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The Spectrum of Non-Convulsive Status Epilepticus in Patients with Cancer

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Abstract

Purpose—Determine incidence, clinical presentation, electrographic correlates, and outcome of NCSE in cancer patients on whom an EEG was performed.

Methods—Retrospective review of 947 EEG reports on 658 patients in whom any type of EEG was performed at Memorial Sloan-Kettering Cancer Center (July 2006 – March 2008). Using the Epilepsy Research Foundation criteria, patients were classified as definite or probable NCSE. Medical records were reviewed for diagnosis, causes of NCSE, response to treatment, and outcome. Mortality was determined for patients with NCSE.

Results—Twenty-six episodes of NCSE were identified in 25 patients (25/658, 4%). Eleven had a primary brain tumor, 12 systemic cancer and two both. At diagnostic EEG, 18 were awake, 3 lethargic, and 5 comatose. EEG revealed a seizure in 62%, PLEDs in 42%, PEDs in 7.7%. Neuroimaging revealed new intracranial pathology in 54%. Seventy-seven percent achieved control; 65% required ≥3 AEDs, and 33% required intubation. Three patients died from NCSE.

Discussion—In our cohort, awake NCSE was more common than comatose NCSE. Treatment was successful in patients with heterogeneous CNS disease. EEG evaluation should be considered in patients with cancer as NCSE is treatable despite a high prevalence of structural brain disease. NCSE control did not always require intubation and burst suppression, but frequently required three or more AEDs.

Keywords

Nonconvulsive status epilepticus; periodic epileptiform discharges; electroencephalogram; cancer; status epilepticus; seizure

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INTRODUCTION

Nonconvulsive status epilepticus (NCSE) is recognized increasingly as a cause of coma or altered mental status and accounts for 25% of all cases of status epilepticus. (Treiman et al., 1998; Privitera et al., 1994) Approximately 55,000 deaths per year in the United States are associated with status epilepticus. (Treiman et al., 1998; Towne et al., 1994; DeLorenzo et al., 1995) In patients with altered mental status, 6% to 33% have NCSE and it can be detected in 8% of all comatose ICU patients without signs of seizure activity. (Towne et al., 1994; Towne et al., 2000; Hosain et al., 2005; Cocito et al., 2001) Prompt diagnosis and treatment is crucial for this treatable but potentially life threatening neurologic emergency which in an ICU setting has a mortality of 57% in some series. (Young et al., 1996; Litt et al., 1998)

Encephalopathy is common in patients with cancer and is usually due to metabolic dysfunction or structural brain disease.(Tuma and DeAngelis, 2000) We sought to identify the frequency of NCSE as a cause of impaired mental status and to ascertain the risk factors for NCSE, its treatment, and clinical outcome in a cancer population who were evaluated with EEG.

METHODS

We reviewed all EEG and long term monitoring (LTM) reports at Memorial Sloan-Kettering Cancer Center (MSKCC) from July 1, 2006 to March 31, 2008 to identify potential patients with NCSE. We then reviewed the patients' medical records to determine who had NCSE, its cause, and response to treatment defined as control of NCSE. We collected information on the patients' history of prior seizures, antiepileptic drugs (AED) and AED levels. We reviewed all available neuroimaging studies. The Epilepsy Research Foundation (ERF) developed a consensus definition that incorporates both EEG and clinical criteria to standardize the diagnosis.(Walker et al., 2005) Using the ERF criteria for NCSE, we classified the patients as either definite or probable NCSE. PEDs, PLEDs and other periodic patterns were classified as NCSE retrospectively if treatment resulted in normalization of EEG with a clinical improvement. All EEGs were performed for a minimum of 20 minutes, using the standard 10–20 system of electrode placement. Reports of all EEGs or LTMs performed after the initial diagnosis of NCSE were also reviewed. All EEG and LTM studies of patients with NCSE were re-reviewed by one of the authors (EA). We determined survival based on the date of NCSE and subsequent follow-up. This retrospective study was approved by the MSKCC Institutional Review Board.

