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Case Report

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# Prolonged Autonomic Dysfunction After Ventricular Ablation, Cardiac Tamponade, and Emergency Open-Heart Surgery: A First-Person Case Report with Integrated Cardiology and Autonomic Analysis

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## Abstract

***Background:*** *Cardiac tamponade is a recognized but uncommon complication of catheter ablation for ventricular arrhythmias. Although its immediate hemodynamic consequences are well described, prolonged autonomic sequelae after tamponade and emergency cardiac surgery remain poorly represented in the cardiology literature.*

***Case Summary:*** *This report presents a first-person longitudinal case of severe multisystem autonomic dysfunction after ablation of premature ventricular ectopy arising from the left ventricular outflow tract, complicated by cardiac perforation, tamponade, and emergency open-heart repair. Post-operatively, the dominant syndrome was not persistent structural cardiac failure, but a prolonged disturbance of autonomic control characterized by marked cardiovascular lability, orthostatic intolerance, profound fatigue, and gastrointestinal dysmotility. Recovery occurred gradually over four years through multidisciplinary, symptom-directed management.*

***Discussion:*** *The observed trajectory is biologically consistent with combined injury to peripheral autonomic pathways, including vagal, sympathetic, and baroreceptor-related mechanisms, compounded by inflammatory activation and ischemia-reperfusion stress associated with hemodynamic collapse and cardiopulmonary bypass. Cardiovascular improvement preceded gastrointestinal recovery, suggesting differential timelines across autonomic territories.*

***Conclusion:*** *This case supports recognition of a prolonged but potentially reversible autonomic syndrome after ventricular ablation complicated by tamponade and emergency sternotomy. Earlier recognition may improve patient counselling, follow-up planning, and interdisciplinary care.*

**Keywords:** Autonomic Dysfunction, Cardiac Tamponade, Ventricular Ablation, Orthostatic Intolerance, Gastrointestinal Dysmotility, Patient Narrative, Recovery Trajectory, Long-Term Recovery

**Abbreviations**

**ANS:** Autonomic Nervous System

**HRV:** Heart-Rate Variability

**LVOT:** Left Ventricular Outflow Tract

**PVC:** Premature Ventricular Contraction

## 1. Introduction

Catheter ablation is an established treatment for selected ventricular arrhythmias and symptomatic premature ventricular contractions. In experienced centers, the procedure is generally safe, yet major complications remain possible, especially when ablation is performed near anatomically complex structures. Cardiac tamponade is among the most feared procedural emergencies because even a brief period of uncontrolled hemodynamic compromise can trigger a wider cascade of inflammatory, ischemic, and neurohumoral injury. The cardiology literature describes the acute management of tamponade well: rapid recognition, pericardial decompression when feasible, surgical rescue when necessary, and post-procedural hemodynamic stabilization. Much less attention is given to what follows in patients who survive the immediate event but do not return to physiological baseline. In such patients, symptoms may span multiple organ systems and appear disproportionate to standard structural findings, creating diagnostic uncertainty and fragmented follow-up. Transient autonomic disturbance after major cardiac intervention is not unexpected. Heart-rate variability may be reduced after surgery, baroreflex sensitivity may be impaired, and recovery may extend beyond the early postoperative period. What is less often captured is a prolonged, multi-year syndrome in which cardiovascular instability, orthostatic intolerance, fatigue, gastrointestinal dysmotility, and secretory dysfunction appear as part of one clinically coherent pattern. This report therefore has two linked aims. First, it documents a rare but clinically relevant post-procedural trajectory after ventricular ablation complicated by tamponade and emergency open-heart surgery. Second, it preserves the patient experience that made the syndrome intelligible over time. The patient perspective is not supplementary material here; it is part of the diagnostic architecture of the case.

## 2. Case Presentation

### 2.1. Pre-Event Baseline

Before the index procedure, I was living with cardiac autonomic neuropathy that was stable, monitored, and functionally manageable. It imposed limits, but it did not define everyday life. I had learned how to pace activity, interpret symptoms, and live within a bodily equilibrium that felt hard won but dependable. The recommendation for ablation of symptomatic premature ventricular ectopy arising from the left ventricular outflow tract was framed as a reasonable next step toward better rhythm control and quality of life. Risk was discussed in the ordinary language of procedural consent: present, real, but relatively remote. The anticipated narrative was one of incremental improvement rather than physiological collapse.

