

Characterizing Sunflower syndrome: a clinical series

James R. Barnett¹, Bradley M. Fleming¹, Kennedy R. Geenen¹, Jo Sourbron², Jason H. Freedman¹, Patricia L. Bruno¹, Elizabeth A. Thiele¹

¹ Pediatric Epilepsy Program, Department of Neurology, Massachusetts General Hospital, Boston, MA, USA

² Department of Development and Regeneration, Section Pediatric Neurology, University Hospital KU Leuven, Leuven, Belgium

Received November 21, 2019; Accepted March 01, 2020

ABSTRACT – *Aims.* To characterize the clinical phenotype of Sunflower syndrome. Sunflower syndrome is a rare photosensitive epilepsy syndrome characterized by highly stereotyped seizures, photosensitivity, and heliotropism.

Methods. We retrospectively reviewed the medical records of patients seen in the Massachusetts General Hospital for Children (MGHfC) pediatric epilepsy program with a history of Sunflower syndrome.

Results. Twenty-four patients were identified; 18 were female. At the time of initial MGHfC evaluation, patients' ages ranged from 6.4 to 25 years, with a median age of 11.5 years. All patients presented with hand-waving episodes (HWEs), although one patient no longer demonstrates this, but now has eye blinking episodes on exposure to light. Four have associated eye fluttering as a component of their most prevalent light-induced seizures. The average age at onset of HWEs was six years. Seventeen developed other symptoms prior to the onset of HWEs. The most prevalent symptom was an attraction to light and possible absence seizures. Light-induced seizures were generally refractory to broad-spectrum antiepileptic drugs (AEDs). Only three patients had a reduction of HWEs with the use of AEDs. Several non-pharmacological strategies reduced seizure frequency, however, efficacy varied. These non-pharmacological strategies included avoiding stimulus, focusing on other tasks, and occupying or restraining the hand that was involved in hand-waving. The use of tinted glasses reduced seizure frequency in 17 patients, however, no patient achieved seizure freedom. Twenty-two patients had available EEGs, 20 of which showed interictal epileptiform discharges. Additionally, many of the patients experienced a negative impact on their self-concept due to anxiety, depression, or negative interactions with peers.

Conclusion. Sunflower syndrome is a generalized, pharmacoresistant epilepsy with childhood onset and remains poorly understood. To improve clinical care and scientific understanding, long-term prospective research exploring the natural history, etiology, and effective treatments for Sunflower syndrome should be conducted. [*Published with video sequence.*]

Key words: refractory seizures, photosensitive, sunflower syndrome, clinical series, self-induced



VIDEO ONLINE

Correspondence:

Elizabeth Thiele
Massachusetts General Hospital,
175 Cambridge Street, Suite 340,
Boston, MA 02114, USA
<EThiele@MGH.harvard.edu>

Sunflower syndrome is a rare, photosensitive epilepsy characterized by stereotyped reflex seizures and attraction to light (Ames and Saffer, 1983; Baumer and Porter, 2018). Individuals with Sunflower syndrome look toward a light source, most commonly the sun, and wave their hand in front of their eyes. The hand-waving episodes (HWEs) associated with Sunflower syndrome are coupled with generalized 3-4-Hz spike-and-wave discharges on electroencephalograms (EEG) (Binnie, 1988). Often, Sunflower syndrome is initially misdiagnosed as a tic or behavioral disorder due to the associated abnormal movements and compulsions to seek out light (Binnie, 1988; Baumer and Porter, 2018). Sunflower syndrome is frequently referred to as a self-induced photosensitive epilepsy (Ames and Saffer, 1983). In 1951, Gastaut interpreted the hand-waving as a mechanism to induce seizures through a strobe-like effect in a patient with photic-stimulated epilepsy that presented similarly to Sunflower syndrome (Gastaut, 1951). However, subsequent clinical series have suggested that hand-waving itself may be an ictal phenomenon (Livingston and Torres, 1964; Ames and Saffer, 1983). In 1983, Ames and Saffer suggested renaming the epilepsy Sunflower syndrome to more appropriately describe the disorder and to highlight its heliotropism, described as a compulsion to seek sunlight (Ames, 1971; Ames and Saffer, 1983). To date, many articles continue to refer to the seizures experienced by individuals with Sunflower syndrome as “self-induced” (Binnie, 1988; Singhi and Bansal, 2004; Baumer and Porter, 2018).

