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Case Report

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Refractory Coronary Vasospasm After Spine Surgery; A Rare Case Report

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ABSTRACT

Refractory vasospastic angina (RVSA) is a rare condition leading to several episodes of constriction of coronary arteries which eventually leads to myocardial ischemia. Calcium-channel blockers (CCBs) and nitrates are usually used, however, sometimes the vasospasm is refractory and recurrent leading to high morbidity and mortality.

A 35-year-old man known case of hypertension underwent two times operation due to T9-T10 discopathy and decompressive laminectomy of three segments T8/T9/T10 following a previous car accident 4 months before this admission. Three days postoperatively he developed chest pain, dyspnea and diaphoresis. Electrocardiography showed inverted T wave in leads I, II and ST elevation in pericardial leads of V1-V4. Left anterior descending (LAD) artery stenosis was present (99%) at mid part that resolved after Trinitroglycerin (TNG) injection during angiography. Totally, he underwent 3 times coronary angiography due to recurrent chest pain refractory to conventional management of Prinzmetal's angina. Coronary stent could not be placed due to severe spasm. Finally, he developed refractory chest pain and dyspnea and cardiac arrest in the CCU despite receiving intravenous high dose TNG, Diltiazem, Nicorandil and Hydrocortisone. He expired after several times of cardiopulmonary resuscitation.

Refractory VSA after spine surgery has not been reported in the literature yet. This patient was resistant to available medications. There is no consensus regarding the treatment unfortunately. Randomized clinical trials have to be done to find ways regarding unconventional treatment options such as alpha-2-agonists, Corticosteroids, rho-kinase-inhibitors, statins and magnesium. Despite the fact, some surgical interventions with sympathetic denervation like left-stellate-ganglion denervation must be assessed.

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Introduction

Refractory vasospastic angina (RVSA) is a rare condition leading to several episodes of constriction of coronary arteries which eventually leads to myocardial ischemia. Several episodes of Prinzmetal's angina or variant angina occurs leading to high morbidity and mortality [1, 2]. It is thought that variant angina occurs due to localized hyper-responsiveness of the vascular smooth muscle cells following non-specific stimuli of vasoconstriction. In addition, autonomic imbalance has been introduced as underlying mechanism of spontaneous vasospasm, as sympathetic or parasympathetic stimulation can provoke coronary spasms [3]. Moreover, there are some reports regarding the role of the carotid sinus in refractory vasospastic angina in the literature [4]. Primary treatment of refractory vasospastic angina is like any other Prinzmetal's angina using calcium-channel blockers (CCBs) and nitrates, however, sometimes the vasospasm is refractory and recurrent which necessitates coronary interventions [5].

Case presentation

A 35-year-old non-smoker man known as a case of hypertension was admitted to the neurosurgery ward following car accident with low back pain and radiation to the lower limbs. Past drug history was Negative. He then underwent a surgery with T9-T10 discopathy. Post-operatively in the operation room, paraplegia developed. He underwent emergency spine MRI and decompressive laminectomy of three segments T8/T9/T10 was planned by trans-thoracic approach. Three days postoperatively in the neurosurgery ward, cardiology consolation was requested due to chest pain, dyspnea and diaphoresis. In history taking patient mentioned recurrent burning retrosternal pain with radiation to the left upper limb which alleviated after 30 minutes. Electrocardiography was performed showing inverted T wave in leads I, II and ST elevation in pericardial leads of V1-V4 (Fig. 1). He was candidate for coronary angiography. Left anterior descending artery (LAD) stenosis was present (99%) at mid part that resolved after nitrates injection (Figs. 2 and 3). Muscle bridges were detected at mid part of LAD. Others were unremarkable. He then transferred to the cardiac intensive care unit and measures were adopted based on the diagnosis of Prinzmetal's angina. Trinitroglycerin (TNG) infusion 5-10 µg/min intracoronary and diltiazem 60 mg daily IV were administered. Echocardiography findings showed reduced LVEF to 30% and hypokinesis was observed in anteroapical and mid septal. After 4 hours, he developed chest pain, TNG was augmented and morphine sulphate was given to patient. Pain was relieved finally. Two hours later, chest pain recurred and EKG showed ST elevation in pericardial leads V1-V6. He then underwent two times emergent coronary intervention due to recurrent chest pain refractory to conventional management of Prinzmetal's angina. LAD stenosis was the main finding responsive to TNG injection in the Cath lab (Figs. 4 and 5). Coronary Stent could not be placed due to severe spasm. Finally, he developed refractory chest pain and dyspnea and cardiac arrest in the CCU despite receiving intravenous high dose TNG, Diltiazem, Nicorandil and Hydrocortisone. He expired after cardiopulmonary resuscitation.

