Available online at www.sciencedirect.com
SCIENCE DIRECT®
Epilepsy & Behavior xxx (2005) xxx–xxx
Epilepsy & Behavior
www.elsevier.com/locate/yebeh

Case Report

Neurosarcoidosis presenting with simple partial seizures and solitary enhancing mass: case reports and review of the literature

Jeffrey L. Sponsler [a], Mary Ann Werz [b,*], Robert Maciunas [c], Mark Cohen [d]

[a] Valley Neuroscience Center, LLC, 3719 E Meridian Loop Suite E. Wasilla, AK 99654, USA
[b] Department of Neurology, University Hospitals of Cleveland, Case Western Reserve University School of Medicine, 11100 Euclid Avenue, Cleveland, OH 44106, USA
[c] Department of Neurosurgery, University Hospitals of Cleveland, Case Western Reserve University School of Medicine, 11100 Euclid Avenue, Cleveland, OH 44106, USA
[d] Department of Neuropathology, University Hospitals of Cleveland, Case Western Reserve University School of Medicine, 11100 Euclid Avenue, Cleveland, OH 44106, USA

Received 3 January 2005; revised 25 February 2005; accepted 26 February 2005

Abstract

A 37-year old woman, who had presented 5 years earlier with suspected simple partial seizures, returned with seizures increasing in frequency and intensity, confirmed by video/electroencephalography (VEEG) monitoring with left frontotemporal onset. A low-grade tumor was suspected, given a magnetic resonance imaging (MRI) study demonstrating enlargement of the left amygdala, anterior hippocampus, and adjacent mesial temporal neocortex, with modest gadolinium enhancement, and a positron emission tomography (PET) scan showing increased metabolism within that region. Surgical resection of the left mesial temporal lobe was performed and pathology revealed pathogen-free granulomas. She was given a diagnosis of sarcoidosis (following chest computed tomography that showed hilar adenopathy). She was treated with oral steroids for neurosarcoidosis with no further epileptic seizures in 19 months of follow-up. The second case was a young man, with known pulmonary sarcoidosis, who developed simple partial seizures and, later, complex partial seizures, with MRI revealing a left insular mass. Stereotactic biopsy again demonstrated pathogen-free granulomas. He has also done well in 4 years of follow-up. Review of the literature suggests that seizures associated with sarcoidosis do not invariably imply a poor prognosis. Certain features—multifocal parenchymal involvement, hydrocephalus, and chronic meningitis—were associated with poor outcome. In contrast, cases with isolated mass lesions often fared well.

Keywords: Neurosarcoidosis; Sarcoidosis; Temporal lobe epilepsy; Seizures; Amygdala; Hippocampus; Positron emission tomography; Brain mass lesion

1. Introduction

Neurosarcoidosis is seen in 5% of systemic sarcoidosis patients, with only about 50% having a prior diagnosis when the nervous system becomes involved [1,2]. Seizures are estimated to occur in 5–20% of neurosarcoidosis patients [1,2]. The neurologic manifestations of sarcoidosis that may contribute to development of seizures are protean and include parenchymal abnormalities including encephalopathy/vasculopathy (5–10%), intraparenchymal mass lesion(s) (5–10%), endocrinopathy with metabolic disturbance including hypothalamic and/or pituitary dysfunction (10–15%), meningeal disease (including aseptic meningitis and meningeal mass) (10–20%), and hydrocephalus (10%) [1]. Most reports indicate a poor prognosis with severe progressing or relapsing course when seizures are

* Corresponding author. Fax: +1 216 844 5066.
E-mail address: maryann.werz@uhhs.com (M.A. Werz).

1525-5050/$ - see front matter © 2005 Elsevier Inc. All rights reserved.
doi:10.1016/j.yebeh.2005.02.016

associated with sarcoidosis [2–4]. We describe two cases of epilepsy and sarcoidosis from our institution who have done well, as well as review the literature.

2. First case history

A 37-year-old white, right-handed woman (Case 1) developed stereotypical episodes 7 months earlier following a "superviral" infection. She had swollen "glands in her neck making it hard to swallow." Her husband came home from work to find her passed out on the floor. She was evaluated in the emergency room and sent home. She then began experiencing brief (40–60 seconds), stereotyped episodes of a "wave" over the back of her head, fuzzy feeling in her face, and a feeling that she might lose her breath. These were initially occurring weekly but, within 6 months, were occurring several times per day. When several occurred in close succession, she noted difficulty in finding words. During an episode she would often stop what she was doing, and people would express concern. She was able to talk throughout and even drove through one event without incident.

