Acute ECG Changes and Chest Pain Induced by Neck Motion in Patients with Cervical Hernia

A Case Report

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ABSTRACT

We report two cases of acute cervical angina and ECG changes induced by anteflexion of the head. Cervical angina is defined as chest pain that resembles true cardiac angina but originates from cervical discopathy with nerve root compression. In these patients, Prinzmetal’s angina, valvular heart disease, congenital heart disease, left ventricular aneurysm, and cardiomyopathy were excluded. After all, the patient’s chest pain was reproduced by anteflexion of head, at this time, their ECGs showed nonspecific ST-T changes in the inferior and anterior leads different from the basal ECG. ECG changes returned to normal when the patient’s neck moved to the neutral position. To our knowledge, these are the first cases of cervical angina associated with acute ECG changes by neck motion.

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Introduction
Cervical angina is defined as chest pain that resembles true cardiac angina but originates from cervical discopathy with nerve root compression. This condition, which is also referred to as pseudoangina, most commonly results from compression of the C6 and C7 nerve roots. We report two cases of acute cervical angina and ECG changes induced by anteflexion of the head. To our knowledge, these are the first cases of cervical angina associated with acute ECG changes by neck motion.

Case Report

Case 1. A 43-year-old man initially presented with anginalike symptoms, including crushing substernal left anterior chest pressure radiating from the left sternum around to the scapula and going down into his left arm. The episodes had come on suddenly and lasted a few seconds to minutes. He reported some nausea and diaphoresis with the episodes. These attacks had started approximately 1–2 days before his admission. His family history was noncontributory. He denied alcohol and tobacco use. Physical examination of the cardiopulmonary system was normal. ST-T changes in the inferior and precordial leads were observed on his ECG. The results of the rest of the laboratory examinations including the complete blood count, thyroid panel, and posteroanterior and lateral chest radiographs were within normal limits. Transient severe chest pain episodes continued after admission. In the light of the clinical information and ECG changes, the condition was interpreted as an unstable angina pectoris, and he was admitted to the coronary care unit. Conventional therapy for unstable angina including beta-blocker, calcium channel blocker, nitroglycerin, aspirin, and heparin were administered. No response was observed to this therapy. Coronary angiogram was performed and showed apparently normal coronary arteries. Echocardiography, treadmill exercise test, and myocardial perfusion scintigraphy showed normal values. Thus, Prinzmetal's angina, valvular heart disease, congenital heart disease, left ventricular aneurysm, and cardiomyopathy were excluded.

After all, the patient reported induction of chest pain by downward pressure on the head and active motion of the head in anterior direction. In addition, hypoesthesia was detected on his left arm in the C7 root distribution. In the light of this information the patient's chest pain was reproduced by anteflexion of his head, at this time, his ECG showed nonspecific ST-T changes.

Figure 1. Resting ECG.
in the inferior and anterior leads (Figures 1 and 2). ECG changes returned to normal when the patient's neck moved to the neutral position. We confirmed reproduction of chest pain and ECG changes several times when the patient flexed his head.

Magnetic resonance imaging revealed cervical discal hernia at the C6–C7 level obliterating epidural space and causing foraminal obstruction due to osteophytes (Figure 3A, B). Based on the patient's pain distribution, the reproduction of symptoms, and signs with head tilt and the imaging studies, the diagnosis of cervical angina was made. He was transferred to the physical therapy department. Because conservative measures failed to alleviate the patient's pain, surgical treatment was recommended, but the patient refused.

Case 2. A 39-year-old woman presented to the Department of Neurosurgery in December 1999 complaining of neck pain that occurred remittently and radiated from left sternum around to scapula and going down into her upper arms. Her pain had started approximately 1–2 months before her admission. She denied alcohol and tobacco use. Physical examination of her cardiopulmonary system was normal. ST-T changes in the inferior and precordial leads were observed on her ECG. The results of laboratory tests, posteroanterior and lateral chest radiographs were within normal limits. Echocardiography, treadmill exercise test, and myocardial perfusion scintigraphy showed normal values.

