only 12% of the control group of baboons without epileptic seizures [16]. However, whether NPE is a facilitating factor or simply a surrogate marker of a severe GCS remains unresolved. In our study, there were no significant differences in the comparison of clinical parameters in patients with GCS with and without signs of NPE. However, the retrospective design of the study and the partly incomplete data must be taken into account. At least one patient with NPE had such a severe respiratory insufficiency that endotracheal intubation was necessary. In this case, a clinically relevant manifestation has to be assumed, and it should be mentioned that the patient had CHD as a comorbidity.

4.1. Limitations of the study design

Data on radiographic signs of NPE are scarce. We are aware of the limitations of the highly selected and biased patient group included in this retrospective and monocentric study. Of course, a serial prospective collection of patients after GCS recorded in a video-EEG monitoring unit would be desirable to investigate true occurrence and its cofactors. In view of the radiation exposition, however, a prospective investigation does not appear justified.

Another limitation is that the diagnosis of seizure was based on clinical symptoms, the medical history of the patient, and the additional examinations performed in the hospital, therefore, the possibility of an incorrect diagnosis remains.

Furthermore, in the case of proven pulmonary edema, we cannot exclude the possibility that it was present before the epileptic seizure, or that it was caused by competing diseases.

4.2. Conclusion

The highly selected and biased patient group warrants caution in the interpretation of the study results. Our data, however, confirm that signs of NPE appear to be rather frequent in patients with GCS. In the study group, approximately 19% of the patients admitted because of GCS showed postictal edema in CT imaging.

Prospective studies with larger case sample sizes are necessary in order to draw more firm conclusions for clinical evaluation.

Potential conflicts of interest

Rainer Surges has received fees as speaker or consultant from Bial, Cyberonics, Desitin, Eisai, LivaNova, Novartis, and UCB Pharma. The other authors declare that they have no potential conflict of interest.

References

[1] Surges R, Sander JW. Sudden unexpected death in epilepsy: mechanisms, prevalence, and prevention. Curr Opin Neurol 2012;25:201–7.
[2] Zhuo L, Zhang Y, Zielke HR, Levine B, Zhang X, Chang L, et al. Sudden unexpected death in epilepsy: evaluation of forensic autopsy cases. Forensic Sci Int 2012;223:171–5.

[3] Sacher DC, Yoo EJ. Recurrent acute neurogenic pulmonary edema after uncontrolled seizures. Case Rep Pulmonol 2018;2018:3483282.
[4] Brambrink AM, Tzanova I. Neurogenic pulmonary oedema after generalized epileptic seizure. Eur J Emerg Med 1998;5:59–66.
[5] Swallow RA, Hillier CE, Smith PE. Sudden unexplained death in epilepsy (SUDEP) following previous seizure-related pulmonary oedema: case report and review of possible preventative treatment. Seizure 2002;11:446–8.
[6] Darnell JC, Jay SJ. Recurrent postictal pulmonary edema: a case report and review of the literature. Epilepsia 1982;23:71–83.
[7] Wayne SL, O'Donovan CA, McCall WV, Link K. Postictal neurogenic pulmonary edema: experience from an ECT model. Convuls Ther 1997;13:181–4.
[8] Kennedy JD, Hardin KA, Parikh P, Li CS, Seyal M. Pulmonary edema following generalized tonic clonic seizures is directly associated with seizure duration. Seizure 2015; 27:19–24.
[9] Burgener FA, Herzog C, Meyers S, Zaunbauer W. Differenzialdiagnosen in der Computertomografie. Stuttgart: Georg Thieme Verlag KG; 2013; 635–9.
[10] Brant WE, Helms CA. Fundamentals of diagnostic radiology. 3rd ed., vol. II Philadelphia: Lippincott Williams & Wilkins; 2007; 417–22.
[11] Ware LB, Matthay MA. Clinical practice. Acute pulmonary edema. N Engl J Med 2005;353:2788–96.
[12] Christe A, Vock P. Radiologic criteria to differentiate pulmonary edema. Ther Umsch 2004;61:665–70.
[13] Romero Osorio OM, Abaunza Camacho JF, Sandoval Briceño D. Postictal neurogenic pulmonary edema: case report and brief literature review. Epilepsy Behav Case Rep 2018;9:49–50.
[14] Zhao H, Lin G, Shi M, Gao J, Wang Y, Wang H, et al. The mechanism of neurogenic pulmonary edema in epilepsy. J Physiol Sci 2014;64:65–72 Jan.
[15] Baumann A, Audibert G, McDonnell J, Mertes PM. Neurogenic pulmonary edema. Acta Anaesthesiol Scand 2007;51:447–55.
[16] Szabo CA, Knape KD, Leland MM, Feldman J, McCoy KJ, Hubbard GB, et al. Mortality in captive baboons with seizures: a new model for SUDEP? Epilepsia 2009;50: 1995–8.