She was the product of a normal gestation, labor, and delivery, including an identical twin, without seizures. There was no history of febrile seizures or significant brain trauma. Her medical history was notable for a monophasic illness a decade earlier with glomerular nephritis with a "lupuslike syndrome" from which she completely recovered. Family history included a teenage daughter with primary generalized epilepsy treated by the authors. A high school graduate, the patient worked as a receptionist and had three children. Her neurologic examination was normal. Magnetic resonance imaging revealed a hazy, enhancing area within the left amygdala and adjacent temporal neocortex. She was started on phenytoin, resulting in reduction of the episodes to one per week. The patient did not follow up until 5 years later, when, while on antiepileptic medications, she experienced a marked increase in seizure frequency from one per week to several per day. She was concerned that her thinking was impaired with some of the episodes. These episodes persisted despite trials of carbamazepine, levetiracetam, and lamotrigine.

VEEG study captured seven events. In five events she reported a "fuzzy" feeling in her head and noted that her stomach felt "weird," with behavioral arrest and minor lip smacking or mouth pursing in two. EEG records during these five events plus two without identified clinical accompaniments exhibited rhythmic theta in the left frontotemporal region at times evolving centrally (Fig. 1).

Neuropsychological testing revealed a wide range of verbal skills including confrontational naming, fluency, and abstraction below expected premorbid abilities. MRI showed enlargement of left amygdala, hippocampal head, and the adjacent temporal neocortical region with bright signal on T2 and FLAIR sequences and patchy gadolinium enhancement, suggestive of low-grade neoplasm (Fig. 2). PET showed increased metabolism in the abnormal structures (Fig. 3).

Surgical resection of the left anterior temporal lobe was performed, and specimens were obtained for study. Neuropathology analysis revealed granulomatous inflammation of brain parenchyma and perivascular spaces

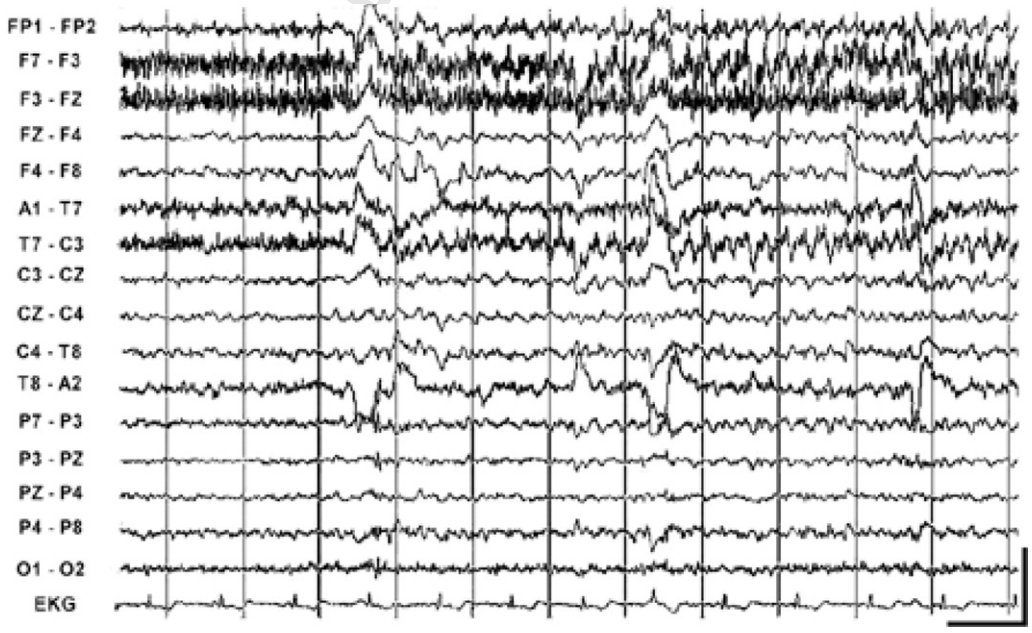

Fig. 1. EEG record showing seizure with left frontotemporal onset. The calibration bar represents 90 μV, 1 second. High-frequency filter was 35 Hz.

