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Atypical Myocardial Infarction Associated with Airbag Deployment: Insights from Electrocardiographic Changes and Intravascular Imaging

CASE REPORT

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INTRODUCTION

Myocardial infarction from blunt chest trauma has been demonstrated in many scenarios, including car crash. However, coronary injury resulting from direct collision with an inflated airbag has rarely been reported. Unawareness of the association could delay the diagnosis in the confounding clinical presentation of multiple traumas.[1,2] We hereby present a case of myocardial infarction associated with airbag deployment whose clinical presentation and intravascular imaging could provide hints to the pathogenesis in this condition.

CASE REPORT

A 56-year-old woman in the front passenger seat without a seat belt fastened encountered a frontal collision with a rapidly inflated airbag in a car crash. She complained of pleuritic-like chest pain accentuated with inspiration and was immediately referred to the local hospital. She had no specific risk factors for atherosclerosis. Her initial 12-lead electrocardiogram revealed 2:1 atrioventricular conduction block (AVB) and left bundle branch block with left axis deviation (Supplementary Figure 1). Forty minutes later, the conduction abnormality evolved into third-degree AVB (Supplementary Figure 2). Two hours after her first medical contact, the conduction disturbances started to regress, while mild ST-segment elevation appeared in the lead III, aVF, and V1 with reciprocal depression in the lead I, aVL, and V2-V6 (Supplementary Figure 3).

At the seventh hour, the patient was transferred to the emergency department of our center for further treatment. She had a normal heart rate and blood pressure on admission. Her lungs were clear, and heart sounds were normal without murmurs or pericardial friction on auscultation. The ST segment remained elevated in the lead III, aVF, and right precordial leads (V1 to V5R) on the 18-lead electrocardiograms for the next 3 hours (Supplementary Figures 4 and 5). She had elevated levels of cardiac specific troponin I (cTnI: 0.910 ng/mL, reference range: 0.010-0.023 ng/mL), D-Dimer (2110 ng/mL, reference range: 80-500 ng/mL), and normal N-terminal pro-B-type natriuretic peptide (NT-proBNP: 222 pg/mL, reference range: 300-450 pg/mL). At the 18th hour, the elevated ST segments resolved without recurrence of atrioventricular conduction disturbances (Supplementary Figure 6).

Her echocardiogram showed normal chamber size without obvious global or segmental ventricular wall motion abnormalities. Computed tomographic angiography was initially performed to delineate possible coronary lesions and exclude aortic root dissection or mediastinal hematoma. Her proximal right coronary artery (RCA) was severely occluded with apparently normal left coronary arteries (Figure 1A). She was prescribed dual anti-platelet therapy after exclusion of internal bleeding. The patient refused to receive invasive coronary angiography and was discharged after spontaneous remission of her symptoms.

