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Review article

Painful tic convulsif: Dual neurovascular compression within the spectrum of cranial nerve hyperactive dysfunction syndromes

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ABSTRACT

Background: Concurrent trigeminal neuralgia (TN) and hemifacial spasm (HFS), historically termed painful tic convulsif, is a rare manifestation of cranial nerve hyperactive dysfunction associated with neurovascular compression. The operative anatomy may involve independent conflicts affecting cranial nerves V and VII or shared compression, particularly in vertebrobasilar dolichoectasia.

Methods: We report a case of medication-refractory TN with ipsilateral HFS treated by microvascular decompression (MVD) and integrate the findings with a focused review of 23 studies addressing combined cranial nerve hyperactive dysfunction and dual neurovascular compression.

Results: A 35-year-old male presented with TN in the V2-V3 distribution followed by progressive ipsilateral HFS. High-resolution MRI showed neurovascular contact of the trigeminal nerve by the superior cerebellar artery and facial nerve compression by a separate posterior inferior cerebellar artery loop. Retrosigmoid MVD confirmed independent dual conflicts. Decompression of both nerves produced immediate resolution of pain and spasms, with sustained symptom-free status at two-year follow-up. Published reports support neurovascular compression as the principal surgically treatable mechanism in painful tic convulsif, with independent dual conflicts and shared ectatic vertebrobasilar compression both described.

Conclusions: Painful tic convulsif is best considered within the spectrum of cranial nerve hyperactive dysfunction. Comprehensive inspection of both the trigeminal and facial nerves during MVD is essential, because missed offending vessels may lead to persistent or recurrent symptoms. When all responsible conflicts are identified and decompressed, single-session MVD can provide durable relief in appropriately selected patients.

1. Introduction

Cranial nerve hyperactive dysfunction syndromes comprise disorders characterized by episodic, stimulus-triggered symptoms, most commonly trigeminal neuralgia (TN), hemifacial spasm (HFS), and glossopharyngeal neuralgia. These conditions are widely attributed to vascular compression at the root entry or exit zone of the affected nerve, a concept initially proposed by Gardner and Miklos and later refined by Jannetta [1,2]. Chronic arterial pulsation at this transition zone may lead to focal demyelination and ephaptic transmission, resulting in neuronal hyperexcitability. Accordingly, microvascular decompression (MVD) has become an established treatment for medically refractory cases, with large surgical series demonstrating durable relief and acceptable morbidity [3–5].

Although TN and HFS are well-established clinical entities, their coexistence in a single patient is uncommon. This combined presentation, traditionally termed painful tic convulsif (PTC), reflects the association of paroxysmal facial pain with involuntary hemifacial contractions. Early clinical and surgical reports showed that posterior fossa lesions or neurovascular compression may affect multiple cranial nerves, supporting the concept of combined cranial nerve hyperactivity rather than a separate nosological entity [6–13].

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A key question is whether concurrent TN and HFS arise from a single offending vessel or from independent neurovascular conflicts. Published operative reports describe both patterns. In non-dolichoectatic cases, the trigeminal nerve is often compressed by the superior cerebellar artery, while the facial nerve is affected by the anterior or posterior inferior cerebellar artery; in other cases, a tortuous or dolichoectatic vertebrobasilar system may contribute to shared or multilevel compression [8,14–17].

Despite advances in high-resolution MRI, accurate preoperative identification of all offending vessels remains challenging. Small arterial branches, venous compression, distal nerve involvement, and complex cerebellopontine angle anatomy may be overlooked [18,19]. As a result, incomplete decompression is a recognized cause of persistent or recurrent symptoms, and systematic intraoperative exploration of both nerves is recommended when dual hyperactive dysfunction is suspected.

Anatomical and developmental factors may further contribute to susceptibility, particularly in younger patients. Variations in posterior fossa morphology, arterial tortuosity, vertebrobasilar dolichoectasia, and cerebellopontine angle crowding have been proposed as predisposing factors [20–26]. These findings suggest that PTC reflects an interaction between structural vulnerability and dynamic neurovascular forces rather than coincidental coexistence alone.

Given its rarity, we believe that well-documented cases remain useful for clarifying mechanisms, diagnostic limitations, and surgical strategy. The present study combines a representative clinical case with a focused literature review to characterize neurovascular compression patterns in concurrent TN and HFS, with particular focus on distinguishing shared from independent conflicts and optimizing surgical management.

