Case report

Traumatic sub-coronary leaflet rupture of bicuspid aortic valve with a type-B aortic dissection

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1. Introduction

Traumatic aortic regurgitation is reported in high-energy non-penetrating thoracic trauma. Treatment is controversial with regards to the timing of surgery. A true dissection of the descending thoracic aorta resulting from blunt chest trauma is a relatively rare injury compared to aortic transection. To the best of our knowledge, a Stanford type-B aortic dissection with concomitant traumatic rupture of the coronary leaflet of a bicuspid aortic valve and complete sparing of the intervening aorta has not been previously reported in English literature.

2. Case report

A 37-year-old man presented with acute onset chest, back and abdominal pain following a car chase where the rear bumper of his car was repeatedly shunted from behind. On admission, he was conscious and oriented with a pulse rate of 95/min and blood pressure of 156/86 mm of Hg. On examination of the precordium, a diastolic murmur was heard. Chest radiogram demonstrated a widened mediastinum and trans-thoracic echocardiography (TTE) revealed severe aortic regurgitation with good bi-ventricular function but a Stanford type-A aortic dissection could not be ruled out. A contrast-enhanced CT scan demonstrated a dissection flap extending from the distal aortic arch to the common iliac vessels without any evidence of transection. The ascending aorta appeared mildly enlarged (3.8 cm), no dissection flap was convincingly seen, but there was a suspicion that motion artefact alone was responsible for an abnormal appearance of the ascending aorta (Fig. 1). An infusion of Labetalol was commenced and a decision was made to proceed with emergency surgery. The prime concern was a dissection involving the ascending aorta in view of severe aortic regurgitation and a descending aortic flap. A Femoro-right atrial cardiopulmonary bypass (CPB) was established and the patient was cooled to 32 °C following median sternotomy. On inspection, the ascending aorta and proximal arch were of normal calibre with no evidence of dissection. A transverse aortotomy was performed and the heart was arrested with direct ostial cold blood cardioplegia. The aortic valve was truly bicuspid and rupture of the sub-coronary leaflet was noticed but there was no intimal tear or flap in the ascending aorta. The valve was excised and replaced with a 27 mm bileaflet mechanical prosthesis (St. Jude Medical, MN, USA). The aorta was then transected at the level of the aortotomy and directly re-anastomosed in order to prevent a possible proximal extension of the Stanford type-B dissection. Post-operative TTE demonstrated the normal functioning of prosthetic aortic valve. The patient made an uneventful recovery and was discharged on anti-hypertensives and Coumadin with a plan to monitor the descending aorta with regular CT scanning.

3. Discussion

Contusion is the most common cardiac consequence of blunt chest trauma. Traumatic valvular damage is uncommon, but when it occurs, affects the aortic and mitral valves most frequently. Traumatic aortic incompetence may be the result of direct injury to the valve itself or a consequence of damage to the ascending aorta. The mechanism of direct valve damage typically involves the application of a compressive force during early diastole. This causes a rise in intra-aortic pressure at a point where the valve is closed with the maximal pressure gradient across the valve. This can lead to a rupture or tear of, most commonly, a single valve cusp. The non-coronary cusp is most often affected, as blood flow behind the coronary cusps lowers the pressure behind the valve leaflets which is relatively protective. Bicuspid aortic valves may be more susceptible to traumatic injury because of their inability to withstand abnormal haemodynamic stress.

Our patient was asymptomatic prior to this event and thus this case describes an acute Stanford type-B dissection associated with acute aortic regurgitation. The diastolic blood pressure remains normal in the acute phase unless the left ventricle decompensates. The trauma from repeated impact to a vehicle from behind is different from the rapid deceleration that occurs after a head-on collision with subsequent aortic injury, previously described in the literature. Isolated injury to the aortic valve and descending aorta without any other injuries is suggestive of a lower energy impact. There is no doubt that increased sympathetic drive and hyperten-
sion may have been a predisposing factor in this case. Combined with repeated collisions, this could have produced an aortic valve rupture and aortic dissection.

In conclusion, cardiac and great vessel injuries are possible following low-energy blunt chest trauma. The attending physicians in the emergency room should be aware of this and should always rule it out by performing a thorough clinical examination. Echocardiography and CT scanning should be promptly available to support the doctors with even minimal suspicion. The presence of a type-B dissection, slight ascending aortic dilatation, aortic incompetence, associated hypertension and the inability to differentiate motion artefact with a dissection flap in the ascending aorta in a post-trauma setting made us undertake emergency surgery in this patient.

References


Fig. 1. Axial CT image demonstrating a motion artefact in ascending aorta mimicking a type-A dissection and a true intimal flap in the descending aorta.