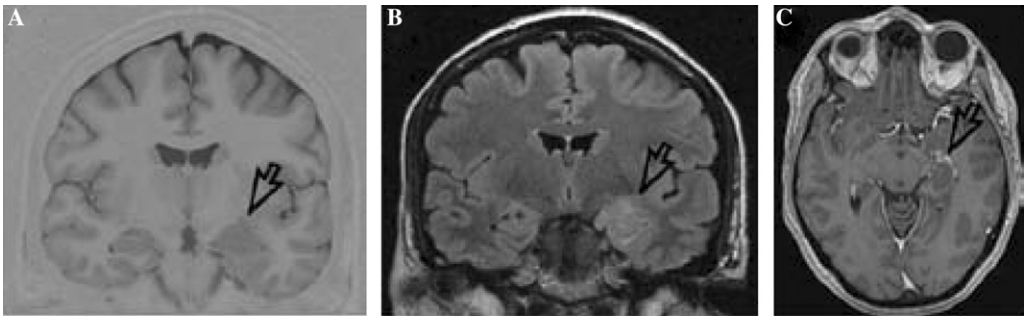

Fig. 2. (A) This MRI image shows enlargement of left amygdala, hippocampal head, and adjacent neocortex on inversion recovery weighted T1 sequence. (B) Bright signal is seen within the abnormal structures on FLAIR. (C) Gadolinium produced patchy enhancement. Arrows identify the abnormal region.

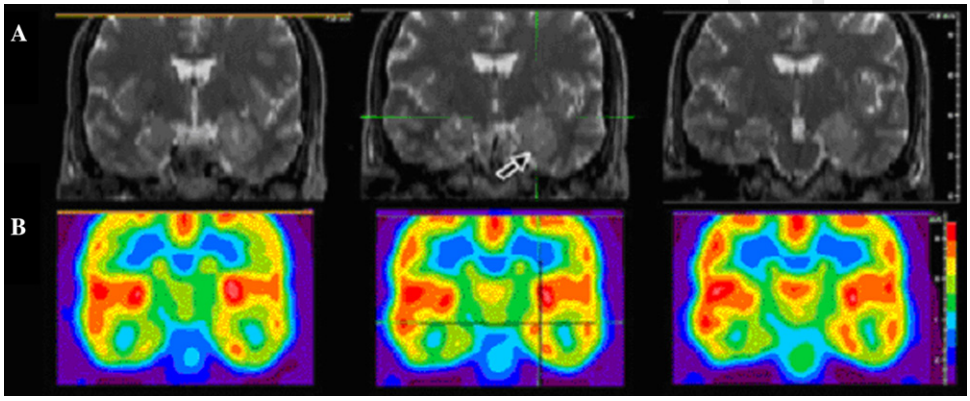

Fig. 3. (A) Successive 3-mm T2-weighted images anterior (left) to posterior (right) showing enlarged amygdala and anterior hippocampus on the left (see arrow). (B) Corresponding PET scans are seen below with hypermetabolism in the abnormal region.

