

Coronary vasospasm in a patient with respiratory failure: A case report and a brief review

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ABSTRACT

Coronary vasospasm is an episodic, augmented, contractile response of coronary smooth muscles to variety of stimuli in the setting of established endothelial dysfunction. Various physiological including cold, stress and pathological factors like smoking, and ethanol have been well known to precipitate vasospasm. Despite these omnipresent factors, coronary vasospasm has become infrequent, in particular due to judicious use of medication including calcium channel blockers, statins and aspirin. We present a case of severe coronary vasospasm resulting in haemodynamic instability, in a patient with hypoxic respiratory failure. Recurrent symptomatic episodes required coronary angiography for the diagnosis and patient was successfully treated with calcium channel blockers.

KEYWORDS

Myocardial infarction; respiratory failure; coronary vasospasm; Diltiazem

Introduction

Myocardial ischemia from coronary artery vasospasm can lead to variety of presentation including stable angina, unstable angina, myocardial infarction and sudden death ¹. Although, pathognomic clinical scenario includes symptom of chest pain, transient ST-segment elevation on the electrocardiogram (ECG), and vasospasm on a coronary angiography, atypical presentations have also been reported ². Various known physiological factors including stress, cold, hyperventilation and pharmacological agents including cocaine, ethanol, 5-Fluouracil, and triptans can precipitate a vasospastic attack. ³⁻⁷. We report a case of ST-segment elevation due to right coronary artery vasospasm, in patient with hypoxic respiratory failure, and successful treatment with calcium channel blockers.

Case description

A 56 year old man was admitted for the repair of a large ventral incisional hernia. The patient had a prior history of morbid obesity, chronic obstructive pulmonary disease (COPD), hypertension and cigarette smoking. The postoperative course was complicated by bilateral pneumonia leading to respiratory failure requiring mechanical ventilation. An electrocardiogram at the time of intubation was essentially normal. Aside from bilateral rhonchi and crackles on lung auscultation, the rest of the physical examination findings were unremarkable. Arterial blood gases at the time of intubation demonstrated PH 7.33, PO₂ 58 mmHg, PCO₂ 65 mmHg, HCO₃⁻ 20 mmol/L, suggestive of hypoxia and concomitant respiratory acidosis. Baseline laboratory studies including cardiac enzymes were within normal limits. The patient was treated with intravenous

vancomycin for methicillin-resistant staphylococcus pneumonia. On postoperative day 4, the patient had recurrent episodes of transient ST-elevation on a bedside monitor (Fig.1).



Figure 1

These episodes lasted for 3-5 minutes and were associated with significant bradycardia and hypotension. In view of recurrent episodes, haemodynamic instability, and underlying risk factors of coronary artery disease, cardiac catheterization was performed. Coronary angiography revealed a 90% stenosis with haziness of the mid-right coronary artery without any other significant epicardial disease. An intravascular ultrasound (IVUS) was performed and was followed by administration of 100 mcg of intracoronary nitroglycerin; the lesion was reduced to almost 20%. (Fig.2). The diagnosis of Prinzmetal's angina was made, based on clinical course and angiographic results and prompt therapy with diltiazem (120 mg per day) was initiated.

The patient had no further recurrences of similar episodes during the hospitalization and on follow up at 3 months.

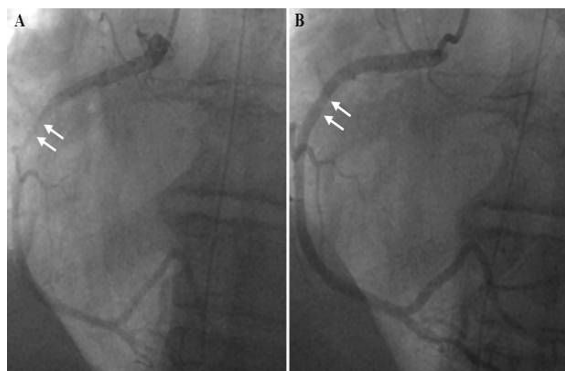


Figure 2

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Discussion

The prevalence of vasospasm has been reported to be higher in Japanese and Korean population as compared to the western population. A recent multi-institute survey in Japan documented spasm in 921 (40.9%) of the 2251 consecutive patients who underwent angiography for angina pectoris⁸. In contrast to the traditional risk factors for atherosclerotic coronary artery disease, the incidence of smoking, age and dyslipidaemia has been reported higher in patients with coronary vasospasm⁹.

