Slow progressing cardiac complications—a case report

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Abstract

Objective: This case presentation describes an uncommon development of complete heart block. Within 48 hours after a motor vehicle accident with the deployment of the air bag against the patient's chest, the patient reported exertional bradycardia and shortness of breath.

Clinical Features: A 51-year-old man was in a motor vehicle accident. After the collision, he noticed a slow onset of chest discomfort with exertion and bradycardia. The patient experienced cardiac difficulty during a stress electrocardiogram. During the 4 months after the motor vehicle accident, symptoms progressed; and a diagnosis of vagal sympathetic reflex was suggested.

Intervention and Outcome: A pacemaker was finally required because of the ventricular pacing of 35 to 40 beats per minute, which was symptomatic of a complete atrioventricular block.

Conclusion: A gradual progression to complete atrioventricular block over a period longer than 3 weeks is unusual. This case demonstrates that a patient manifesting exertional bradycardia and shortness of breath shortly after chest trauma should be regularly monitored until all symptoms are resolved.

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Introduction

Cardiac injury due to nonpenetrating chest trauma or thoracic trauma is very common in motor vehicle accidents and deployment of air bags.^{1,2} Many of these injuries go unsuspected and in many cases result in no long-term disability.³ However, some do progress to manifest themselves with arrhythmias, heart rupture,

septal rupture, and other types of cardiac problems. Despite the large number of cases that are now being documented in the literature, there is no standardization and much disagreement on how to diagnosis and treat the cardiac injury. Furthermore, there is great discrepancy in reporting the incidents and implications as to nonpenetrating heart trauma.^{4–9}

The purpose of this paper is to describe an abnormal presentation and development of a complete atrioventricular (AV) block after an automobile accident. The apparent precipitating mechanism was deployment of the air bag against the patient's chest resulting in a blunt force injury, causing a suspected contusion of the heart.

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Case report

A 51-year-old man, before an auto accident, walked 4 miles each day in 60 minutes with no shortness of breath, no chest discomfort, and no heart distress. There was no history of heart disease. He had no history of shortness of breath, chest discomfort, arrhythmias, or AV block. An electrocardiogram (ECG) done in the past was read as normal.

The patient was involved in an auto accident. He was on a straightaway, and a pickup truck approaching from the opposite direction started to slow but turned directly in front of him. The front center of the patient's car struck the right front corner of the truck as the truck turned at approximately 35 mph. The driver of the auto and the passengers were taken to the hospital for evaluation, treated, and released. The patient reported being stiff the day after, so he did not go for his daily walk. Two days after the accident, he started back for his typical 4-mile walk; however, he noted that he had to slow his pace because of some chest discomfort. The slower pace allowed the discomfort to ease off until there was no discomfort whatsoever. As he progressed, he found that he would regularly have to slow his pace; and by the eighth day, he was only able to walk 2 miles in 60 minutes. On the evening of the 12th day, he noted a severe drop in heart rate, 40 to 45 beats per minute, which held at this level for several hours and then returned to normal. Because the heart rate returned to normal rate and rhythm, he did not go to the emergency department but called his family physician. He was unable to get an appointment until 2 weeks later.

An examination was performed, and Cardiolite GXT (Bristol-Myers Squibb, North Billerica, MA) was ordered with a stress ECG. While he waited for the evaluation, he was generally stable with occasional incidents of bradycardia. Two weeks after his visit to the family doctor, he was seen at a local medical clinic. During the stress test, the examiners noted a sudden drop from maximum heart rate to 90 beats per minute. The patient became lightheaded and pale, and the test was stopped. The result of the perfusion study was normal, showing no ischemia or infarct. Based on the information from the examination, the findings of the family doctor were that the patient was having a transitional or a self-limiting condition that would probably resolve because there was no blockage or electrical conduction abnormality noted. As a follow-up, the patient saw a second specialist 2 months later at a different clinic. During the interval, the incidents of bradycardia had increased in number. It was suggested

that the patient was having a vasovagal reflex response. If the condition did not resolve, a pacemaker would be required. The physician decided to allow further time for normalization of the vagal sympathetic response.