Sunflower syndrome appears to be refractory to most broad-spectrum antiepileptic drugs (AEDs) (Robertson, 1954; Hutchison et al., 1958; Ames, 1971; Aicardi and Gastaut, 1985; Baumer and Porter, 2018). AEDs commonly used to treat other photosensitive epilepsies, such as sodium valproate, are usually not effective in Sunflower syndrome patients (Baumer and Porter, 2018). Anecdotal evidence has suggested certain non-pharmacological interventions, such as tinted glasses, to be effective in reducing HWEs in Sunflower syndrome. However, the efficacy of these interventions has not been tested in large sample sizes (Ames, 1971; Ames and Saffer, 1983; Belcastro and Striano, 2014).

Here, we review 24 patients presenting with symptoms consistent with Sunflower syndrome; the largest series of patients with Sunflower syndrome seen at a single site.

Methods

We retrospectively reviewed the medical records of patients with Sunflower syndrome seen in the Massachusetts General Hospital for Children (MGHfC)

pediatric epilepsy program from 2014-2018. Approval from the Partners Human Research Committee was attained; the requirement for written patient consent was waived.

Patients with a history of being attracted to bright lights and having seizures involving hand-waving were identified. Information analyzed included medical history, family history, age at onset, earlier symptoms, seizure types, seizure frequency, electroencephalogram (EEG) and MRI results, and treatments. The level of patient cognitive function was based on clinical notes detailing developmental milestones and academic performance.

Results

Patients and family history

Twenty-four patients with Sunflower syndrome were identified (*table 1*). Eighteen were female (75%). The average age at onset of HWEs was 6.2 years (median=5.5, first Q=4.0, third Q=8.2). Eight patients had a family history of epilepsy (33.3%), five of which were a generalized epilepsy (62.5%), including absence epilepsy and juvenile myoclonic epilepsy (JME). None had a family history of Sunflower syndrome.

Seventeen of the 24 patients who experienced hand-waving seizures exhibited symptoms prior to the onset of HWEs (70.8%). These included attraction to light (25%), eye rolling (20.8%), lateral head deviations/head shaking (12.5%), blinking (8.3%), lapse of awareness in sunlight (8.3%), eye fluttering (8.3%), myoclonic jerks (4.2%), tonic-clonic seizures (4.2%), and aversion to light (4.2%). Eye rolling, lateral head deviations/head shaking, blinking, a lapse of awareness in sunlight, and eye fluttering were interpreted as possible absence seizures. These symptoms occurred on average 1.6 years prior to the onset of HWEs for the nine patients with reported symptoms.

Seizure characteristics

All 24 patients experienced light-induced seizures and HWEs (*table 1*). Patient 16 had HWEs until the age of 12, which then transitioned to eye blinking episodes (BE). Four patients, Patient 10, 11, 23, and 24 (16.7%), also exhibited eye rolling or eye fluttering with their HWEs.

Patient handedness was not strongly associated with the hand involved in HWEs (*table 2*). Every patient consistently used the same hand during their HWEs.

Videos, available on parents' phones for most of the patients, and parents' descriptions showed that the HWEs were particularly stereotyped and occurred in

Table 1. Patient characteristics.