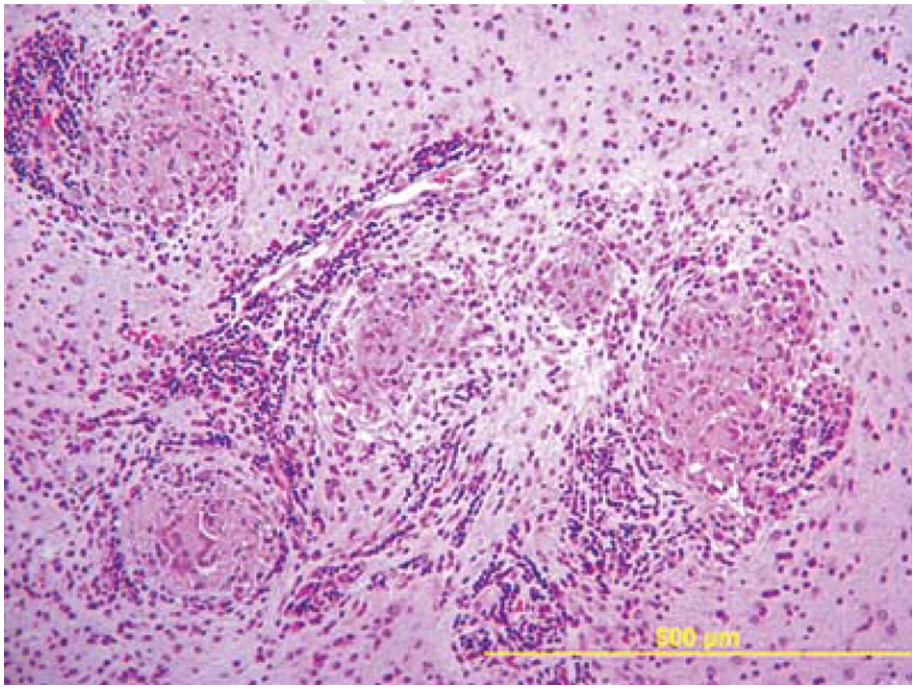

Fig. 4. Confluent areas of granuloma within the amygdala consistent with sarcoidosis.

without evidence of vasculitis within the amygdala, hippocampus, and temporal cortex (Fig. 4).

No organisms were seen on acid-fast stain or gram stain and no polarizable foreign material was observed. Serum fluorescent treponemal antibody and lyme titers were negative. Serum and cerebrospinal angiotensin-converting enzyme levels were normal. Gallium scan did not show abnormal uptake. Computed tomography of the chest revealed subcarinal, mediastinal, and hilar lymphadenopathy. Pulmonary function tests were normal.

Prednisone, 40 mg daily, was prescribed for the CNS sarcoidosis for 1 year, followed by a taper. Her neuropsychological functioning and neuroimaging tests, showing typical postoperative changes, have remained stable for more than 17 months. She has had no further epileptic seizures while maintained on her preoperative dose of lamotrigine, though nonepileptic events documented by VEEG monitoring occurred in association with reports of recurrent sexual abuse involving minors by her father and brother.

3. Second case history

We have identified a second case of neurosarcoidosis presenting with seizures. A 28-year-old white man complained of fatigue and mouth breathing. He underwent reduction of nasal turbinates but also had a chest X-ray consistent with hilar adenopathy. He was diagnosed with sarcoidosis and started on 5 mg of prednisone daily. At age 31, he developed episodes of anxiety and a metallic taste in his mouth lasting for seconds every several weeks. He saw a neurologist 3 years later when the episodes progressed to confusion and speech arrest lasting 10–30 seconds two to three times daily. MRI revealed a lesion with increased signal on T2 and FLAIR sequences in the left superior frontal insula and subinsular region, with mild mass effect on the lateral ventricle and gadolinium enhancement. Pathology of a subinsular stereotactic biopsy identified pathogen-free, noncasseating granuloma. Prednisone was increased to 10 mg per day. He has done well during 4 years of follow-up, though simple partial seizures occur about once per week on carbamazepine therapy. MRI 3 years after biopsy showed marked improvement, though a ribbon of abnormal signal remains in the superior frontal insula with modest enhancement.

4. Literature review of neurosarcoidosis and seizures

Delaney [3] reviewed reports published over 50 years, through 1980, of 500 patients with neurosarcoidosis, of 54 whom had seizures (11%). Of those with seizures, 60% died and an additional 15% had significant morbidity. The mean survival was 9 months when three patients who survived more than 249 months were excluded; half the deaths occurred in 4 months. He noted that eight patients did well and six were lost to follow-up. "Nearly all had granulomatous meningitis," but 11 (20 %) had a space-occupying mass lesion. He commented that those with solitary mass lesions seemed to "fare better after craniotomy."

More recent case series continue to show markedly variable mortality in neurosarcoid but consistently demonstrate higher mortality when seizures manifest (Table 1). Mortality is increased 20% to nearly 10-fold when seizures occur in neurosarcoidosis [4–7]. How does neurosarcoidosis presentation affect prognosis? We have identified 277 cases of neurosarcoidosis in the literature, 43 of whom had seizures (16%). In only 40 cases were clinical, neuroradiologic, or pathologic descriptions sufficient. Of these 40 cases, only 28 were followed up either to death or, for survivors, to a minimum of 1 year. These 28 cases plus the 2 from our institution are detailed in Table 2 and summarized in Table 3.