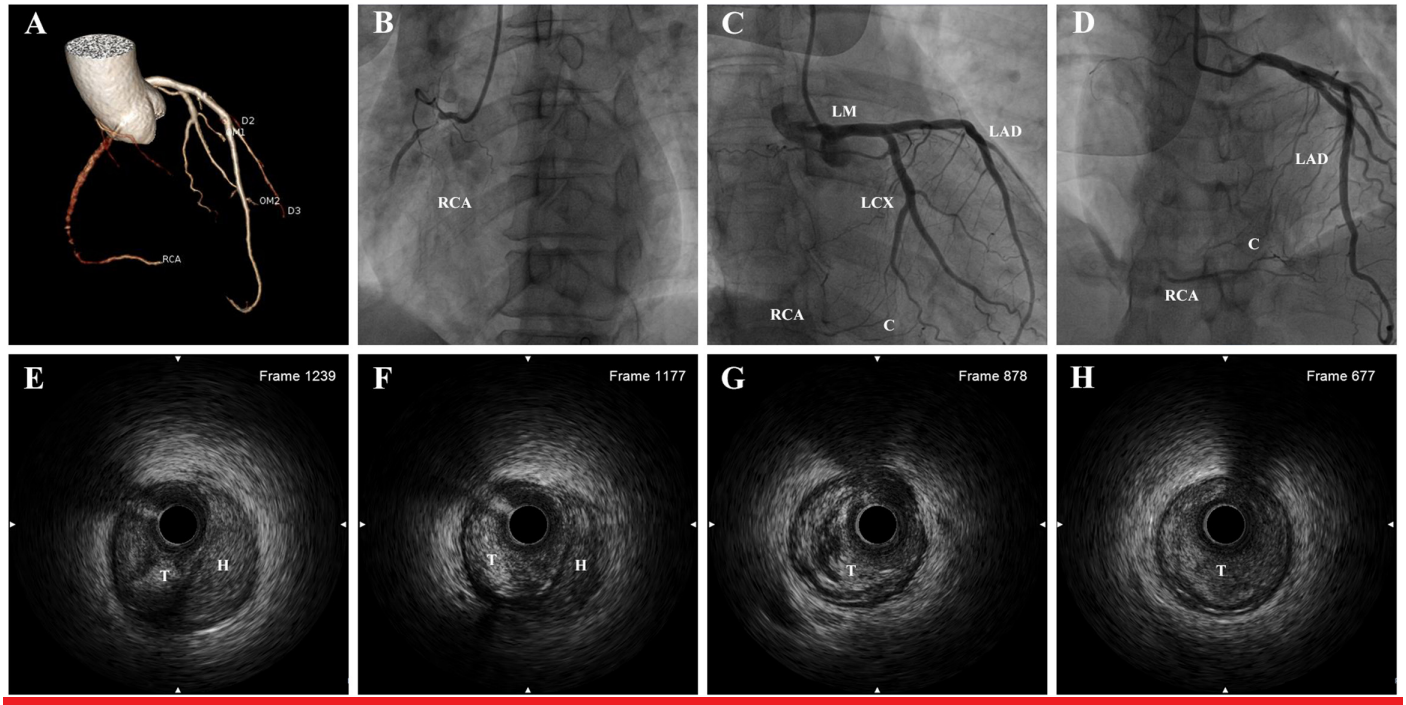

Figure 1. Computed tomographic angiography (A) shows severe occlusion in the proximal RCA with apparently normal left coronary arteries. Invasive coronary angiography demonstrates a severely occluded RCA (B), with coronary collaterals from the left coronary arteries (C, D). Intravascular ultrasound imaging of the RCA shows a large intramural hematoma in the proximal stenotic segment and luminal thrombosis (E, F) with partial recanalization in the distal stenotic segment (dark area, G, H). C, coronary collaterals; H, hematoma; LAD, left anterior descending coronary artery; LCX, left circumflex coronary artery; LM, left main coronary artery; RCA, right coronary artery; T, thrombi.

A week after presentation, the patient returned to our center for persistent hypotension. Invasive coronary angiography revealed subtotal occlusion of the RCA with thrombolysis in myocardial infarction (TIMI) flow grade 1 and coronary collaterals from the left coronary arteries (Figure 1B-D, Video 1-3). After pre-dilatation, an intravascular ultrasound (IVUS) catheter was introduced into the RCA, showing a large intramural hematoma in the proximal stenotic segment and luminal thrombosis with distal partial recanalization (Figure 1E-H, Video 4). The proximal RCA was treated successfully with a 3.0*29 mm drug-eluting stent with postprocedural TIMI flow grade 3 (Video 5). The patient was asymptomatic and in good condition during the 6-month follow-up.

DISCUSSION

Automatic airbag deployment can be life-saving during a car crash. However, this case indicates that blunt chest trauma from rapid airbag inflation can result in severe coronary injury and myocardial infarction. The affected individual may have different clinical and pathological presentations than the common atherothrombotic myocardial infarction.

According to angiographic imaging or autopsy, several underlying mechanisms have been proposed for myocardial infarction from blunt chest trauma in a car crash, including external compression of the coronary artery from an epicardial hematoma,[3,4] coronary dissection,[5] and thrombotic occlusion.[6] However, coronary angiography provides limited information on the pathogenesis in this case. IVUS imaging enables detailed visualization of the vessel wall with high spatial resolution and discloses a more confirmative underlying mechanism for this rare scenario than coronary angiography. An intimal tear or vasa vasorum rupture can occur in the vessel wall as a result of blunt chest trauma, and an intramural hematoma forms with accumulating blood in the media. In addition, intimal dissection can also induce local thrombosis in the lumen. The bulking intramural hematoma and thrombus lead to lumen narrowing and final coronary occlusion.

