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CASE REPORT

Complete heart block and asystole following blunt cardiac trauma

Mohamed Morsy, MD, Nephertiti Efeovbokhan, MD and Sunil K. Jha, MD*

Department of Cardiology, University of Tennessee Memphis, Memphis, TN, USA

Cardiac contusion is a well-recognized complication of blunt chest trauma. Various conduction system disorders have been reported in association with this condition, the most common being right bundle branch block. Complete heart block (CHB) is seen rarely. Most cases of CHB are transient. We present the case of an 80-year-old woman who developed CHB and asystole following blunt cardiac trauma. Malignant cardiac arrhythmias such as CHB can be associated with blunt cardiac trauma. In most cases, CHB is transient resolving in days to weeks. In rare cases, however, CHB leads to asystole. Close monitoring and prompt intervention is thus required.

Keywords: *complete heart block*; *post trauma*; *cardiac contusion*

*Correspondence to: Sunil K. Jha, 7945 Wolf River Blvd Ste 120, Germantown, TN 38138, USA, Email: sjha@uthsc.edu

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Cardiac contusion is a known complication of blunt chest trauma. The exact incidence is difficult to determine as different criteria exist for its diagnosis, but it is estimated to occur in between 8 and 76% of blunt chest trauma (1). Various conduction system disorders have been associated with cardiac contusion. We present the case of an 80-year-old woman who sustained complete heart block (CHB) and asystole following blunt chest trauma.

Case report

An 80-year-old woman presented to the emergency room with complaints of left-sided chest pain after being involved in a head on motor vehicle collision. She was a restrained driver in one of the vehicles. Review of systems was otherwise negative. On physical examination, she was alert in no distress. She was tender to palpation on her left lateral chest wall, with clear lung sounds. Cardiovascular examination revealed normal first and second heart sounds with no murmurs or rubs. Chest X-ray showed fractures of the left 6, 7 and 8th ribs. Twelve lead electrocardiogram showed ectopic atrial rhythm at a rate of 70 beats/min, with 2–3 mm ST elevation in Leads V1–V3 (Fig. 1). Pertinent findings on her basic metabolic profile were: potassium level 4 mmol/L, magnesium level 1.8 mg/dL,

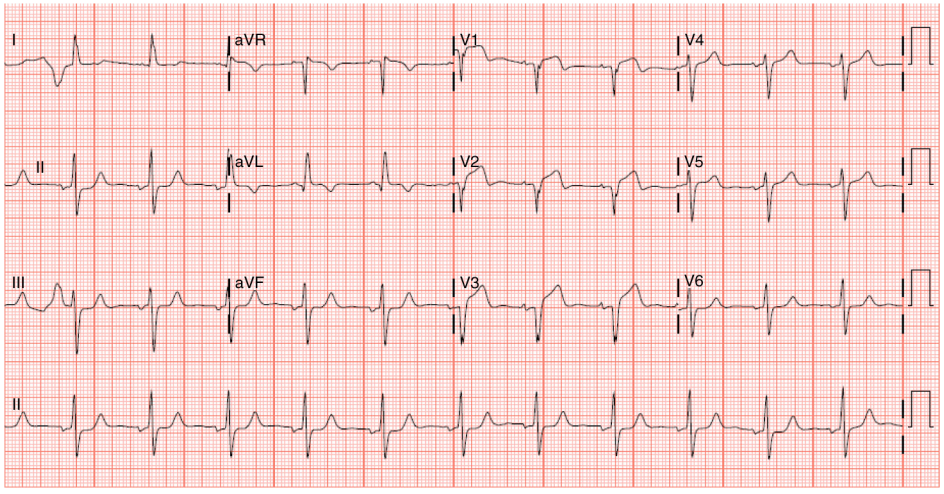

Fig. 1. EKG at presentation showing ectopic atrial rhythm with ST elevations in Leads V1–V3.

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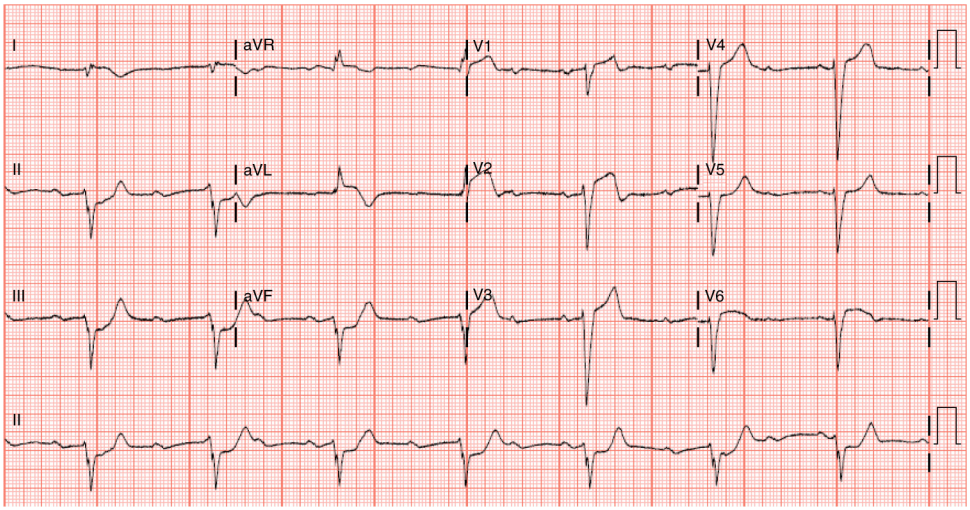

Fig. 2. EKG showing complete atrio-ventricular heart block with AV dissociation and underlying sinus tachycardia on day 2.

