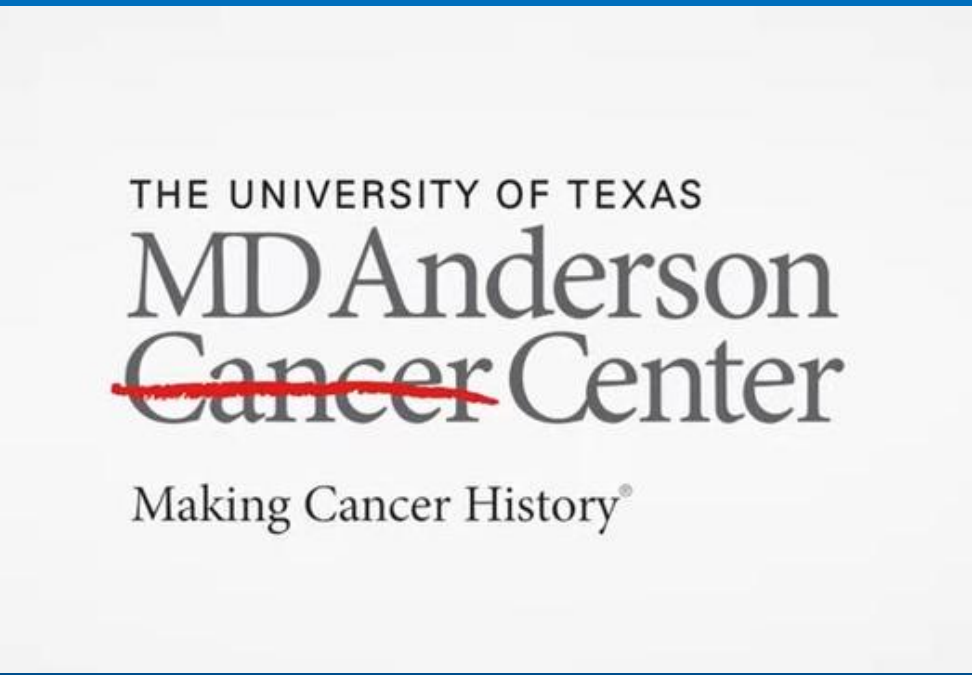
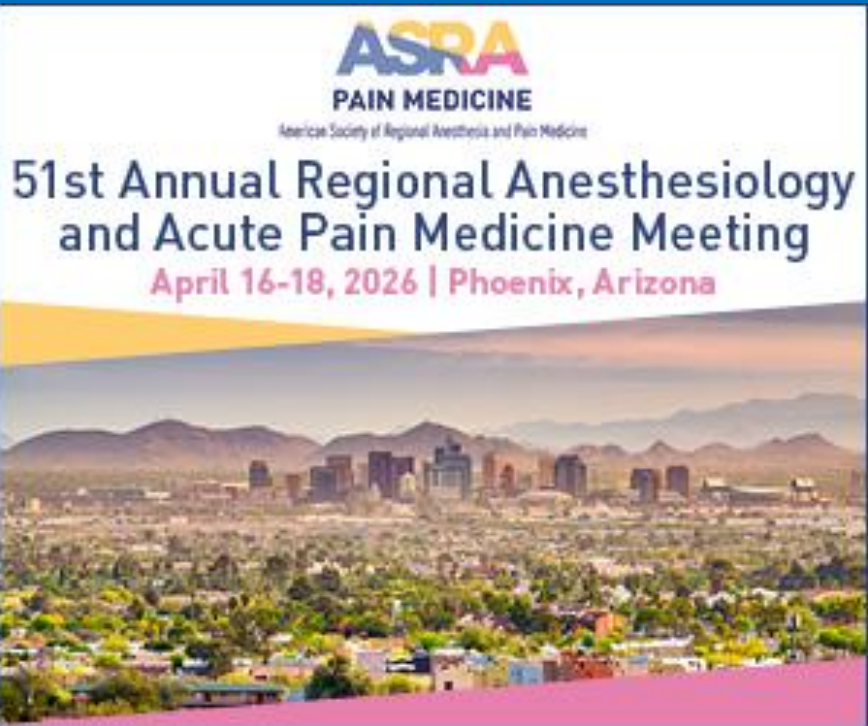