2. Case Presentation

Clinical History and Examination: A 35-year-old right-handed man with no relevant medical history presented with a 2-year history of left-sided trigeminal neuralgia in the V2–V3 distribution. The pain was brief, electric-shock-like, and triggered by chewing, speaking, toothbrushing, and cold exposure. Carbamazepine up to 800 mg/day provided only transient partial relief, while gabapentin and baclofen were ineffective. Eight months after the onset of trigeminal neuralgia, he developed intermittent ipsilateral hemifacial spasm, initially involving the periorbital region and later progressing to the perioral muscles. The spasms became frequent and were exacerbated by stress and fatigue. Neurological examination demonstrated preserved trigeminal sensation, intact corneal reflexes, symmetric facial motor function, but visible left hemifacial spasm. No additional cranial nerve, cerebellar, or long-tract deficits were identified.

Preoperative Imaging: High-resolution MRI demonstrated contact between the superior cerebellar artery and the root entry zone of the left trigeminal nerve (Fig. 1A). A separate posterior inferior cerebellar artery loop was seen compressing the facial nerve at its root exit zone (Fig. 1B). No shared arterial loop was identified. There was no evidence of a mass lesion, demyelinating disease, abnormal venous structures, cerebellopontine angle crowding, or vertebrobasilar dolichoectasia.

Surgical Procedure: Microvascular decompression was performed via a standard retrosigmoid approach under general anesthesia. The patient was positioned in the park-bench position, with the head flexed and rotated to optimize exposure. A limited retrosigmoid craniotomy measuring approximately 2.5-3 cm was performed. After cerebrospinal fluid release, microsurgical arachnoid dissection was carried out. The trigeminal nerve was explored first, followed by systematic inspection of the facial nerve regardless of imaging findings, to identify both independent and shared neurovascular conflicts. Offending vessels were mobilized away from the nerve root entry or exit zones and separated using Teflon pledgets to maintain durable decompression. Adjacent cranial nerves and venous structures were inspected for additional compression. Hemostasis was achieved, and the dura was closed in a watertight fashion. Postoperative care included serial neurological examinations and follow-up assessment of symptom resolution.

