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Epileptic Disorders

REVIEW ARTICLE

Subclinical rhythmic EEG discharge of adults (SREDA) in pediatric population: A case series with systematic review of the literature

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Abstract

Subclinical rhythmic electrographic discharge of adults (SREDA) is one of the rarest and most challenging non-epileptic electroencephalographic variants. Although the pathogenesis of this activity is unclear, an association with vascular insufficiency and cerebral hypoxia has been proposed. SREDA usually occurs in adulthood, but there are few reports in the pediatric population. We performed a systematic review of the literature, confirming the rarity of this condition in children, and added 5 more subjects. We report on a total of 16 children with SREDA. Sufficient data are available for 15 patients. The mean age at first detection of SREDA was 11.5 years. We observed that 67% (10/15) of the subjects had previous seizures: 80% (8/10) of them had an epilepsy diagnosis and 38% (3/8) had generalized epilepsy. Moreover, 8 of 13 subjects whose medical history was available (61%) had a neurodevelopmental disorder. From an electroencephalographic point of view, we noted a prevalence of bilateral SREDA with atypical localization and abrupt onset and end. Since SREDA can be incorrectly interpreted as an epileptic discharge, with possible therapeutic implications, it is important to consider its possible occurrence also in pediatric patients, perhaps more frequently in those with neurodevelopmental disorders.

KEYWORDS

electroencephalogram, neurodevelopmental disorders, nonepileptic variants, pediatric neurophysiology, subclinical rhythmic discharge of adults

Abbreviations: ASM, anti-seizure medication; CAE, childhood absence epilepsy; CBZ, carbamazepine; CCM, cerebral cavernous malformation; CLB, clobazam; EEG, electroencephalogram; ESM, ethosuximide; HV, hyperventilation; IPS, intermittent photic stimulation; IQ, intelligence quotient; LEV, levetiracetam; MRI, magnetic resonance imaging; OXC, oxcarbazepine; PB, phenobarbital; PHT, phenytoin; PNES, psychogenic nonepileptic seizures; PRISMA, Preferred Reporting Items for Systematic Reviews and Meta-Analyses; SREDA, subclinical rhythmic EEG discharge of adults; TPM, topiramate; VGB, vigabatrin; VPA, valproic acid.

1 | INTRODUCTION

The electroencephalogram (EEG) is the major diagnostic tool for diagnosing epilepsy. However, a clear distinction between epileptiform EEG patterns and artifacts or non-epileptic variants may be tricky.[1]

Amin et al. recently published a comprehensive review that analyzes and classifies all non-epileptiform EEG patterns, distinguishing between artifacts (physiological and extra-physiological), ordinary rhythms, and variants of uncertain significance.[2,3]

First described in the 1960ies, Subclinical Rhythmic EEG Discharge of Adults represents a rare non-epileptiform pattern in adulthood.[4,5] The term was subsequently changed to Subclinical Rhythmic Electrographic Discharges of Adults (SREDA) and its benign nature and uncertain significance were later confirmed.[6]

SREDA is an unusual EEG pattern characterized by rhythmic sharp waves in the theta band with abrupt or gradual onset and symmetric or asymmetric distributions mainly in the temporal and parietal regions. Typically, the duration ranges from a few seconds to several minutes. Furthermore, an atypical form of SREDA has been defined in the literature by the presence of unusual topography, morphology, and frequency, including frontal localization, focal distribution, notched waveforms, prolonged duration, and evolution from delta to theta frequency.[5–7] SREDA differs significantly from other unusual EEG patterns, e.g. with respect to "mid-temporal rhythmic discharges", SREDA is less confined to the mid-temporal region, has a longer duration and occurs mainly during wakefulness.[2,5,6]

SREDA has been described mostly in older adults,[8] especially in the wake state.[5]

Although the pathogenesis of SREDA is not completely understood, several hypotheses have been proposed, mainly suggesting a possible correlation with hypoxic states and intracranial vascular insufficiency in adults.[4,9] However, the occurrence of this EEG variant even in childhood suggests that a clear neurophysiological and etiologic hypothesis is still lacking.

Here we report a case series and a systematic review of the literature to provide an overview of the current knowledge on the occurrence of SREDA in pediatric patients.

Key points

- Subclinical rhythmic electrographic discharge of adults (SREDA) is rare non-epileptic electroencephalographic variant that can also occur in pediatric age.
- Physicians must recognize the SREDA EEG pattern and differentiate it from epileptic discharge to avoid inappropriate treatment.

