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The role of cannabis in epilepsy illustrated by two case reports

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ABSTRACT

Cannabis use is increasingly prevalent among individuals with epilepsy, yet its impact on seizure control remains poorly understood. While cannabidiol (CBD) has demonstrated antiseizure properties and gained FDA approval for specific epileptic syndromes, tetrahydrocannabinol (THC), the primary psychoactive compound in cannabis, may alter neuronal excitability and potentially exacerbate seizure activity. We present two illustrative case reports of male patients with focal epilepsy and chronic cannabis use who underwent treatment with antiseizure medications and responsive neurostimulation (RNS). In both cases, cannabis use was temporally associated with breakthrough seizures and poor seizure control. These cases highlight the complex and multifactorial relationship between cannabis use and seizure outcomes, including potential pharmacokinetic interactions with antiseizure medications (ASM) and the possibility that cannabis may blunt the neuromodulation effects of RNS. Given the retrospective data and limited detail on cannabis use, these findings should be interpreted with caution. As cannabis use rises among individuals with epilepsy, further research is needed to clarify its potential effects on seizures and treatment response, including neuromodulation.

1. Background

Within the realm of medicinal plants, Cannabis, commonly known as marijuana, draws a significant interest, mainly due to its primary components, tetrahydrocannabinol (THC) and cannabidiol (CBD), due to their distinct and impactful psychological effects. THC, primarily found as tetrahydrocannabinol acid (THCA), requires decarboxylation to convert into the pharmacologically active Δ9-THC, known for its psychoactive effects [1]. On the other hand, CBD, which does not exhibit euphoric psychoactive effects, has gained popularity as an anti-seizure agent [2]. Both components are part of a broader group of 125 cannabinoids identified in Cannabis, highlighting the plant's complex chemical profile [3]. While socially touted as a way to treat anxiety, depression, and other neuropsychological ailments, chronic THC use at high doses has been found to have pronounced anxiogenic effects, increased hyperactivity, decreased slow-wave sleep, and disruption of regular circadian cycles [1,2,4]. Whereas high-dose CBD has been found to increase sleep duration, improve sleep quality, and have sedating effects [4,5].

In conjunction with research into the psychological effects of cannabis use across the various constituent compounds, recent studies on cannabis-derived compounds, especially CBD, have broadened the spectrum of treatment options for refractory seizures. CBD has shown a promising result in reducing seizure frequencies without the psychoactive effects associated with THC. The growing acceptance of CBD in medical settings underscores the significance of embracing cannabis-derived therapies with drugs such as Epidiolex, a highly purified CBD-derived therapy, being FDA-approved for use in the treatment of Lennox Gastaut, Dravet Syndrome, and reports of successful treatment in other epileptic syndromes [6,7].

Abbreviations: **ASM**, Antiseizure Medication; **CBD**, Cannabidiol; **CB1**, Cannabinoid Receptor Type 1; **CB2**, Cannabinoid Receptor Type 2; **CNS**, Central Nervous System; **ED**, Emergency Department; **EMU**, Epilepsy Monitoring Unit; **FA**, Focal Aware; **FBTC**, Focal to Bilateral Tonic-Clonic; **FIA**, Focal Impaired Awareness; **ICU**, Intensive Care Unit; **PWE**, Patients with Epilepsy; **RNS**, Responsive Neurostimulation; **SEEG**, Stereo Electroencephalography; **THC**, Tetrahydrocannabinol; **THCA**, Tetrahydrocannabinol Acid.

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While the potential benefits of CBD in managing refractory seizures are promising, concerns remain regarding the levels of THC in widely available cannabis products. Recent data indicates that a significant number of cannabis products, especially those accessible in recreational and medical dispensaries, possess high THC concentrations, often exceeding 15 %, which can be highly intoxicating and may exacerbate risks associated with cannabis use [8,9]. Moreover, the evolving cannabis market has seen a rise in strains with higher THC-to-CBD ratios, potentially increasing the likelihood of adverse effects rather than providing the anticipated therapeutic benefit. Additionally, recent studies reveal significant mislabeling in commercially available CBD products, with only a portion meeting their label claims for full spectrum and CBD isolate, emphasizing the need for stricter testing requirements for commercially available cannabinoid products [10,11]. This current dearth of accurate reporting on the chemical makeup of commercially available cannabinoids poses unique challenges for patients attempting to receive the therapeutic benefits of CBD consumption without the potential downside of THC consumption.

