Cervical Angina: An Overlooked Source of Noncardiac Chest Pain

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Abstract

Cervical angina has been widely reported as a cause of chest pain but remains underrecognized. This series demonstrates the varied clinical presentation of patients with cervical angina, the delay in diagnosis, and the extensive cardiac examinations patients with this condition typically undergo prior to a definitive diagnosis. Recognition of this condition in patients with acute chest pain requires a high index of suspicion and an awareness of the common presenting features and clinical findings of cervical angina.

Keywords

cervical angina, noncardiac chest pain, cervical pain

Introduction

Each year, more than 7 million patients present to emergency departments with chest pain.¹ The majority of these patients are admitted for in-hospital cardiac “rule out”; however, only 15% to 25% of patients with acute chest pain will actually have acute coronary syndrome.^{2,3} The risk of fatally misdiagnosing acute coronary syndrome often results in noncardiac causes of chest pain being overlooked. In many cases, patients remain underdiagnosed.⁴

The prevalence of noncardiac chest pain is estimated to be more than 50% of all cases with chest pain that present to the emergency department.⁵ Cervical angina is one potential cause of noncardiac chest pain and originates from disorders of the cervical spine. Although the exact incidence is unknown, in 1 series of 241 patients with C7 radiculopathy undergoing anterior cervical discectomy, 16% of the patients had associated chest pain or subscapular pain.⁶ In another series of 706 patients with cervical pathology requiring surgery, 1.4% exhibited symptoms of cervical angina.⁷ Despite being first described in 1934, the diagnosis of cervical angina remains underrecognized.^{7,8} Here, we present a series of 6 cases of cervical angina and review the symptomatology and clinical approach to this unusual presentation of cervical pathology.

Methods and Results

Our institutional review board approved this study. A retrospective chart review was performed. All patients presented to Saint Vincent Hospital (Worcester, Massachusetts) between January 2008 and December 2011. Using *International Classification*

of Diseases, Ninth Revision codes, patients who were admitted and diagnosed with both atypical or noncardiac chest pain and cervical pathology were identified. For inclusion, the patient also had to be seen by a neurologist during the admission and undergo magnetic resonance imaging (MRI) of the cervical spine (Figure 1). Of the 44 patients initially identified, only 6 patients (5 males, 1 females; mean age 58.3 years, ranging from 39 to 86 years) had concordant physical examination findings and cervical imaging.

None of the 6 patients had chest pain that satisfied the definition of true angina pectoris. For pain to meet the criteria for angina pectoris, it had to satisfy 3 criteria: (1) constricting discomfort in the front of the chest or in the neck, shoulders or arms; (2) symptoms that are precipitated by exertion or emotion; and (3) relieved by rest.⁹ Two patients had atypical chest pain, defined as the presence of 2 of the 3 criteria. Four patients were diagnosed with noncardiac chest pain, with 0 to 1 criteria. The chest pain was located substernally in 2 cases and in the left anterior chest wall in 3 cases. The exact location of chest pain was not documented in 1 case. The mean duration of symptoms was 20.2 months (median 5 months). The characteristics of the chest pain are outlined in Table 1.

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Figure 1. Flow diagram of patient screening and inclusion.

All 6 patients included in this series were seen by a neurologist during the hospital admission and diagnosed with cervical angina by the neurologist. The reason for neurology consultation varied, but all were for “secondary” symptoms initially considered unrelated to the primary complaint of chest pain. In 3 cases, the consultation was for left upper extremity pain or numbness. The other 3 consults were for symptoms of headaches, dizziness, and syncope. All patients had cervical pathology on MRI (Table 1). Cervical spondylotic radiculopathy or cervical disk disease causing neuroforaminal narrowing was present in 4 patients, and 2 patients had cervical myelopathy on MRI.

Varying degrees of cardiac workups were performed (Table 2). Prior to the most recent admission, all 6 patients had previously undergone cardiac stress testing or angiogram. None of the patients had been evaluated by a neurologist during these prior admissions. During the current hospitalization, all patients had an electrocardiogram, chest x-ray, and cardiac enzymes tested, which were unremarkable. Of the 4, 2 patients who had stress testing showed potential ischemic changes and underwent coronary angiograms. One additional patient underwent coronary angiography without a stress test. All 3 coronary angiograms performed were normal.

Illustrative Cases

A 50-year-old male presented with 1 month of neck pain radiating down his left arm, with associated tingling and weakness. During his stay in the emergency department, he developed substernal chest pain and experienced moderate relief with morphine. The patient had an extensive history of anginal symptoms, with associated left upper extremity radicular symptoms, extending back over 5.5 years. During prior admissions, the patient had 3 negative cardiac catheterizations and 2 negative stress tests.