### 2.2. Procedural Catastrophe and Rescue

During the procedure, acute cardiac perforation occurred with rapid deterioration consistent with cardiac tamponade. The event moved from routine electrophysiology to surgical emergency within moments. Emergency open-heart surgery was performed, hemopericardium was evacuated, and the cardiac injury was repaired. Cardiopulmonary bypass was required.

*Emergency surgery saved my life. That fact has remained uncomplicated, even while much else has not.*

### 2.3. Early Postoperative Phase

In the immediate postoperative period, the most obvious threat had passed. There was no major central neurological deficit and no dominant residual respiratory complication. Yet within weeks a different syndrome began to declare itself. The problem was not simply delayed convalescence after sternotomy. Rather, the dominant features were instability of physiological regulation: marked fluctuations in pulse and blood pressure, orthostatic vulnerability, severe fatigue, exercise intolerance, gastrointestinal dysfunction, and impaired tear production. The symptoms did not present as isolated inconveniences. They were persistent, cross-system, and internally patterned. Cardiovascular regulation felt unreliable; standing, exertion, meals, and environmental stressors could provoke disproportionate responses. The gastrointestinal tract seemed to lag furthest behind, with dysmotility becoming one of the most disabling persistent domains.

### 2.4. Clinical Phenotype of Autonomic Dysfunction

#### 2.4.1. Cardiovascular Manifestations

The cardiovascular picture was characterized by reduced tolerance of postural change, bidirectional blood-pressure lability, altered heart-rate responsiveness, and impaired exercise recovery. The syndrome was not easily reducible to a single static measurement because the most significant abnormality was loss of buffering. Normal physiology dampens perturbation. Here, perturbation propagated.

#### 2.4.2. Systemic and Functional Manifestations

Fatigue became systemic rather than situational. Capacity could not be inferred from motivation. Activity pacing ceased to be a lifestyle preference and became a physiological necessity. Recovery after exertion was prolonged and at times out of proportion to the immediate task performed.

#### 2.4.3. Gastrointestinal and Secretory Manifestations

Gastrointestinal dysmotility persisted longest. Clinical management over time included pancreatic enzyme replacement, gastric per-oral endoscopic myotomy for refractory dysfunction, and targeted treatment for eosinophilic oesophagitis. Ocular dryness, consistent with autonomic involvement of lacrimal secretion, provided further support for a broader dysautonomia picture.

*Each specialty could name part of the problem. What was missing was a frame large enough to hold the whole of it.*

### 2.5. Timeline

| Period | Clinical Course |
|---|---|
| **Day 0** | Left ventricular outflow tract premature ventricular contraction ablation complicated by cardiac perforation and acute tamponade. |
| **Day 0** | Emergency open-heart surgery with evacuation of hemopericardium and surgical repair under cardiopulmonary bypass. |
| **Weeks to months** | Emergence of severe autonomic instability: blood-pressure lability, orthostatic intolerance, profound fatigue, and gastrointestinal dysmotility. |
| **1-3 years** | Gradual cardiovascular autonomic recovery with continued symptom-directed, multidisciplinary management. |
| **3-4 years** | Functional improvement and restoration of day-to-day cardiovascular stability; residual gastrointestinal symptoms remain the slowest to resolve. |

### 2.6. Patient Experience as Clinical Evidence

From the patient side of the bed, the most difficult feature was not only suffering, but fragmentation. Cardiology assessed rhythm and anatomy; gastroenterology assessed motility; neurology considered signaling and autonomic function. Each consultation generated valid observations. None, on its own, was sufficient to make sense of the full trajectory. That gap matters clinically. When no clinician integrates the whole pattern, the patient becomes the integrator by necessity, often while exhausted and functionally impaired. This does not mean that lived experience should replace formal medical analysis. It means that in complex, multisystem recovery, patient narrative may reveal the continuity that organ-based pathways obscure.