In the following section, we present two case studies that demonstrate the relationship between cannabis use and epilepsy management. These cases underscore the need for further research into the pharmacological properties of cannabis, particularly its interactions with the central nervous system (CNS), antiseizure medications (ASM) and responsive neurostimulation in the context of seizure control.

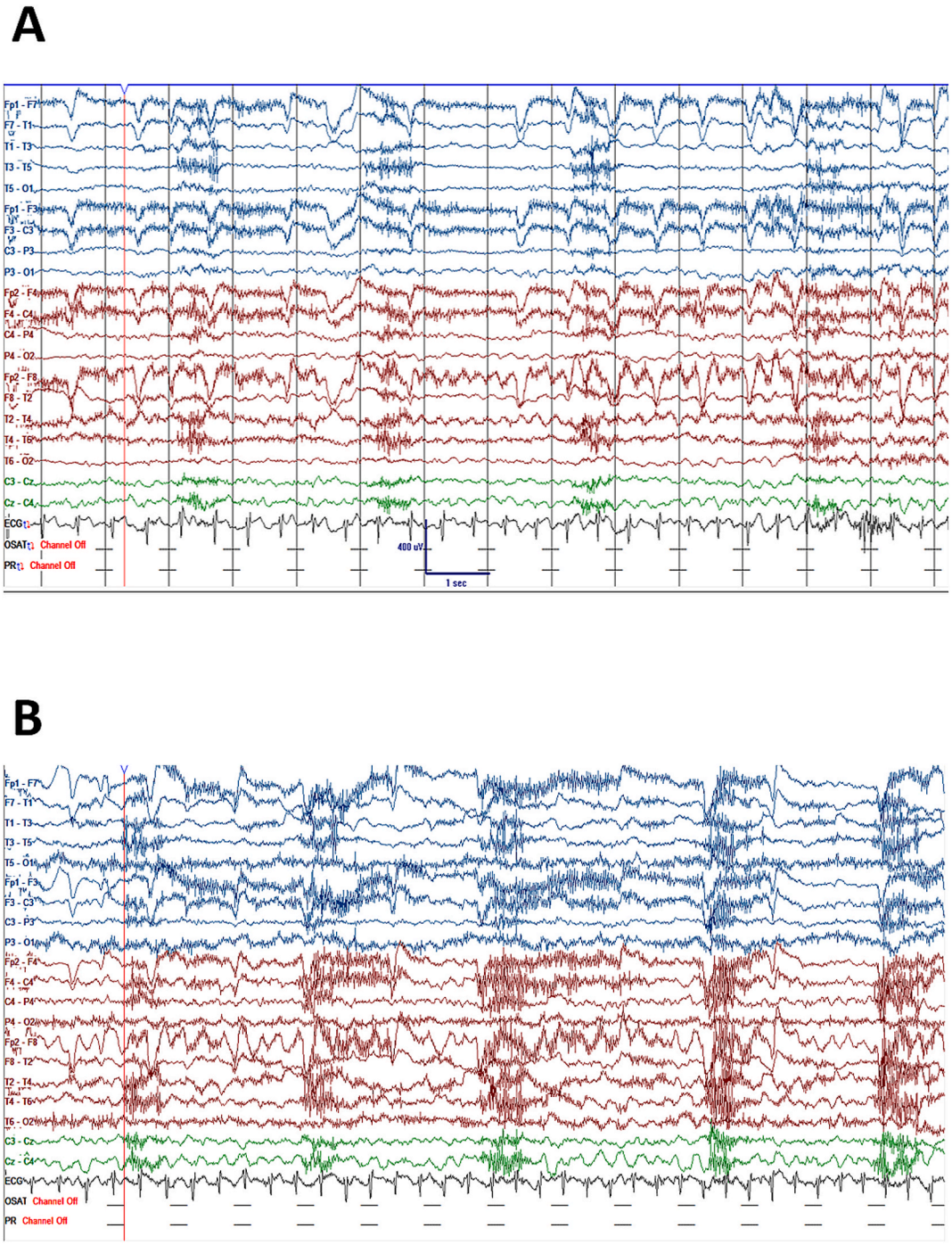

Fig. 1. SEEG. A) Stereo-EEG (sEEG) recording demonstrating ictal activity in the left temporal lobe. **B)** Another segment of the sEEG from the same patient showing left temporal discharges, further confirming the diagnosis of left temporal lobe epilepsy.

2. Case reports

2.1. Case 1

A 25-year-old male patient who first experienced seizures at the age of 14, medical history is negative for developmental delays, febrile seizures, and central nervous system infections, as well as familial history of epilepsy. His medical history is positive for polymicrogyria and heterotopia, a condition of neurodevelopment affecting the gyration of the cerebral cortex. The patient also reported a mild concussion that did not require medical care two months prior to the onset of seizures.

Over the next three years, the patient remained seizure-free while on lamotrigine as the sole ASM. At the age of 17, he had a focal to bilateral tonic clonic (FBTC) seizure which was potentially brought on by the start of daily use of cannabis. This seizure event necessitated a trip to the emergency department (ED) since it lasted from 3 to 5 min. Upon presentation to the ED and a full history of the events leading up to it, it was determined that there were no additional factors that precipitated the re-emergence of seizure activity. The patient reported that he was smoking upwards of 0.5 g/day of the cannabis flower.

Following this revelation of cannabis use, the patient was referred to the Center for Addictive Disorders; however, follow-up was inconsistent as the patient appeared to struggle with committing to appointments. In the months and years that followed, the patient experienced multiple FBTC seizures accompanied by focal aware (FA) and focal impaired awareness (FIA) events despite several medication regimens, including lamotrigine, oxcarbazepine, lorazepam, levetiracetam, zonisamide, and clobazam. During each appointment the patient indicated that daily use of cannabis was ongoing and was reminded about the use of cannabis potentially increasing seizure burden, and despite this the patient continued the use of the product. These seizure events were associated with cannabis use, as reported by the patient and confirmed by toxicology screens during emergency department (ED) and intensive care unit (ICU) admissions.