RESULTS

Clinical Features

A total of 947 EEG/LTM procedures were performed on 658 patients during the study period (Table 1). There were 26 episodes (18 definite and 8 probable) of NCSE in 25 patients comprising 2.7% of the total EEGs, and 4% of the total patient cohort. Patients classified as probable NCSE all had clinical seizures leading up to diagnosis and improvement in clinical symptoms and EEG after treatment with AEDs. Most episodes of

NCSE (69%) occurred in patients who had an awake mental status with associated confusion, agitation or new neurologic deficit at the time of the EEG; 12% were lethargic and 19% were comatose. Of the 69 EEGs on comatose patients, 5 (7.3%) had NCSE, and of the 13 EEGs on lethargic patients, 3 (23%) had NCSE. Lethargy or coma was more common in the NCSE cohort (8/26, 31%) than the group as a whole (82/947, 9%).

The 25 patients with NCSE included 10 men and 15 women, with a median age of 59 years (Table 1). Twenty-two (88%) patients had an intracranial tumor; 13 had a primary brain tumor and 9 had brain metastases. Of the 13 patients with primary brain tumors, eight (32%) had a glioblastoma (GBM) and one each (4%) an anaplastic astrocytoma, low grade oligodendroglioma, primary central nervous system lymphoma (PCNSL), meningioma, and medulloblastoma. There were 14 patients with systemic cancer, two of whom also had a GBM. Four patients (16%) had lung cancer, three (12%) breast cancer, and one each (4%) had T-cell lymphoma, gastric cancer, colon cancer, ovarian cancer, retinoblastoma, renal carcinoma, and squamous cell carcinoma of the vocal cords.

The clinical presentation of patients with NCSE included confusion (50%), aphasia (31%), face or hand twitching (38%) and myoclonus (8%) (Tables 1 and 2). Sixteen (62%) patients had a prior lifetime history of seizures with 10 having had a seizure (generalized tonic-clonic seizure or complex partial seizure) immediately prior to the diagnosis of NCSE. Of these 16 patients, only one had a subtherapeutic AED level (for those drugs with established therapeutic levels) at the time of the diagnostic EEG. In the 9 patients without a prior lifetime seizure, NCSE was their first seizure episode.

EEG/LTM Findings

Twenty-two patients had routine EEGs, 13 of whom also had LTM. Four episodes of NCSE were diagnosed on LTM alone based on the clinical suspicion of NCSE. Therefore, 17/26 (65%) episodes of NCSE were managed with LTM. Of the 9 patients who never had LTM at MSKCC, seven had follow up EEGs. One had withdrawal of care and one was transferred to another facility for LTM. Of the 26 episodes of NCSE, electrographic partial seizures were seen in 16 and the remainder had periodic patterns on EEG. Several patients with recorded seizures also had periodic patterns on EEG. Resolution of seizures or periodic patterns on EEG determined control of NCSE.

Causes of Nonconvulsive Status Epilepticus

All patients had neuroimaging with each episode of NCSE. Ninety-six percent of our patients had new or pre-existing CNS neoplastic (88%) or vascular (23%) disease as a cause of NCSE (Table 3). All but one of our patients had structural CNS pathology and 15 (58%) patients had a new intracranial lesion defined as progression of neoplastic disease or a new unrelated lesion. Two patients with brain metastases also had confirmed HSV encephalitis. Five patients developed NCSE in the immediate post-operative period following craniotomy for tumor removal; all were receiving post-operative AEDs. One patient was admitted with alcohol withdrawal as well as hyperammonemia. This patient’s mental status declined despite medical therapy, culminating in a GTC seizure and postictal coma caused by NCSE found on EEG; MRI was normal. Five patients had hyponatremia at the time of NCSE

diagnosis, but it was usually chronic and only one patient had a sudden drop. No patient had hypoglycemia and only one patient had a blood glucose level >300 mg/dL (309 mg/dL).

Treatment

Seventeen (65%) of the 26 episodes of NCSE occurred in a patient taking at least one AED. All patients received AEDs for their NCSE; phenytoin and levetiracetam were the most commonly used drugs both prior to NCSE and at the time of NCSE. Seventeen episodes (65%) of NCSE required three or more AEDs to achieve control including the baseline drugs. Levetiracetam was used in 85% of patients, phenytoin in 69%, lorazepam in 38%, and phenobarbital and valproic acid in 35% each. Levetiracetam, phenytoin or a benzodiazepine were newly added in 9/26 episodes and control was achieved in six (6/26, 23%), none of whom required intubation. Nine patients were intubated and treated with IV lorazepam, midazolam, or propofol drips, five of whom were treated with pharmacologic coma to a burst suppression pattern on EEG.