Of the 30 patients with neurosarcoidosis and seizures, 14 (47%) died. Death occurred 1 month to 10 years following onset of CNS symptoms, with average and median times to death of 2.7 and 2.0 years, respectively. Thirty-three percent had good neurologic function for up to 19 years. Follow-up in survivors was 5.0 years on average, with a median of 3.5 years. Seizure type did affect survival. Focal seizures (focal sensory, focal motor, or complex partial seizures) with or without secondary generalization were described in 10 patients; 9 had only generalized tonic–clonic seizures. Death was more frequent (about doubled) for those with only generalized tonic–clonic seizures (40%) compared with focal seizures (20%). Timing of seizures did not affect outcome as patients with seizures within the first 6 months of neurosarcoidosis fared no better than those with later onset of seizures. There were no reports that seizures were a direct cause of any deaths. Only Krumholz et al. [5] described seizure control. Eight of ten had complete seizure control, and of these, six did well. Of the two

Table 1
Mortality in neurosarcoidosis with and without seizures in four case series

Reference	Neurosarcoid with seizures		Neurosarcoid without seizure	
	Patient No.	No. of deaths (%)	Patient No.	No. of deaths (%)
Delaney, 1977 [4]	6	6 (100)	10	8 (80)
Krumholz et al., 1991 [5]	13	2 (15)	66	1 (2)
Sharma, 1996 [6]	2	2 (100)	26	4 (26)
Tahmoush et al., 2002 [7]	1	1 (100)	7	4 (57)

Table 2
Clinical and anatomical manifestations of neurosarcoidosis associated with seizures and affect on clinical outcome

Patient #	[Ref.] (year)	Age/race/ gender	Sarcoid initial presentation (neurologic/ systemic)	Systemic involvement	Onset time of seizure in CNS sarcoid[a]	Seizure class[b]	Isolated focal anatomic dist. (MRI/CT/ PATH)	Multifocal anatomic dist.[c] (MRI/CT/PATH)	Clinical-focal anatomic dist. (MRI/CT/ PATH)	Meningitis	Hydrocephalus	Encephalopathy	Follow-up (years)	Status at follow-up[d]
1	[8] (1965)	30/?/F	Neurologic	Lungs, lymph nodes	NR[e]	GTC	No	Brain, PIT, MEN, SC	Yes	Yes	NR	NR	NR	Died
2	[9] (1965)	23/W/M	Neurologic	Hilar lymph nodes	Late	NR	Not isolated	NR	Yes	Yes[f]	NR	Yes	4	Poor
3	[4] (1977)	30/B/F	NR	Lungs, lymph nodes, liver	NR	NR	No	BAS MEN, PIT, HYP, CN1,CN2	Yes	Yes	NR	NR	6	Died
4		66/B/M	Neurologic	Lungs, lymph nodes, liver, kidneys	Early	NR	No	HYP,BAS MEN, hemispheres, BS, CER	Yes	Yes	NR	Yes	0.1	Died
5		30/B/M	Neurologic	Hilar lymph node, lungs, spleen	NR	NR	No	BAS MEN, CN2, BS, HYP, PIT, temporal lobe, 3rd ventricle	Yes	Yes	Yes	Yes	1.5	Died
6		33/B/M	NR	Lungs, lymph nodes, liver, eye	NR	NR	No	BAS MEN, HYP, PIT, CN7, Optic Chiasm	Yes	Yes	NR	No	4	Died
7		28/B/M	Neurologic	Lymph node, Lung, liver, spleen	Early*	NR	No	BAS MEN, aqueduct, CER, caudate	Yes	Yes	Yes	Yes	0.5	Died
8		29/B/F	Neurologic	Lymph node, lung, spleen, heart	NR	NR	No	BAS MEN, HYP, PIT, ventricles	Yes	Yes	Yes	Yes	1	Died
9	[10] (1983)	61/B/M	Sarcoid	Lymph node, spleen	Late	GTC	No	MEN, CN	Yes	Yes	Yes	Yes	3	Died
10		36/B/M	Sarcoid	Lymph nodes, liver, spleen, pancreas	Early*	FS, GTC	No	MEN, HYP, PIT, CN, SC, ventricles	Yes	Yes	Yes	Yes	10	Died
11		29/B/F	Sarcoid	Lungs, uveitis	Late	GTC	No	BAS MEN, HYP, CN	Yes	Yes	Yes	Yes	2	Died
12	[11] (1986)	33/B/F	Sarcoid	Eye, lungs, liver, spleen, heart	Late	NR	No	BAS MEN, ventricles, HYP, PIT, CN, SC	Yes	Yes	Yes	Yes	0.75	Died
13	[5] (1991)	58/B/M	Neurologic	Lung	Early*	FM, GTC	Not isolated	Diffuse plus temporal mass	Yes	NR	Yes	Yes	1	Died
14		21/B/F	ND	Skin, lymph node	Early*	GTC	NR	NR	Yes	NR	Yes	Yes	4	Died
15		20/W/M	Neurologic	None	Early*	FS, GTC	Parietal mass	NR	NR	NR	NR	NR	3	Good
16		35/B/M	NR	Eye	Late	GTC	Dural mass	NR	NR	NR	NR	NR	3	Good
17		25/W/F	Neurologic	None	Early*	FS, GTC	NR	NR	CN2,CN7	NR	NR	Yes	3	Poor
18		30/B/F	NR	Skin, lung, liver, stomach	Late	GTC	Not isolated	CER mass	Yes	NR	Yes	Yes	1	Good
19		25/M/B	Neurologic	Lymph node, nasal sinuses	Early*	GTC	No	Yes	CN2	NR	NR	Yes	4	Vision loss
20		17/B/F	NR	Lymph nodes, eye, lung	Late	GTC	Not isolated	CN7, plus temporal mass	Yes	Yes	Yes	NR	2	Good
21		31/W/M	Neurologic	None	Early*	FS, GTC	NR	CN7, SC	Yes	NR	Yes	Yes	19	Good
22		23/B/F	NR	Lung, eye	Early*	FS,GTC	NR	NR	NR	Yes	NR	NR	3	Good
23	[6] (1996)	61/W/F	Neurologic	Lacrimal, glands, spleen	NR	"Seizure"	ND	NR	CN 7, proptosis	NR	NR	NR	10	Good
24		36/W/F	NR	Parotid, lymph nodes	Early*	"Seizure"	NR	MRI "abnormal"	NR	NR	NR	NR	5	Good
25	[12] (2001)	61/M	NR	NR	Early*	FS	NR	NR	NR	NR	NR	NR	>5	Minor
26		35/M	NR	NR	Early*	GTC	NR	MRI "supertentorial," cerebellar	Yes	NR	NR	NR	>5	Poor
27		39/F	NR	NR	Early*	FS	NR	Supra- and infratentorial	Yes	NR	NR	NR	>5	Minor