Four years after the initiation of cannabis use and refractory seizures, a stereo electroencephalography (SEEG) study was performed to evaluate the onset zone of seizure activity and elucidate potential new therapeutic options. SEEG confirmed a left temporal lobe seizure onset, and the patient subsequently underwent implantation of a responsive neurostimulation (RNS) device to reduce seizure frequency. (Fig. 1).

The patient remained seizure-free for nine months following the RNS implantation until the epilepsy team received a call from the family reporting a new FBTC seizure. Over the next two years, the patient continued to experience FBTC seizures, with at least one event every few months, requiring multiple ED admissions and telemedicine consultations. During these encounters, the patient admitted to daily cannabis use. The turning point occurred after a significant FBTC seizure during a weightlifting session. Following this event, the patient agreed with healthcare providers to discontinue cannabis use entirely. Since ceasing cannabis use, the patient has remained seizure-free for the past 18 months, as confirmed by long-term follow-up visits, hospital records, and telemedicine encounters.

2.2. Case 2

A 29-year-old male developed intractable epilepsy following a motor vehicle accident. He remained seizure-free for three months until a housemate found him actively seizing. His medical history includes familial epilepsy—his father had idiopathic seizures, and his stepsister experienced a single seizure episode unlinked to any diagnosis. He has no known CNS abnormalities, infections, or developmental delays. He has smoked 1.5 packs of cigarettes daily since the age of twelve.

Approximately one month after the seizure onset, the patient referred to daily use of cannabis, which soon became a clear trigger for seizure exacerbations. Clinician evaluations and patient interviews consistently linked drug exposure to seizure flare-ups, complicating seizure control. Despite this correlation, he continued to use cannabis products. Initially treated with levetiracetam, his condition worsened over time, requiring additional ASMs, including valproic acid and clobazam, with intermittent use of benzodiazepines during acute events. exacerbations.