Outcome

The time to control NCSE was determined electrographically when EEG was available, and by clinical symptoms when EEG was unavailable; 20 episodes were controlled and 6 were not. In the 6 patients who were not controlled, a decision to withdraw care was made in all after the initial attempt to treat NCSE was unsuccessful. In seventeen of the 20 controlled patients, resolution of NCSE was confirmed electrographically. In the remaining 3 patients, electrographic resolution of NCSE was not documented for the following reasons: two patients with probable NCSE improved clinically with treatment and follow-up EEG was not obtained, and one patient was transferred to another facility for treatment of NCSE after diagnostic EEG, and returned to MSKCC after NCSE was controlled but we did not have the LTM available for review. Of the 21 episodes of NCSE that were controlled, the median time to control was 2 days; the range was 0 to 9 days. In the 5 patients treated with burst suppression, control was defined as the onset of burst suppression on EEG with resolution of electrographic seizures or periodic patterns; 4 achieved EEG control.

Of the 21 patients in whom NCSE was controlled, nine (43%) had no new neurologic sequelae and 11 patients had new deficits. One patient never recovered from coma; this patient’s NCSE was controlled with burst suppression at eight days, with subsequent EEG 10 days later without seizures.

Three patients (12%) died with NCSE as a major contributing factor. In all three patients NCSE was present on EEG but a decision was made not to escalate treatment of this condition and all patients had comfort measure care. For all 26 episodes of NCSE, all-cause mortality was 12% at two weeks, 28% at one month, and 65% at six months with a median overall survival of 5 months.

DISCUSSION

The majority of status epilepticus is convulsive, but a recent study found that NCSE accounted for 25%–50% of all status epilepticus,(Knake et al., 2001) indicating that it is widely under diagnosed. In our retrospective study, NCSE was present in 4% of patients

who had an EEG; all patients in this cohort had cancer and the majority had a focal brain lesion as a contributing factor to NCSE. While this frequency is less than that reported in non-cancer populations with NCSE, 91% of our patients who had an EEG were awake and there was no clinical suspicion for NCSE for most. The most unexpected finding in our study is that 69% of patients with definite or probable NSCE occurred in patients who were awake at the time of EEG. This is particularly striking given the fact that all but one had significant structural brain pathology.

The prevalence of NCSE has been described and best studied in the ICU population where it is a contributing cause of unexplained persistent coma in 8% to 33% of adults and children. (Towne et al., 1994; Towne et al., 2000; Hosain et al., 2005; Cocito et al., 2001; Claassen et al., 2004) A prior study done in patients with systemic cancer identified NCSE in 6% of those with an impaired mental status and no brain metastases.(Cocito, 2001) Another study identified NCSE in 6 patients with systemic cancer although none had new intracranial pathology, including brain metastasis. (Drislane, 1994a). Ambulatory NCSE has been ascribed to generalized seizures such as absence epilepsy or myoclonic epilepsy which do not cause substantial changes in alertness. Ambulatory NCSE from complex partial status epilepticus can cause significant alteration in mental status and unresponsiveness. (Drislane, 2000) In contrast, 25/26 episodes had EEGs with focal features consistent with partial status epilepticus. Only 19% of our patients were comatose and 3 out of 5 had generalized features on EEG. Eighty-one percent were awake or lethargic, many of them ambulatory, and met criteria for NCSE. Because our patients were not profoundly impaired, there is the likely possibility that many such affected patients were missed because EEG was not obtained.