2 | METHODS

2.1 | Case series

A retrospective review of EEGs performed at the Child Neuropsychiatry Unit of Gaslini Children Hospital was conducted between 2010 and 2023 by experts in pediatric neurology (LB, SB, EC, MMM, and GN). Data were collected from our database in May 2023. Each subject was younger than 18 years at the time of EEG recording. We reviewed both routine EEG and video-EEG monitoring during clinical activity. EEGs were performed for several indications (seizure, epilepsy, head trauma, headache, differential diagnosis from seizures, neurodevelopmental disorders). Patients with acute encephalopathy or a post-ictal state were excluded.

2.2 | Systematic review

The present study was performed according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.[10]

2.3 | Search strategy

We performed a comprehensive search of the PubMed/MEDLINE, Scopus, and EMBASE databases for studies published between January 1981 and July 2024. The search terms used were as follows: (A) "EEG", and (B) "SREDA" or "Subclinical rhythmic EEG discharge of adult" or "subclinical rhythmic epileptiform discharge of adults" or "subclinical rhythmic electrographic discharges of adults" or "decharges paroxystiques du carrefour".

2.4 | Inclusion and exclusion criteria

We selected studies published in any language in peer-reviewed journals. We included manuscripts with at least one pediatric subject with SREDA, as defined by Westmoreland and Klass, and Dash et al.[5,7] Adult subject reports, reviews, meta-analyses, or papers without any pediatric subjects were excluded. Studies not available online or those in which critical data could not be pulled out were discarded (Figure 1).

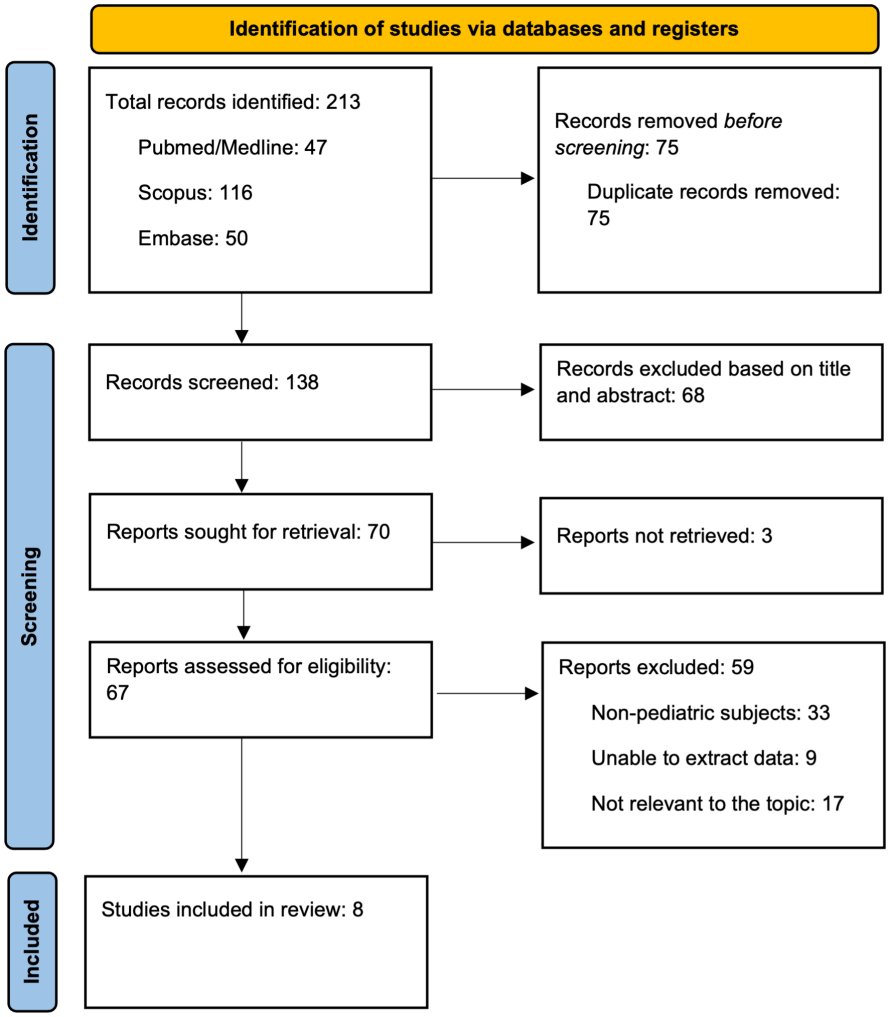

FIGURE 1 Systematic review flowchart.

2.5 | Study selection

Two reviewers (LB and GN) independently analyzed all titles and abstracts for eligibility and evaluated the full texts of the papers potentially suitable for inclusion.