and creatinine of 0.9 mg/dL. Initial troponin-I level was 0.056 ng/mL, peak was >40 ng/mL (normal level 0.0–0.045 ng/mL). She was taken emergently to the cardiac catheterization laboratory. She was noted to have severe native three-vessel disease with patent left internal thoracic artery graft to the left anterior descending artery, patent venous grafts to the posterior descending and obtuse marginal arteries. Echocardiogram showed mid and basal anteroseptal wall akinesia with an estimated ejection fracture of 35–40%. On hospital day 2, while on the floor she was noted to be bradycardic with heart rates in the thirties. Telemetry review showed CHB, EKG showed complete atrio-ventricular heart block with AV dissociation and underlying sinus tachycardia (Fig. 2). She was started on dopamine infusion while plans for a temporary pacemaker were on the way. At the time of insertion of the temporary pacemaker, she went into asystole. A pulse was regained after she received 1 mg of atropine. She was subsequently intubated. On hospital day 3, electrophysiology was consulted for consideration of permanent pacemaker insertion as her CHB persisted. On hospital day 4, she underwent dual-chamber pacemaker insertion. On hospital day 6, 12 Lead EKG showed resolution of CHB with the development of sinus tachycardia (Fig. 3). Unfortunately, she was unable to be weaned off mechanical ventilation. A computed tomography of the head showed an acute left parietal stroke, which was thought to be secondary to transient ischemia she sustained when she went into asystole.

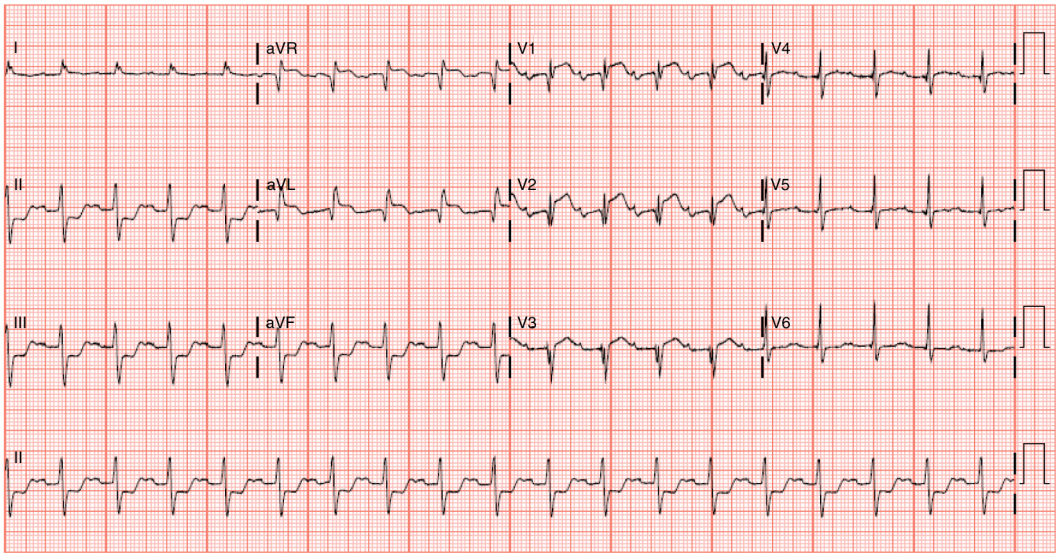

Fig. 3. EKG showing sinus tachycardia with 1:1 conduction on day 6.

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Despite aggressive measures, her neurologic function continued to deteriorate. In agreement with her family, she was terminally weaned off ventilator on day 16.

Discussion

The clinical presentation of cardiac contusion varies ranging from transient or persistent arrhythmias to mechanical complications such as valvular or free wall rupture leading to pericardial tamponade (2). Of the conduction disorders associated with cardiac contusion, right bundle branch block is most commonly encountered (2), whereas CHB has rarely been reported (3–5). The postulated pathologic mechanisms underlying conduction disorders in blunt cardiac trauma include myocardial injury, hemorrhage, or stunning of specialized conduction tissues; local release of metabolic substances with depressant effects on conduction tissues; and excitation of cholinergic reflexes (3–5). New mid and basal anteroseptal wall akinesia with significant troponin-I elevation in the absence of coronary artery stenosis was noted in our patient, making myocardial injury or hemorrhage of specialized conduction tissue a plausible mechanism for the development of CHB. As seen in other reported cases following blunt cardiac trauma, our patients CHB was transient resolving in about 1 week. Our patient's presentation was unique, however, unlike other reported cases, her CHB led to asystole with resulting brain injury. Although attempts were made to support her cardiac status, they proved abortive. We conclude that CHB following blunt cardiac trauma may lead to life-threatening conditions such as asystole. Close monitoring and prompt intervention is required to prevent untoward consequences.

Conflict of interest and funding

The authors have no conflict of interest to disclose.

References

1. El-Chami MF, Nicholson W, Helmy T. Blunt cardiac trauma. J Emerg Med 2008; 35: 127–33.
2. Plautz CU, Perron AD, Brady WJ. Electrocardiographic ST-segment elevation in the trauma patient: Acute myocardial infarction vs myocardial contusion. Am J Emerg Med 2005; 23: 510–16.
3. Benitez RM, Gold MR. Immediate and persistent complete heart block following a horse kick. Pacing Clin Electrophysiol 1999; 22: 816–18.
4. Lazaros GA, Ralli DG, Moundaki VS, Bonoris PE. Delayed development of complete heart block after a blunt chest trauma. Injury 2004; 35: 1300–2.
5. Brennan JA, Field JM, Liedtke AJ. Reversible heart block following non-penetrating chest trauma. J Trauma 1979; 19: 784–8.