

EDITORIAL COMMENT

Spontaneous or Traumatic?

Differences, Commonalities, and Lessons Learned About Nonatherosclerotic Coronary Artery Dissections*



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Traumatic coronary artery dissections are uncommon and life threatening. Unlike spontaneous coronary artery dissections (SCAD) or dissections related to percutaneous coronary intervention, they are typically associated with distinct episodes of blunt trauma to the chest wall. Myocardial infarction (MI) related to nonpenetrating chest trauma has been described since at least the 1940s, initially with findings on postmortem examination, and later with coronary angiography. Coronary angiography, if performed, was often delayed sometimes days to weeks after the event, with variable angiographic findings, from intracoronary thrombus to “normal” coronaries (1). The hypothesized causes of traumatic MI without obstruction ranged from in situ thrombus that subsequently lysed, vasospasm, extrinsic vessel compression, and dissection, but there was insufficient evidence to implicate any of these mechanisms because of the delay in coronary imaging (2). By the 1980s, case reports of MI associated with blunt chest trauma accompanied by coronary angiographic evidence of dissection were increasingly reported, including cases requiring revascularization for ongoing symptoms or ischemia (3-6). Complete healing of the vessel on subsequent coronary imaging, not unlike that seen in patients with SCAD, has been described (3,7).

The most common causes of traumatic coronary artery dissection include motor vehicle accidents,

caused either by deceleration and traction or direct impact with the steering wheel or other fixed objects (5); during sports (8), related to impact with a ball or another player’s body part; and in other situations in which there is a severe crush or abrupt impact to the chest, such as falls from a height, farming and other workplace accidents; and, rarely, by attacks or altercations with other people or large animals (9).

The case presented in this issue of *JACC: Case Reports* by Kurklu and Tan (10) identifies yet another potential mechanism of coronary artery injury: a blast injury from a detonated bomb accompanied by penetrating shrapnel injury that reportedly did not directly affect the heart. The authors hypothesize that the blast shockwave resulted in disruption of coronary intima-medial integrity. This has been previously suspected but unconfirmed in other bomb blast-associated cardiac injury (11) and MI (12), and this case may be the first in which dissection was confirmed angiographically.

The case also demonstrates some of the diagnostic challenges associated with traumatic coronary dissections, especially in the setting of substantial chest trauma or multiple injuries. The patient may be unable to report symptoms of myocardial ischemia because of mental status changes or sedation, the patient may have generalized chest pain that could signify a broad range of differential diagnoses or affected structures, and the index of suspicion is often low among the initial treating healthcare team so that testing that might have diagnosed myocardial injury or infarction may not be performed or available. Among patients presenting with recent chest trauma and describing chest pain or dyspnea or who have evidence of significant thoracic trauma (e.g., multiple rib fractures), there should be a high degree of suspicion for potential cardiac injury (Table 1). Appropriate diagnostic testing includes 12-lead

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electrocardiogram (ECG), chest x-ray, determination of troponin levels, and, in most cases, a period of continuous ECG monitoring. There should be consideration of early echocardiography and/or chest computed tomographic imaging to exclude injury to the heart or great vessels. Patients with angina-like chest pain, persistent or progressive dyspnea, arrhythmias, and/or elevated troponin levels should be admitted, and coronary artery imaging, either CT or invasive angiography should be performed.

Many challenges to optimal care are highlighted in the current case (10), where not only were what are considered “simple” diagnostic tests, such as imaging and troponin assays, unavailable, but there were barriers to care as a result of nearby armed conflict. The case demonstrates the importance of assessing persistent or new symptoms consistent with ischemia. Although the patient described having chest pain and dyspnea at the time of presentation and had persistent typical exertional angina, some patients with traumatic coronary artery dissections have been reported to experience angina-like symptoms several days to weeks after the injury in the absence of evidence of myocardial injury or dysfunction at acute presentation (6). This observation may represent extension of an initial traumatic dissection, a phenomenon that occurs in $\leq 15\%$ of patients with SCAD (13). The treatment of patients with traumatic coronary dissection should be individualized. Thrombolysis is relatively contraindicated because of associated injuries, and conservative management has been reported to have good outcomes in patients who are in stable condition (5). Stenting and coronary artery bypass grafting have also been reported to result in good outcomes in individual case reports (5,6,10).

