Research Article

Closed heart injury in medical practice: Review of literature

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ABSTRACT

Aim: This paper reviews the publications and analyzes modern literature on pathogenesis, classification, clinical features, diagnosis, and treatment of closed heart injury. Closed heart injury is a heart damage, which develops in the immediate period after an injury or as a result of metabolic disorders arising from the injury. Its severity depends on the nature of injury, the phases of cardiac activity at the time of injury, and the pre-injury state of the myocardium and coronary vessels. At the same time, there are hemorrhages, degenerative, and necrotic changes in the myocardium, as well as metabolic disorders observed in the heart. **Materials and Method:** The following main factors are identified in the pathogenesis of closed heart injury: Heart compression with increased pressure in the cardiac cavities, heart damage with the fragments of ribs, heart displacement in the chest, stress disorder of the autonomic regulation of the heart, and myocardial metabolic disturbance; development of cardiac arrest is possible. Clinical manifestations of closed heart injury are either concussion or contusion of the heart, myocardial rupture, and traumatic myocardial infarction. **Results:** Heart concussion starts with arrhythmias in the form of premature ventricular contraction, atrial fibrillation or flutter, bradycardia, and conduction disorders. Heart contusion can simulate angina and be accompanied by weakness, shortness of breath, pallor, Crocq's disease, and rhythm disorders. Traumatic myocardial infarction is more common in the elderly against the background of coronary artery disease, and more severe, it may be complicated by pulmonary edema. The detachment of the intima of the coronary arteries is also possible to occur, leading to a heart attack in the absence of atherosclerosis. A traumatic outer myocardial rupture promotes hemopericardium and cardiac tamponade, resulting in death, as well as cardiac arrhythmias. An internal myocardial rupture leads to acute heart failure in combination with traumatic shock that makes an unfavorable prognosis. An acute valvular insufficiency is typical of the initially modified valves. The aorta may also be damaged in the form of break at the point of attachment to the spine due to the intercostal arteries. Possible consequences of a closed heart injury are also a myocardial dystrophy, which ends in either complete recovery or persistent arrhythmias. **Conclusion:** Knowing the pathogenesis and clinical manifestations of a closed heart injury is an important element of the competence of a modern physician.

KEY WORDS: Classification, Closed heart injury, Diagnostics, Treatment

INTRODUCTION

Thorax injuries are a major cause of mortality in 20–25% of cases and, even in a half of the cases, significantly aggravate the patient's condition. Closed chest injuries in >70% of cases are accompanied by damage to the heart, which leads to death in 45–62% of patients. Closed heart injury is a damage to the heart, developing in the immediate period after an injury or as a result of post-traumatic metabolic disorders.[1]

The severity of closed heart injury depends on the nature of injury, the phases of cardiac activity at the time of injury, and the pre-injury state of the myocardium and coronary vessels. At the same time, there are hemorrhages, degenerative and necrotic changes in the myocardium, as well as metabolic disorders observed in the heart. The following main factors are identified in the pathogenesis of closed heart injury: Heart compression with increased pressure in the cardiac cavities, heart damage with the fragments of ribs, heart displacement in the chest, stress disorder of the autonomic regulation of the heart, and myocardial metabolic disturbance.[2] Injuries, not accompanied by severe damage to vital organs, may promote the development of cardiac arrest as follows: Apnea, vasovagal reflex, and primary ventricular fibrillation. The most likely mechanism is primary atrial fibrillation. Clinical manifestations of closed heart injury are either concussion or contusion of the heart, myocardial rupture, and traumatic myocardial

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infarction.[3] The cause of death in heart injuries is often arrhythmias occurring due to severe myocardial damage.[4] Late diagnosis of cardiac injury reaches 55% of cases and is associated with the severity of associated trauma.[5] Closed heart injuries are often revealed only at postmortem examination. In this context, the diagnosis of closed heart injuries is an actual clinical problem.[6]

METHODS

We conducted the analysis of scientific publications devoted to the pathogenesis, classification and progress features, and diagnosis and treatment of closed heart injury.