(*continued on next page*)

Table 2 (*continued*)

Patient #	[Ref.] (year)	Age/race/ gender	Sarcoid initial presentation (neurologic/ systemic)	Systemic involvement	Onset time of seizure in CNS sarcoid[a]	Seizure class[b]	Isolated focal anatomic dist. (MRI/CT/ PATH)	Multifocal anatomic dist.[c] (MRI/CT/PATH)	Clinical-focal anatomic dist. (MRI/CT/ PATH)	Meningitis	Hydrocephalus	Encephalopathy	Follow-up (years)	Status at follow-up[d]
28	[7] (2002)	36/B/F	Sarcoid	Parotid, lung	NR	GTC	NR	"Brain" on MRI	CN7	No	NR	NR	2	Died
29	Case 1	37/W/F	Neurologic	Hilar lymph node	Early*	FS	Temporal Lobe	No	No	No	No	No	6.5	Good
30	Case 2	33/W/M	Sarcoid	Lung	Early*	FS	Insular Cortex	No	No	No	No	No	7	Good

[a] Early, first 6 months; Early*, seizures were present as initial neurologic manifestation.
[b] FS, focal sensory, focal motor, or complex partial; GTC, generalized tonic–clonic.
[c] PIT, pituitary; MEN, meninges; SC, spinal cord; BAS MEN, basal meninges; HYP, hypothalamus; CN, cranial nerves; BS, brainstem; Cer, cerebellum.
[d] Good = improvement with no significant neurologic dysfunction; poor = significant encephalopathy and, needing some but not continuous care in daily living; minor = symptoms not interfering with lifestyle [12].
[e] NR, not reported.
[f] Four years earlier and then right temporal parenchymal and meningeal mass.

intractable patients, one had continued generalized tonic–clonic seizures and neurologic deterioration, and died within 4 years. The second had persistent complex partial and generalized tonic–clonic seizures with significant dementia. One of our two cases was also completely controlled on medication, though only after temporal lobectomy. Our second case had only simple partial seizures on medication.