Endothelial dysfunction is now considered to be the major inciting factor in the pathogenesis of the vasospasm¹⁰. Vasospastic angina (VA) with normal coronary arteries on the angiography, impaired endothelial-dependent and endothelial-independent vasodilatation has been frequently observed in these patients. Vascular tone is normally regulated by production of vasodilator factors like nitrous oxide (NO), prostacyclin and vasoconstricting agents like endothelin-1. In the presence of dysfunctional endothelium the agents that normally cause vasodilatation lead to paradoxical vasoconstriction, due to direct muscle stimulation, like acetylcholine.

Stress, whether physical or mental stress has been shown to induce coronary vasospasm and myocardial ischemia. In a study by Kim et al, coronary spastic angina was diagnosed in 292 patients out of 672 coronary spasm provocation tests. Among 292 patients, 21 (7.2%) had myocardial infarction and 14 out of these 21 had experienced severe emotional stress before the event¹¹. Recently, animal studies have also shown that high circulatory level of stress hormones (cortisol) exaggerate coronary vasoconstriction through Rho-Kinase activation¹². Hypoxia has been seen in animal models to predispose to

vasospasm through superoxide formation, which leads to loss of vasodilator function of NO.⁽¹³⁾

The ECG changes that occur during attack include ST-segment elevation, and or peaking of T wave from total or subtotal coronary occlusion¹. In some cases spasm can involve more than one artery leading to ST-segment elevation in multiple leads, which may predispose to ventricular tachycardia or fibrillation¹⁴. Coronary spasm is diagnosed by angiography, and spasm can occur at the site of atherosclerotic plaque or in normal segment of the coronary artery. In patients with equivocal diagnosis, provocative tests including administration of acetylcholine, hyperventilation to induce spasm may be required for the diagnosis.

Current first line therapy involves use of calcium channel blockers (CCB) alone or in combination with long acting nitrates. In a study comparing the effect of long acting nitrates (Isosorbide dinitrate 40mg/day) versus calcium channel blockers (amlodipine 5mg/day or long acting nifedipine 20mg/day) on coronary endothelium and vasoconstriction between patients with normal or minimally diseased coronary artery, treatment with long acting nitrates was associated with less favourable effects on coronary endothelial functions¹⁵. Sudden withdrawal of CCB in patients with known vasospasm can lead to rebound of symptoms and may prove dangerous. In patients with refractory symptoms alpha-blockers, nicorandil have been used. Although beta blockers are believed to enhance vasospasm, Betaxalol, a selective beta-1 blocker, has been found to be effective in the treatment of variant angina due to its vasorelaxing effects¹⁶. In addition, elimination of or control of all other risk factors or precipitants is very important for successful treatment. In drug refractory cases the percutaneous coronary intervention or coronary artery bypass graft has been performed for the ischemia relief¹⁷.

Our patient had multiple precipitating factors for vasospasm. Endothelial dysfunction from severe physical illness and sepsis could have precipitated the VA. Furthermore, hypoxia from respiratory failure could have also been an inciting agent and cannot be ruled out. It is worth mentioning that intensive care unit patients frequently have coexistence of both the underlying risk factors and the precipitating factors for vasospasm, yet VA as a clinical syndrome is uncommonly seen or reported.

Conclusion:

The clinician needs to be aware of coronary artery vasospasm as it can pose a serious medical threat. Early diagnosis and treatment may result in improved outcomes from vasospastic angina.

Competing Interests

None Declared

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