| Patient number | Sex | Family history of seizures | Age at onset (y) | Current Age (y) | Light-induced seizures | Other seizure types | Inducing stimulus | Estimated frequency of HWEs |
|----------------|-----|--|------------------|-----------------|------------------------|---------------------|--------------------|-----------------------------|
| 1 | F | | 6 | 11 | HW | A | N | 150/week |
| 2 | F | | 9 | 11 | HW | A, TC | N, Ar | Daily |
| 3 | M | | 3 | 7 | HW | | N, Ar ^Δ | 200 by midday |
| 4 | F | 2 Paternal aunts with isolated seizures | 2 | 10 | HW | | N | 50/week |
| 5 | M | | 4 | 7 | HW | | N, Ar | 1000/week |
| 6 | F | | 5 | 10 | HW | TC* | N | 15/day |
| 7 | F | | 6 | 21 | HW | TC | N | 10/week |
| 8 | M | | 7 | 8 | HW | | N, Ar | 700/week |
| 9 | F | Maternal uncle with TC seizures | 3 | 11 | HW | A, TC* | N, Ar ⁺ | 100/week |
| 10 | F | Paternal grandfather with childhood absence epilepsy; mother with history of seizures in setting of cerebral palsy | 4 | 15 | HW+S+EF | aA, M | N, Ar | Less than daily |
| 11 | F | | 10 | 15 | HW+EF | TC | N | 100/week |
| 12 | F | Father with childhood-onset TC seizures, sister with JME, cousin treated for epilepsy | 9 | 12 | HW | TC* | N, Ar | 1000/day |
| 13 | F | | 8 | 25 | HW | A, TC* | N, Ar | Less than daily |
| 14 | F | | 5 | 15 | HW | TC | N, Ar | 70/week |
| 15 | M | Mother with childhood-onset absence and TC seizures | 4 | 11 | HW | | N, Ar | Daily |
| 16 | M | Mother's cousin with childhood absence seizures | 12 | 15 | BE (previously HW) | NEE | N | Less than daily |
| 17 | F | | 5 | 7 | HW | | N, Ar | "Constant" |
| 18 | F | | 5 | 6 | HW | | N, Ar | |
| 19 | F | | 8 | 15 | HW | A, TC | N | Daily |
| 20 | F | Father with febrile seizures in early childhood | 4 | 7 | HW | A | N, Ar | 50/week |
| 21 | F | | 5 | 9 | HW | | N, Ar | |
| 22 | M | | 8 | 15 | HW | A, TC | N | |
| 23 | F | | 9 | 19 | HW+EF | TC | N, Ar | |
| 24 | F | Maternal cousin, details unknown | 7 | 22 | HW+EF | At, M, TC* | N | |

P: patient; F: female; M: male; HW: hand-waving; S: staring; EF: eye fluttering; HS: head shaking; BE: blinking; A: absence; TC: tonic-clonic; aA: atypical absence; M: myoclonic; At: Atonic; JME: juvenile myoclonic epilepsy; NEE: non-epileptic event; N: natural light; Ar: artificial light.

*Report TC with prolonged HW.

^ΔHWEs in artificial light occurred two years after onset of HWEs in natural light.

⁺Only had HWEs in artificial light when tired or stressed.

Table 2. Specific hand waving activity.

| Patient number | Handedness | Hands involved in hand waves | Waves predominantly with dominant hand |
|----------------|------------|------------------------------|--|
| 1 | Right | Both | Yes |
| 2 | Right | Both | No |
| 3 | Right | Both | Yes |
| 4 | Right | Right | Yes |
| 5 | Right | Both | No |
| 6 | Right | NC | NC |
| 7 | Right | Left | No |
| 8 | Right | Right | Yes |
| 9 | NC | NC | NC |
| 10 | NC | NC | NC |
| 11 | Left | Left | Yes |
| 12 | Right | Left | No |
| 13 | Right | Left | No |
| 14 | Right | Right | Yes |
| 15 | Right | Right | Yes |
| 16 | NC | NC | NC |
| 17 | Right | Both | NC |
| 18 | Right | Right | Yes |
| 19 | Right | Left | No |
| 20 | Left | Right | No |
| 21 | Right | Left | No |
| 22 | Right | Right | Yes |
| 23 | Left | Right | No |
| 24 | Right | Right | Yes |

NC= Information not collected.

short clusters with brief pauses between episodes (see *video sequence*).

The frequency of light-induced seizures was available for 19 patients (79.2%) (*table 1*). Frequency varied and was generally influenced by stimulus exposure. Patients 3, 5, 12, and 17 (16.7%) experienced “almost constant” seizure activity when exposed to light. Patients 7, 10, and 16 (12.5%) reported HWEs on a less than daily basis.

The detailed description of the light stimulus that triggered their light-induced seizures (*table 1*) was available for all patients. All 24 patients had seizures induced by natural sunlight. Fifteen (62.5%) also had seizures induced by artificial light.

Stress and anxiety increased seizure frequency in eight patients (33.3%), and fatigue increased seizure frequency in five patients (20.8%).