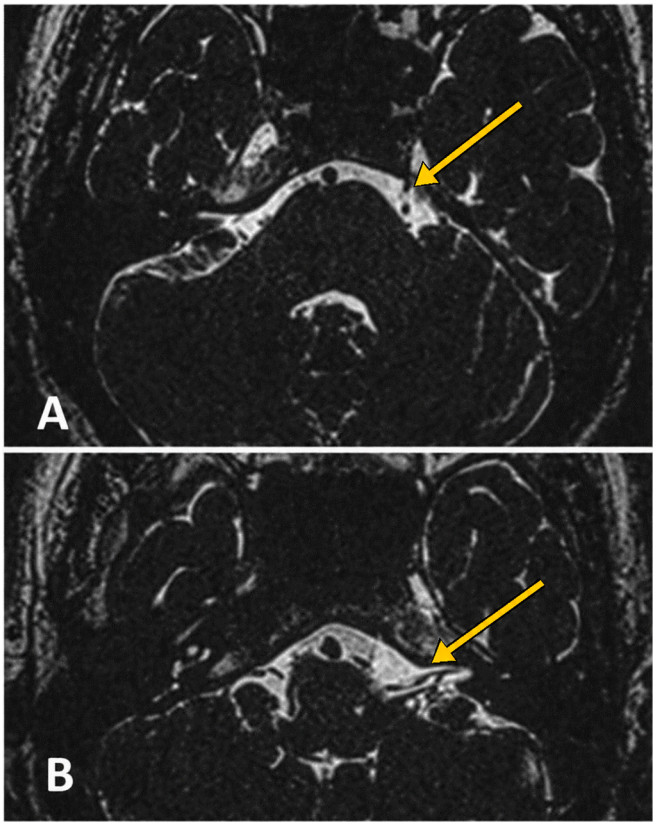

Fig. 1. Preoperative high-resolution MRI demonstrating neurovascular compression. (A) Superior cerebellar artery contacting the trigeminal nerve root entry zone; the yellow arrow marks the neurovascular conflict. (B) Posterior inferior cerebellar artery contacting the facial nerve root exit zone; the yellow arrow marks the neurovascular conflict. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Intraoperative Findings: Intraoperative findings confirmed independent neurovascular compression of the trigeminal and facial nerves. The trigeminal nerve was compressed by the superior cerebellar artery (Fig. 2A), and the facial nerve was compressed by a separate posterior inferior cerebellar artery loop (Fig. 2C). Both vessels were mobilized and separated from the respective nerves using Teflon interposition (Fig. 2B, D). No venous compression or shared vascular complex was identified.

Postoperative Outcome and Follow-up: Pain and hemifacial spasm resolved immediately postoperatively. The postoperative course was uneventful. Follow-up MRI confirmed maintained decompression. The patient remained symptom-free at 2-year follow-up.

3. Structured Review: Methods

3.1. Literature search and study selection

A focused literature review was conducted to identify studies describing the coexistence of trigeminal neuralgia (TN) and hemifacial spasm (HFS), commonly referred to as painful tic convulsif. Two authors (T.N. and N.T.) independently searched PubMed/MEDLINE, Scopus, and Web of Science without restriction on October 25, 2025. Only studies involving human subjects published in English were included. Reports of mixed etiologies were considered if neurovascular compression cases were clearly delineated.

Search terms included combinations of “painful tic convulsif,” “trigeminal neuralgia AND hemifacial spasm,” “combined cranial nerve

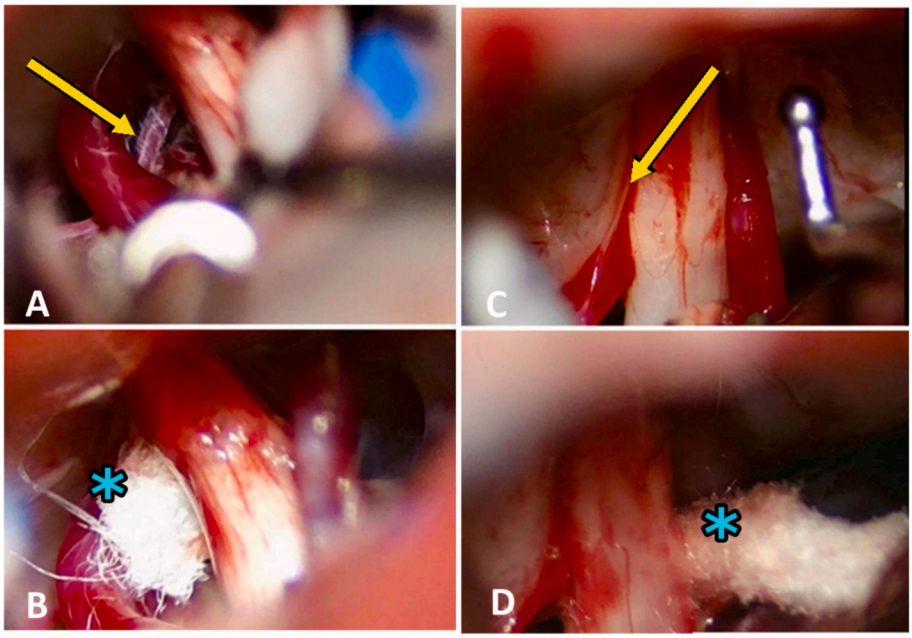

Fig. 2. Intraoperative findings demonstrating independent neurovascular compression of the trigeminal and facial nerves. (A) Trigeminal nerve (cranial nerve V, yellow arrow) compressed by the superior cerebellar artery. (B) Trigeminal nerve after microvascular decompression; the cyan asterisk marks the Teflon interposition. (C) Facial nerve (cranial nerve VII, yellow arrow) compressed by a loop of the posterior inferior cerebellar artery. (D) Facial nerve after microvascular decompression; the cyan asterisk marks the Teflon interposition. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

hyperactive dysfunction," "dual neurovascular compression," and "microvascular decompression," along with anatomical terms such as "root entry zone" and "cerebellopontine angle." Reference lists were manually screened, and forward citation tracking was performed to identify additional studies.