The unique clinical feature in this case is the dynamic electrocardiographic changes in the absence of typical ischemic symptoms. The hallmarks in electrocardiograms involve early evolution and regression of AVB, and the late occurrence of ST segment elevation in the inferior and right precordial leads with final resolution. Both the AVB and ST segment elevation patterns indicate the RCA as the infarct-related artery. In the literature, the most commonly affected artery from blunt chest trauma in a car crash is the left anterior descending artery,[1,2,7] followed by RCA,[5] and the left main coronary artery.[8] In this case, the anterior location of the proximal RCA renders it more vulnerable to direct frontal collision. Moreover, the electrocardiographic evolution reflects the gradual but progressive process of coronary injury, including bulking intramural hematoma, thrombotic occlusion, and late spontaneous recanalization with collateral formation. Therefore, ST segment elevation myocardial infarction indicative of coronary artery occlusion can occur a few hours or days after the indexed accident.[1,2,7]

Electrocardiogram can provide the first signs suggestive of coronary injury and should be recorded at the first medical contact and monitored serially for the next few days, irrespective of the symptoms.

Coronary hematoma often occurs as an iatrogenic complication during percutaneous coronary intervention and rarely secondary to spontaneous dissection or trauma. There is no consensus on the optimal management of traumatic coronary hematoma. Experience with spontaneous coronary artery dissection favors revascularization if high-risk features (ongoing/recurrent ischemia, hemodynamic instability, TIMI flow grade 0 to 1, or sustained ventricular arrhythmias) are present.[7,9] In view of TIMI flow grade 1 and recurrent hypotension in this case, interventional treatment with coronary stenting is indicated as the first-line therapy.

CONCLUSIONS

Myocardial infarction resulting from blunt chest trauma during rapid automatic airbag deployment is a rare condition, with intramural coronary hematoma and thrombotic occlusion as the underlying mechanism. Serial electrocardiographic monitoring is necessary to reveal latent coronary injury even in the absence of apparent ischemic symptoms. Moreover, this case reiterates the indispensability of wearing seat belt fastened for both drivers and passengers.

Informed Consent: Written informed consent was obtained from the patient and her family for the publication of this case report.

Declaration of Interests: The authors have no conflict of interest to declare.

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Video 1: Invasive coronary angiography shows a severely occluded right coronary artery.

Video 2: Invasive coronary angiography shows coronary collaterals from the apparently normal left coronary arteries.

Video 3: Invasive coronary angiography shows coronary collaterals from the apparently normal left coronary arteries.

Video 4: Intravascular ultrasound imaging of the right coronary artery demonstrates a large intramural hematoma in the proximal stenotic segment and luminal thrombosis with partial recanalization in the distal stenotic segment.

Video 5: Postprocedural right coronary angiography.