Successful Use of Suzetrigine for Treatment of Refractory Trigeminal Neuralgia in Four Patients

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Introduction

- Suzetrigine selectively blocks the Nav1.8 voltage-gated sodium channel in peripheral neurons [1].
- Trigeminal neuralgia (TN) is a neuropathic pain syndrome affecting the trigeminal nerve and is traditionally treated with non-selective sodium channel blockers.
- Animal and human models have implicated altered Nav1.8 expression in TN and neuropathic pain disorders[2-5], though pattern and degree require more study.
- No studies have evaluated Suzetrigine for TN, but potential involvement of Nav1.8 in TN signaling may represent a novel therapeutic target.

Goal

- We describe four cases of patients with refractory TN who responded to Suzetrigine therapy. As Suzetrigine is currently only FDA approved as an analgesic in the U.S. for adults with moderate-to-severe acute pain, this is an off-label use of the medication.

Methodology

- In all cases, other diagnoses were ruled out via neurological exam, MRI brain, dental exams and/or eye exams. Table 1 describes unsuccessful interventions trialed before consideration of Suzetrigine. All information was gathered via direct patient interview and Electronic Medical Records review.
- As the case report is devoid of patient identifiable information, it is exempt from IRB requirements as per MD Anderson Cancer Center policy. Patient informed consent was obtained.

Case Report

Case 1: 58-year-old female with hypertension and hypothyroidism was diagnosed with classic TN per ICHD-3 criteria. We initiated Suzetrigine 50 mg BID and titrated to 100 mg BID, with >70% reduction in paroxysms at 8 weeks.

Case 2: 76-year-old male with atrial fibrillation on apixaban and CKDIII presented with lancinating facial pain in left V1–V2 distribution with tactile triggers and brief duration, meeting TN criteria. Constant background burning suggested mixed phenotype. Started Suzetrigine 50 mg BID and titrated to 100 mg BID, with 60% reduction in paroxysms and background dysesthesia at 6 weeks.

Case 3: 34-year-old female with clinically distinct migraine without aura and generalized anxiety disorder presented with classic TN symptoms. Started suzetrigine at 50 mg BID with improvement after 2 weeks and >80% reduction in attack severity and frequency at 3 months.

Case 4: 52-year-old male with diabetes and obstructive sleep apnea presented with right-sided V1–V2 shock-like TN paroxysms following shingles 9 months earlier. Dermatology confirmed healed zoster. Allodynia on exam but no dense sensory loss, making neuropathic deafferentation unlikely. Suzetrigine started at 50 mg BID and titrated to 100 mg BID with decrease in paroxysms from 15 to 3-4/day and improved allodynia at 3 months.

Table 1: Interventions Trialed before Initiation of Suzetrigine

Patient	Intervention	Reason for Stopping
1	Carbamazepine	Transaminitis
	Oxcarbazepine	Hyponatremia
	Pregabalin	Extreme fatigue
	Microvascular decompression	Partial relief
2	Carbamazepine	Conduction issues
	Gabapentin	Minimal Improvement
	Gamma Knife Radiosurgery	Minimal Improvement
3	Carbamazepine	Severe cognitive slowing
	Oxcarbazepine	Intolerance
	Topiramate	Paresthesias
	Peripheral Nerve Blocks	No improvement
	Duloxetine	No symptomatic benefit
4	Gabapentin	Minimal improvement

Conclusions

- We present a novel use of Suzetrigine for treatment of refractory TN. We describe four patients with varying symptomatology of TN and failed responses to pharmacological and interventional treatments who exhibited notable improvements with Suzetrigine therapy.
- Till date, there were no adverse effects of Suzetrigine in these four patients at our institution. Further study is required in larger populations regarding safety and efficacy of long-term Suzetrigine use for treatment of TN

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