The patient noted more bradycardia incidents over the next 6 weeks. By the end of the fifth month, he noted a complete drop of heart rate that maintained between 30 and 45 beats per minute. He then requested another stress ECG, which revealed a third-degree heart block; and the next day, a Guidant dual-chamber pacemaker (Boston Scientific, Indianapolis, IN) was implanted. His response was very good; the relief of the bradycardia enables him to function and perform his daily activities. As required, exercise was limited to a 10-pound lift; and walking was reinitiated very quickly after being released from the hospital. He was off work for 2 weeks, in which time he recovered from surgery and had the pacemaker calibrated for his lifestyle. The patient's return to his daily activity progressed very well, and he was able to return to his daily walks and gradually was able to increase his pace. In fact, he was able to reach maximum heart rate without any interference. On a follow-up echocardiogram, a mild mitral valve prolapse was noted. Four months later, during the scheduled device check, the atrial lead was malfunctioning and needed to be replaced. This was performed 1 month later. Three days after this procedure, pericarditis developed; and he was hospitalized and administered an antibiotic and prednisone to reduce the inflammation. After release from the hospital, gradual progress was made; and he was able to return to work. The pacemaker was recalibrated for his lifestyle. At the time of this writing, the patient was doing well, exercising on a daily basis and able to walk at a rate of 1 mile per 15 minutes. After the 3-month lifting and weight restrictions, he was able to start back to controlled resistive exercise using Cybex equipment (Cybex International Inc., Medway, MA) to limit stretch and strain to the chest area.

Discussion

The atypical presentation and development of periodic bradycardia and vasovagal reflex response to complete AV heart block demonstrate the need to monitor patients after chest trauma. This case, as is noted in the literature, first identified an arrhythmia within 48 hours after the trauma. Unlike most cardiac injuries that progress rapidly, this one developed slowly and did not manifest any objective data until 5 months later.

Five mechanisms of injury

Trauma to the thoracic cavity can result from 5 different mechanisms of injuries. The first involves direct injury to the chest and thoracic wall, which then causes direct damage to the heart, lungs, or great vessels.^{5,10} The second mechanism is an indirect trauma, such as trauma to the abdominal cavity, causing movement of internal organs and resulting in a ram effect upon the heart and the lungs. The ram effect, when it damages the heart or lungs, usually results in damage to the abdominal viscera and the diaphragm.^{5,10,11} A third mechanism is compression of the thoracic cavity. This type of injury includes the flailed chest, which is the fracture of the ribs bilaterally. There is direct compression of the heart and lungs between the sternum and the vertebral column.^{5,10} The fourth mechanism is an acceleration/deceleration phenomenon, which creates a swinging effect of the heart against vertebrae and/or sternum. Because the heart is free moving, it can swing upon the great vessels and stretch them. The movement can also result in direct impact of the heart on the sternum or vertebral column, causing bruising or deformation of the heart itself.^{5,10} The last mechanism of injury is a combination of any of the above in which forces in 2 or more directions cause damage to the heart. In a motor vehicle accident, there can be a combination of compression due to the air bag against the chest compressing the thoracic cavity between the air bag and the back of the seat.^{5,10} In addition to compression, there would be rapid deceleration, forcing the body forward. The deployment of the air bag stops the forward motion of the body; but the heart continues to swing forward, coming in contact with the sternum.^{5,11} Damage may vary depending upon the cardiac cycle and the volume of blood present in the heart at the time of injury. Blood, being a fluid, is relatively noncompressible⁵ and may be a factor in the rupture of the cardiac structure.

There is a well-defined description of the mechanism of injuries resulting from stress waves affecting the body and its organs. A stress wave causes inward movement of the thoracic wall, compressing the heart. The intensity of the stress wave is dependent upon the magnitude of the thrust. Stress waves are described as longitudinal pressure waves that travel at or slightly faster than the velocity of sound in the tissue and differ from sound in that they have a higher amplitude. Shock waves are described as¹⁰ “waves of higher pressure characterized by an effectively instantaneous wave front propagated through the underlying tissue at a velocity faster than the velocity of sound in tissue.” A

shock wave can be considered a special form of a stress wave.¹⁰ There are also shear waves. According to Cooper and Taylor,¹⁰ “These are transverse waves of long duration and low velocity producing gross distortions of the tissue and organs. These waves are rather like the pulse produced by the whipping of a loose rope.”¹⁰ The last is the crush injury¹⁰ “... that is not rate dependent but simply a consequence of applying effective static intense loads to the tissue.” Researchers conclude that most impact injuries to the torso encountered in civilian medical practices, such as those resulting from road traffic accidents, are produced by “shear” waves because they are long-duration, low-velocity, and high-momentum impacts producing severe but slow distortions of the body wall.^{10,12}