*Patients do not always need certainty. We do need honesty about what is known, what is not known, and how we will move forward together.*

### 2.7. Pathophysiological Analysis

#### 2.7.1. Anatomical Vulnerability

The left ventricular outflow tract lies in close relation to the aortic root, pericardium, and cardiac autonomic plexuses. In this territory, several forms of neural vulnerability converge: cardiac vagal branches involved in chronotropic and dromotropic regulation; thoracic sympathetic fibres, including contributions linked to the stellate ganglia; baroreceptor-related afferent pathways near the aortic arch; and vagal efferent projections relevant to upper gastrointestinal function. A catastrophic event in this region therefore has the potential to produce an autonomic syndrome that is anatomically plausible rather than speculative.

#### 2.7.2. Mechanisms of Injury

Four overlapping mechanisms appear particularly relevant. First, mechanical stretch and compression during tamponade may distort or injure adjacent autonomic structures and may also amplify baroreflex failure through abrupt hemodynamic compromise. Second, ischemia-reperfusion stress during collapse, rescue, and bypass may affect neural tissue even when overt infarction is absent. Third, cardiopulmonary bypass and major surgery generate a substantial inflammatory response with cytokine signaling capable of altering autonomic regulation. Fourth, local thermal and electrical exposure from ablation may contribute to peripheral neural disturbance in an already vulnerable field. The case is not presented as proof of one single mechanism. Rather, the strength of the interpretation lies in convergence: the procedural geography, the clinical phenotype, the order of symptom emergence, and the prolonged but incomplete reversibility all point toward a combined autonomic insult.

#### 2.7.3. Why the syndrome lasted so long

A prolonged trajectory is consistent with peripheral nerve regeneration and central recalibration rather than with a brief postoperative disturbance alone. Autonomic recovery is not necessarily synchronous across tissues. Cardiovascular regulation may improve earlier because some compensatory pathways are more redundant or more rapidly reconditioned. Enteric and upper gastrointestinal manifestations may persist longer, particularly when dysmotility, nutritional stress, inflammation, and altered visceral feedback loops reinforce one another over time.

### 2.8. Management

Management was necessarily longitudinal, adaptive, and multidisciplinary. The therapeutic aim was not the unrealistic promise of rapid symptom eradication, but physiological stabilization while recovery occurred.

### 2.9. Cardiovascular Stabilization

A defining challenge was bidirectional instability. Orthostatic vulnerability required strategies to support blood pressure and reduce venous pooling, while episodic hypertensive or adrenergic surges required cautious modulation. Medication use therefore had to be dynamic rather than protocol-driven in a rigid sense. Functional stability mattered more than a single idealized target.

### 2.10. Mechanical, Postural, and Behavioural Measures

Compression garments, abdominal support, careful hydration, head-of-bed elevation, pacing of activity, and avoidance of prolonged orthostasis became core self-preservation measures. These interventions may look simple on paper, but in complex dysautonomia they often determine whether the patient can sustain day-to-day life with less physiological penalty.

### 2.11. Gastrointestinal Management

The gastrointestinal domain required some of the most substantive intervention. Pancreatic enzyme replacement supported digestion and nutritional resilience. Gastric per-oral endoscopic myotomy became necessary for refractory motility impairment. Treatment for eosinophilic oesophagitis addressed a parallel inflammatory burden that otherwise magnified dysphagia and upper gastrointestinal distress. Together these measures helped restore enough nutritional and functional stability to support broader rehabilitation.

### 2.12. The Role of Education and Clinical Language

Clinical language became part of therapy. When uncertainty was acknowledged honestly, trust improved and symptom interpretation became less adversarial. When reassurance was offered in a way that minimized lived experience, the burden of illness intensified. Education that framed symptoms as physiological warning signals rather than moral weakness or anxiety was protective in its own right.

### 2.13. Outcome and Follow-Up

By approximately four years after the event, cardiovascular autonomic stability had substantially improved. Exercise tolerance was much better, quality of life had risen, and day-to-day function was no longer dominated by cardiovascular lability. Gastrointestinal symptoms remained the slowest to settle but had become more manageable. The overall pattern was therefore one of meaningful recovery rather than complete restoration to the pre-event baseline.

*Recovery was not a return to 'before'. It was a negotiated new equilibrium.*

## 3. Discussion

This case is clinically important for several reasons. First, it extends the usual discussion of tamponade beyond acute rescue and into the neglected territory of long-term autonomic sequelae. Second, it shows that persistent post-procedural symptoms may have a coherent physiological basis even when they are distributed across multiple specialties. Third, it demonstrates why the patient perspective is not merely emotive context but a source of longitudinal pattern recognition. The distinction between survival and recovery deserves emphasis. In procedural cardiology, successful rescue understandably focuses clinical attention on immediate mortality and structural outcomes.