All records were imported into a citation management system, and duplicates were removed. Titles and abstracts were screened independently by two reviewers, followed by full-text assessment of eligible studies. The review was designed as a focused literature review rather than a formal systematic review; therefore, no PRISMA flow diagram, risk-of-bias assessment, or meta-analysis was performed, and findings were summarized descriptively.

3.2. Eligibility criteria and data extraction

Studies were included if they reported patients with concurrent TN and HFS confirmed clinically, radiologically, or intraoperatively, and provided operative findings or clinical outcomes. Both case reports and surgical series were eligible for inclusion.

Studies were excluded if symptoms were secondary to tumors, cysts, aneurysms, multiple sclerosis, or trauma; if neurovascular compression was insufficiently documented; or if postoperative outcomes were not clearly reported.

Data extraction was performed independently by two authors and cross-checked for accuracy. Extracted variables included demographics, symptom laterality and sequence, offending vessels for cranial nerves V and VII, presence of shared versus independent compression, imaging findings, surgical technique, outcomes, recurrence, follow-up duration, and complications. Due to heterogeneity in study design and reporting, results were summarized descriptively without quantitative analysis.

4. Results

Database searches identified 214 records. After removal of 63 duplicates, 151 abstracts were screened, and 59 full-text articles were assessed. Twenty-three publications met the inclusion criteria. Because of heterogeneity, small sample size, and partial overlap among historical reports, results were summarized descriptively without quantitative analysis. Table 1 summarizes selected reports with operative or outcome details.

Early descriptions of painful tic convulsif appeared in clinical and surgical reports of combined cranial nerve hyperactive dysfunction and neurovascular compression [7–13]. Larger contemporary reports and pooled analyses support neurovascular compression as the principal surgically treatable mechanism, while broader reports also describe secondary causes such as tumors, epidermoid cysts, demyelination, and vertebrobasilar dolichoectasia in selected cases [14–17].

Demographic and Clinical Characteristics: Most patients in published reports were middle-aged or older, although younger cases have been described [14–17]. Symptom sequence varies; pooled data suggest that HFS-first presentations are common, whereas TN-first and simultaneous presentations are also reported [17].

Neurovascular Compression Patterns: Operative findings consistently show that both cranial nerves V and VII must be evaluated when TN and HFS coexist. Independent compression by separate vessels is a recurring pattern in non-dolichoectatic cases, whereas a single ectatic vertebrobasilar system may cause shared or multilevel compression in another subset [8,14–17,20–23].

Imaging-Operative Discordance: Although MRI frequently demonstrates neurovascular contact, small arterial branches, venous conflicts, and distal compression sites may be missed [18,19]. This discrepancy explains differences between imaging and operative findings and supports systematic exploration of both nerves when dual dysfunction is suspected.

Surgical Outcomes: Retrosigmoid MVD was the primary treatment across surgical reports and generally resulted in high rates of symptom resolution, with recurrence reported in a minority of cases [8,14–17,27–29]. Selected surgical outcomes are summarized in Table 2.

Vertebrobasilar Dolichoectasia and Anatomical Predisposition: A subset of patients involved vertebrobasilar dolichoectasia or multivessel crowding, where enlarged or tortuous arteries caused broader

Table 1
Selected reports with operative and outcome details in painful tic convulsif and related combined cranial nerve hyperactive dysfunction.

Study	Year	Patients (n)	Age	TN offending vessel / cause	HFS offending vessel / cause	Compression configuration	Outcome
Cook & Jannetta [8]	1984	11	NR	CN V vascular compression in most cases	CN VII vascular compression in operative series	Dual neurovascular compression	8/11 pain-free and 8/11 spasm-free at mean 6 years 2 months
Tsuyumu & Kohmo [10]	1991	1	77	SCA	AICA/PICA	Independent dual compression	Pain- and spasm-free at 18 months
Grigoryan et al. [11]	1991	1	NR	Contralateral vertebral artery	Contralateral vertebral artery	Shared vertebrobasilar compression	Complete postoperative relief reported
Zhong et al. [14]	2011	9	NR	Variable	Variable	Predominantly vertebral artery-related compression causing coexistent ipsilateral HFS and TN,	Immediate or delayed improvement in both symptoms
Zhang et al. [15]	2012	4	NR	Variable; three NVC cases	Variable; three NVC cases	Three neurovascular compression cases; (one epidermoid cyst case reported separately)	Good postoperative relief in surgical cases
Wang et al. [16]	2014	6	NR	Looped VBA / crowded posterior fossa	Looped VBA / crowded posterior fossa	Multinerve compression with looped VBA/crowded posterior fossa	TN resolved immediately in 4/6 and within 2 weeks in 2/6; HFS stopped immediately in 4/6 and within 3 months in 2/6
Yin et al. [17]	2021	192	Median 54	VBA common among single-vessel causes	VBA common among single-vessel causes	Mixed patterns; VBA common among single-vessel causes	MVD cure rate > 80% in pooled analysis