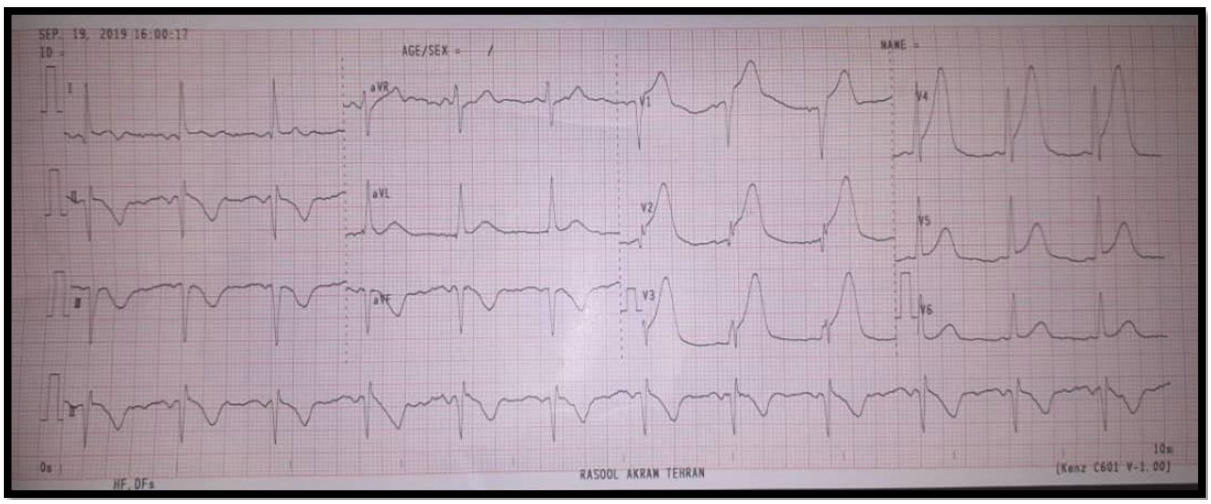

Fig. 1. ECG at the time of chest pain

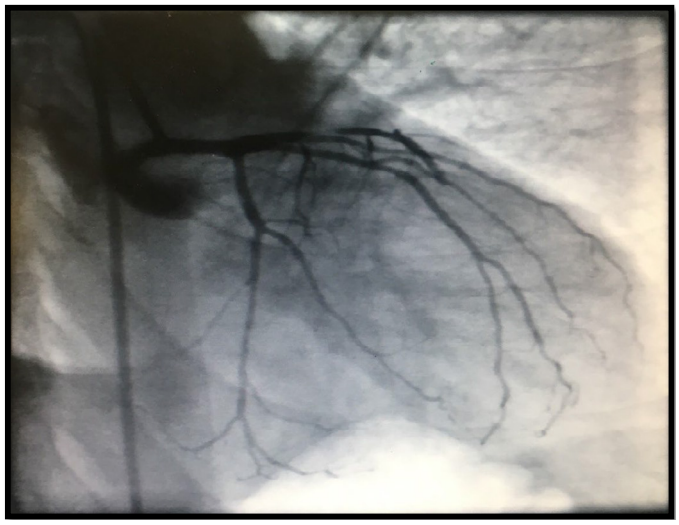

Fig. 2. LAD stenosis in angiography

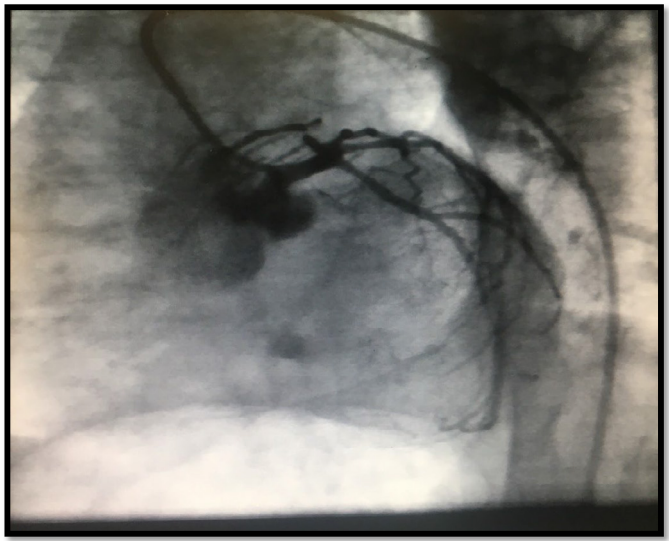

Fig. 3. LAD stenosis relief after using nitrate

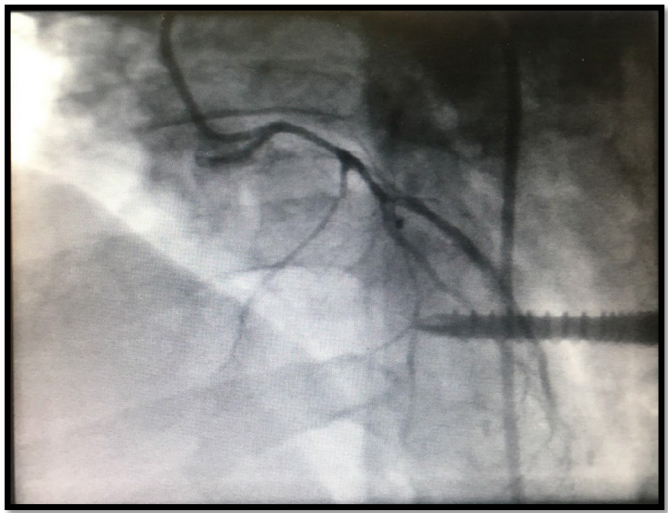

Fig. 4. LAD stenosis in the second angiography (after 2 hours)

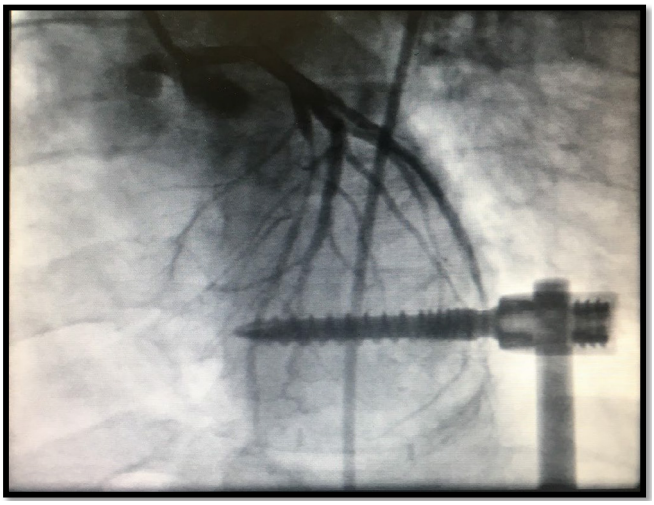

Fig. 5. LAD stenosis relief after using nitarate

Discussion

For the first time, Dr. Myron Prinzmetal in 1959, introduced a variant form of angina pectoris namely "Prinzmetal's angina". Signs and symptoms include chest pain at rest, ST-elevation and pain relief by using sublingual nitroglycerin [3]. Using coronary angiography, arterial spasm as the cause of Prinzmetal's angina has been confirmed. Conventionally, calcium channel blockers and long-acting nitrates are main treatments in coronary vasospasm. However, statins, clonidine, and Rho-kinase inhibitors have been reported to be efficient in some reports.

In this patient, three episodes of marked ST-segment elevation occurred and patient underwent coronary angiography three times showing diffuse spasm in LAD. It responded well to TNG during the procedure. However, due to diffuse spasm, and to avoid metal jacket phenomenon, stent placement was not possible. The muscle bridge detected in the first angiography was minor and unremarkable, whereas significant diffuse spasm in the mid to distal part of the LAD artery was the main finding leading the patient to a life-threatening state. Due to the critical state of the patient all first and second-line therapy of vasoconstriction as TNG, Diltiazem, nicorandil, and beta-blockers were administered. Also, to prevent additional adverse effects of anesthetic drugs, a high dose corticosteroid was given.