In cases of disagreement, a third review, EC, resolved discrepancies. Foreign language articles were translated by the authors.

2.6 | Data extraction and synthesis

We found 213 publications; after screening and eligibility assessment, only 8 studies were included (Figure 1). We extracted the following data for each subject:

- Clinical features: Age at SREDA finding, sex, seizure type, eventual epilepsy diagnosis or syndrome, diagnosis other than seizure/epilepsy, ongoing drug treatment, reported treatment side effects, indication for EEG, and all comorbidities.
- Electroencephalographic features: SREDA in wakefulness (during hyperventilation, HV, and intermittent photic stimulation, IPS, and transition from wakefulness to sleep), SREDA in sleep (in different stages, N1, N2, N3, and REM), localization, frequency, laterality, symmetry, synchronism, onset and end pattern of SREDA.

3 | RESULTS

3.1 | Case series

Among the 18.980 EEG recorded in children in the Child Neuropsychiatry Unit of the Giannina Gaslini Institute between January 2010 and June 2023, we found 5 cases of SREDA.

Below we describe the clinical histories of these five subjects. The clinical findings and EEG features are summarized in Tables 1 and 2.

3.1.1 | Case 1

A 17-year-old female with a diagnosis of neurodevelopmental disorders (autism spectrum disorder level I,

TABLE 1 Clinical features of pediatric SREDA cohort.

Author, year	Case	Age of SREDA detection (years)	Sex	Epilepsy diagnosis	Age at first seizures (years)	Type of epilepsy or cause of seizures	Indication for EEG	SREDA misdiagnosis	ASMs at the time of SREDA detection	ASMs after SREDA detection	Side effects of ASM	Comorbidity and additional findings
Gil et al., 2019[11]	1	17	M	No	NR	No	Zoning out episode	Yes	VPA	VPA	Behavioral worsening	Marijuana abuse, attention deficit
Hoppe et al., 2005[12]	2	14	F	Yes	7	Epilepsy with generalized and focal seizures of unknown origin	Seizures	No	NR	NR	NR	Delayed neuromotor development; anxiety and somatization disorder
	3	17	M	Yes	15	Focal epilepsy of unknown cause	Seizures	No	NR	NR	NR	No
Goeden et al., 2017[13]	4	10	F	Yes	8	Childhood absence epilepsy (CAE)	Seizures	No	ESM	ESM	No	NR
Dash et al., 2012[14]	5	5	M	No	NR	Febrile seizures	Febrile seizures	Yes	CBZ	CBZ	NR	No
Nagarajan et al., 2001[15]	6	11	F	No	NR	Acute symptomatic seizures during hemolytic uremic syndrome	Seizures	No	PHT	CBZ, VGB, CLB, PHT	Rush (CBZ); Behavioral worsening (VGV)	Hemolytic uremic syndrome
	7	10	F	No	NR	No	Headache	Yes	No	VPA	No	Learning difficulties, headhache
Santoshkumar et al., 2009[16]	8	Between 6 and 15	M	NR	NR	NR	NR	NR	NR	NR	NR	NR
Ramesha et al., 2010[17]	9	13	M	No	NR	No	Post-traumatic amnesia	No	No	No	NR	NR
Thet et al., 2023[18]	10	NR	NR	Yes	NR	Focal Occipital epilepsy	Seizures	No	NR	NR	NR	Developmental delay and significant neurological conditions not better defined
	11	NR	NR	Yes	NR	Focal Occipital Epilepsy	Seizures	No	NR	NR	NR	Developmental delay and significant neurological conditions not better defined
Case 1	12	12	F	Yes	7 months	Generalized epilepsy	PNES	No	LEV	PB, OXC, LEV	NR	Developmental disorder (Autism, ADHD, borderline intellectual functioning), anxiety and mood disorder
Case 2	13	10	F	No	NR	No	Loss of consciousness episodes	No	No	No	NR	Specific learning disorder
Case 3	14	11	M	No	NR	No	Investigation for neurodevelopmental disorder	No	No	No	NR	Mild intellectual disability
Case 4	15	13	M	Yes	7	Symptomatic epilepsy (cavernomatosis)	Seizures	No	TPM, LEV	TPM, LEV	Behavioral worsening (LEV)	PNES, emotional disorders
Case 5	16	7	F	Yes	2	Generalized epilepsy	Epilepsy	No	LEV	LEV	No	Agenesis of the septum pellucidum

Abbreviations: ASMs, antiseizure medications; CAE, childhood absence epilepsy; CBZ, Carbamazepine; CLB, clobazam; ESM, ethosuximide; F, female; LEV, levetiracetam; M, male; NR, not reported; OXC, oxcarbazepine; PB, phenobarbital; PHT, phenytoin; PNES, psychogenic nonepileptic seizures TPM, topiramate; VGB, vigabatrin; VPA, valproic acid.