RESULTS AND DISCUSSION

There are five major factors distinguished in the pathogenesis of CHI: (1) A sudden cardiac compression with increasing pressure in its cavity, (2) a sudden blow to the heart region with its damaging with the fragments of ribs, (3) heart displacement on chest injury, (4) the impact of stress of central nervous system on the heart, and (5) myocardial metabolic disorders as a result of polytrauma. Possible mechanisms of death in patients having suffered CHI are apnea, deep vasovagal reflex, or primary ventricular fibrillation.[1,7]

Most often, clinical manifestations of closed heart injury are heart concussion, heart contusion, traumatic myocardial infarction, myocardial rupture, acute valvular insufficiency, and myocardial dystrophy.[8-11]

Heart concussion starts with arrhythmias in the form of premature ventricular contraction, atrial fibrillation or flutter, bradycardia, and conduction disorders. The pain is rare and short-lived. The pain is rare and short-lived, so characteristically, the lowering blood pressure and voiceless heart tones. This is accompanied by cerebral disorders: Dizziness, muscle tone disorders, and consciousness disorders. In 50% of cases, the collapse develops immediately; however, it may occur after a period of consciousness preservation. The electrocardiogram (ECG) shows subepicardial changes and cardiac arrhythmias. Differential diagnosis is conducted in case of myocardial infarction and viral myocarditis. The key ones are the relationship of symptoms with a blow to the chest and the absence of changes in the heart that can cause death.[9-11]

Heart contusion is accompanied by the following symptoms: Pain at the site of injury or behind the sternum; palpitation, weakness, shortness of breath, paleness, and acrocyanosis; rhythm disturbance (tachycardia, bradycardia, and arrhythmia); low volume pulse and labile pressure tending to reduction; and possible muffled heart tones, systolic murmur at the heart apex, gallop rhythm, and pericardial murmur. The ECG can reveal sinus tachycardia or bradycardia, arrhythmia, atrial fibrillation or atrial flutter, various types of tachycardia, atrioventricular blocks, intraventricular conduction disturbances, signs of ischemia, and metabolic disorders. The acute period may be complicated by cardiogenic shock, occurring against the background of the traumatic one. On days 3–10 after injury, after the state stabilization, there are the regrowing clinical and ECG signs of myocardial damage caused by the development of deep degenerative and inflammatory changes in the hemorrhage areas.

Heart injuries are classified as follows: [1,9] (1) By severity: Mild - no hemodynamic disorders, short-lived rhythm and conduction disturbances, and pronounced changes on ECG; moderate (anginal) - persistent rhythm and conduction disturbances and transient hemodynamic disorders; and severe (infarction-like) - persistent and progressive hemodynamic disorders; (2) by disease stages: Primary traumatic disorders (first 3 days), traumatic myocarditis (up to 25 days), recovery of the impaired functions (up to 25 days), and outcome; and (3) by nature of morphological disorders: First period - acute (2–3 days), second period - reparative regeneration (up to 14 days), and third period - post-traumatic cardiosclerosis (over 14 days).

Traumatic myocardial infarction is more common to older people with coronary artery disease. It is more severe than non-traumatic myocardial infarction: Intense chest pain immediately after the injury, arrhythmias, and cardiogenic shock. It is characterized by paleness, cyanosis, cold sweats, tachycardia, low volume pulse, and hypotension. The examination revealed muffled heart tones and maximum systolic murmur at the heart apex. In the acute period, ECG shows ST-segment elevation, pathological Q-wave over the destructed area, various arrhythmias, and conduction disorders. The course may be complicated by cardiac asthma and pulmonary edema. The detachment of the intima of the coronary arteries is also possible to occur, leading to a heart attack in the absence of atherosclerosis.

Post-traumatic myocardial rupture can be both external and internal.[1,8] The injured are pale, with severe shortness of breath, filiform pulse, collapse, and wide heart borders. Severe arrhythmias may occur. In case of internal ruptures, the integrity of the ventricular or atrial septum, as well as valves, tendon threads, and papillary muscles is impaired. It is characterized by shortness of breath, cyanosis, tachycardia, hypotension, and rough systolic murmur. An acute right ventricular (in case of ventricular septal defect) or left ventricular (in case of damaged papillary muscles or mitral valve) heart failure may occur in combination with the traumatic shock. The prognosis is unfavorable.

An acute valvular insufficiency occurs due to damage to the valves, papillary muscles, and chords and is characteristic of initially modified valves. The aortic valve is most vulnerable. Damage to the valves can occur on the appearance of noise, hypotension, and increasing pulmonary edema. Pansystolic noise also appears in rupture of the interventricular septum. An acute tricuspid regurgitation is better tolerated and manifested in swelling of legs and ascites. Aorta often suffers during CHI, in the form of bursting or tearing, which leads to death.

