Thoracic Outlet Syndrome Mimicking Angina Pectoris with Elevated Creatine Phosphokinase Values*

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Four patients with elevated creatine phosphokinase (CPK) values and recurrent chest pain were found to have thoracic outlet syndrome. This association of abnormal CPK levels and chest pain due to thoracic outlet syndrome has not been previously reported. Symptoms and CPK values improved with anti-inflammatory medications and or proper posture instruction. It is proposed that CPK values become elevated by ischemic or neurologic compromise of muscles supplied by the subclavian artery or brachial plexus respectively. Accordingly, chest pain in the same dermatomal distribution as that of angina pectoris may be simulated by ischemic skeletal muscle. Thoracic outlet syndrome therefore should be suspected in any patient with chronically abnormal CPK values and chest pain in whom no other etiology can be determined.

The etiology of chest pain in patients admitted to cardiac care units is often not determined. Approximately 7 percent to 10 percent of patients undergoing coronary arteriography for angina symptoms have not only normal coronary arteries but also no other basis found for their chest pain.

Fourteen such patients have been referred to us over a one-year period of time with persistent chest pain of unknown etiology. Ten of these patients were found to have either costochondritis or chest wall syndromes. The other four were observed to have chronic episodic chest pain and persistently elevated creatine phosphokinase (CPK) values. All CPK isoenzymes were 100 percent mm band. In these four patients, a thoracic outlet syndrome manifested primarily by chest pain appeared to be responsible for the angina-like pain and was associated with abnormal CPK values.

**Patients and Methods**

Four patients were seen at the Arthritis Center of Memorial Hospital of Long Beach between August 1980, and April 1981, with recurrent chest pain and abnormally elevated CPKs. All patients were men. Two had left-sided chest pain with radiation down the left arm. One had substernal and left-sided chest pain. The fourth had interscapular pain which radiated toward the anterior chest. Their pain was described as tightness, pressure, or dull ache. No shoulder, arm, or hand symptoms typical of thoracic outlet were present. Three subjects, when questioned closely, admitted to mild paresthesias in two to three fingers. Two had sustained chest injuries at four and ten years prior to evaluation. Duration of symptoms ranged from 12 to 48 months and averaged 24 months.

None had a history of thyroid disease, muscle disease, or alcohol abuse to account for elevated CPKs. There was no history of factors known to elevate CPKs, such as heavy exercise, muscle trauma, loss of consciousness, or head trauma within 72 hours of each measured CPK.

Three had completed treadmill stress tests which were all negative for coronary artery disease. Two had undergone coronary arteriography which were both normal. One had a five-vessel bypass with repeat arteriography demonstrating widely patent grafts. The upper gastrointestinal radiographs, chest x-ray films, and Bernstein's test for esophagitis were all normal.

The CPKs were done with the DuPont automatic clinical analyzer which utilized a modification of the UV enzymatic determination. The normal range established at our laboratory was 20 to 50 IU/L for male patients. The CPK isoenzymes were done by electrophoresis with quantitation by densitometry. Vasographs were done with plethysmography with positioning for thoracic outlet as shown in Figure 1.

**Case Reports**

Case 1 was a 64-year-old white man who had multiple admissions for chest pain after having a successful five-vessel coronary artery bypass for coronary artery disease. His chest pain was over the left anterior chest with radiation down the left arm. Repeat angiography of the grafts showed them all to be widely patent. A past history was elicited of an auto accident prior to his coronary artery bypass with neck and chest pains noted thereafter. An aortic arch study done prior to his bypass had demonstrated 60 percent stenosis of the left subclavian artery. He admitted to mild intermittent paresthesias of the left first, second, and third fingers. On physical examination, he had a loud bruit over the left subclavian artery which radiated down to the shoulder. Pulses on the left were one fourth compared to three fourths on the right upper extremity. Thoracic outlet maneuvers were positive with costoclavicular compression positions. The CPK values over a two-year period ranged from 142 to 170 IU/L on three determinations. Anti-inflammatory medications were declined. Therapeutic measures were therefore limited to instruction in proper muscle posture. During the successive four months, there was gradual decline of his CPK values to 32 IU/L with decreased frequency and intensity of chest pain. His bruit persisted, although there was substantial improvement in his left upper extremity pulses to 2/4 on physical examination.

Case 2 was a 31-year-old Nigerian man with a four-year history of...
improved symptomatically while receiving indomethacin, 50 mg orally three times daily and proper muscle positioning with his CPK values improving from a high of 350 IUL to 247 IUL.

Case 3 was a 33-year-old white male mechanic who did much of his work with his shoulders hyperabducted to reach overhead. He complained of interscapular chest pain with radiation forward to the anterior chest. He also had intermittent mild hypesthesia of his left fourth and fifth fingers and lateral left arm. A bone scan of the thoracic spine was normal. Thoracic outlet maneuvers and vasograms demonstrated bilateral hyperabduction and costoclavicular syndromes. His CPK values averaged 126 IUL. Although he improved receiving naproxen, 250 mg orally three times a day, his job as a mechanic aggravated his pain particularly when working overhead. He was recommended for job retraining rather than pursuing invasive studies and possible vascular surgery. His last CPK value was improved at 77 IUL while receiving phenylbutazone, 200 mg orally three times daily.

Case 4 was a 41-year-old white man with multiple admissions for chest pain. His chest pain was left inframammary and substernal and felt like a pressured weight. His cardiac catheterization and coronary angiography were normal. The CPK values ranged from 99 to 195 IUL over a three-year period of time. Maneuvers for costoclavicular syndrome elicited a positive response. The patient improved receiving phenylbutazone, 200 mg orally three times daily (after failing to respond to indomethacin) with decline in his CPK value to 54 IUL.

