

Migrainous Thoracalgia in a Patient with Chronic Migraine and Coronary Vasospasm: A Case Report

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Abstract: Migrainous thoracalgia (MT) refers to chest pain (CP) potentially arising from a neurologic etiology, often temporally linked to migraine. We present a 57-year-old woman with chronic migraine who developed recurrent CP typically following migraine attacks. Despite multiple cardiac evaluations, including cardiac MRI and catheterizations, she was diagnosed with fibromuscular dysplasia, spontaneous coronary artery dissection, and coronary microvascular disease. Her CP partially responded to nitroglycerin and improved modestly with migraine control using ubrogepant and calcium channel blockers. During admission for refractory migraine, she received intravenous lidocaine, magnesium, ketorolac, and neuroleptics, resulting in several months of CP remission despite brief migraine relief. Longitudinal follow-up demonstrated that eptinezumab was associated with improvement in both migraine and CP symptoms. This case highlights the diagnostic challenge of MT and the overlap between migraine and coronary vasospastic or microvascular processes. The observed improvement in CP following migraine-directed therapies raises the possibility of a shared neurovascular mechanism, although causality cannot be established. Greater awareness of MT and interdisciplinary management may improve outcomes in similar patients.

Plain Language Summary: This report describes the case of a 57-year-old woman who experienced repeated episodes of chest pain that appeared to be linked to her migraines. Although her heart tests showed some past heart conditions—including a tear in one of her coronary arteries and small blood vessel abnormalities—doctors could not identify an ongoing cardiac cause that fully explained her symptoms. Notably, her chest pain almost always occurred shortly after a migraine attack.

Over several years, the patient tried multiple treatments, including migraine medications, heart medications, and nitroglycerin. Some provided temporary relief, but the chest pain continued to recur. In 2022, she was admitted to a headache clinic and received a combination of intravenous treatments for severe migraine. This led to a four-month period without chest pain—the longest relief she had experienced—despite only short-term improvement in her headaches.

She was later started on eptinezumab (Vyepti), a medication given every few months to prevent migraines. With this treatment, both her headaches and chest pain became less frequent and less severe.

This case highlights a lesser-known condition called “migrainous thoracalgia”, in which chest pain may be related to migraine. While it is not possible to determine cause and effect from a single case, the patient’s improvement with migraine-directed therapies suggests there may be a link between migraine and chest pain in some individuals. Greater awareness of this possible connection may help clinicians better recognize and manage similar cases through collaboration between neurology and cardiology specialists.

Keywords: migraine, chest pain, migrainous thoracalgia, coronary vasospasm, CGRP, case report

Introduction

Migrainous thoracalgia (MT) refers to chest pain (CP) that may arise from a neurologic etiology, potentially linking migraine and cardiovascular conditions.¹ While migraine has been associated with increased acute coronary syndrome (ACS) risk,² MT remains a diagnosis of exclusion that requires a full cardiologic workup.³ In a study published in the New England Journal of Medicine, Miller et al famously found a 26% prevalence of migraines among patients with

angina,⁴ with Heupler et al reporting similar numbers in patients with known coronary arterial spasm.⁵ These findings further suggest an overlap between migraine and cardiovascular dysfunction, though the pathophysiology remains unclear, and no definitive treatment protocol exists.^{6–9} The following case presentation involves an individual with recurrent episodes of MT in association with chronic migraine (CM) and coronary vasospasm that improved following inpatient treatment of migraine. The concept of migrainous thoracalgia remains incompletely defined and is not widely recognized as a distinct clinical entity, with current understanding largely derived from isolated case reports and small series. The relationship between migraine and chest pain is likely multifactorial and remains an area of clinical uncertainty, particularly in patients with coexisting cardiovascular disease.^{7,10}

Case Presentation

A 57-year-old woman with a history of chronic migraine (CM) since her 20s began experiencing new episodes of severe, transient chest pain (CP) in 2018, typically following migraine attacks by minutes to hours. Initial cardiac evaluation, including electrocardiograms and troponin levels, was unremarkable, but persistent symptoms prompted further testing. She was ultimately diagnosed with a spontaneous coronary artery dissection (SCAD) in a distal obtuse marginal artery (OM2) and treated for non-ST segment elevation myocardial infarction (NSTEMI). Further evaluation confirmed fibromuscular dysplasia.

Despite appropriate medical management with antiplatelet therapy and calcium channel blockers, the CP persisted, occurring weekly and often following migraine headaches, leading to repeated emergency department visits. Over subsequent years, her CP followed a recurrent pattern, frequently occurring in clusters and nearly always preceded by migraine attacks. She reported associated symptoms, including cold, pale hands resembling Raynaud's phenomenon as well as cough and shortness of breath shortly after CP onset. While most CP episodes were temporally linked to migraine attacks, she occasionally experienced isolated CP, particularly after exercise, introducing potential alternative triggers unrelated to migraine activity.

Extensive cardiac workup—including electrocardiograms, echocardiograms, cardiac magnetic resonance imaging (CMRI), multiple left heart catheterizations (LHCs), and positron emission tomography scans—revealed microvascular disease and evidence of prior infarction without significant atherosclerotic coronary artery disease. Repeat LHCs demonstrated healed SCAD and mild myocardial bridging without evidence of active ischemia. Stress CMRI in 2023 identified a small, discrete perfusion defect in the mid-inferolateral segment at both rest and stress, consistent with coronary microvascular dysfunction (CMD). Despite her history of SCAD and NSTEMI, her cardiologist opted against further invasive workup, given the vasospastic nature of her symptoms and their consistent relationship to migraine headaches.