TABLE 2 EEG features of SREDA pediatric court.

		SREDA onset during EEG			SREDA EEG features:							
Author, year	Case	Awake – IPS – HV – Drowsiness	NREM I/ NREM II/ NREM III	REM	Distribution	Rhythm frequency	Lateralization	Symmetry	Synchrony	Onset	End	Additional findings
Gil et al., 2019[11]	1	Yes/NR/NR/Yes	Yes/Yes/No	Yes	TP	6 Hz	Bilateral	No	No	Abrupt	Abrupt	SREDA disappears during active wakefulness; no changing of SREDA after VPA withdrawal
Hoppe et al., 2005[12]	2	Yes/NR/NR/Yes	Yes/Yes/NR	NR	FCP, Extension to T regions in sleep	5–6 Hz	Bilateral	Yes	Both synchronous and asynchronous	Abrupt	Abrupt	SREDA disappears during active wakefulness
	3	Yes/NR/NR/NR	Yes, during sleep; stage not reported	NR	FT	4–5 Hz	Bilateral	No	Both synchronous and asynchronous	Abrupt	Abrupt	SREDA disappears during active wakefulness
Goeden et al., 2017[13]	4	Yes/NR/Yes/NR	NR	NR	TO	5–6 Hz	Unilateral	No	No	NR	NR	No
Dash et al., 2012[14]	5	Yes/NR/Yes/No	NR	NR	PT	4–5 Hz	Bilateral	Yes	No	Abrupt	Abrupt	No
Nagarajan et al., 2001[15]	6	Yes/Yes/Yes/NR	Yes/Yes/NR	NR	FT, tendency to diffusion	5–7 Hz	Bilateral	Both symmetric and asymmetric	NR	Both gradual and abrupt	Both gradual and abrupt	No
	7	Yes/NR/Yes/Yes	Yes/NR/NR	NR	TO and P	5–7 Hz	Bilateral	No	NR	Gradual	Both gradual and abrupt	No changing of SREDA with or without ASM
Santoshkumar et al., 2009[16]	8	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR
Ramesha et al., 2010[17]	9	Yes/Yes/Yes/Yes	Yes/No/NR	NR	T	6–18 Hz	Bilateral	NR	NR	NR	NR	NR
Thet et al., 2023[18]	10	Yes/NR/NR/Yes	Yes/No/NR	NR	F	NR	NR	NR	NR	NR	NR	NR
	11	Yes/NR/NR/Yes	Yes/No/NR	NR	F	NR	NR	NR	NR	NR	NR	NR
Case 1	12	Yes/Yes/No/Yes	Yes/No/NE	No	TO	5–6 Hz	Unilateral	No	No	Abrupt	Abrupt	No
Case 2	13	Yes/Yes/NE/Yes	No	NE	Right TP and left FO	5–7 Hz	Bilateral	No	Both synchronous and asynchronous	Abrupt	Abrupt	No
Case 3	14	Yes/No/Yes/NE	NE	NE	OP	5–6 Hz	Bilateral	Yes	Yes	Gradual	Gradual	No
Case 4	15	Yes/No/NE/Yes	Yes/Yes/NE	NE	FT	6 Hz	Bilateral	No	No	Abrupt	Abrupt	No
Case 5	16	Yes/No/NE/Yes	Yes/Yes/NE	NE	Right T and left TO	5–7 Hz	Bilateral	No	Yes	Gradual	Gradual	No

Abbreviations: ASM, antiseizure medication; C, central; F, frontal; HV, hyperventilation; IPS, intermittent photic stimulation; NE, not executed; NR, not reported; O, occipital; P, parietal; T, temporal.

attention deficit hyperactivity disorder, borderline intellectual functioning), generalized epilepsy, anxiety, and mood disorders.

Seizures started at the age of 7 months, with generalized motor episodes, responsive initially to phenobarbital (PB) and then to levetiracetam monotherapy (LEV). At the age of 6 years, a brain MRI revealed an intrasellar cystic lesion, and the patient underwent cyst fenestration via the trans-sphenoidal approach. Genetic investigations were performed: the results of Fragile X analysis and next-generation sequencing (NGS) of the epileptic encephalopathy panel were normal.