Yet for the patient, the later burden of autonomic dysfunction may define the true recovery period. A patient can leave hospital alive and still enter a prolonged phase of profound physiological disability. The case also supports a pragmatic clinical lesson: when persistent symptoms after cardiac intervention involve cardiovascular lability, orthostatic intolerance, gastrointestinal dysmotility, fatigue, and secretory disturbance, clinicians should consider an integrated autonomic formulation early. Such a formulation need not claim more certainty than the evidence allows. Its value lies in organizing care, validating symptoms, and guiding interdisciplinary follow-up.

This report has limitations. It is a single case, written in the first person, and it cannot establish incidence or mechanism with the certainty of controlled physiological testing across every domain. However, single cases remain valuable when they illuminate an under-recognized trajectory, generate clinically useful hypotheses, and preserve details that are commonly lost in more abstract reporting. In this sense, the case sits at the intersection of cardiology, autonomic medicine, and narrative-based clinical understanding. It asks clinicians to hold two truths together: emergency surgery saved the patient's life, and life afterwards required years of adaptation to a multisystem disorder likely shaped by the same event.

### 3.1. Learning Points

- Cardiac tamponade during ventricular ablation can lead to consequences that extend beyond the acute haemodynamic emergency.
- A prolonged syndrome of cardiovascular lability, orthostatic intolerance, fatigue, and gastrointestinal dysmotility is biologically plausible after tamponade and emergency cardiac surgery.
- Patient narrative can materially improve clinical interpretation when symptoms are multisystem, longitudinal, and difficult to integrate within single-specialty care.
- Recovery may occur over years rather than weeks, with cardiovascular improvement preceding enteric recovery.

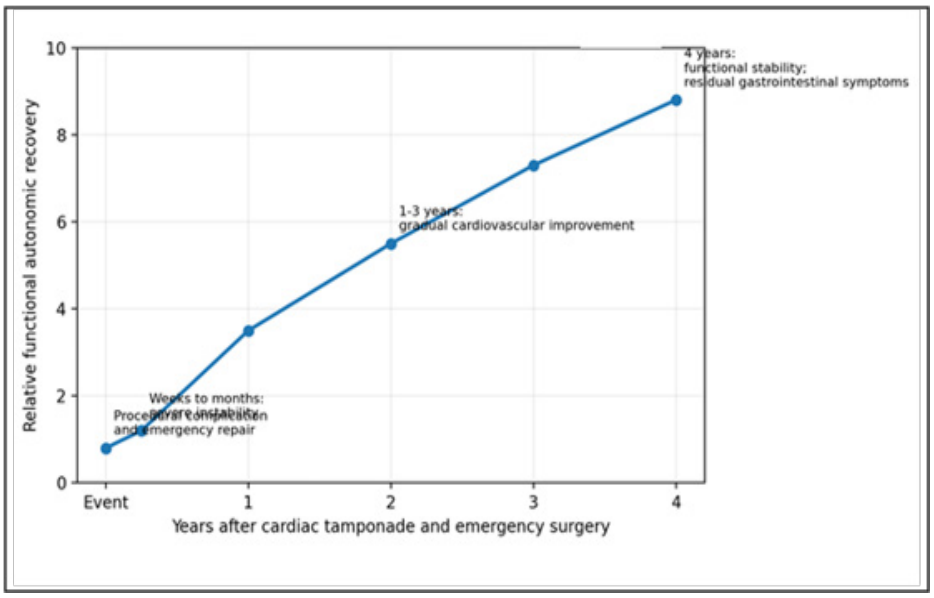



**Figure 1:** Conceptual Longitudinal Recovery Trajectory

The graph is illustrative rather than quantitative. It summarizes the patient-reported pattern of profound early instability, gradual cardiovascular improvement through years 1 to 3, and later functional consolidation with residual gastrointestinal symptoms.

