

Cardiac Pain Syndrome

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Cardiac pain is a warning symptom for any doctor. Reflex pain syndrome should be a consideration in the presence of chest pain with negative findings on cardiac physical examination and ECG.

Coronary vasomotor and cardiac dystrophic changes might result as a reflex response to irritated receptors from disturbed cervical or thoracic vertebrae and connected sympathetic structures.

The heart is innervated by upper, middle and lower heart nerves coming from the cervical sympathetic ganglia. Pathology of the sympathetic plexus, vertebral artery and/or other sympathetic structures could influence the state of the heart. The integrity of vegetative innervation of the cervical vertebral discs should also be taken into consideration. Sympathetic fibers go to cervical nodes from the lateral horns through rami communicantes albi of the upper segments. This shows the possible role of pathology of the cervical and upper thoracic vertebrae in cardiac vasomotor dystrophic disturbances (vertebrogenous discirculatory cardiopathy).

Disorders of cervical or upper thoracic vertebrae could be the cause of coronary pain or might provoke it, especially in the case of coronary pathology when the localization of coronary pain might be atypical. These vertebral disorders might also serve as a contributing factor to pain with a more complicated etiology (coronary, sternal and vertebral).

Thus, thoracogenous impulses might imitate heart pathology through pain coming from the anterior chest wall, and modify the true heart pain by competing with it. What is the difference between vertebrogenous pseudocardiac pain (false angina) and true anginal pain? They differ by location, duration, dependence on vertebral position, and by the effectiveness of nitroglycerin at relieving the pain. Such cases might be described as anterior chest wall syndrome (ACWS). This is a vertebrogenic syndrome, and it might be influenced by previous diseases of coronary vessels, myocardium and brain tissue. The main source of pathological impulses is usually the thoracic and cervical vertebral areas. ACWS is often combined with other types of back, neck and cranial pains.

Three variants of ACWS might be considered; they are respectively caused by cervical, thoracic and cervical-and-thoracic pathology. All of these variants present with pain, muscular-and-tonic, dystrophic and neurovascular changes in the pectoris major and other tissues of the anterior chest wall. This pain has its onset after physical challenge of the anterior chest muscles, and head, neck and thoracic twists, but never after emotional and general physical challenges or food intake as happens in the case of anginal pain. The patient experiences long-term (hours, days, months) dull pain sometimes with burning sensations. One might find widespread soreness on palpation and trigger points at muscle attachments. The most painful zones are usually located on the middle axillary line at the level of the 3rd-4th ribs, parasternally at the costochondral junctions of these same ribs, and on the inferior border of the pectoralis major. ACWS might be explained by reflex tension and dystrophic changes of several muscle groups such as sternocleidomastoids, scalenes, and subclavicular muscles. Involvement of the last one might lead to the anomaly of the rib-to-clavicle gap and consequent symptoms.¹

Dystrophic-and-degenerative changes of the spine inevitably lead to the disturbed trophic supply of

paravertebral sympathetic nodes. The paravertebral sympathetic nodes suffer from dystrophic changes as well. All that might lead to the insufficient synthesis of norepinephrine in sympathetic neurons, and, as a compensation for that, increased discarding of epinephrine from the adrenal medulla. Excess of epinephrine might in turn trigger cardiac arrhythmias. Consequently, early determination and prevention of dystrophic-and-degenerative changes of the spine through manipulation might decrease the patient's risk for cardiac pathology.²

In case we are dealing with true heart problems, viscerosomatic reflex patterns are involved in the presentation of characteristic findings. The ones which deal with coronary artery disease or ischemic conditions of the heart consist of the involvements of two or more adjacent spinal segments from T1 to T5, with T2 and T3 being most frequently involved. Changes are characterized by deep muscle splinting reaction over the lateral half of the transverse process, extending an equal distance laterally beyond the tip of the transverse process, along with a resistance to segmental spinal motion.³ Data presented in a later review by the same author show peak incidence of spinal findings in the C2-C3, T2-T4, T9-T10, and L5-S1 areas in 76 patients diagnosed with coronary artery disease.⁴

Clinical findings of the ECG improvements following spinal manipulations (normalization of sinus rhythm, decrease of tachycardia and premature ventricular contractions) support the evidence of somatovisceral patterns and show the effectiveness of spinal manipulations for certain heart disorders.^{5,6}

References

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