At 12 years of age, psychogenic nonepileptic seizures (PNES) were recorded during video EEG monitoring. During this exam, for the first time, we detected the presence of SREDA, which was not simultaneous with clinical PNES (Figure 2). SREDA persisted in subsequent EEGs, despite levetiracetam (LEV) discontinuation, whereas seizures and PNES completely disappeared during follow-ups.

3.1.2 | Case 2

Healthy 10-year-old girl born from a full-term pregnancy with no family history of neurological or psychiatric disorders. Neurodevelopmental milestones were achieved at the proper age. She received a specific learning disorder diagnosis. When she was 10, we recorded SREDA (Figure 2)

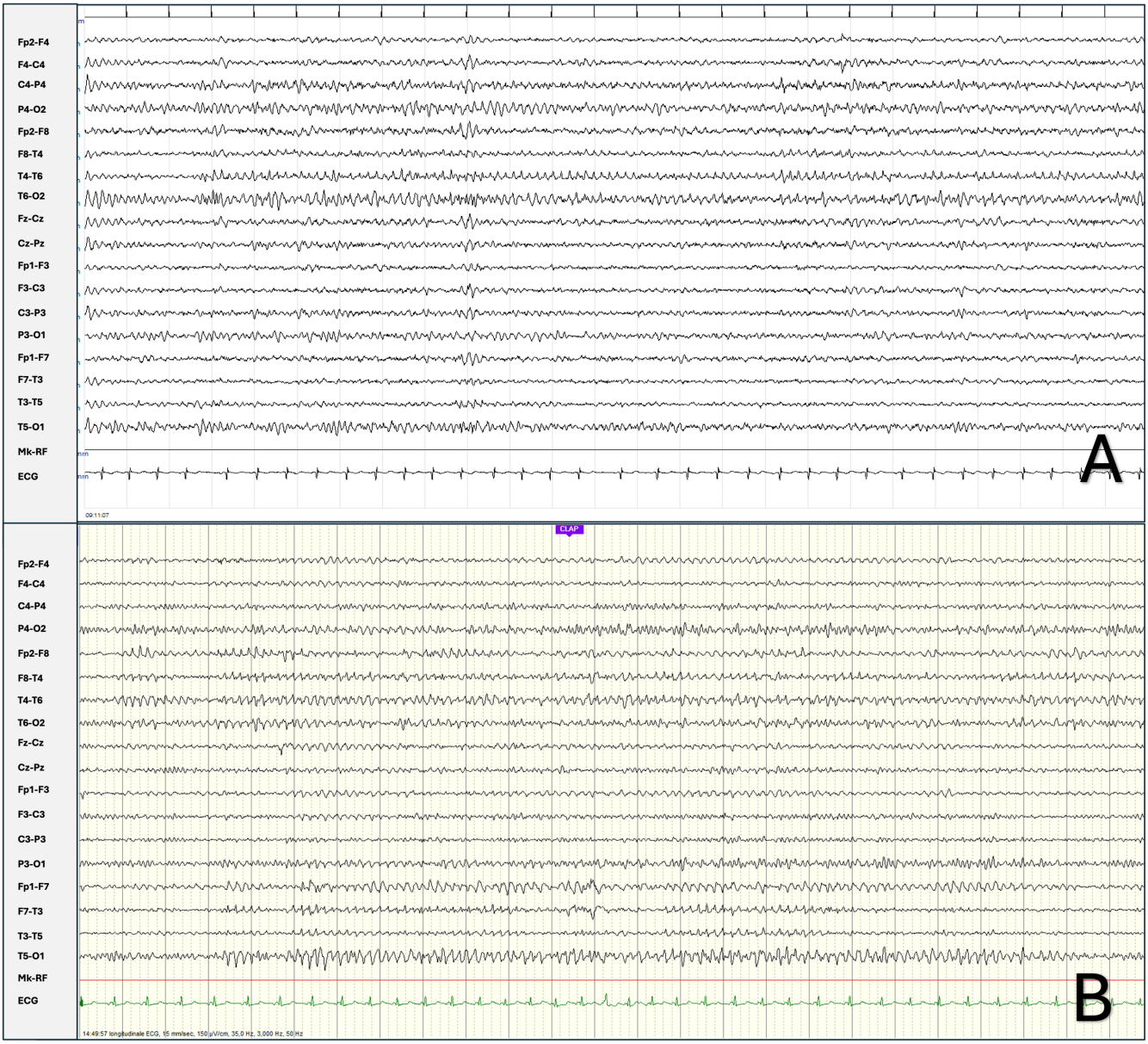

FIGURE 2 (A) Case 1 scalp EEG: Bipolar montage. SREDA in wakefulness. 25-Second epoch, sensitivity 7 μV/mm; (B) Case 2 scalp EEG: Bipolar montage. SREDA in wakefulness. 25-Second epoch, sensitivity 15 μV/mm.

during an EEG performed to investigate episodes of loss of consciousness further diagnosed as syncope.