Outcome varied with neurologic presentation. Meningitis and hydrocephalus were associated with the highest mortality, 79 and 75%, respectively. Mortality was also high in multifocal disease (57%) and in widespread disease indicated by encephalopathy (73%). In contrast, isolated mass lesions occurred in only four patients (three parenchymal and one dural), but none died and all had good outcome. Three additional patients without hydrocephalus, meningitis, encephalopathy, or multifocal disease also had good outcomes. Meningitis may present variably as an acute monophasic illness, a relapsing course, or a chronic progressive illness. The last clinical course was confirmed on autopsy in more than 11 of 14 patients with meningitis, with the remainder presumably less aggressive. Though mortality was high when neurosarcoidosis was associated with hydrocephalus, this was not absolute and one such patient was followed 19 years.

5. Discussion

We described a woman followed more than 6 years with slow worsening of seizures and also enlargement of a left temporal abnormality seen on MRI with modest enhancement. This lesion was hypermetabolic on PET scan. Though the time course and neuroimaging characteristics were interpreted to be most consistent with a low-grade neoplasm, analysis of resected brain tissue revealed pathogen-free granulomas. Subsequently, hilar and mediastinal adenopathy was identified, consistent with a diagnosis of sarcoidosis. As in our case, lung is commonly also affected (74–94% of cases). Other organ systems are less commonly affected: skin (30%), eyes (37%), liver (13%), and heart (11%) [1].

Neurologic manifestations and MRI imaging characteristics are myriad. Magnetic resonance imaging is abnormal in 80% and will show periventricular T2 or FLAIR signal abnormalities (46%), multiple supratentorial and infratentorial lesions (36%), solitary intraaxial mass lesions (9%), solitary extraaxial mass (5%), and leptomeningeal enhancement (36%) [13,14].

The temporal mass diagnosed by MRI in our case was shown to be hypermetabolic on PET. Systemic sarcoidosis is hypermetabolic on PET scan and may also lead to an initial working diagnosis of neoplasm [15]. However, [^{18}F]fluorodeoxyglucose ([^{18}F]FDG) PET may have a role in diagnosis and follow-up of sarcoidosis and neurosarcoidosis [1,16–18]. Increased cellular up-

Table 3
Summary of neurosarcoidosis and seizures

	N	Died	Major morbidity	Minor morbidity	Good
Seizures	30	14(47%)	3(10%)	3(10%)	10(33%)
Seizure characteristics					
Early seizures	16	5(31%)	2(12%)	3(19%)	6(38%)
Late seizures	7	3(43%)	1(14%)	0(0%)	3(43%)
GTC (without FS)	9	4(44%)	1(11%)	1(11%)	3(33%)
FS (with/without GTC)	10	2(20%)	1(70%)	2(20%)	5(50%)
Effect of neurologic presentation on outcome					
Isolated focal lesion	4(13%)	0(0%)	0(0%)	0(0%)	4(100%)
Multifocal abnormalities	23(77%)	13(57%)	3(13%)	2(9%)	5(22%)
No multifocal abnormalities	7(23%)	0(0%)	0(0%)	1(14%)	6(86%)
Meningitis	14(47%)	11(79%)	1(7%)	0(0%)	2(14%)
No meningitis	16(53%)	3(19%)	2(12%)	3(19%)	8(50%)
Hydrocephalus	12(40%)	9(75%)	0(0%)	0(0%)	3(25%)
No hydrocephalus	18(60%)	5(27%)	3(17%)	3(17%)	7(40%)
Encephalopathy	15(50%)	10(73%)	2(13%)	1(6%)	2(13%)
No encephalopathy	15(50%)	4(27%)	1(7%)	2(13%)	8(53%)

take of [^{18}F]FDG is related to inflammatory cell infiltrates that are composed of lymphocytes, macrophages, and epithelioid cells. [^{18}F]FDG accumulates in lung and hilar lymph nodes in sarcoidosis patients. Uptake by lung tissue has been shown to be concordant with histologic activity of pulmonary sarcoidosis and decreases after high-dose steroid therapy.

Similarly, one group found that sarcoidosis activity in the heart was best followed by PET (compared with gallium scan and SPECT) [18]. Though whole-body FDG-PET may be useful in assessing systemic involvement and response of sarcoidosis to therapy, the effects of neurosarcoidosis on brain metabolism are complex, with Dubey et al. reporting hypometabolism of temporal lesions [19].