Types of injuries resulting from chest trauma

In a nonpenetrating chest trauma, there can be the development of a cardiac contusion. The contusion can be evaluated and assessed either during an autopsy after death or during emergency department care. The ECG may demonstrate arrhythmias,^{5,7,13} similar to a cardiac infarct with an elevated creatine kinase–MB fraction.^{2,14–16} Histologically, after impact or injury, there are release of free blood,¹⁷ some cell necrosis, and red cell incorporation into the myocardial fibers. As the heart tissue heals, there will be reabsorption of the injured tissue and the potential for scar formation. There is the potential for long-term effects that may mimic infarct-type responses on an ECG; there can be arrhythmias and further necrosis and impaired cardiac function due to the healing and to the fibrotic changes. In most of the mild to moderate type of contusions, there is complete healing, with only temporary or no residual effects.^{5,18–20}

During recovery from a mild cardiac infarct in which heart muscle dies because of ischemia, there is a period during which fibrous material will develop. Typically, with heart muscle, the tissue is going to die off or become functional again over a period of a few days to about 3 weeks; but during this time, fibrous tissue is going to develop, replacing and strengthening that portion of the heart. Wherever there is ischemia or necrosis of tissue in the heart muscle, there is a stimulation of fibroblasts to try to normalize and prevent rupture of the cardiac tissue.²¹ The subendocardial muscle of the heart typically has a slower blood flow as compared with the outer surface portions and is thus more susceptible to infarct. Because an infarct leads to necrosis,²⁰ these authors

suspect that myocardial contusions may have a greater effect on the subendocardial tissue. The necrosis, inflammation, or fibrotic changes in this layer may lead to the progression seen in some of the studies where there is a slow, gradual development of arrhythmias and other cardiac responses due to the blunt injury effect.⁹ For some patients with known cardiac contusion, angina-like pain gradually developed over hours to days after the initial impact.⁵

Reviewing the anatomy of the heart and the pericardium, it is noted that the heart is fixated only partially. It is suspended in the thoracic cavity from the aorta; and the left pulmonary aorta hooks around the left bronchus, whereas the right pulmonary artery hooks around the right bronchus, forming a sling for the heart to be suspended from. In the pericardium, the fibrous and the sierras pericardia are attached to the central tendon of the diaphragm on the lower end; and on the superior aspect, the fibrous pericardium blends with the tunica adventitia of the great vessels, supporting it from the superior aspect and giving the heart the ability to move and sway within that pericardial sack.²⁰ Under high-speed serial radiography and flash radiography, the distortion of and displacement of the heart during impact on anesthetized pigs have shown the movement of the heart and contact that the heart makes with the sternum and the vertebral column.^{6,10,12,17} Animal studies demonstrated a variety of cardiac injuries depending on the rate of speed the animal was moving and the mass of the animal. Injuries included structural damage and conduction changes.²²

Dealing with the suspected cardiac injury, physicians often encounter coinjuries including rib fractures, pulmonary contusion, pneumohemothoracic/clavicular fractures, widening of the mediastinum, spinal fractures, ruptured diaphragms, sternal fractures, closed head injury, pelvic fractures, liver injury, and spleen injuries. All have been factors that have led to the diagnosis of cardiac injury resulting from compression, either direct or indirect (a secondary compression that will have a ram effect into the thoracic cavity).⁴ The actual physical damage to the heart has included injuries to the pericardium, causing rupture and hematoma; lacerations or ruptures of the coronary arteries and the great vessels; and rupture of the right or left ventricle, right or left atrium, and the septum. Cardiac impact injury may also damage the valves themselves, the tricuspid, mitral, pulmonary, and aortic, as well as ruptures and tears of the papillary muscles.