3.1.3 | Case 3

Thirteen-year-old male with no family history of neurological or psychiatric disorders, suffering from mild intellectual disability. Brain MRI was not informative. At 11 years of age, an EEG was performed, as provided by the Gaslini Children's Hospital diagnostic protocol for neurodevelopmental disorders. During this recording, we detected SREDA (Figure 3).

3.1.4 | Case 4

Seventeen-year-old boy with a negative family history of neurological and psychiatric disorders. He showed normal psychomotor development and IQ. At the age of 7 years, he developed focal seizures due to left occipital hemorrhagic cerebral cavernous malformation (CCM). Topiramate (TPM) was started, and the CCM was subsequently surgically removed. At the age of 11, the boy developed psychiatric symptoms (emotional dysregulation, aggressiveness), which increased in frequency and duration when attempting to switch from TPM to LEV. During routine EEG, at the age of 13 years, SREDA was detected. It persisted at further follow-up visits, even after treatment withdrawal (Figure 3).

3.1.5 | Case 5

Eight-year-old girl, born prematurely. Neurodevelopmental milestones were achieved in time, and she had a normal IQ. Generalized seizures first appeared during febrile episodes and then even without fever when she was years old. LEV was started leading to complete seizure control. Brain MRI revealed agenesis of the septum pellucidum. From a genetic point of view, Array-CGH and panel sequencing of mTOR pathway-related genes were normal. HESX1, SOX2, and OTX2 analyses revealed the c.359 T > C p.Ile120Thr variant in the HESX1 gene, classified as a variant of uncertain significance. During a routine EEG at the age of 7 years, SREDA was recorded (Figure 3).

3.2 | Systematic review of the literature

In addition to our 5 cases, we identified a total of 11 pediatric subjects with this uncommon EEG variant. The clinical and electrophysiological data of the entire cohort are available in Tables 1 and 2. In the study by Santoshkumar et al.[16], no clinical and electrophysiological data were available. Beneath, we report the percentages of patients for whom specific clinical data were available.

Fifty percent (7/14) of the subjects were male; the mean age at first detection of SREDA was 11.5 years (age range 7–17 years, data available for 13 subjects). Fifty-three percent (8/15) of patients had a diagnosis of epilepsy, and 67% (10/15) had at least one seizure.

In 13% (2/15) of the children, SREDA was interpreted as epileptiform activity. These subjects started ASM because of the misinterpretation of SREDA and, one of them had a drug-related side effect.

In 12 cases (12/15, 80%), EEG was performed to better investigate potential seizure; one subject underwent EEG during the headache diagnostic workup, one due to post-traumatic amnesia, and the last one to further evaluate his neurodevelopmental disorder. The diagnosis of a neurodevelopmental disorder was concomitant in at least 8 subjects (Table 1).

Regarding the EEG pattern, 100% (15/15) of the subjects presented SREDA during wakefulness; when the EEG technician provided a simple motor or verbal task to the patient, this bioelectrical activity ended in 4 cases but persisted in one child. In 91% (10/11) of the subjects, SREDA appeared during the transition from wakefulness to sleep; in 86% (6/7) of patients during HV, and in 57% (4/7) during IPS. SREDA persisted during sleep in 92% (11/12) of the subjects: we detected SREDA more frequently during stage N1 than during N2. SREDA during stage N3 was not observed. Only one subject had SREDA reported during REM sleep (Table 2).

Eighty-five percent (11/13) of the patients had bilateral SREDA while in two subjects it was unilateral. SREDA was symmetric in 25% (3/12) of cases; synchronous in 20% (2/10); onset was abrupt in 64% (7/11), and the end was abrupt in 64% (7/11). All the cases had a theta-band frequency and 53% (8/15) of the children had an "atypical" SREDA distribution (over frontal or occipital areas) (Table 2).