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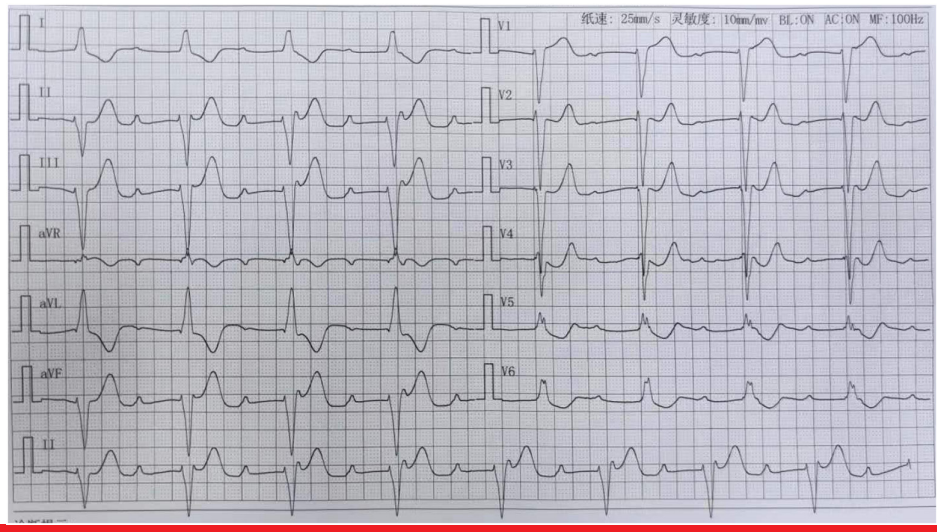
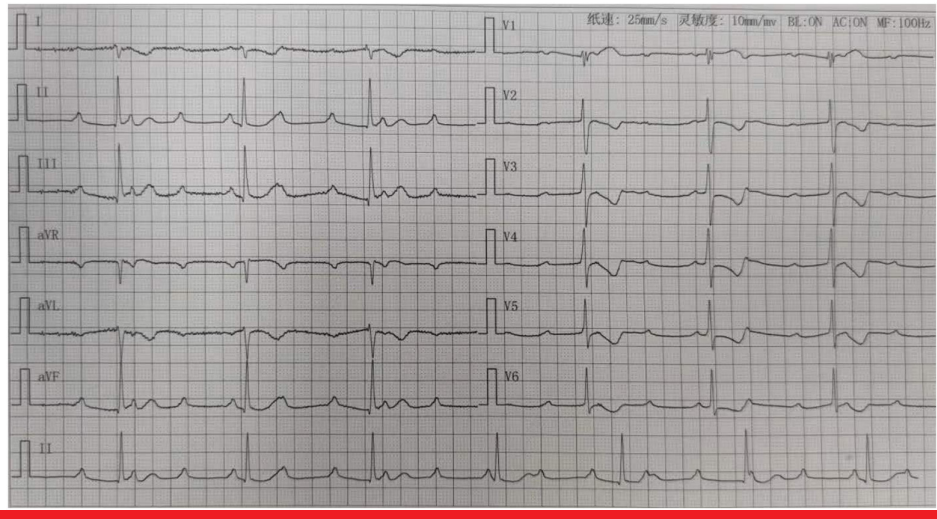
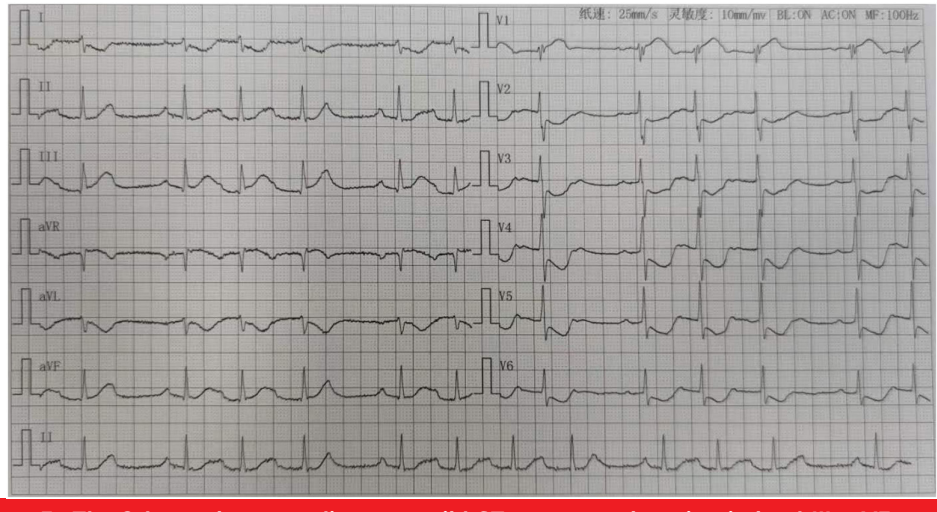