Her CP was partially responsive to nitroglycerin and exacerbated by triggers, such as cold weather and emotional stress. Improved migraine control with medications, including ubrogepant and calcium channel blockers, correlated with a reduction in CP frequency. However, this effect was incomplete, with daily recurrences. Episodes persisted despite interventions, necessitating escalating doses of nitroglycerin and occasional hospital admissions for nitroglycerin infusions, with variable response lasting several days to weeks.

In 2022, the patient was admitted to the Jefferson Headache Center for refractory migraine management. A multimodal intravenous regimen of lidocaine, magnesium, ketorolac, and neuroleptics was initiated, and nitroglycerin was withheld during the admission. Dihydroergotamine was also withheld given her cardiac and suspected vasospasm history. Remarkably, she experienced a four-month remission of CP despite only several days of migraine symptom relief, although the contribution of concurrent therapies and the natural variability of symptoms cannot be excluded. This interval represented her longest CP-free period since symptom onset. Approximately 1.5 years later, the patient reported worsening CP episodes coinciding with an increase in migraine frequency. While her symptoms gradually returned over the following year, neurology and cardiology follow-up in 2025 revealed that the chest pain remained closely linked to migraine activity. With initiation of eptinezumab, along with abortive therapy with ubrogepant and intranasal ketamine, she experienced a marked reduction in headache frequency and severity, and her chest pain became milder and less frequent. Nitroglycerin no longer provided benefit, and no further emergency evaluations were required.

Discussion

This case highlights a potential relationship between migraine headaches and CP. The patient's recurrent, stereotyped episodes of CP, shortness of breath, and cough inconsistently responded to migraine abortive therapies and notably, became less responsive

over time. While some relief was observed with cardiac interventions, the effectiveness of migraine-directed treatments remained incomplete, and her CP episodes continued despite improved migraine control. Notably, she required escalating doses of nitroglycerin over time, suggesting a dynamic interplay between her cardiac and neurologic symptoms rather than a straightforward diminishing response to migraine therapy. Unlike prior reports of MT,^{1,6} which describe stereotyped thoracic band-like pain without headache and completely normal cardiac, gastrointestinal, and neurologic evaluations, this case raises the possibility of overlapping cardiopulmonary symptoms that may reflect a migraine postdrome or associated feature, similar to migraine-associated abdominal pain.^{10–12} Conversely, cardiac cephalgia, a condition in which patients with significant heart disease report headaches, offers another possible mechanism for this presentation.^{7,9} Importantly, alternative explanations for chest pain remain plausible, including coronary microvascular dysfunction, prior SCAD, and fibromuscular dysplasia. Her shortness of breath and cough symptoms could also mirror a similar case of pulmonary migraine, previously reported.¹³ The concept of corpalgia—a generalized pain syndrome associated with migraine—has been proposed as a contributing factor, potentially due to central sensitization processes that also underpin the frequent coexistence of fibromyalgia and migraine.^{11,14} Further investigation into the pathophysiology of coronary vasospasm and migraine may reveal further pathophysiologic and treatment paradigms for consideration.

Treatment approaches and outcomes of MT have been sparsely reported. Unlike the findings of Roldan et al, who reported that nitroglycerin was ineffective for CP relief while metoclopramide was more successful, our patient found nitroglycerin to be the most effective treatment for CP following migraine attacks, with migraine-specific abortive therapies leading to partial relief.¹¹ The long-lasting CP relief following combination migraine infusions is particularly noteworthy. While lidocaine infusion has been shown to produce at least short- and medium-term benefits in the treatment of refractory migraine,¹⁵ it is not typically used to treat anginal pain. This remarkable response suggests that termination of the migraine pain, even if not long-lasting, may have disrupted the feed-forward loop of pain and vasospasm, thereby achieving significant remission in CP.

In the longer term, the patient's chest pain symptoms improved substantially with the use of eptinezumab, a monoclonal antibody targeting CGRP. Not only did her headache frequency and severity decline, but her CP episodes became milder and less frequent, and she no longer required nitroglycerin or emergency evaluation. While this observation is notable, it is limited to a single patient and cannot establish causality. The relationship between CGRP signaling and coronary vasoreactivity remains incompletely understood, and both vasodilatory and vasoconstrictive effects have been described in different contexts.¹⁶ This durable response to CGRP-targeted therapy highlights the possibility that her chest pain was driven—at least in part—by migraine-related mechanisms. Given the vasodilatory role of CGRP^{17,18} and its involvement in both migraine and vascular tone regulation, this case highlights the potential utility of migraine preventive therapies in patients with overlapping neurovascular syndromes.

This report has several limitations. As a single-patient observational case, causal relationships cannot be established. The presence of multiple vascular comorbidities, including spontaneous coronary artery dissection, fibromuscular dysplasia, and coronary microvascular dysfunction, introduces potential confounding factors in interpreting the origin of chest pain. Additionally, treatment effects cannot be isolated due to the use of multiple simultaneous interventions and the natural variability of migraine symptoms.

Conclusion

This case highlights the diagnostic complexity of recurrent chest pain in a patient with chronic migraine and coexisting vascular disease. The temporal relationship between migraine and chest pain, along with symptom improvement following migraine-directed therapies, suggests a possible neurovascular association. However, given the presence of multiple potential cardiac contributors, this relationship should be interpreted cautiously. This case underscores the importance of considering migraine-associated chest pain in select patients and supports the value of interdisciplinary management in complex neurovascular presentations.

Ethics Approval

Institutional approval was not required for publication of this single-patient case report in accordance with the policies of Thomas Jefferson University Hospital.

Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying details.

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Disclosure

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