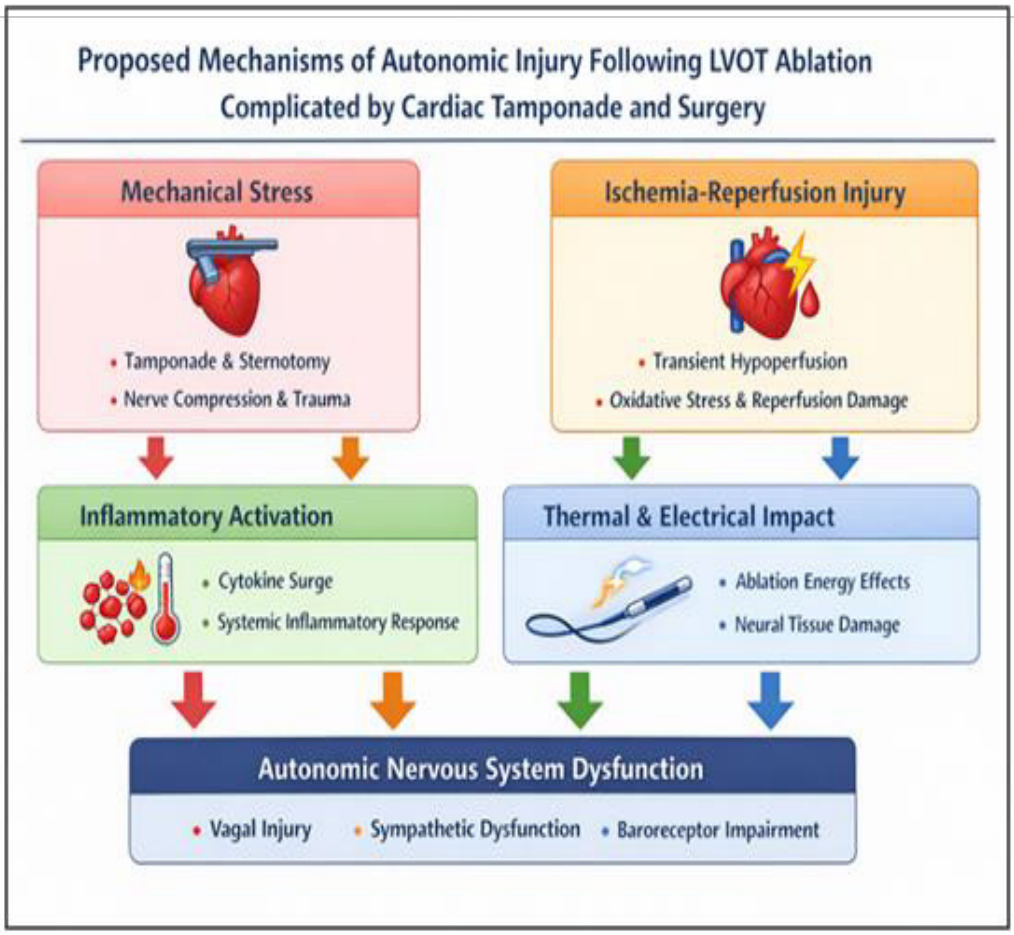
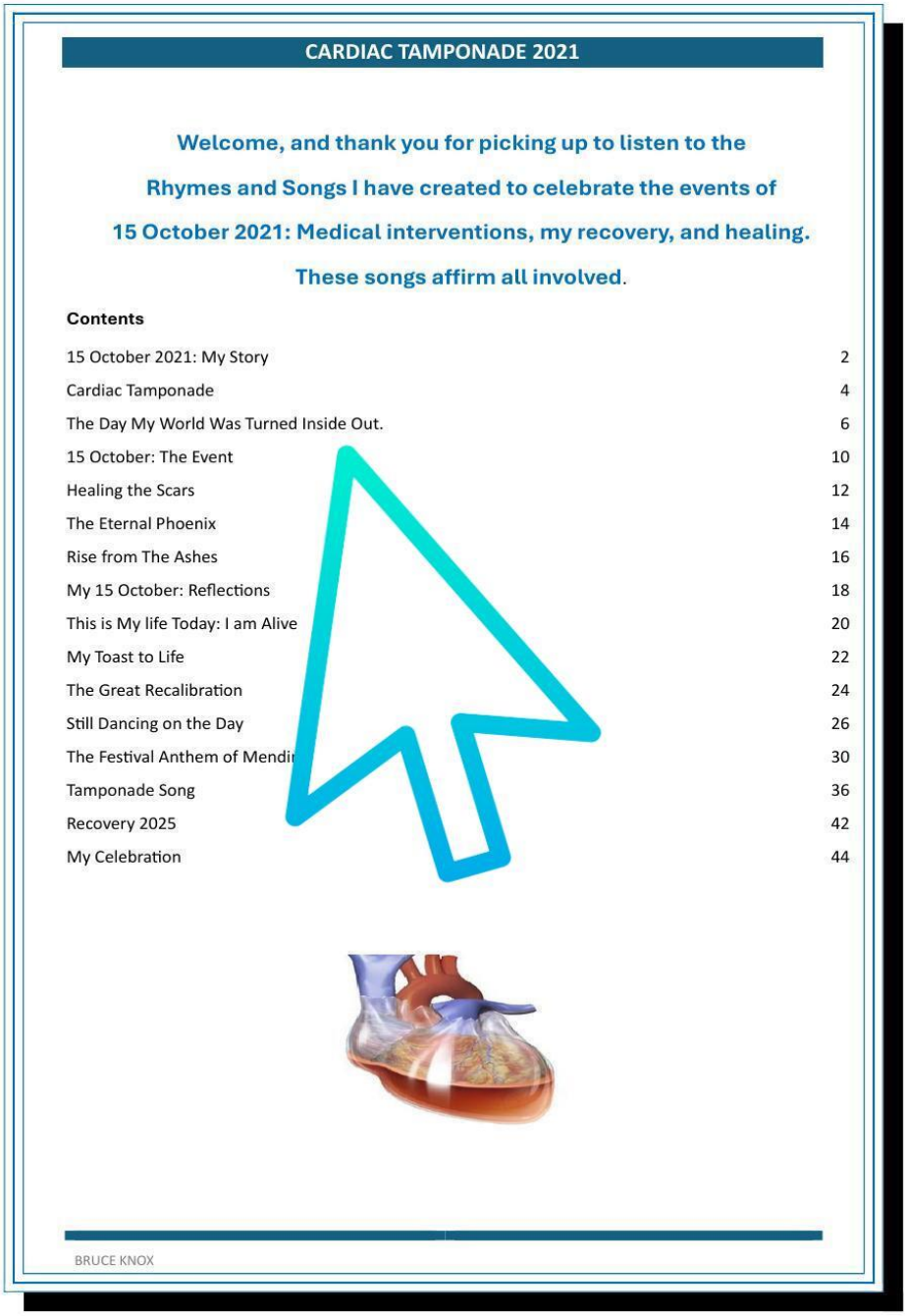



