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Case Report

Spontaneous coronary vasospasm due to polytrauma

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ABSTRACT

Coronary vasospasm is characterized by focal or diffuse spasm of an epicardial coronary artery. Definitive diagnosis is usually made with coronary angiography, when resolution of stenosis is observed after administration of intracoronary vasodilators. Coronary vasospasm is rarely a consequence of a blunt force injury to the chest. Among trauma induced cardiac complications, coronary vasospasm has been the least common with only one other reported case of coronary vasospasm induced by trauma. We report a rare case of severe spontaneous coronary vasospasm in a patient with polytrauma successfully treated with intracoronary, intravenous and oral vasodilator therapy. The mechanism is thought to be due to compensatory catecholamine response to trauma, and coronary vasospasm should be strongly suspected in trauma patients with unexplained hypotension, new conduction abnormalities or evidence of ischemia on the ECG.

Introduction (background)

Coronary vasospasm is characterized by focal or diffuse spasm of an epicardial coronary artery, often resulting in transient transmural myocardial ischemia depicted as ST segment elevation on electrocardiogram [5,8]. In the United States, the frequency of vasospastic angina is among the lowest in the world, with only 4% of patients who undergo coronary angiography actually showing evidence of focal coronary vasospasm [4]. Episodes of vasospastic angina promptly respond to short acting nitrates and are thought to stem from vascular smooth muscle hyper-reactivity [8]. Focal spasms are attributable to sites of atherosclerotic plaques but can also occur in normal coronary vessels as well. We report a rare case of severe spontaneous coronary vasospasm in a patient with polytrauma.

Case presentation

A previously healthy 49-year-old man was admitted as level 1 trauma activation for a rollover motor vehicle collision. The patient sustained multiple injuries — C7 transverse processes fractures, left rib fractures, left pneumothorax, and left subclavian artery disruption. The patient was taken emergently to the operating room for repair via placement of interpositional graft and resection of damaged vascular segment. He was emergently sedated and intubated thus history was unobtainable. Initial vital signs were heart rate 110, blood pressure 70/50 mm Hg, and 100% oxygen saturation on the ventilator. Cardiac examination was notable only for sinus tachycardia and weak pulses bilaterally.

During surgery, the patient had recurring 2:1 atrioventricular (AV) block with labile blood pressures requiring aggressive volume resuscitation and norepinephrine drip. As part of the initial evaluation, a 12-lead electrocardiogram demonstrated ST elevations in

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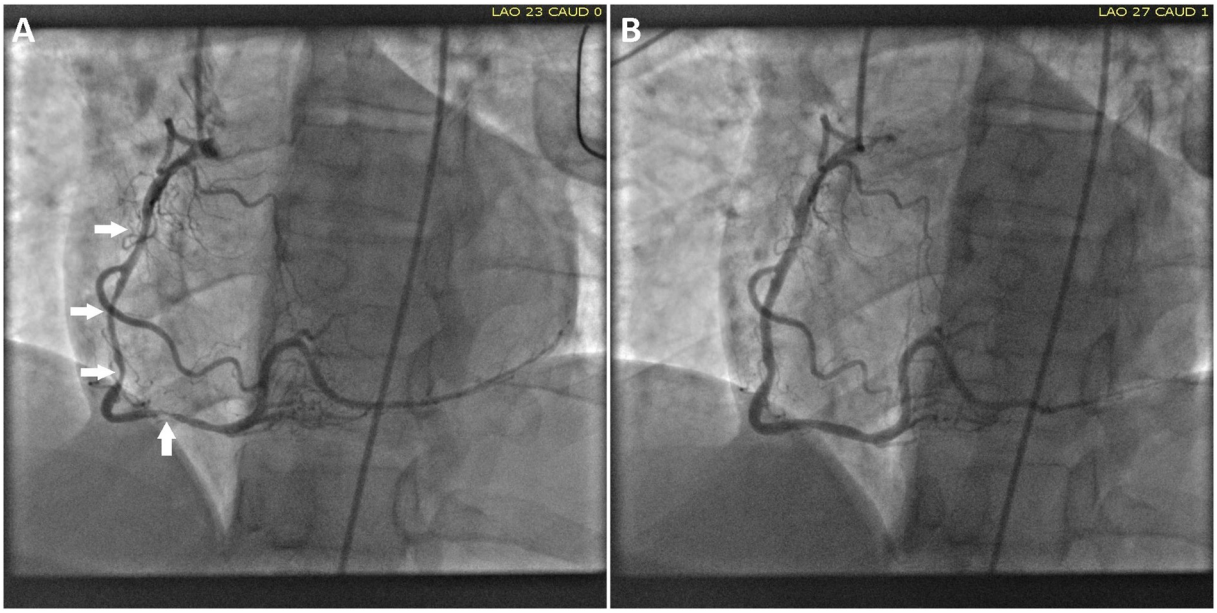

Fig. 1. Left anterior oblique (LAO) view — right coronary artery. A White arrows indicate areas of vasospasm. B Left anterior oblique (LAO) view — right coronary artery post intra-coronary nitroglycerin.

the inferior leads with reciprocal changes (Fig. 2). As surgery concluded, the patient was taken emergently to cardiac catheterization laboratory.