Sixteen patients (66.7%) had other seizure types, including absence, tonic-clonic, and myoclonic seizures (*table 1*). Twelve patients had a history of tonic-clonic seizures; of those, five had tonic-clonic seizures following a prolonged HWE. Patient 10 noted that prolonged HWEs resulted in absence seizures.

Other evaluations

EEG reports were available for 22 patients; 20 had interictal abnormalities. All 20 of the abnormal EEGs showed generalized epileptiform activity, the majority characterized by spike and polyspike-and-wave discharges with frequencies ranging from 1 to 4 Hz (*figure 1*). Many of these discharges had bifrontal or bioccipital predominance. Hyperventilation was performed for 10 patients, and seven of these patients had associated changes on EEG. Three of the seven displayed epileptiform activity during or shortly following hyperventilation; four revealed generalized slowing during hyperventilation. Photostimulation was performed for 14 of the patients, nine of whom demonstrated a photoparoxysmal response (*figure 2*). One patient, Patient 6, had a significant consistent photoparoxysmal response with eyes closed, which abated when eyes opened during continued photic stimulation. All available EEGs showed normal background activity characterized by age-appropriate posterior dominant rhythm, with good anterior-posterior organization.

Thirteen of the 24 patients had available brain imaging. Ten patients had brain MRIs which were normal. Three patients had head CTs that were also normal. One patient underwent epilepsy gene panel testing that did not identify any disease-causing mutation.

Twenty-two of the 24 patients had normal cognitive development. Patient 5 had mild speech, language, and gross motor delays and Patient 22 had autism. For all patients, neurological exams were normal.

Epilepsy treatment

At the time of initial evaluation at the MGHfC, patients were being treated with an average of 1.4 AEDs (Median=1.5, first Q= 1.0, third Q=2.0, Max=3.0, Min=0.0) (*table 3*). Sixteen patients (66.7%) had previously discontinued at least one medication due to lack