Abbreviations: TN – trigeminal neuralgia; HFS – hemifacial spasm; SCA – superior cerebellar artery; AICA – anterior inferior cerebellar artery; PICA – posterior inferior cerebellar artery; NR – not reported; NVC – neurovascular compression; MVD – microvascular decompression; VBA – vertebrobasilar artery.

Table 2
Selected surgical outcomes reported in painful tic convulsif and related combined cranial nerve hyperactive dysfunction.

Study	Patients (n)	Resolution of TN	Resolution of HFS	Recurrence/complications
Cook & Jannetta [8]	11	8/11 pain-free; 1/11 mild discomfort; 2/11 recurrence	8/11 spasm-free; 2/11 trace residual spasm	Mean follow-up 6 years 2 months
Tsuyumu & Kohmo [10]	1	Pain-free	Spasm-free	No recurrence at 18 months
Zhong et al. [14]	9	Immediate or delayed relief reported	Immediate or delayed cessation reported	No major recurrence or complications reported
Zhang et al. [15]	4	Good postoperative relief in surgical cases	Good postoperative relief in surgical cases	One case due to epidermoid cyst rather than NVC
Wang et al. [16]	6	TN disappeared immediately in 4/6 and within 2 weeks in 2/6	HFS stopped immediately in 4/6 and within 3 months in 2/6	Favorable outcomes, no recurrence or cranial nerve dysfunction reported
Yin et al. [17]	192	Overall PTC MVD cure rate > 80%	Overall PTC MVD cure rate > 80%	Older age associated with recurrence risk

posterior fossa compression and required more complex surgical strategies [20–23,30]. Occasional younger cases suggest a role for developmental or structural factors, including reduced posterior fossa dimensions, cerebellopontine angle crowding, and variant arterial anatomy [24–26].

5. Discussion

Painful tic convulsif (PTC), defined as the ipsilateral coexistence of trigeminal neuralgia (TN) and hemifacial spasm (HFS), is an uncommon manifestation within the spectrum of cranial nerve hyperactive dysfunction syndromes. Early neurosurgical observations established that vascular compression within the cerebellopontine angle can involve multiple cranial nerves, producing combined symptom complexes rather than isolated disorders [6–13]. Despite the frequency of TN and HFS individually, their coexistence remains rare and represents only a small proportion of patients undergoing MVD [14,17].

A central finding across surgical reports is that neurovascular compression is usually present when PTC is treated operatively, but the configuration varies. Independent compression of the trigeminal and facial nerves is frequently reported in non-dolichoectatic cases, whereas shared or multilevel compression from an ectatic vertebrobasilar system accounts for an important subgroup [8,14–17,20–23]. In the present case, the trigeminal nerve was compressed by the SCA and the facial nerve by a separate PICA loop, consistent with an independent dual-compression pattern.

This distinction has important surgical implications. Limiting exploration to the nerve responsible for dominant symptoms risks missing a second compression site. Therefore, systematic intraoperative inspection of both the trigeminal and facial nerves is essential whenever dual hyperactive dysfunction is suspected [14,17]. In this case, sequential exploration enabled identification and effective decompression of both neurovascular conflicts.

Although high-resolution MRI improves visualization of neurovascular relationships, it has important limitations. Small arterial branches, venous compression, and distal nerve involvement may not be detected, and discrepancies between imaging and intraoperative findings are well documented [18,19].

The relatively young age of this patient is notable. Most reported patients are middle-aged or older [14–17], suggesting that earlier onset may reflect underlying anatomical predisposition. Factors such as reduced posterior fossa volume, cerebellopontine angle crowding, and variant vascular anatomy may increase susceptibility to symptomatic neurovascular compression at a younger age [24–26].