4 | DISCUSSION

SREDA represents an uncommon benign EEG variant. Considering it is almost exclusivity in the adult population, there is a broad risk of misinterpretation when it occurs in children. Gil et al. and Nagarajan et al.[11,15] reported two cases of misdiagnosis of epileptic discharge instead of SREDA. Using EEG source analysis, Zumsteg et al.[9] reported that SREDA discharge occurs predominantly in

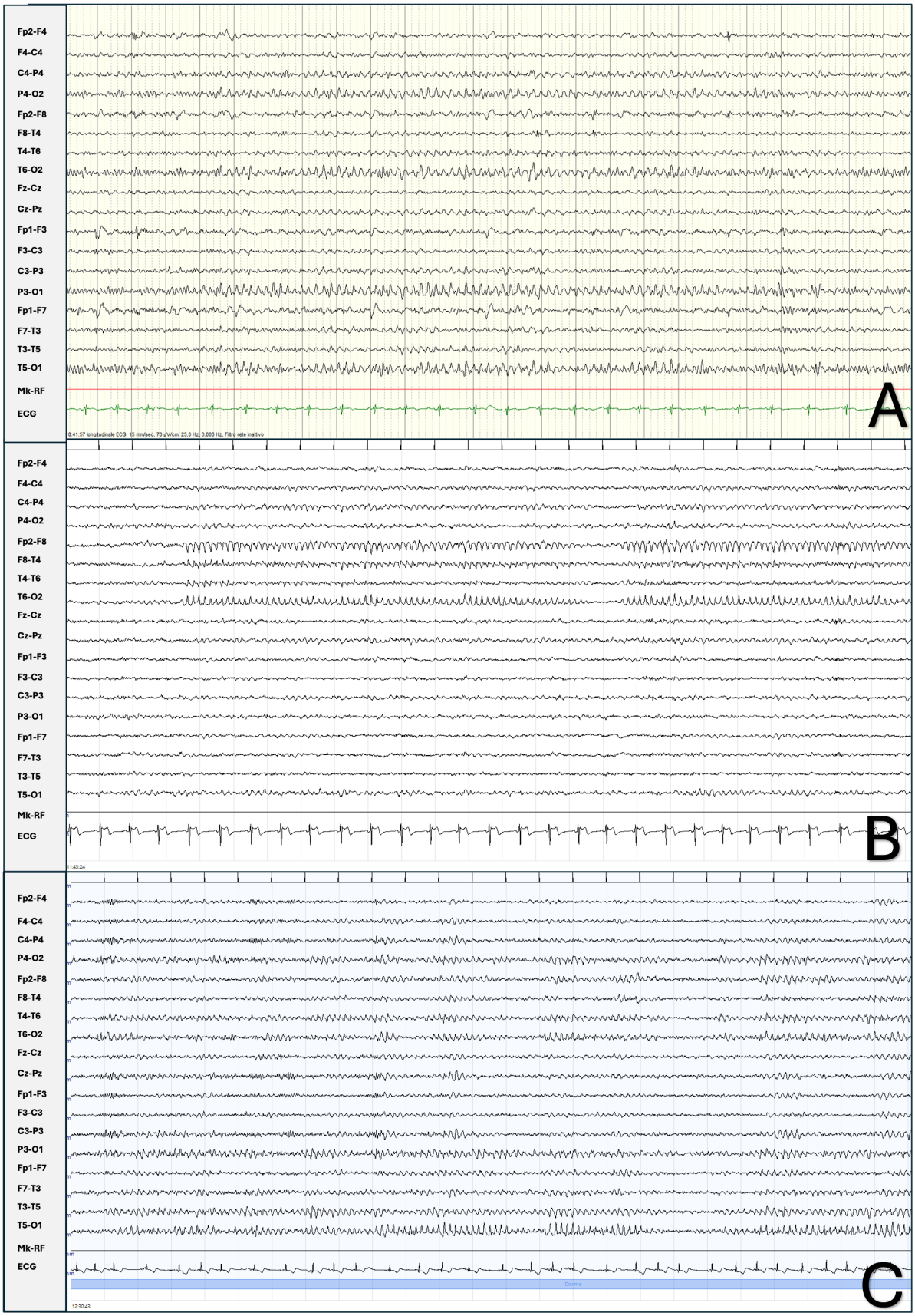

FIGURE 3 (A) Case 3 scalp EEG: Bipolar montage. SREDA in wakefulness. 25-Second epoch, sensitivity 7 μV/mm; (B) Case 4 scalp EEG: Bipolar montage. SREDA in wakefulness. 25-Second epoch, sensitivity 10 μV/mm; (C) Case 5 scalp EEG: Bipolar montage. SREDA in wakefulness. 25-Second epoch, sensitivity 14 μV/mm.

brain regions straddling the distribution territory of the anterior, middle, and posterior cerebral arteries, supporting hypoxic pathogenesis.

Carson et al.[19] instead reported that benzodiazepine administration (lorazepam) resulted in SREDA ending, similar to what occurs in epileptic discharges. In addition, Goeden et al.[13] suggested that SREDA in the pediatric population may be more frequent in subjects with generalized epilepsy.