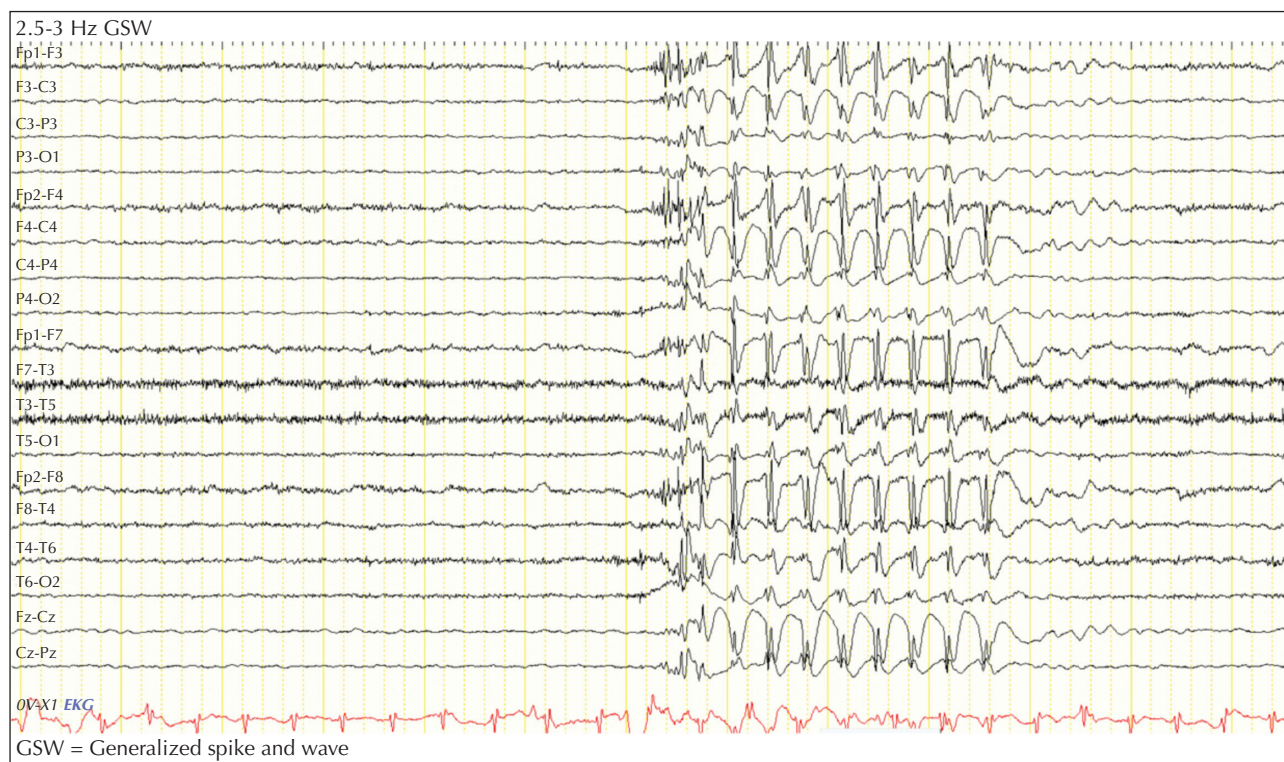


Figure 1. Sunflower syndrome interictal EEG.

of efficacy or tolerability; these patients had been on an average of 2.5 AEDs.

Medications used included sodium valproate, levetiracetam, lamotrigine, ethosuximide, topiramate, artisanal formulation of cannabidiol (CBD) oil, purified CBD (Epidiolex®), clonazepam, lorazepam, clobazam, and diazepam.

While taking at least one AED, the frequency of HWEs was reduced for Patients 10, 15, and 4. Patient 10's HWEs reduced from multiple times a day to one per week while on a combination of levetiracetam, sodium valproate, and clonazepam. However, this decrease occurred concurrently with an increase in the frequency of absence seizures. Patient 15's HWEs reduced from multiple times a day to a total of six HWEs over the course of a summer while on levetiracetam and sodium valproate. Patient 4's HWEs reduced from multiple times a day to approximately one per day while on levetiracetam, however, previous higher doses of levetiracetam had had no effect.

Three patients, Patients 6, 8, and 12, tried low glycemic index treatment (LGIT). The HWE frequency of Patient 6 and 12 decreased on dietary therapy; Patient 8's HWE frequency did not change.

Other interventions that reduced the frequency of HWEs included avoiding bright light, focusing on other tasks, and restricting or occupying the hand that was

involved in hand-waving. Seventeen patients (70.8%) reported that reducing light exposure via a hat or tinted glasses reduced the frequency of HWEs. Patient 5's HWE frequency decreased with the use of both tinted and non-tinted glasses. Five patients (20.8%) reported that focusing on a task, while in the presence of the sun or bright light, reduced the frequency of episodes. For example, Patient 4 did not have HWEs while competing in athletic competitions, and Patient 7 did not have HWEs while driving. Eight (33.3%) of the 24 patients reported that occupying one or both of their hands could reduce the number of HWEs. No strategy led to complete seizure freedom and in most cases provided only limited benefit.

Seven of the patients (29.2%) were originally diagnosed with tics after developing HWEs. Patient 12 was prescribed clonidine and Patient 15 was prescribed guanfacine; neither was effective. Patient 9 was also treated for tics; pharmacologic treatment was not effective.

Burden and stigma of disease

Nine of the patients (37.5%) described experiencing negative psychosocial impact due to the HWEs. Six patients (25%) were teased and bullied in school. Patients 4, 10, 12, and 16 (16.7%) reported low self-