Coronary angiography was notable for severe spontaneous spasm in mid to distal right coronary artery (RCA).The severe vasospasm within the RCA was resolved with intracoronary nitroglycerin (Fig. 1). Minimal coronary atherosclerosis and hyperdynamic left ventricular systolic function with ejection fraction > 75% was found. The patient was started on low-dose nitroglycerin drip to prevent further vasospasm (Fig. 2). Attempts to wean the patient off nitroglycerin were unsuccessful within the first 24 h due to recurrence of inferior ST elevations. He was weaned of the nitroglycerin drip over several days with initiation of oral calcium channel blocker.

The patient required intense surgical and physical rehabilitation and was discharged from the hospital to skilled nursing facility for acute rehabilitation without any permanent cardiac maladies. His discharge regimen included amlodipine 10 mg daily and as needed sublingual nitroglycerin.

Discussion

This case demonstrates coronary vasospasm as a rare sequela of a blunt force injury to the chest. Other documented cardiac complications of trauma include arrhythmias, coronary dissection, valvular rupture and ventricular rupture. Among these complications, coronary vasospasm has been the least common with only one other reported case of polytrauma induced coronary vasospasm [1]. Similar to the case entitled *Asymptomatic Coronary Spasm due to Polytraumatism* [1]; our patient had transient ST elevations secondary to coronary spasm with resolution by nitroglycerin and coronary angiography with minimum coronary arthrosclerosis. In contrast, our patient required prolonged use of intravenous nitroglycerin drip combined with oral calcium channel

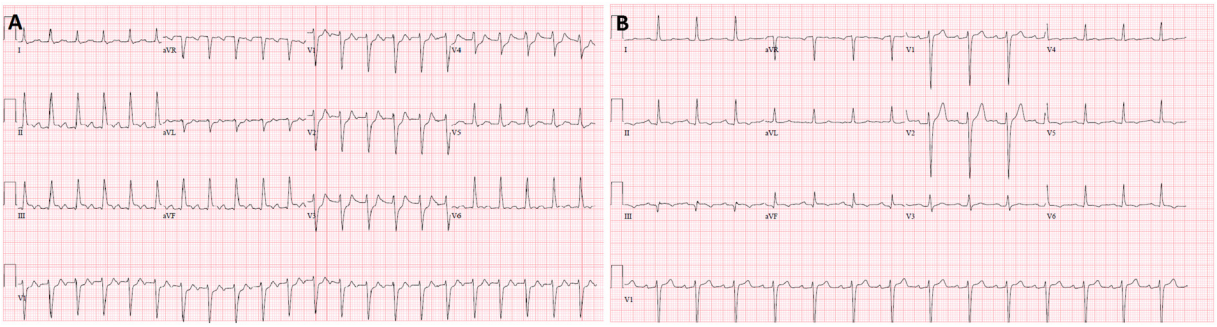

Fig. 2. A. ECG showing ST elevation consistent with acute inferoposterior injury. B. ECG demonstrating improvement in ischemic ST abnormalities after administration of coronary vasodilators.

blockers.

The pathophysiology of a coronary vasospasm centers on an exaggerated constrictor response from a systemic stimulus [6] leading to a focal coronary hyper-reactivity, often fostered by an imbalance between parasymphathetic and sympathetic tone. Many studies have shown acetylcholine, serotonin, histamine, norepinephrine, and dopamine elements can spur on vasospasm [9,10]. Vasospasms may occur in angiographically normal coronary vessels, but is more common at sites of atherosclerotic plaque with variable severity [4]. In this case, we hypothesize the catecholamine stimuli resultant from trauma played a central role which led to the patient's vasospasm. In addition, myocardial oxygen demand due to large volume blood loss and the use of norepinephrine for hemorrhagic shock played a concomitant role [6].

Other types of vascular spasms have been associated with trauma as well, most specifically traumatic brain injuries with or without subarachnoid hemorrhage (SAH). The mechanism for TBI related vasospasm is similar to coronary spasm in that an imbalance between vasoconstrictor and vasodilator substances is implicated. However, other mechanisms proposed include the presence of blood breakdown products in extravascular space and mechanical alterations of the cerebral vasculature via stretch or direct impact from injury, both resulting in release of vasoactive substances ergo cerebral vasospasm [2,3,7].

Coronary vasospasm can lead to high-grade obstruction which can evolve into a myocardial infarction and/or arrhythmias with either atrioventricular or ventricular conduction abnormalities [4]. In this case, the patient developed 2:1 atrioventricular (AV) block. The pathophysiology is quite complex which can lead to a wide spectrum of clinical findings which may make the initial diagnosis difficult. Additional questions remain to be answered as more cases are documented to help direct infarct-free survival in patients such as utility of long term calcium channel blockers, recurrence of future coronary artery spasm, and additional precautions when the obvious provocation is polytrauma.

Conclusions

To conclude, spontaneous coronary vasospasm can arise in trauma patients due to compensatory catecholamine response and should be strongly suspected in those with unexplained hypotension, new conduction abnormalities or evidence of ischemia on the ECG. Treatment should focus on hemodynamic stabilization, prompt initiation of vasodilator therapy such as nitroglycerin drip, and slow wean with initiation of oral calcium channel blockers.

Conflict of interest statement

All authors have no conflicts of interest.

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