After all, the patient's chest pain was reproduced by anteflexion of her head, at this time, her ECG showed nonspecific ST-T changes in the inferior and anterior leads different than the basal ECG. ECG changes returned to normal when the patient's neck moved to the neutral position. Reproduction of transient neck pain, with the synchronous nonspecific ECG changes, occurred repeatedly when the patient flexed her head. Thus, we confirmed sudden reproduction of ECG changes several times when the patient flexed the head.

Magnetic resonance imaging revealed cervical discal hernia at the C3–C4, C4–C5, and C6–C7 levels and spondylosis. Epidural space was obliterated and cord and root were compressed. Based on the patient's pain distribution, the reproduction of symptoms and signs with head tilt and the imaging studies, the diagnosis of cervical angina was made.

Discussion

The syndrome of chest discomfort in the absence of significant coronary artery obstruction has been an enigma for many years. Approximately
Figure 3. Cervical spinal MRI demonstrated C6-C7 discal hernia obliterating epidural space and right foraminal compression. (A) Sagittal view. (B) Axial view.

20% to 30% of patients, who undergo coronary arteriography for the evaluation of chest pain, are found to have normal coronary arteries.\(^4\)

Cervical angina or pseudoangina, which is caused by cervical spondylosis and nerve root compression, is an uncommon entity that may explain the presence of chest pain despite a negative cardiac work-up. In cervical angina, chest or arm pain can be induced by active neck motion combined with downward head pressure.\(^2,3\)

Patients with cervical angina can present with anginalike pain in association with nausea, diaphoresis, and shortness of breath. Interestingly, Masters\(^5\) observed that in some instances
pseudoangina pain could be relieved by the use of nitroglycerin. For these similarities, the coexistence of coronary artery disease should be ruled out in cervical angina. Treatment includes intermittent cervical traction, physical therapy, nonsteroidal antiinflammatory drugs and muscle relaxants. If these measures fail to alleviate the patient's pain, surgical intervention to decompress the nerve root may be indicated.

The pathogenesis of cervical angina can be explained by the fact that cervical neural roots from C4 to C8 contribute to the sensory and motor innervation of the anterior chest wall. Neural root compromise at the cervical foramina can produce several symptoms, including radicular pain. Patients with true cervical angina are more likely to have disease at the C6 and/or C7 level. Divisions of the anterior roots of C5 to T1 give origin to a number of individual nerves that supply motor, sensory, and autonomic fibers to the upper thorax, shoulder, and arm. Autonomic symptoms, including nausea and diaphoresis, can occur and are mediated through the sympathetic nervous system, although the exact mechanism is not known. The patient may or may not have neurologic deficit, depending on the severity and the chronicity of the discopathy. A useful diagnostic maneuver is the lateral head tilt. If the cause of the syndrome is truly cervical discopathy, the maneuver should reproduce the pain.

For reasons which are not understood, several types of acute disturbance in the central nervous system give rise to electrocardiographic changes. ECG changes can be seen in relation to infections or tumors in the central nervous system, after head injuries, and after neurosurgery, but are most commonly seen in association with subarachnoid hemorrhage and intracranial hemorrhage. Also, T-wave changes have been seen in cervical angina, as shown in this case. It is well known that electrocardiographic changes, such as, T-wave inversion, ST segment elevation or depression, prolongation of the QT interval, can be observed in central nervous system diseases. The most generally accepted explanation of the pathogenesis of ECG changes in central nervous system disease is that the changes occur as a result of sympathetic or parasympathetic activity resulting from damage in central areas rich in autonomic connections.

Conclusion

These cases have demonstrated that ST-T alterations can develop during chest pain due to cervical discopathy; however, the exact mechanism is not understood. Although we confirmed acute reproduction of ECG changes several times when the patient flexed the head, further studies need to explain the pathogenesis of ECG changes in patients with cervical hernia.

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