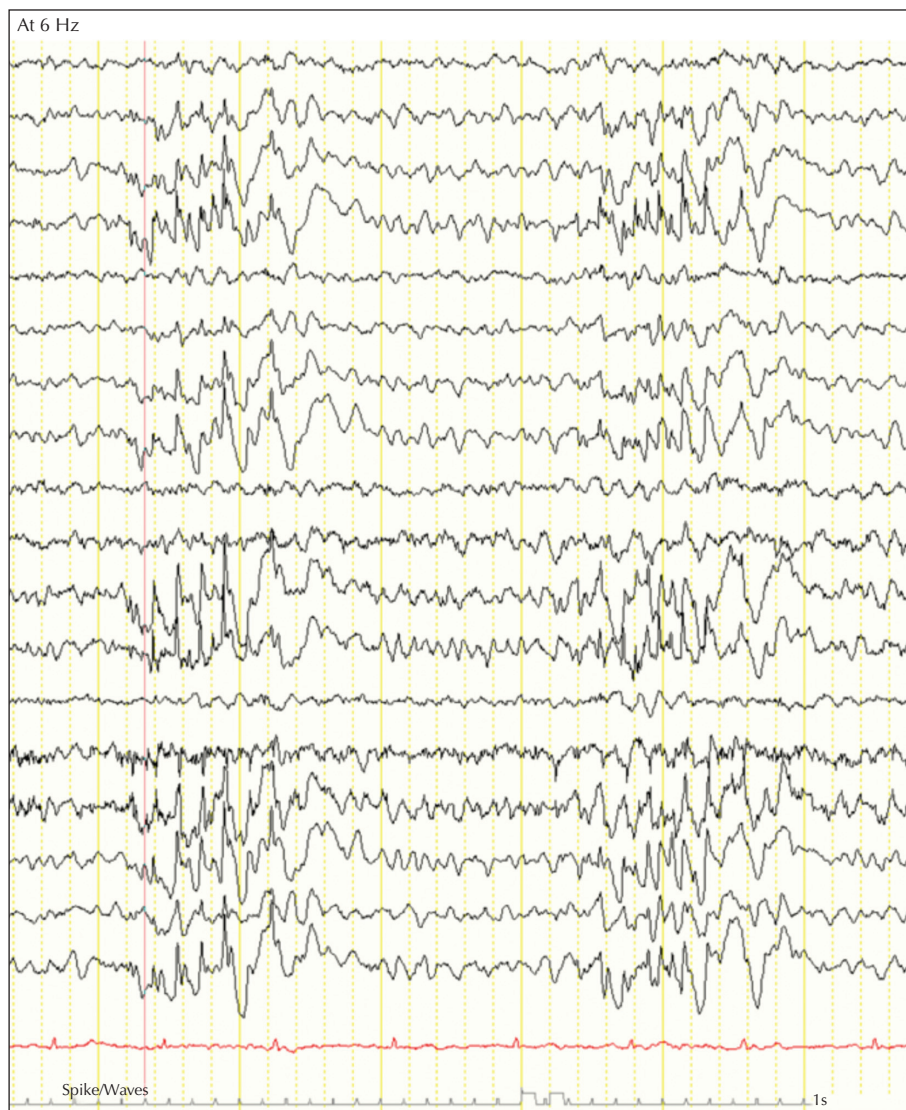


Figure 2. Sunflower syndrome photoparoxysmal response on EEG.

esteem and self-efficacy. Patients 12 and 16 reported symptoms of anxiety and depression, and Patient 16 was prescribed escitalopram for these symptoms.

Discussion

In 1951, Henri Gastaut published the first possible case of Sunflower syndrome. He reported two children who appeared to induce seizures by searching out light and waving their hands in front of their eyes. In 1983, this epilepsy, which consists of heliotropism, photosensitivity, and abnormal upper limb movements during seizures, was renamed “Sunflower syndrome” (Ames and Saffer, 1983). Several case series of probable

Sunflower syndrome have been published, however, the disorder remains poorly characterized and understood (Robertson, 1954; Hutchison *et al.*, 1958; Whitty, 1960; Chao, 1962; Andermann *et al.*, 1962; Livingston and Torres, 1964; Ames, 1971; Ames and Saffer, 1983). We present the largest case series of Sunflower syndrome patients evaluated at a single center.

Similar to previous case series (Andermann *et al.*, 1962; Ames, 1971; Ames and Saffer, 1983; Belcastro and Striano, 2014; Baumer and Porter, 2018), our Sunflower syndrome population was predominantly female and developed HWEs within the first decade of life. No patient had a family history of Sunflower syndrome, but eight of the 24 patients had a family history of epilepsy. Andermann *et al.* also reported that over one

Table 3. Current and previous treatments.