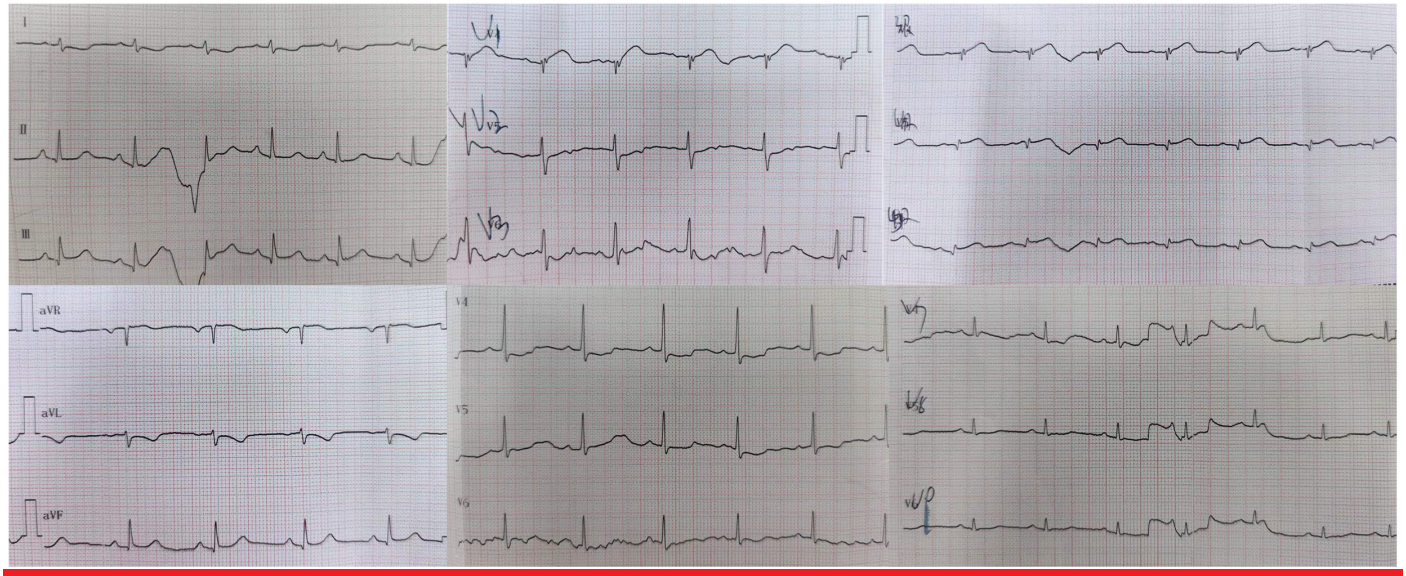
Supplementary Figure 1. Initial 12-lead electrocardiogram: 2:1 atrioventricular conduction block and left bundle branch block with left axis deviation.



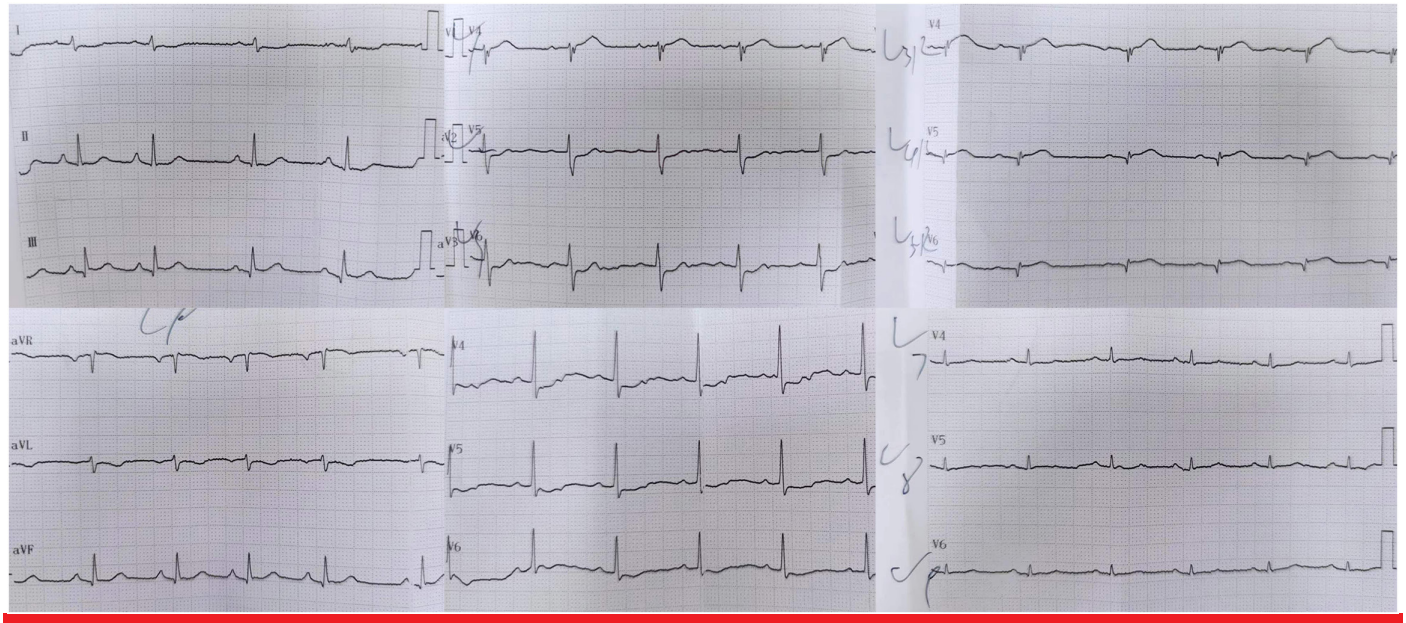
Supplementary Figure 2. The 40-minute electrocardiogram: third-degree atrioventricular conduction block.