Despite these reports, the literature data still do not seem sufficient to support a clear diagnostic hypothesis, nor an association with epilepsy or other neurological diseases.

In our Child Neuropsychiatry Unit, EEGs were performed during the diagnostic work-up for seizures, epilepsy, neurodevelopmental disorders, head trauma, and headache.

Therefore, attempting to associate SREDA with some anamnestic comorbidities may introduce sample selection bias. Considering this bias, SREDA was observed in 67% (10/15) of the subjects with previous seizures, including 80% (8/10) of the patients with an epilepsy diagnosis, 38% (3/8) of whom had a generalized epilepsy.

In our case series, which was collected at the Giannina Gaslini Institute, 3 out of the 5 subjects had a diagnosis of epilepsy, specifically: case 1 had generalized seizures that started at the age of 7 months; she never had any focal seizures, and epileptiform abnormalities on EEG were clearly differentiated from SREDA. Case 4 developed SREDA only after neurosurgical intervention (left occipital lesionectomy); EEG epileptiform abnormalities, before surgery, were left posterior on EEG, while SREDA was bilateral but prevalent on the right side; after surgery, this patient developed PNESs, confirmed by Video-EEG recording; the PNES episodes were unrelated to the occurrence of SREDA on EEG. Finally, case 5 was a child with generalized seizures and generalized interictal epileptic abnormalities, independent from SREDA on EEG recording.

Interestingly, we detected a high frequency of neurodevelopmental disorders (62%, 8/13 of cases), and several neuropsychiatric comorbidities have been described, e.g., neuromotor developmental delay, attention deficit, specific language disorder, specific learning disorder, mild intellectual disability, ADHD, and autism spectrum disorder.

We found only one pediatric case with a possible relationship between the presence of SREDA and an episode of transient amnesia, in contrast to what was reported in the adult population.[20,21]

Concerning electrophysiological findings, all the subjects (15/15) presented SREDA during wakefulness, and 92% (11/12) also during sleep. We observed a higher prevalence of abrupt onset and end of discharge than gradual ones. Topographically, SREDA occurred predominantly bilaterally and was localized in atypical regions (frontal and occipital).

The prevalence of this atypical variant appears to be higher than that in the adult population.[6]

In agreement with Rathore et al.[22], we also noted some cases in which SREDA was interpreted as epileptic discharges however, considering their rarity in children and the limited sample analyzed, the rate of diagnostic errors does not seem to differ from that of the adult population.

5 | CONCLUSION

SREDA may be present in the pediatric population, despite being considered a benign EEG variant typical of adulthood. Clinicians should recognize the SREDA EEG pattern and distinguish it from epileptic discharge to avoid inappropriate treatments.

We found a possible connection with neurodevelopmental disorders, but our data are insufficient to assume a correlation between the presence of this EEG pattern and a specific comorbidity or triggering cause. Further studies with homogeneous and larger patient samples are needed to clarify its significance. Finally, considering the increase in this EEG variant not only in adulthood but also in pediatrics, we propose to change the term SREDA to SRED-A for adults and SRED-P for pediatrics.

AUTHOR CONTRIBUTIONS

LB, MM, GN and EC: conception and design of the study, literature review and article selection, manuscript writing, and realization of figures and tables. LS, SB, GP and LN: participation in the writing of the manuscript. All authors contributed to the article and approved the submitted version.

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CONFLICT OF INTEREST STATEMENT

This study was partly conducted in the framework of the ERN EpiCARE. The authors declare no conflict of interests.

DATA AVAILABILITY STATEMENT

The original contributions presented in the study are included in the manuscript, further inquiries can be directed to the corresponding author/s.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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Test yourself

1. What are the typical EEG features of SREDA?
   A. Rhythmic sharp waves in the theta band with distributions mainly in the temporal and parietal regions
   B. Rhythmic sharp waves in the alpha band with distributions mainly in the frontal and temporal regions
   C. Rhythmic polispikes discharge with distributions mainly in the temporal and parietal regions
2. What is the age range in which the SREDA electroencephalographic variant is most commonly described?
   A. Childhood
   B. Young adults
   C. Older adults
3. In children with SREDA, what is the comorbidity most frequently described in the scientific literature?
   A. Epilepsy
   B. Neurodevelopmental disorder
   C. Cerebrovascular disease
4. The atypical variant of SREDA is more prevalent in which group of subjects?
   A. Children
   B. Older adults
   C. Prevalence is approximately the same among children and older adults

Answers may be found in the supporting information.