| Patient number | Current treatments | Previous treatments |
|----------------|--------------------|----------------------------------|
| 1 | LTG, VPA | CLB, ETX, BRV |
| 2 | LEV | CBD oil |
| 3 | VPA | |
| 4 | LEV | |
| 5 | LTG | |
| 6 | | CBD oil, LGIT |
| 7 | ETX | LTG, VPA |
| 8 | LEV | LGIT |
| 9 | VPA | |
| 10 | LEV, VPA, CLZ | ZNS |
| 11 | VPA, TPM | LEV, ZNS, CBD oil, LTG |
| 12 | LTG, LGIT | LCS |
| 13 | TPM, ETX | VPA, LTG |
| 14 | LTG | TPM, LEV |
| 15 | LEV, VPA | ETX, LTG, GFN, CLN |
| 16 | | |
| 17 | VPA | ETX |
| 18 | | |
| 19 | LEV, LTG | ETX, RFM, CLB, VPA, TPM, CBD oil |
| 20 | LEV | |
| 21 | | |
| 22 | CLB, CBD oil*, VNS | ZNS, VPA, LEV, LTG, CLZ, DZP |
| 23 | CBD oil | TPM, LTG, LEV |
| 24 | CBD oil | LEV |

LTG: lamotrigine; VPA: sodium valproate; LEV: levetiracetam; CBD: cannabidiol; ETX: ethosuximide; LZP: lorazepam; DZP: diazepam; CLB: clobazam; ZNS: zonisamide; CLZ: clonazepam; TPM: topiramate; LGIT: Low Glycemic Index Treatment; CLN: clonidine; LCS: lacosamide; GFN: guanfacine; RFM: rufinamide; VNS: vagus nerve stimulator.

*Purified CBD oil

third of their “self-induced photosensitive epilepsy” patients had a positive family history of seizures; significantly higher than the one in 13 patients with a positive family history in their cohort of patients with other photosensitive epilepsies (Andermann *et al.*, 1962). In a previous study, a case series was described with a mother and daughter pair both with Sunflower syndrome (Ames, 1971).

The highly stereotyped presentation of seizures in this patient population, as well as family histories of other generalized epilepsies, suggest a possible genetic component to Sunflower syndrome. To date, no comprehensive genetic analyses have been conducted and no underlying genetic etiology has been identified.

Although the presentation of consistent unilateral hand-waving during seizures may suggest a focal epilepsy, the EEG characteristics are suggestive of a generalized epilepsy. Previous studies have shown 3-4-Hz generalized spike and wave activity during HWEs (Belcastro and Striano, 2014). The interictal EEG data from our patients, which show generalized discharges, and the high prevalence of a family history of generalized epilepsy suggest that Sunflower syndrome is likely to occur in families with other genetic generalized epilepsies (Kim *et al.*, 2015). Sunflower syndrome shares characteristics with Jeavons syndrome (epilepsy with eyelid myoclonia) (Smith *et al.*, 2018). However, given that patients with Jeavons syndrome do not have associated HWEs, Sunflower syndrome should be considered distinct from Jeavons syndrome.

Sunflower syndrome appears to be pharmacoresistant (Kwan and Brodie, 2010) for the majority of patients. Few patients in the literature, and none of our patients, attained seizure freedom after pharmaceutical intervention (Baumer and Porter, 2018). Broad spectrum medications such as levetiracetam and sodium valproate were only reported to be effective in a minority of the patients who received treatment. Dietary therapy with LGIT appeared effective in a small number of our patients. LGIT has proven effective for other refractory epilepsies (Muzykewicz *et al.*, 2009) and could be considered as a possible treatment option for Sunflower syndrome. However, effective treatments for individuals with Sunflower syndrome are currently limited.

Other strategies to reduce the frequency of light-induced seizures, such as the use of sunglasses, have been reported in the medical literature (Andermann *et al.*, 1962; Ames, 1971; Ames and Saffer, 1983; Belcastro and Striano, 2014; Baumer and Porter, 2018). In one case, a patient’s photoparoxysmal response and light-induced seizures stopped with the use of Zeiss Clarlet F133 Z1 tinted lenses (Belcastro and Striano, 2014). Although avoiding the sun or bright light did not prevent seizures completely, 70.8% of our patients reported that the use of a hat or tinted glasses

noticeably reduced the frequency of light-induced seizures. Interestingly, one patient had fewer seizures even while wearing non-tinted glasses.

One additional patient was identified who did not meet the inclusion criteria for this series due to a lack of hand-waving. However, the subject had episodes of head shaking and was strongly drawn to light, similar to the other patients in this series. This suggests a possible variability in the phenotypic presentation of Sunflower syndrome.