The specific neurologic manifestations of sarcoidosis associated with seizures profoundly affect outcome. Seizures accompanying meningitis or hydrocephalus are highly associated with poor outcome, as is multifocal disease including encephalopathy. In contrast, isolated mass lesions, though relatively infrequent, appear often to be associated with good outcome. Simple partial and complex partial seizures with or without secondary generalization also are associated with better outcome than generalized tonic–clonic seizures only. This finding is likely again to be related to focal dysfunction compared with more widespread parenchymal damage. Seizures in neurosarcoidosis are often easy to control with medication. Death in neurosarcoidosis correlates with an aggressive relapsing or progressive course.

6. Conclusions

Our literature review yields several trends regarding neurosarcoidosis and seizures. Seizures are often associated with a higher occurrence of morbidity and death. Poor outcome is associated with hydrocephalus, chronic meningitis, and multifocal parenchymal disease. In contrast, an isolated mass lesion is associated with good outcome, though this pathology is relatively infrequent. Neurosarcoidosis mass lesions may mimic tumors by showing gadolinium enhancement and increased metabolism on PET scan.

Acknowledgments

This work was done while Dr. Jeffrey L. Sponsler was a clinical neurophysiology fellow at University Hospitals of Cleveland. We thank Dr. Ted Westbrook for providing us with the second case. We thank Tari Rienzo for help with the manuscript.

References

[1] Stern BJ. Neurological complications of sarcoidosis. Curr Opin Neurol 2004;17:311–6.
[2] Gullapoli D, Phillips LH. Neurologic manifestations of sarcoidosis. Neurol Clin 2002;20:1–17.
[3] Delaney P. Seizures in sarcoidosis: a poor prognosis. Ann Neurol 1980;7:494.
[4] Delaney P. Neurologic manifestations in sarcoidosis: review of the literature, with a report of 23 cases. Ann Intern Med 1977;87:336–45.
[5] Krumholz A, Stern BJ, Stern EG. Clinical implications of seizures in neurosarcoidosis. Arch Neurol 1991;48:842–4.
[6] Sharma O. Neurosarcoidosis: a personal perspective based on the study of 37 patients. Chest 1996;112:220–8.
[7] Tahmoush AJ, Amir MS, Connor WW, et al. Sarcoidosis Vasc Diffuse Lung Dis 2002;19:191–7.
[8] Wiederholt WC, Siekert RG. Neurologic manifestations of sarcoidosis. Neurology 1965;15:1147–54.
[9] Silverstein A, Feuer MM, Siltzbach LE. Neurologie Sarcoidosis. Arch Neurol 1965;12:1–11.

[10] Manz HJ. Pathobiology of neurosarcoidosis and clinicopathologic correlation. Can J Neurol Sci 1983;10:50–5.
[11] Schlitt M, Duvall ER, Bonnin J, Morawetz RB. Neurosarcoidosis causing ventricular loculation. Surg Neurol 1986;26:67–71.
[12] Ferriby D, DeSeze J, Stojkovic T, et al. Long-term follow-up of neurosarcoidosis. Neurology 2001;57:927–9.
[13] Fels C, Riegel A, Javaheripour-Otto K, Obenauer S. Neurosarcoidosis findings in MRI. J Clin Imaging 2004;28:166–9.
[14] Pickuth D, SPielmann RP, Heywang-Kobrunner' SH. Role of radiology in the diagnosis of neurosarcoidosis. Eur Radiol 2000;10:941–4.
[15] Ludwig V, Fordice S, Lamar R, Martin WH, Delbeke D. Unsuspected skeletal sarcoidosis mimicking metastatic disease on FDG positron emission tomography and bone scintigraphy. Clin Nucl Med 2002;28:176–9.
[16] Zhuang H, Alavi A. 18-Fluorodeoxyglucose positron emission tomographic imaging in the detection and monitoring of infection and inflammation. Semin Nucl Med 2002;32:47–59.
[17] Aberg C, Ponzo F, Raphael B, Amorosi E, Moran V, Kramer E. FDG positron emission tomography of bone involvement in sarcoidosis. Am J Roentgenol 2004;182:975–7.
[18] Yamagishi H, Shirai N, Takagi M, Akioka K, Takeuchi K, Yoshikawa J. Identification of cardiac sarcoidosis with ^{13}N-NH_3/^{18}F-FDG PET. J Nucl Med 2003;44:1030–6.
[19] Dubey N, Miletich RS, Wasay M, Mechtler L, Bakshi R. Role of fluorodeoxyglucose positron emission tomography in the diagnosis of neurosarcoidosis. J Neurol Sci 2002;205:77–81.