Post-traumatic myocardial dystrophy occurs frequently and is divided into three periods: Acute (3–5 days), subacute (7–14 days), and recovery (from 15–30 days to 1–2 months). The complete recovery is possible, but arrhythmias or angina pectoris often occurs in the future. Arrhythmias, muffled tones, and hemodynamic disorders are typical of this condition. A carefully collected history and the detection of traces of chest injury are highly important in diagnosing.[1,8]

Diagnosis of closed chest injuries should be carried out according to the following scheme: (1) Clarification of the circumstances and mechanism of injury (direct stroke and chest compression), (2) evaluation of external clinical signs (deformation of the chest, the nature and frequency of respiration, cyanosis, etc.), and (3) carrying out a thorough objective examination. Local pain, crepitation, the "interrupted inhalation" symptom, Payr's sign (tenderness when bending to the healthy side), soreness with axial loads, subcutaneous emphysema, changes in percussion tone, auscultatory findings, and changes in vocal tremor are characteristic. In the presence of experience, a thorough examination, palpation, and auscultation can be done in 5 min. It is urgent to identify life-threatening conditions and establish their cause. Examination may reveal the presence of cyanosis - a sign of increasing hypoxemia due to respiratory failure. If only the face, neck, and upper half of the chest (a symptom of "decollete") have the cyanotic color, one must suspect traumatic asphyxia arising from chest compression. Traumatic asphyxia is also characterized by pinpoint hemorrhages in the skin, mucous membranes, and conjunctiva. The presence of independent breathing, paradoxical breathing (the final fracture of the ribs with flotation of the thoracic wall), and unilateral respiratory movements (rupture of the bronchus, pneumothorax, and unilateral hemothorax) is revealed. Swollen soft tissues, especially the eyelids and necks (subcutaneous emphysema), are a sign of damage to the lung or the main bronchus. Attention should be payed to unusual breathing noises, "sucking" wounds of the chest wall. In case of penetrating wounds, both the front and back surface of the trunk were examined (a hole can be found on the back). Swollen non-pulsating cervical veins are a sign of a developing cardiac tamponade. In addition, the swelling of the cervical veins is observed during the agony.

Rapidly palpate the neck, chest, arms, and stomach. Subcutaneous emphysema is a sign of a strained valve pneumothorax or bronchial rupture. Consistently palpate ribs and sternum lightly press the chest in all directions. In case of fractures of the ribs with displacement injuries of the thoracic organs with acute bone fragments are possible.

Measurements of blood pressure, heart rate, and pulse are mandatory. Pulse and blood pressure are measured on each arm. The absence of a pulse may be due to damage to a large vessel.

During auscultation of the lungs, respiratory noises in the right and left lungs are compared. If they differ, percussion is performed. Dullness of percussion sound on the affected side means either hemothorax or atelectasis. Loud tympanic sound over one lung, especially in case of penetrating injury on this side, is a sign of pneumothorax. Intense pneumothorax is possible. It is often difficult to perform auscultation of the heart, but it is absolutely necessary. Heart murmurs can indicate damage to one of the valves, rupture of papillary muscles, or septum. If, during the diastole, a noise resembling "crunching" (pericardial friction noise) is present, air can be in the pericardial cavity.

ECG is performed to all injured, especially when referring to a blunt trauma - a strong blow to the chest, a fall on the blunt object. The following typical ECG changes are distinguished: (1) Rhythm disturbance, various conduction disorders, up to a complete blockade, (2) changes in the T wave (coronary), (3) shift in the ST segment, and (4) increase in the amplitude of the P wave. At the same time, the absence of changes on the ECG does not completely exclude injuries.

The prognosis in closed heart injuries varies. Either complete recovery and rehabilitation or incomplete recovery is possible (pain associated with physical activity or arrhythmia may occur in the future). Death in the closed heart injury can occur in case of the development of ventricular fibrillation, asystole, heart failure, or myocardial rupture. Under impaired consciousness and traumatic shock, the diagnosis of this disease is considerably difficult.

A high rate of late diagnosis leads to both delay in the indicated treatment and increased mortality. Closed heart injury has different clinical manifestations at different stages of its flow.

SUMMARY

Chest injuries are often accompanied by damage to the heart. Closed heart injury is a damage to the

heart, developing in the immediate period after an injury or as a result of post-traumatic metabolic disorders. Diagnosis of closed heart injury is a relevant clinical problem. The cause of death in closed heart injuries is often arrhythmias occurring due to traumatic myocardial damage. Late diagnosis of closed heart injury happens in more than half of the cases which is associated with the severity of an associated trauma. The following main factors are important in the pathogenesis of closed heart injury: Heart compression, heart damage with the fragments of ribs, abrupt heart displacement in the chest, stress disorder of the autonomic regulation of the heart, and myocardial metabolic and coronary flow disturbance. Closed heart injury is characterized by hemorrhage and degenerative and necrotic changes in the myocardium, as well as its metabolic disorders.

CONCLUSIONS

Knowing the pathogenesis and clinical manifestations of a closed heart injury is an important element of the competence of a modern physician.

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