A distinct subgroup of patients presents with vertebrobasilar dolichoectasia, in which enlarged or tortuous arteries produce multilevel compression within the posterior fossa [20–23,30]. These cases may require more complex surgical strategies, including vascular mobilization or transposition, due to reduced vessel mobility and broader anatomical involvement.

MVD remains the main surgical treatment for symptomatic neurovascular compression refractory to medical therapy. Large surgical series report high rates of symptom resolution and durable long-term outcomes when decompression is complete [3–5,27–29]. In patients

with PTC, outcomes are favorable when both compression sites are identified and treated during a single procedure [8,14–17]. In broader trigeminal neuralgia care, multidisciplinary assessment can help confirm the clinical phenotype, exclude mimics, optimize medical therapy, and support informed selection of surgical or non-surgical options in refractory presentations [31].

Technical factors may influence surgical success. Teflon interposition is widely used to maintain nerve-vessel separation, but excessive material or suboptimal placement can lead to persistent symptoms, inflammatory reactions, or granuloma formation [32,33]. In persistent or recurrent HFS, missed compression zones, adhesions, and Teflon-related factors may require revision surgery [34,35]. Delayed symptom resolution has also been reported and likely reflects gradual recovery after decompression [36].

Beyond vascular contact alone, current models emphasize intrinsic nerve vulnerability, particularly at the root entry or exit zone. Focal demyelination, indentation severity, and structural susceptibility are thought to contribute to pathological hyperexcitability in TN and HFS [37–39]. Recent imaging and anatomic reviews further support careful interpretation of neurovascular contact, emphasizing the vulnerable transition zone/central myelin segment and the greater relevance of nerve deformity or displacement than simple contact alone [40,41]. Larger combined-HDS series confirm that combined syndromes are uncommon and heterogeneous, while bilateral PTC case literature illustrates that multiple independent neurovascular conflicts may occur in the same patient [42–45]. Within this framework, PTC can be viewed as part of a broader continuum of cranial nerve hyperactive dysfunction driven by both anatomical predisposition and neurovascular interaction.

This study has several limitations. The available literature is largely composed of retrospective series and case reports with heterogeneous methodology, limiting direct comparison and precluding meta-analysis. Nevertheless, the recurring anatomical patterns and favorable surgical outcomes across reports support the practical value of systematic exploration of both nerves. Overall, PTC is best approached as a combined cranial nerve hyperactive dysfunction syndrome in which all potential neurovascular conflicts must be identified and treated.

6. Conclusion

Painful tic convulsif is best understood as part of the spectrum of cranial nerve hyperactive dysfunction syndromes rather than the coincidental coexistence of two unrelated disorders. Neurovascular compression is the dominant surgically treatable mechanism, but the offending anatomy may involve either independent compression of the trigeminal and facial nerves or shared/multilevel compression from an ectatic vertebrobasilar system. Although modern imaging contributes to surgical planning, definitive operative confirmation and treatment depend on careful intraoperative inspection of the cerebellopontine angle. When all offending vessels are identified and decompressed, single-session microvascular decompression can provide effective and durable relief of both trigeminal neuralgia and hemifacial spasm.

Patient consent for publication

Written informed consent was obtained from the patient for publication of anonymized clinical information and images.

Author contribution

Iago Tsertsvadze performed the surgical procedure and contributed the clinical case details, operative findings, and intraoperative interpretation included in the case presentation.

Declaration of Generative AI and AI-assisted technologies in the writing process

ChatGPT was used during the final revision to improve readability, concision, and language clarity. The authors reviewed and approved the final manuscript and take full responsibility for its content.

CRediT authorship contribution statement

Tariel Natsvaladze: Methodology, Writing – original draft. **Iago Tsertsvadze:** Visualization, Data curation. **Lika Burduli:** Validation, Resources. **Andrii Netliukh:** Project administration, Formal analysis, Writing – review & editing. **Nana Tchantchaleishvili:** Supervision, Software, Methodology, Conceptualization, Writing – review & editing. **Mario Ganau:** Writing – review & editing.

Ethics approval

Ethics approval was waived for this case report and focused literature review.

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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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