Months later, he began experiencing monthly FBTC seizure events lasting 5–10 min, frequently followed by postictal aggression and confusion. Initial ASM treatment provided temporary relief, but over the next 18 months, seizure frequency and severity increased. As a result of poor seizure control, the patient was admitted to the epilepsy monitoring unit (EMU) for an inpatient stay in which the seizures were tracked using EEG. These were found to be localized in the right temporal region (Fig. 2**A and 2B**). Due to the progression of his condition, a RNS device was implanted 26 months post-accident. While RNS led to a reduction in seizure severity, its impact on frequency was limited.

Frequent seizures led to multiple hospitalizations for inpatient monitoring and medication adjustments. Toxicology screens repeatedly confirmed cannabis exposure during these episodes. Over time, his condition deteriorated into recurrent status epilepticus, requiring ICU admissions and intubation. Both healthcare providers and family members suspected a strong association between cannabis use and seizure exacerbation, based on repeated clinical patterns and toxicology results. Family members and medical providers strongly suspected cannabis as a key seizure trigger, based on both clinical observations and toxicology findings.

Despite clear medical evidence linking drug use to worsening seizures, the patient has been unable to maintain abstinence. Healthcare providers and family members have repeatedly emphasized the risks, yet he has struggled with adherence to recommendations. His continued cannabis use contributes to ongoing medical crises, frequent ED admissions, and persistent management challenges, making his epilepsy particularly difficult to control.

3. Discussion and Conclusion

These two case reports illustrate differing clinical contexts in which cannabis use coincided with exacerbated seizure activity. The first patient, a 25-year-old male with polymicrogyria, reported increased seizure frequency and clinical burden, that improved substantially following cannabis cessation. Conversely, the second patient, a 29-year-old male without any prior CNS pathology, developed a seizure disorder closely associated with his use of cannabis products. These cases raise concerns that, despite the therapeutic use of cannabinoids in certain contexts [11], cannabis, particularly THC-containing products, may play a role in seizure management for some individuals [12]. The exacerbation of seizure by cannabis in these instances invites a critical examination of the substance's neurological outcomes despite having a family or personal history of neurological conditions [13].

The varying responses to cannabis in the two cases illustrate the complex and potentially divergent effects of cannabinoids on seizure thresholds [2,6,14]. In the first patient, cessation of cannabis use led to a marked decrease in seizure frequency and severity, suggesting that cannabis may have been lowering the seizure threshold or interfering with the efficacy of the ASM from epilepsy related to an underlying structural etiology. The second patient's epilepsy remains cryptogenic, but his persistent use of cannabis in the context of worsening seizure control raises the possibility that cannabinoids served as an aggravating factor. That said, his inability to sustain a cannabis-free period limits any definitive conclusions regarding causality. This possibility is further complicated by the occurrence of status epilepticus episode temporally linked to cannabis exposure [15], particularly in the context of inconsistent use patterns and the lack of standardized thresholds for dosage amounts associated with increased seizure risk [16].

The diverse effects of cannabis on seizure activity can be attributed to its complex interaction with the CNS. THC, the primary psychoactive component of cannabis, acts on the brain's cannabinoid receptors, CB1