During this hospitalization, cardiac workup included a negative myocardial perfusion study and cardiac catheterization, which showed no evidence of coronary artery disease. Neurology was consulted for the patient’s headaches. On examination, the patient had a positive Spurling maneuver and findings suggestive of cervical radiculopathy. A cervical MRI was recommended, and neuroforaminal stenosis was found at the left C4-5 and C5-6 levels (Figure 2). The patient was prescribed a cervical collar, and the patient was discharged home with outpatient follow-up.

Electrodiagnostic studies were consistent with C5 and C6 radiculopathy, and the patient underwent anterior cervical

Table 1. Symptoms and Clinical Findings.

Case No.	Age and Sex	Characteristics of Chest Pain						Relieving Factors	Diagnosis	MRI Findings (Affected Level)	Affected Nerve Root
		Duration	Location	Quality and Intensity	Associated Symptoms	Autonomic Symptoms					
1	64 M	4 mo	LAC	Sharp (10/10)	LUE (u,p,w), NP	Nausea, diaphoresis	NTG Opiates	NTG Opiates	Radiculopathy	CSR/CDD (C5-6)	C6
2	52 F	2 days	Unspecified	Pressure, tingling	LUE (p,h), NP, HA	Nausea	NTG	NTG	Radiculopathy	CDD (C6-7)	C7
3	39 M	17 mo	SS	Sharp (7/10)	None	Nausea, vertigo diaphoresis	None	None	Radiculopathy	CSR/CDD (C6-7)	C7
4	86 M	6 mo	LAC	Pressure, burning	HA	Nausea, vertigo	None	None	Myelopathy	CSM (stenosis from C3-4 through C6-7)	C4-C7
5	50 M	90 mo	SS	Pressure, tingling (10/10)	LUE (p,w), NP, HA	Dyspnea	Opiates	Opiates	Radiculopathy	CSR/CDD (C5-6)	C6
6	59 M	4 mo	LAC	Pressure (2/10)	LUE (p,w)	Vertigo	None	None	Myelopathy	Lipomatosis mass (C8-T2)	C8

Abbreviations: Localization of CP; SS, substernal; LAC, left anterior chest; associated symptoms: LUE, left upper extremity symptoms (u, unspecified pain; p, paresthesia, h, heaviness; w, weakness); NP, neck pain, HA, headaches; relieving factors: NTG, nitroglycerin. MRI Findings: CSR, cervical spondylotic radiculopathy; CDD, cervical disk disease; CSM, cervical spondylotic myelopathy; CP, chest pain; MRI, magnetic resonance imaging; mo, months; M, male; F, female.

Table 2. Cardiac Findings and Prior History.

Case No.	Age and Sex	Cardiology Consult	Stress Test	Coronary Angiogram	TIMI Risk Score	Prior Stress Test	Prior Coronary Angiogram	Cardiac Risk Factors
1	64 M	Yes	No	No	0	MPI, 3mo prior	none	HTN, HL, tobacco use
2	52 F	Yes	MPI	Yes	0	DSE, 21mo prior	none	None
3	39 M	No	MPI	No	0	MPI, 12mo prior	none	Tobacco use
4	86 M	Yes	DSE	No	0	MPI, 27mo prior	none	HTN
5	50 M	Yes	MPI	Yes	1	2 prior MPI; 12 and 23mo prior	3 prior angiographically normal catheterizations, 12, 23 and 67mo prior	HTN
6	59 M	Yes	No	Yes	0	MPI, 4mo prior	none	None

Abbreviations: Stress test: MPI, myocardial perfusion imaging; DSE, Dobutamine Stress Echo; HTN, hypertension; HL, hyperlipidemia; TIMI, thrombolysis in myocardial infarction; M, male; F, female.

Figure 2. Axial image showing a left-sided osteophyte–disk complex encroaching on the C5-6 neuroforamen.

microdisectomy of the C4-5 and C5-6 intervertebral disks. Intraoperatively, the patient was found to have significant narrowing of the left C5-6 foramen due to an osteophyte–disk complex. Postoperatively, the patient symptoms resolved.

Discussion

This series demonstrates the varied clinical presentations of patients with cervical angina. Although the true incidence and prevalence of this condition are unknown, many patients with cervical angina undergo extensive cardiac evaluations and often experience a delay in diagnosis.⁶ In many cases, the neurological signs and symptoms are often overlooked. Without a strong sense of suspicion in patients with inadequately explained chest pain, the clinician can mistake symptoms of chest pain and any associated neurological signs or symptoms as dual clinical entities. Prompt diagnosis of this underrecognized syndrome requires an awareness of the common presenting features and clinical findings of cervical angina.