The etiology of NCSE in all but one patient was due to structural brain disease, most often neoplastic but also vascular. Only one patient with de novo brain metastasis presented with NCSE, which differs from other reports.(Blitshteyn and Jaeckle, 2006; Carota et al., 2009; Broderick and Cascino, 1987; Steg et al., 1993) Two of our patients had HSV encephalitis in addition to brain metastases. Only one of our comatose patients with NCSE had a cardiac arrest, although this is the most frequent etiology of coma with generalized epileptiform discharges reported in the literature. (Drislane and Shomer, 1994b; Treiman, 1995; Young et al., 1996; Akman, 2010)

Treatment of NCSE has been based on therapies for convulsive status epilepticus.(Treiman et al., 1998; Treiman, 1993) Seventy seven percent of our patients achieved control of NCSE despite advanced CNS disease in most. Successful treatment did not always require aggressive management with intubation and pharmacologic coma. NCSE in patients with absence epilepsy does not result in neuronal injury and aggressive treatment is not always warranted.(Akman, 2010) However, patients with NCSE and periodic patterns such as PLEDS, PEDs, may have secondary neuronal injury.(Chong and Hirsch, 2005) In these patients treatment and control of NCSE is warranted but the degree of intervention has to be considered on an individual basis. Because many of our patients were awake with NCSE, intubation was not required. Furthermore, in some of our most critically ill patients care was withdrawn so aggressive management was not always pursued.

The prognosis of NCSE in the general population is heterogenous and depends upon the subtype and etiology of the NCSE as well as the age and comorbidities of the patient. Comatose NCSE is associated with a worse prognosis than complex-partial or absence NCSE. In a study of comatose patients, administration of AEDs resulted in clinical improvement in only two of 33 patients.(Drislane and Shomer, 1994c) Mortality in non-cancer comatose NCSE patients was 18% in one study and was defined by the NCSE etiology, severity of coma, and development of complications. Increased mortality in all types of NCSE is associated with seizure duration, delay in diagnosis, and decreased consciousness at diagnosis. (Lowenstein and Alldredge, 1998; Shneker and Fountain, 2003) Mortality in our population was primarily due to the patients' underlying malignancy. (Stupp et al. 2005, Gaspar et al. 1997)

Limitations of our study include its retrospective nature and the selection of patients based on an EEG. We reviewed all patients undergoing EEG at a major cancer center and all episodes of NCSE were diagnosed on inpatients at the time of diagnostic EEG. It is likely that there were other patients with NCSE who were not evaluated with EEG and therefore missed. Awake NSCE was more common than comatose NCSE despite most patients having structural brain disease and the diagnosis being unsuspected based their clinical manifestations. The EEG was essential in diagnosis and management of these patients. NCSE control did not always require aggressive management with sedation and intubation but was usually reversible with AED treatment.

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Table 1

Summary of Patient Characteristics

	N=947 EEGs			*N*=25 patients with NCSE	
Mental Status	**No.**	**%**		**No.**	**%**
Awake	865	91	**Age (median, range)**	59	5–79
Lethargic	13	1.4	**Men**	10	40
Comatose	69	7.3	**Women**	15	60
NCSE on EEG	26	2.7	**1° Brain tumors**	13	52
Definite	18	1.9	GBM	8	32
Probable	8	0.8	Other	5	20
Mental Status	***N*=26 EEGs with NCSE**		**Systemic cancer**	14	56
Awake	18	69	Lung	4	16
Lethargic	3	12	Breast	3	12
Comatose	5	19	Other	7	28
Clinical Presentation			**Brain mets**	9	36
Confusion	13	50			
Twitching	10	38			
Aphasia	8	31			

Abbreviations: GBM – *glioblastoma multiforme;* GTCS – *generalized tonic-clonic seizure;* NCSE – *nonconvulsive status epilepticus*