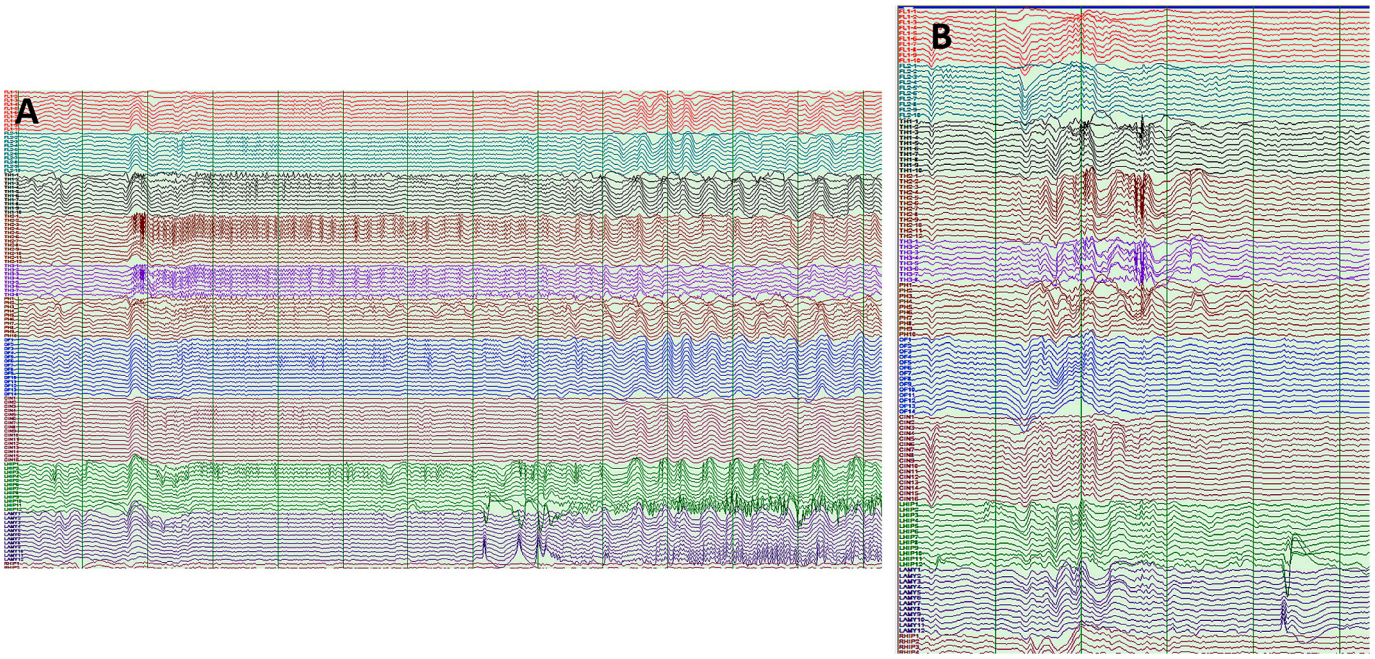

Fig. 2. EEG. A) Standard EEG recording at the EMU showing focal onset of right temporal seizure activity. **B)** Another segment demonstrating further propagation of seizure activity with high-amplitude rhythmic activity.

and CB2. The activation of CB1 receptors in the CNS can modulate neurotransmitter release and may disrupt the balance between excitatory and inhibitory pathways, critical for maintaining neuronal stability [3,17,18]. CB1 interacts with delta-9-THC and its derivatives, activating a negative feedback state. This interaction supports a theory suggesting that THC may induce an excitatory mechanism, potentially contributing to excitatory impulses resulting in seizure activity. For instance, in the second patient, the continued use of cannabis products may have contributed to increased CB1 receptor engagement, correlating with episodes of severe seizures, suggesting a potential potentiation of neuronal excitability.

Furthermore, the role of cannabis in alternating the GABAergic and glutamatergic functions could provide another layer of complexity in patients with epilepsy (PWE) who consume cannabis products. THC's potential to reduce GABA activity while enhancing glutamate activity may exacerbate abnormal neuronal firing, leading to seizures [14,16,19]. CBD, in contrast, does not engage CB1 with high affinity and appears to exert antiseizure effects through multiple non-cannabinoid pathways, including calcium channels, TRPV1 receptors, and adenosine modulation [16,20]. This mechanistic distinction may help explain why both of our cases, which involved daily cannabis use, were associated with persistent seizure activity despite medical and device-based interventions. In addition to these receptors and neurotransmitters effects, cannabis may also influence seizure control through interactions with pharmacologic treatments, particularly ASMs.