Regarding the electrical injuries that have been associated with nonpenetrating cardiac injuries, these are ventricular fibrillation, sinus bradycardia, ventri-

cular extrasystoles, atrial fibrillation, supraventricular tachycardia, supraventricular extrasystoles, complete AV block, and AV block with idioventricular rhythms, ventricular fibrillation, loss of sinus or P-wave activity, multifocal preventricular contractions, and various arrhythmias and various degrees of AV block.^{7,11,18,19,23} One explanation of this may be the jarring of the heart that disrupts the vagal sympathetic response, or a direct interference to the neuromuscular mechanisms of the heart resulting in cardiac standstill or ventricular arrhythmias. This information was reviewed in a discussion of animal studies.¹⁹ These animal studies indicated that direct trauma to the sternum with minimal or no indication of injury to the heart at death and autopsy may be the mechanism of electrical disruption to the heart. In some cases, the trauma led to core pulmonal, right-side heart failure; but in other cases, the trauma led to various arrhythmias and AV blocks.^{19,23-25} When dealing with electrical disturbances of the heart, there have been numerous cases where there have been transient alterations of rhythm and conduction,^{14,26} including right bundle-branch block,²⁷ complete heart block,^{28,29} ST abnormalities, and ventricular fibrillation. These cases had been transient and over a period of days to a week regressed back to normal; however, in one case, a continual complete left-bundle branch block remained. However, the normal rhythm had been restored; and a pacemaker was not inserted at the emergency department.^{12,14,21,25}

There are a number of cases revealing long-term sequelae of blunt trauma to the chest and heart with delayed complications. Some of the long-term problems are chest wall deformity, dysemia, chronic neck pain, depression, fibril thorax headache, idiopathic chronic pain, impaired exercise intolerance, impaired pulmonary function, neuralgias, occupational disability, paresthesia, persistent chest wall pain, restrictive ventilatory defects, sternal pain, and temporal mandibular disorders.^{11,28-32} There are also cases of arrhythmias, after the initial injury, developing even as late as a month afterward. These include bradycardia, emboli, and atrial ventricular dissociations. In some cases, there was development of arrhythmias 48 to 72 hours after the initial injury that progressed to require medical intervention. There is a documented case in which ventricular tachycardias resulting from intermittent left anterior hemiblock developed into a left bundle-branch block. In this case, the tachycardias and other symptoms did not manifest themselves until 6 days after the injury.^{33,34}

The central point of this recitation is that heart injury may not manifest itself at the time of injury. Typically,

the first 48 hours are the most crucial; but even as far out as a month from the trauma, there have been cases where arrhythmias have developed and progressed into the need for some form of medical intervention. The caveat to everything is that in some rare cases (0.1%), patients initially thought to be noninjured can develop cardiac problems. Patients older than 45 years had a higher incidence of dysrhythmias as a result of chest and thoracic injury.³⁵

Assessment of the heart after trauma

Evaluation of the heart after blunt chest trauma or blunt cardiac trauma is very difficult and vaguely defined in the literature. The sole form of heart trauma that is well documented is the rupture of the cardiac muscle, the septum, or the chordae tendineae. These are easily picked up on scans and other studies that can reveal the direct trauma-related physical damage. However, when dealing with arrhythmias, vagal sympathetic reflexes,¹⁹ and other abnormal profusions and electrical factors of the heart, there is very little consensus on the way to test and evaluate a patient presenting to the hospital after chest trauma. Studies that depend on using ECGs alone show very little correlation with direct heart trauma. Creatine phosphokinase, the MB isoenzyme, also has been inconsistent in identifying heart injury. Echocardiograms are inconsistent because of false positives and self-limiting electrical abnormalities. In one study, a very low-grade creatine kinase–MB isoenzyme was used with 2-dimensional echocardiograph; and this seemed to have some correlation to the altered electrical impulses resulting from cardiac trauma.³⁶ Because there may be delayed presentation of heart symptoms resulting from heart trauma, this evaluation was also inconsistent.^{17,33,34} In addition, holding a patient with any type of chest trauma for 48 to 72 hours for observation and monitoring is cost ineffective.³⁷ Patient education may be the most effective approach for delayed symptom onset,^{4,15,36} and good documentation of injury to the chest wall will be necessary to associate late effects.³⁸

Limitations of this case study

The limitation of this case study is the lack of supporting data indicating the potential relationship of trauma or vasovagal reflex response to the complete AV heart block. One supporting factor was the early presentation of an arrhythmia, and a second is the fact that there was no evidence of cardiomyopathy

before the injury and all evaluations demonstrated normal perfusion and electrical conductivity even after the injury and onset of symptoms.

Conclusion

This is an unusual case in which an arrhythmia was an intermittent factor that appeared to be a vagal-vagal reflex response causing a sudden drop in heart rate. This case suggests that there may be progressive sequelae to the cardiac trauma without indications until later. The deployment of the air bag saved his life, but may have introduced stress factors on the heart that eventually developed into a complete heart block and a potential mitral valve prolapse.

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