EMERGENCY CASEBOOK

Blunt traumatic dissection of the proximal left anterior descending artery

D Leong, M Brown

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Coronary artery dissection secondary to blunt chest trauma is a rare occurrence recently reported in the *Emergency Medicine Journal*. The case here is reported of a left anterior descending artery dissection after blunt chest trauma to illustrate a different, more precipitous presentation of this condition after severe trauma. This case report highlights how clinical history can alert to the presence of this complication and how electrocardiography is fundamental to risk stratification after trauma, and discusses management alternatives for this condition in the setting of multi-trauma.

We report the case of a 50-year-old, otherwise healthy man brought to The Royal Adelaide Hospital (Adelaide, South Australia, Australia) after a motor-cycle accident. On arrival his blood pressure was 120/70 mm Hg, heart rate 100 beats/min and respiratory rate 26/min. Glasgow Coma Score was 15/15. He had indigestion-like chest pain that was disproportionately severe to the level of apparent musculoskeletal injury. He also had severe facial injuries and a compound right femoral fracture. Initial electrocardiography featured hyperacute anterior T waves and inferior ST depression (fig 1A), which progressed to anterior segment ST elevation with anterior Q waves (fig 1B). Computed tomography of his abdomen, chest and head showed right upper lobe lung contusion with no evidence of haemorrhage or aortic dissection. Transthoracic echocardiography showed severe distal septal hypokinesis, mild to moderate distal anterior hypokinesis and apical akinesis. There was no pericardial effusion, and right ventricular and valvular functions appeared normal. Immediately after completion of these preliminary investigations, the patient developed shock. Adrenaline infusion was started. He was intubated and mechanically ventilated. Urgent coronary angiography showed proximal left anterior descending artery dissection with superimposed thrombosis, resulting in 80% vessel stenosis. Other coronary arteries were angiographically normal. After infusion of 70 U/kg unfractionated heparin, a bare metal stent was deployed in the proximal left anterior descending artery, and post-dilated to 15 atm. Abciximab was considered but not infused initially because of the risk of bleeding associated with his head and leg injuries. Subsequent stent thrombosis at the completion of the case required re-angioplasty and infusion of abciximab. Aspirin and clopidogrel were infused via nasogastric tube. An intra-aortic balloon pump was inserted. The time interval between the accident and first balloon inflation was 6 h. The patient made a good haemodynamic recovery: inotropic support was ceased and balloon pump removed within 48 h of percutaneous coronary intervention (PCI).

DISCUSSION

Coronary artery dissection has been reported after non-penetrating trauma,^{1 2} although it is a rare complication.^{3 4}

Prompt recognition is paramount because of incremental myocardial damage with delay to revascularisation. The potential mechanisms of coronary artery dissection include intimal tearing from deceleration injury, compression of the artery between the heart and the sternum, and coronary spasm. Coronary flow may be impaired by the dissection flap or superimposed thrombosis. The differential diagnosis of cardiogenic shock in the setting of severe trauma includes myocardial contusion, cardiac tamponade, ventricular rupture, coronary artery occlusion by proximal aortic dissection, and valvular disruption. Electrocardiography remains pivotal in screening for cardiac complications of blunt chest trauma, particularly when the patient is unable to communicate symptoms. The earliest feature of coronary occlusion is the development of hyperacute T waves in the affected territory, followed by ST segment elevation, and ultimately the development of Q waves. Echocardiography can identify cardiac tamponade and valvular disruption. It may show regional abnormalities in wall motion, although this cannot reliably distinguish cardiac contusion from ischaemia associated with coronary artery dissection. Computed tomography coronary angiography may potentially have a diagnostic role in suspected traumatic coronary dissection; however, high heart rates associated with trauma will result in suboptimal radiographic images with current technology. Computed tomography aortography can identify aortic dissection involving coronary artery ostia. Aortic dissection has implications for the risk and feasibility of cardiac catheterisation: if the dissection flap is entered with a guide wire or cardiac catheter, the dissection may be propagated. If coronary artery dissection is suspected, both diagnostic and therapeutic cardiac catheterisation may be adopted. PCI is the treatment modality most often undertaken in reported cases.¹⁻³ The use of adjunctive anticoagulants and antiplatelet agents must be individualised depending on the perceived risk of haemorrhage versus the likelihood of stent thrombosis. Dual antiplatelet treatment with aspirin and clopidogrel is thought critical in preventing acute stent thrombosis. Abciximab has been proved efficacious in reducing ischaemic events at 30 days in acute myocardial infarction as an adjunct to PCI. Whether abciximab's benefit can be generalised to trauma victims is unclear given the risk of bleeding from associated injuries. Thrombolysis is generally inadvisable because of the risk of bleeding from associated injuries.

Authors' affiliations

D Leong, M Brown, Cardiovascular Investigation Unit, Royal Adelaide Hospital, North Terrace, Adelaide, South Australia, Australia

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Correspondence to: D Leong, Cardiovascular Investigation Unit, Royal Adelaide Hospital, North Terrace, Adelaide, SA 5000, Australia; dleong@mail.rah.sa.gov.au

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Abbreviation: PCI, percutaneous coronary intervention

Figure 1 Initial electrocardiography.

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