RESULTS

The average pretreatment CPK values ranged from 126 to 264 IUL or 2.5 to 5.3 times the upper limit of normal. The CPKs were mm fraction in all cases (Table 1).

On physical examination, no chest wall pain could be elicited on compression or palpation of the chest wall or costochondral junctions. Three had positive thoracic outlet maneuvers for vascular compromise confirmed later by vasographs. Maneuvers included costoclavicular, hyperabduction, and scalenus anterior compression positions. The one patient with normal vascular maneuvers had a positive EMG for a left C7 lesion with a normal CAT scan of the cervical spine. This would be compatible with a thoracic outlet impingement of the C7 nerve.

All four patients obtained both significant (75 percent to 100 percent) relief of their pain and improvement of the CPK values with anti-inflammatory medications and/or proper muscle positioning. This included avoidance of those positions re-creating shoulder hyperabduction (arms overhead), shoulder

Table 1—Summary of Data on Four Patients with Elevated CPKs and Chest Pain

<table>
<thead>
<tr>
<th>Age</th>
<th>Average Pre-RX CPK†</th>
<th>Average Post-RX CPK†</th>
<th>TMST‡</th>
<th>Coronary Angiography</th>
<th>Thoracic Outlet Maneuvers and Vasographs</th>
<th>EMG/NCV‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>64</td>
<td>131</td>
<td>47</td>
<td>N</td>
<td>P</td>
<td>Positive</td>
<td>N</td>
</tr>
<tr>
<td>31</td>
<td>264</td>
<td>247</td>
<td>N</td>
<td>N</td>
<td>Negative</td>
<td>Positive</td>
</tr>
<tr>
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<td>126</td>
<td>77</td>
<td>ND</td>
<td>ND</td>
<td>Positive</td>
<td>N</td>
</tr>
<tr>
<td>41</td>
<td>145</td>
<td>57</td>
<td>N</td>
<td>N</td>
<td>Positive</td>
<td>ND</td>
</tr>
</tbody>
</table>

*N is normal; ND, not done; P, patent grafts; and normal CPK in males, 20 to 50 IUL/L.
†All PCKs were 100 percent mm fraction on CPK isoenzyme testing.
‡TMST = Treadmill stress test
§Electrolymogram/nerve conduction velocity.
hyperextension (military posture) or scalenus anticus compression (cervical lateral rotation).

DISCUSSION

Thoracic outlet syndrome has been well described.\textsuperscript{10-13} This syndrome occurs when there is compression of the subclavian artery or the brachial plexus within the thoracic outlet. This can be caused by compromise from such structures as the scalenus anticus muscle, congenital fibrous bands, cervical ribs and deformed clavicles. Symptoms usually involve the upper extremity with pain and paresthesias noted in the C8-T1 distribution. Chest pain has been a less frequent and minor secondary symptom.\textsuperscript{10-13} Abnormal CPK values associated with chest pain due to thoracic outlet syndrome have not been reported previously.

Pseudoangina may occur because the usual thoracic outlet arm pain is referred to the chest wall similar in dermatomal distribution to the referred pain of angina pectoris.

With clinical presentation of chest rather than shoulder or arm pain predominating, the diagnosis of thoracic outlet is often overlooked. Persistent elevations of CPK values may provide a clue to the presence of this syndrome. These values may be abnormal from ischemic or neurologic compromise of muscles supplied by the subclavian artery or brachial plexus respectively. As pointed out in several studies,\textsuperscript{14-18} vasographs and vascular maneuvers are not always positive. Nerve conduction and electromyography are often necessary as in case 2 to detect abnormalities in the thoracic outlet and even they may have only a detection rate of 61 percent.\textsuperscript{10-13}

Of interest in two of the four patients reported here is the remote past history of chest trauma. The possibility exists that adhesions and alteration of anatomy around the thoracic outlet secondary to this trauma might contribute to this syndrome.

The data here suggest that there is a correlation between improvement in clinical symptoms and CPK values, but the small number of patients does not allow for statistical significance. If this observation is also noted with larger numbers of patients, then CPK values may serve as a useful objective parameter to evaluate therapeutic response. Unfortunately, none of the previously published studies on thoracic outlet which we reviewed mentioned CPK values and thus no other data are available.

Urschel et al\textsuperscript{14} previously described a pseudoangina syndrome in 44 patients with thoracic outlet syndrome. The patients had substernal and left-sided chest pain with radiation into the neck, left shoulder, or left parascapular posterior chest wall. The authors did not report CPK values and advocated conservative physical therapy unless ulnar nerve conduction was slower than 55 meters per second. However, transaxillary resection of the first rib was performed.

McGough et al\textsuperscript{15} reported that conservative treatment of thoracic outlet syndrome was successful in 90.6 percent of a total of 1,200 patients. The authors did not describe any patients, however, who had chest pain and did not report CPK values. Patients improved doing exercises designed to strengthen the shoulder girdle muscles and instruction in proper exercises against gravity. The goal was to elevate and slightly abduct the shoulder girdle thereby decreasing compressive forces.

It appears from this initial collection of four patients that thoracic outlet compromise of either the vascular or neural structures can produce chest pain mimicking angina in dermatomal distribution associated with elevation of CPK to 2.5 to 5.3 times the normal values. This syndrome should be suspected whenever patients persist with chest pain and no other cause can be determined. Chronically abnormal muscle enzyme values should further make one suspect of this syndrome. Pain can be relieved by anti-inflammatory preparations and conservative muscle positioning. Surgical correction may be warranted in resistant cases.

ACKNOWLEDGMENT: Drs. Paul Greenberg and John Messenger referred the patients, and Laurel Mae Wilhelm assisted in preparing the manuscript.

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