The novel mechanism and unique presentation of this case should not overshadow the far more common occurrence of SCAD in young and middle-aged women. Estimated to be the number 1 cause of MI in women under the age of 50 years, over the past decade, SCAD has increased in its recognition and understanding of the pathophysiology and optimal management (7,14-16). There are correlates and contrasts between traumatic coronary artery dissection and SCAD. By definition, SCAD presents without prior physical trauma or coronary manipulation/injury. Well over 90% of patients presenting with SCAD are women, whereas most cases of traumatic dissection

TABLE 1 Cardiac Injuries Associated With Nonpenetrating Chest Trauma

Myocardium: contusion, rupture, laceration
Great arteries: laceration, dissection, disruption, thrombosis, pseudoaneurysm
Coronary arteries: dissection, disruption, external compression
Valves: rupture, disruption
Pericardial effusion, tamponade
Arrhythmia

occur in men. The presenting symptoms of SCAD are those of MI, including sudden cardiac death. The diagnosis of SCAD is confirmed at coronary angiography or, in some cases, coronary CT angiography (17). Patients with SCAD may require intracoronary imaging to differentiate SCAD from alternative causes of coronary obstruction such as atherosclerosis or in situ thrombus (7,15,16). Establishing an accurate initial diagnosis is important in SCAD. SCAD patients in stable condition should not routinely undergo percutaneous interventions because these procedures are more likely to be complicated and less often successful than those performed for atherothrombotic obstruction, and healing of the affected vessel is common, occurring in 80% to 90% of conservatively-managed vessels (18).

Among patients with either traumatic coronary artery dissection or SCAD, an accurate diagnosis is important to provide optimal management, improve outcomes, and avoid iatrogenic harm. In both conditions, a heightened index of suspicion of coronary artery injury, specifically of dissection, should be informed by the patient’s history, presenting symptoms, and demographics. Although patients with traumatic coronary dissection are rare, their treatment may ultimately benefit from the lessons learned from the short-term and longer-term management of SCAD.

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REFERENCES

1. Christensen MD, Nielsen PE, Sleight P. Prior blunt chest trauma may be a cause of single vessel coronary disease; hypothesis and review. *Int J Cardiol.* 2006;108:1-5.
2. Liedtke AJ, DeMuth WE Jr. Nonpenetrating cardiac injuries: a collective review. *Am Heart J.* 1973;86:687-697.
3. Grady AE, Cowley MJ, Vetrovec GW. Traumatic dissecting coronary arterial aneurysm with subsequent complete healing. *Am J Cardiol.* 1985;55:1424-1425.
4. Kohli S, Saperia GM, Waksmonski CA, Pezzella S, Singh JB. Coronary artery dissection secondary to blunt chest trauma. *Cathet Cardiovasc Diagn.* 1988;15:179-183.
5. Lobay KW, MacGougan CK. Traumatic coronary artery dissection: a case report and literature review. *J Emerg Med.* 2012;43:e239-e243.
6. Colombo F, Zuffi A, Lupi A. Left main dissection complicating blunt chest trauma: case report and review of literature. *Cardiovasc Revasc Med.* 2014;15:354-356.
7. Hayes SN, Tweet MS, Adlam D, et al. Spontaneous coronary artery dissection: JACC state-of-the-art review. *J Am Coll Cardiol.* 2020;76:961-984.
8. Van Mieghem NM, van Weenen S, Nollen G, Ligthart J, Regar E, van Geuns RJ. Traumatic coronary artery dissection: potential cause of sudden death in soccer. *Circulation.* 2013;127:e280-e282.
9. Kawahito K, Hasegawa T, Misawa Y, Fuse K. Right coronary artery dissection and acute infarction due to blunt trauma: report of a case. *Surg Today.* 1998;28:971-973.
10. Kurklu HA, Tan TS. Blast injury: a very rare cause of left coronary artery dissection. *J Am Coll Cardiol Case Rep.* 2021;3:1898-1902.
11. Parmley LF, Manion WC, Mattingly TW. Nonpenetrating traumatic injury of the heart. *Circulation.* 1958;18:371-396.
12. Keren A, Stessman J, Tzivoni D. Acute myocardial infarction caused by blast injury of the chest. *Br Heart J.* 1981;46:455-457.
13. Waterbury TM, Tweet MS, Hayes SN, et al. Early natural history of spontaneous coronary artery dissection. *Circ Cardiovasc Interv.* 2018;11:e006772.
14. Tweet MS, Hayes SN, Pitta SR, et al. Clinical features, management, and prognosis of spontaneous coronary artery dissection. *Circulation.* 2012;126:579-588.
15. Hayes SN, Kim ESH, Saw J, et al. Spontaneous coronary artery dissection: current state of the science: a scientific statement from the American Heart Association. *Circulation.* 2018;137:e523-e557.
16. Adlam D, Tweet MS, Gulati R, et al. Spontaneous coronary artery dissection: pitfalls of angiographic diagnosis and an approach to ambiguous cases. *J Am Coll Cardiol Interv.* 2021;14:1743-1756.
17. Tweet MS, Akhtar NJ, Hayes SN, Best PJ, Gulati R, Araoz PA. Spontaneous coronary artery dissection: acute findings on coronary computed tomography angiography. *Eur Heart J Acute Cardiovasc Care.* 2019;8:467-475.
18. Hassan S, Prakash R, Starovoytov A, Saw J. Natural history of spontaneous coronary artery dissection with spontaneous angiographic healing. *J Am Coll Cardiol Interv.* 2019;12:518-527.

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