The clinical management of PWE using cannabis presents several challenges, as highlighted by the case studies. Cannabis can complicate seizure control due to its variable effects on neuropharmacology and potential interactions with ASMs. For instance, cannabis and certain ASMs may share pharmacokinetic pathways, particularly implicating cytochrome enzymes, leading to altered drug levels and efficacy [21]. In the cases presented, while one patient experienced improved control after discontinuing cannabis, suggesting an interaction that impeded ASM efficacy, the other continued to suffer severe episodes despite multiple medication adjustments.

The effectiveness of responsive neurostimulation (RNS) in managing focal epilepsy has been well documented, particularly over long-term follow-up, with seizure reductions ranging from 48 % to 66 % and significant quality-of-life improvements among treatment-resistant patients. [22,23] While seizure frequency often improves gradually over months or even years as stimulation parameters are optimized, clinical outcomes are highly variable and depend on factors such as seizure onset zone, lead placement, and underlying network dynamics. [24] In this context, it is plausible that external factors, including chronic cannabis use, may alter the trajectory of RNS efficacy. Cannabinoids have known neurophysiological effects [2,4,8,12,17], and ongoing exposure could modulate excitatory/inhibitory balance or affect the device's responsiveness to network patterns [16,19,20]. Our first patient's marked improvement following cannabis cessation raises the possibility that ongoing cannabis use may have interfered with the neuromodulation effects of the device.

Emerging models of RNS suggest that its therapeutic benefit may be more dependent on long-term network modulation than on acute seizure termination [24]. If cannabis use disrupts neural plasticity, delays seizure network reorganization, or alters neurostimulation thresholds, it may blunt the long-term efficacy of RNS [4,12,14,22]. Although some preliminary data suggest that certain patients may experience decreased epileptiform activity during periods of cannabis use, these findings are based on single-subject, uncontrolled reports and lack product standardization [25]. In our second case, continued cannabis consumption coincided with persistent seizure activity and eventual progression to status epilepticus, despite RNS implantation and medication adjustments.

This report is subject to several limitations, including its retrospective nature, reliance on patient self-report, and lack of standardized cannabis product characterization. Neither patient maintained a detailed seizure log from the onset of symptoms, and associations between cannabis use and seizure activity were drawn from clinical impressions and toxicology results rather than structured experimental data. Information regarding cannabis formulation, dosage, and mode of administration was also incomplete. In addition, variability in RNS stimulation parameters and adjustments were not consistently documented, making it difficult to assess potential interactions between neuromodulation and cannabis exposure with confidence. Given these limitations, the observations described should be interpreted cautiously, and any apparent associations should be viewed as hypothesis-

generating rather than conclusive.

These cases may reflect the complex and individualized nature of cannabis effects on seizure activity. While some patients may derive therapeutic benefit, others could experience less favorable outcomes, possibly due to a range of contributing factors such as underlying comorbidities, medication interactions, or cannabinoid composition. The contrasting outcomes observed in these patients may underscore the variability in individual responses, although causality remains uncertain. The findings tentatively suggest a need for more nuanced consideration of cannabis use in epilepsy care, particularly in patients undergoing device-based therapies such as RNS. Further prospective research is warranted to better understand whether, and under what conditions, cannabis might influence seizure dynamics or interact with neuromodulatory treatments.

CRediT authorship contribution statement

Santiago Philibert-Rosas: Writing – original draft, Resources, Data curation. **Cameron J. Brace:** Writing – original draft, Resources, Data curation. **Sanaa Semia:** Writing – original draft, Resources, Data curation. **Barry E. Gidal:** Writing – review & editing, Validation. **Bradley T. Nix:** Writing – review & editing, Validation. **Anne F. Josiah:** Writing – review & editing, Validation. **Melanie Boly:** Writing – review & editing, Validation. **Aaron F. Struck:** Writing – review & editing, Validation, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Ethical Statement

A waiver of consent was approved by the Institutional Review Board (IRB), as the research involves minimal risk, does not require procedures beyond standard clinical care, and all patient data have been fully de-identified in compliance with HIPAA regulations. The waiver was granted in accordance with institutional policies and ethical guidelines to protect patient privacy and confidentiality.

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