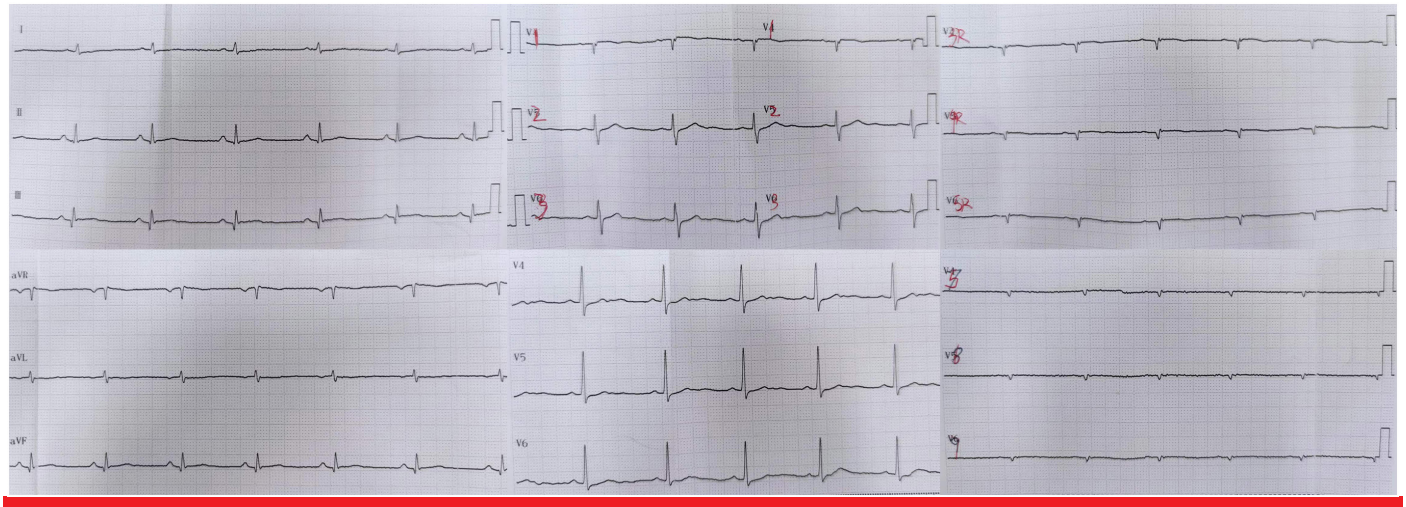
Supplementary Figure 3. The 2-hour electrocardiogram: mild ST segment elevation in lead III, aVF, and V1 with reciprocal depression in the leads I, aVL and V2-V6.



Supplementary Figure 4. The 7-hour electrocardiogram: ST segment elevation in lead III, aVF, and right precordial leads (V1-V5R).



Supplementary Figure 5. The 10-hour electrocardiogram: ST segment elevation in lead III, aVF, and right precordial leads (V1-V5R).



Supplementary Figure 6. The 18-hour electrocardiogram: ST segment resolution without atrioventricular conduction disturbances.