Trauma-Induced Conduction Disturbances

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Background: Electrical disturbances following blunt cardiac injuries are rare but can be caused by electrical or structural damage to the heart. We present the case of a patient who had conduction abnormalities following blunt traumatic injury that were incidentally detected on telemetry.

Case Report: A 64-year-old female with no history of cardiac disease was brought to the emergency department after a motor vehicle collision that resulted in chest wall bruising. The patient was found to have L-spine fractures and was admitted for observation. During her hospitalization, the patient had multiple episodes of heart block. A temporary pacemaker was inserted because of the recurrent episodes, and a dual-chamber permanent pacemaker was placed on day 4 of her hospitalization.

Conclusion: Heart block as a consequence of blunt cardiac injury is rare; however, it needs to be recognized as early as possible. Permanent pacemaker placement is usually indicated for patients with prolonged or recurrent episodes.

Keywords: Arrhythmias, heart block, trauma

INTRODUCTION
Blunt chest trauma can result in a variety of cardiac injuries that may involve all the structures of the heart, including the pericardium, the myocardium, and the valves. Depending on the extent of the chest trauma, such injuries can cause varying amounts of electrical dysfunction. Electrical disturbances following blunt cardiac injuries are rare but can be caused by electrical or structural damage to the heart. We present a case of recurrent episodes of heart block that were incidentally found on cardiac monitoring.

CASE REPORT
A 64-year-old female with a medical history of hypertension, renal transplantation on immunosuppression therapy, and posterior communicating artery aneurysm status post clipping that was complicated by postoperative seizure disorder was brought by emergency medical services to the emergency department after she reversed her car into her neighbor’s house. The patient was wearing a seatbelt, but the vehicle airbag did not deploy during the accident. Upon examination, the patient was afebrile, her blood pressure was 115/70 mmHg, and her heart rate was 89 bpm. She had two small lacerations on her left forehead without active bleeding. She had moderate tenderness upon palpation of the anterior chest wall over the sternum and bruising from the seatbelt across the sternal and lower abdomen area. Cardiac examination revealed normal heart sounds without murmurs, gallops, or rubs. The patient’s medications were levetiracetam, mycophenolate mofetil, pantoprazole, phenytoin, tacrolimus, and hydralazine. Laboratory tests were normal except for a slight and transient increase in highsensitivity troponin. Electrocardiogram (ECG) on admission was unremarkable with normal sinus rhythm. Computed tomography of the head showed no evidence of hemorrhage. Chest x-ray showed no presence of pneumothorax, hemothorax, contusion, rib fracture, clavicular fracture, scapular fracture, or pneumomediastinum.

The patient was found to have a transverse process L-spine fracture (L1 and L2) and was admitted to the surgical intensive care unit for observation. No acute surgical intervention was recommended for the fractures, and she was transferred to the general floor.

On the third day of the patient’s hospitalization, two episodes of heart block that occurred during sleep were detected incidentally on cardiac telemetry. The first occurrence was a 5-second episode of ventricular standstill (Figure 1). The second episode was a high-grade atrioventricular (AV) block with fixed PR intervals (Figure 2). The patient denied feeling any palpitation or chest pain and was hemodynamically stable. Review of previous 12-lead ECGs did not reveal any rhythm abnormalities. Echocardiogram showed no valvular abnormalities, normal left ventricular systolic function with an estimated ejection fraction of 55%, and no wall motion abnormalities.
Although bradycardia and high-degree heart block could reasonably be explained by the vagal effect of significant trauma, recurrence of the patient’s heart block made this explanation less likely. To rule out coronary artery disease as a cause of the patient’s symptoms, coronary angiography was performed and showed no evidence of obstructive artery disease or coronary artery dissection.

Given the patient’s recurrent episodes of heart block—she had multiple similar episodes that were noticed on telemetry; some episodes were brief—a temporary transvenous pacemaker was placed, and the patient was transferred to the cardiac intensive care unit for close monitoring. Because of the intermittent pacing requirements, a dual-chamber permanent pacemaker (PPM) was placed on day 4 of the patient’s hospitalization. The procedure was uneventful. The patient was monitored closely for 4 days, remained asymptomatic, and was discharged to subacute rehabilitation.

DISCUSSION

The spectrum of blunt cardiac injury varies greatly; patient presentation can range from being asymptomatic with minor ECG changes to developing heart block and life-threatening abnormalities.

Despite being rare, a variety of conduction disturbances have been reported with traumatic blunt cardiac injury.\(^1\) High-grade AV block has also been reported in human studies and animal models following nonpenetrating injury and myocardial contusion.\(^2,3\) Most of these anomalies occur immediately after or within 72 hours of blunt cardiac injury.\(^1\)

While high-grade AV block after blunt cardiac injury is generally transient, and normal conduction returns after a period of a few days, in some patients—such as ours—the AV clock recurs or persists for more extended periods, requiring PPM insertion.\(^1\) In our case, the decision to proceed with PPM placement was related to the recurrence of the episodes.

The underlying etiology of traumatic AV block, whether infra-Hisian or at the AV node, remains elusive. Multiple mechanisms have been proposed to explain this phenomenon, including injuries of cardiac structures that are in close anatomic relation with AV conductive systems, such as tricuspid valve damage and interventricular septum through acceleration/deceleration phenomenon or a direct injury to the chest.\(^1,4\) Other explanations might be the disruption of the vagal sympathetic response or direct interference to the neuromuscular mechanisms of the heart, resulting in cardiac standstill and other conduction disturbances.\(^1,4\)

Echocardiograms can show abnormal septal motion in addition to right ventricle or tricuspid valve injuries; nevertheless, an unremarkable echocardiogram with relevant abnormalities is not uncommon.\(^1\)

PPM placement is usually indicated in patients with prolonged or recurrent high-grade AV block.\(^1\) Similar to its indication in cases of high-grade AV block after cardiac surgery,\(^2\) temporary pacing is usually preferred before proceeding with PPM implantation to assess the pacing dependence, thus assisting the decision process regarding PPM implantation.

![Telemetry strip shows normal sinus rhythm followed by ventricular standstill (asystole) where no QRS complexes are seen after P waves (arrows) for a few seconds.](image1)

![Telemetry strip demonstrates a high-grade atrioventricular block with fixed PR intervals.](image2)
CONCLUSION
Heart block as a consequence of blunt cardiac injury is rare; however, it needs to be recognized as early as possible. PPM placement is usually indicated for patients with prolonged or recurrent episodes.

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