Table 2

Details of the 25 patients with NCSE

Pt	Age	Sex	Cancer	Clinical presentation	EEG findings	Cause of NCSE	Days to control
PROBABLE NCSE							
1	61	M	GBM	Twitching, aphasia	Continuous PLEDs	Progression of GBM	6
2	69	F	Metastatic Lung	Aphasia → R motor sz → GTCS, post-ictal lethargy	PLEDs	Brain mets	1
3	69	M	Metastatic RCC	Confusion	biPLEDs	Hemorrhagic brain mets HSV encephalitis Hyponatremia	N/C
4	57	F	GBM	Confusion	Runs of focal sharp and slow waves	GBM	2
5	74	F	GBM	Agitation, disorientation	Partial seizures, PEDs, TW	GBM	6
6	58	F	GBM, Breast cancer	Lethargy, confusion	Partial seizures	GBM	3
7	71	M	GBM	Lethargy & confusion, followed by GTCS	Partial seizures, PLEDs	GBM	2
8	65	F	PCNSL	Fluctuating mutism	PLEDs	Progression of PCNSL	1
DEFINITE NCSE							
9	62	F	LG Oligo	Auditory hallucinations, déjà vu, ideas of reference	Partial seizures	Low Grade Oligodendroglioma	1
10	79	F	Metastatic Lung	Lethargy, twitching	Partial seizures	Brain mets, post-craniotomy	Unknown
11	51	F	Metastatic Breast	Lethargy, twitching	Periodic focal slow waves	Ischemic stroke New leptomeningeal mets, hyponatremia	N/C
12	52	F	Metastatic Breast	Confusion, aphasia → R motor sz → GTCS → postictal aphasia	Partial seizures, PLEDs	Progression of brain mets HSV encephalitis, hyponatremia	8
13	69	F	GBM, T-cell lymphoma	Confusion, aphasia, twitching	Partial seizures, PLEDs	New GBM	3
				Lethargy, twitching	Continuous PLEDs	Progression of GBM, postcraniotomy	N/C
14	77	M	GBM	Aphasia, lethargy, GTCS	Partial seizures, PLEDs	GBM, post-craniotomy	2
15	60	F	Gastric CA	GTCS followed by coma	Continuous biPLEDs, occasional biPEDs	Hemorrhagic stroke	2
16	76	M	Metastatic Lung	Lethargy, twitching	Partial seizures	Brain mets, post-craniotomy Subdural and subarachnoid hemorrhage	1
17	37	M	Anaplastic Astrocytoma	Confusion, staring spells, poor concentration	Continuous partial seizure	Progression of astrocytoma	5
18	65	F	Meningioma	Confusion, lethargy, twitching	Rhythmic focal sharp and slow waves	Meningioma, post-craniotomy Hemorrhagic infarct Subdural hemorrhage	4

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Pt	Age	Sex	Cancer	Clinical presentation	EEG findings	Cause of NCSE	Days to control
19	64	F	Metastatic Lung	Lethargy, aphasia, GTCS	Continuous partial seizure	Brain mets, post-craniotomy	9
20	5	F	Metastatic retinoblastoma	Coma, twitching	Partial seizures, biPLEDs	Progression of brain mets Ischemic stroke	N/C
21	68	M	Vocal cord SCC	Confusion followed by GTCS & post-ictal coma	Partial seizures, TW	EtOH withdrawal Hyperammonemia	1
22	60	F	Metastatic ovarian	Confusion, twitching	Continuous partial seizure	Progression of brain mets	0
23	55	M	GBM	Confusion & twitching, later global aphasia	Partial seizures	Progression of GBM, hyponatremia	N/C
24	64	M	Colon CA	Coma, myoclonus	Continuous PEDs	Anoxic brain injury	N/C
25	30	M	Medulloblastoma	Confusion	Partial seizures	Ischemic strokes, hyponatremia Stable medulloblastoma	2

Abbreviations: GBM – *glioblastoma multiforme*, POD – *progression of disease*, (bi)P(L)EDs – *(bilateral independent) periodic (lateralized) epileptiform discharges*, R – *right*, GTCS – *generalized tonic-clonic seizure*, RCC – *renal cell carcinoma*, mets – *metastases*, N/C – *not controlled*, TW – *triphasic waves*, PCNSL – *primary central nervous system lymphoma*, SCC – *squamous cell carcinoma*

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Table 3

Causes of NCSE in 26 Episodes

	No.	%
Intracerebral Neoplasm	23	88
Primary Brain Tumor	13	50
Brain Metastasis	9	35
Stroke (ischemic, hemorrhagic, global anoxia)	7	27
Post-craniotomy	5	19
Hyponatremia	5	19
HSV encephalitis	2	8
Hyperglycemia	1	4
Alcohol withdrawal, hyperammonemia	1	4
Anoxic Injury	1	4
>1 cause	11	42