Currently, Sunflower syndrome is poorly understood, and research is needed to characterize the syndrome's etiology, pathophysiology, and natural history. The treatment approach should also address the psychological comorbidity which is commonly seen. Since Sunflower syndrome appears to be refractory to broad-spectrum AEDs, there is a need to identify effective treatments. Therefore, prospective studies including possible medication trials, better characterizing the role of dietary therapy, and other treatment options should be considered. □

Legend for video sequence

Video demonstrating a typical hand-waving episode seen in Sunflower syndrome.

Key words for video research on
www.epilepticdisorders.com

Phenomenology: photosensitive, generalized

Localisation: generalized

Syndrome: sunflower syndrome

Aetiology: unknown

Acknowledgements and disclosures.

The authors of this paper would like to thank the patients and their families for cooperating in the study and for allowing their data to be used.

Elizabeth A. Thiele, M.D., Ph.D. has served as a paid consultant for GW Pharmaceuticals, Zogenix Pharmaceuticals, Upsher Smith, West Therapeutics, Aquestive and Biocodex. The remaining authors have no conflicts of interest to disclose.

References

- Aicardi J, Gastaut H. Treatment of self-induced photosensitive epilepsy with fenfluramine. *N Engl J Med* 1985;313(22):1419.
- Ames FR. "Self-induction" in photosensitive epilepsy. *Brain* 1971;94(4):781-98.
- Ames FR, Saffer D. The sunflower syndrome. A new look at "self-induced" photosensitive epilepsy. *J Neurol Sci* 1983;59(1):1-11.
- Andermann K, Berman S, Cooke PM, et al. Self-induced epilepsy. A collection of self-induced epilepsy cases compared with some other photoconvulsive cases. *Arch Neurol* 1962;6:49-65.
- Baumer FM, Porter BE. Clinical and electrographic features of sunflower syndrome. *Epilepsy Res* 2018;142:58-63.
- Belcastro V, Striano P. Self-induction seizures in sunflower epilepsy: a video-EEG report. *Epileptic Disord* 2014;16(1):93-5.
- Binnie CD. Self-induction of seizures: the ultimate non-compliance. *Epilepsy Res Suppl* 1988;1:153-8.
- Chao D. Photogenic and self-induced epilepsy. *J Pediatr* 1962;61:733-8.
- Gastaut H. L'épilepsie photogénique. *Rev Prat* 1951;1(2):105.
- Hutchison JH, Stone FH, Davidson JR. Photogenic epilepsy induced by the patient. *Lancet* 1958;1(7014):243-5.
- Kim SH, Korff CM, Kim AJ, et al. A practical, simple, and useful method of categorizing interictal EEG features in children. *Neurology* 2015;85(5):471-8.
- Kwan P, Brodie MJ. Definition of refractory epilepsy: defining the indefinable? *Lancet Neurol* 2010;9(1):27-9.
- Livingston S, Torres IC. Photic epilepsy: report of an unusual case and review of the literature. *Clin Pediatr (Phila)* 1964;3(5):304-7.
- Muzykewicz DA, Lyczkowski DA, Memon N, et al. Efficacy, safety, and tolerability of the low glycemic index treatment in pediatric epilepsy. *Epilepsia* 2009;50(5):1118-26.
- Robertson EG. Photogenic epilepsy: self-precipitated attacks. *Brain* 1954;77(2):232-51.
- Singhi PD, Bansal D. Self induced photosensitive epilepsy. *Indian J Pediatr* 2004;71(7):649-51.
- Smith KM, Youssef PE, Wirell EC, et al. Jeavons syndrome: clinical features and response to treatment. *Pediatr Neurol* 2018;86:46-51.
- Whitty CW. Photic and self-induced epilepsy. *Lancet* 1960;1(7136):1207-8.

TEST YOURSELF



- (1) What is the seizure semiology of Sunflower syndrome?
- (2) What are the clinical features of Sunflower syndrome?
- (3) What are the EEG features seen in Sunflower syndrome?

Note: Reading the manuscript provides an answer to all questions. Correct answers may be accessed on the website, www.epilepticdisorders.com, under the section "The EpiCentre".