**Figure 2:** Proposed Pathophysiological Model

This schematic links the procedural event to plausible mechanisms of autonomic injury and to the observed multisystem phenotype. It is presented as an explanatory framework to support clinical reasoning rather than as proof of a single causal pathway.


CARDIAC TAMPONADE 2021

Welcome, and thank you for picking up to listen to the

Rhymes and Songs I have created to celebrate the events of

15 October 2021: Medical interventions, my recovery, and healing.

These songs affirm all involved.

Contents



BRUCE KNOX

## 4. Conclusion

This case supports recognition of a prolonged but potentially reversible autonomic syndrome after ventricular ablation complicated by tamponade and emergency sternotomy. Earlier recognition may improve patient counselling, follow-up planning, and interdisciplinary care [1-15].


## Acknowledgements

The author acknowledges the multidisciplinary clinicians involved in his care across cardiology, electrophysiology, autonomic medicine, gastroenterology, ophthalmology, and immunology. Their contributions were central to survival, stabilization, and long-term recovery.

## Ethics

This manuscript is a self-authored patient case report. No third-party identifiable patient data are included.

## Author Declaration

Submission of this manuscript implies that the work has not been published previously in this form and is not under consideration elsewhere. The author prepared the manuscript and approved the final version.

## Author Contribution

Bruce H. Knox conceived the manuscript, integrated the clinical narrative, interpreted the case, and wrote the final text.

## Consent for Publication

Written informed consent for publication was obtained from the patient-author.

## Data availability

All material relevant to this case is contained within the manuscript; no external dataset was generated.

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