The majority of cases with cervical angina, up to 70%, have been attributed to cervical nerve root compression.¹⁰ Radicular pain is likely mediated by compression of the C4 to C8 nerve roots, which supply the sensory and motor innervation to the anterior chest wall through the medial and lateral pectoral nerves. The most frequently affected levels were C5-C6 (37%), C6-C7 (30%), C4-C5 (27%), and C3-C4 (4%).¹⁰

A few cases have been reported in which cervical angina was attributed to cervical myelopathy,⁷ spinal cord infarction,^{11,12} spinal cord tumors,¹³ cervical instability,¹⁴ and intervertebral disk disease.⁶ In these cases, the mechanism of pain is controversial. Degenerative disk disease and facet joint pathology may

refer via the sinuvertebral nerves, independent of root compression. Myelopathic pain and sympathetic-mediated pain have also been suggested as potential mechanisms.⁷ Although the exact mechanism is unclear, pain may be mediated by the sympathetic afferent fibers to the heart and coronary arteries, which originate in the dorsal root ganglia of C8 to T9. In cervical myelopathy, lesions of the dorsal horn or disruption of the ascending cardiac spinothalamic tracts may create the sensation of anginal pain.^{11,12}

History and Physical Examination

Cervical angina often presents with anterior chest pain and has been described as sharp, achy, or crushing in quality. Some patients may even experience relief with nitroglycerin.¹⁵ Symptoms may be present at rest or exacerbated by physical activity. Associated neck pain, stiffness, headaches, shoulder, or arm pain may be present. Up to 50% to 60% of patients experience autonomic symptoms (dyspnea, vertigo, nausea, diaphoresis, pallor, fatigue, diplopia, and headaches), but the mechanism is not well explained.^{7,10} The severity and chronicity of the discopathy often dictate the degree of deficits on physical examination. Except in cases of obvious nerve root compression, neurologic findings are often absent or nonfocal.¹⁰ Although cervical angina can present in varied ways, there are certain features in the patient's history and physical examination that should increase suspicion for cervicogenic chest pain (Table 3). The Spurling maneuver, performed by rotating the cervical spine toward the symptomatic side while providing a downward compression through the patient's head, has been shown to reproduce symptoms of cervical angina in case reports.^{10,15} In a series of 257 patients with cervical nerve root pathology, a positive Spurling maneuver correlated with findings on computer tomography with a sensitivity of 95% and specificity of 94%.¹⁷

Diagnostics and Treatment

Once coronary artery disease has been adequately excluded, cervical imaging can be helpful in defining anatomic abnormalities. Plain radiographs or MRI may demonstrate degenerative cervical changes, including disk space narrowing, osteophyte formation, or neuroforaminal encroachment. Magnetic resonance imaging can also rule out any myelopathic changes, fractures, tumors, or infections that may require emergency intervention. Electromyography may corroborate imaging.

Concordant examination and radiographic findings support the diagnosis of cervical angina. When there is a discrepancy between the clinical, radiographic, and neurophysiologic findings, additional testing may be necessary. Degenerative changes are frequently found in the asymptomatic population, and functional tests may be helpful in confirming the etiology of cervical angina.¹⁸ Diagnostic nerve block and/or provocative discography remain the best tools to make a functional

Table 3. History and Physical Examination Findings Useful in Diagnosing Cervical Angina.^{7,10,15,16}**History**

- History of cervical radiculopathy, subjective upper extremity weakness or sensory changes, occipital headaches, or neck pain
- Pain induced by cervical range of motion or movement of the upper extremity
- History of cervical injury or recent history of manual labor (lifting, pulling or pushing, such as with yard work or lifting heavy objects)
- Pain persisting for >30 minutes or less than 5 seconds (although exceptions exist)

Examination

- Restricted cervical motion and/or paraspinal tenderness
- Positive Spurling maneuver
- Radicular symptoms associated with a specific dermatome or myotome, although these may be only present with persistent disease.
- Radiologic evidence of degenerative changes in the cervical spine
- Negative cardiac workup

diagnosis, but discography is not without risk and typically reserved for patients contemplating surgery.^{7,19}

Conservative treatment is successful in the majority of cases and includes symptom management directed at the underlying pathology.¹⁵ The majority of patients with cervical angina from cervical radiculopathy will respond to conservative care.¹⁰ Surgical intervention may be indicated if conservative therapy fails or in cases where neurological compromise is evident. Some case series report that 90% of patients experienced long-term relief following cervical discectomy and fusion.^{6,15}

There are limitations to this study, including those inherent in a retrospective chart review. The sample size was small, and the 6 cases likely underestimates the total number of patients seen at our institution with cervical angina. The narrow inclusion criteria also limited the sample to only those patients with both clinical and radiographic findings of cervical angina. The review was limited to inpatient records, but it is possible that patients completed the workup in the outpatient setting.

Nevertheless, this series demonstrates the varied presentation of patients with cervical angina, and the extensive cardiac workup these patients often undergo. There should be a strong suspicion for cervical angina in any patient with inadequately explained noncardiac chest pain, especially, when neurologic signs and symptoms are present. A greater awareness of this unusual radiating pattern for cervical pathology will hopefully led to early diagnosis and a recognition that this symptoms pattern is not due to dual clinical entities but unified by the diagnosis of cervical angina.

Declaration of Conflicting Interests

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