The etiology of refractory coronary spasms in our patient is not well elucidated. Certainly, various factors were synergically involved in this process. However, sympathetic and parasympathetic nerve branches manipulation in the thoracic cavity during the operations and adverse reaction to anesthetic drugs might have led to an autonomic imbalance. This condition causes sympathetic nerve over-riding and a storm of spasmodic reactions in the LAD artery. To our knowledge, it is the first report of refractory coronary vasospasm after spine surgery.

Choi et al. reported a case of refractory angina during radical neck dissection under general anesthesia [4]. They postulated that recurrent vasospasm has been occurred due to carotid sinus manipulation. The coronary artery spasm probably has been induced following vagal activation due to carotid sinus activation during general anesthesia. In our case, vagal activation might have been occurred as well, but not precisely known.

In another case report, a 24-year-old man was reported by vasospasm after blunt thoracic trauma. They mentioned that trauma could provoke coronary arteries spasm leading to myocardial ischemia and finally infarction. This hypothesis is supported by coronary spasm in patients with thoracic trauma with no previous history of coronary atherosclerosis. This patient survived one month later in follow-up with verapamil [6].

A recent case report and reviewing the literature regarding refractory vasospasm in 2019 indicated that refractory vasospasm is called when intermittent vasospasm continues, despite receiving a combination of two medications at least. They assessed its pathophysiology including 4 main domains of A) Smooth muscle hypersensitivity due to vasoconstriction or increased calcium sensitivity, B) Endothelial dysfunction due to decreased NO production, C)

Micro vascular dysfunction due to spasm or the Steal phenomenon and D) Autonomic nervous system due to decreased parasympathetic tone or upregulation of alpha adrenergic receptors [7]. As smooth muscle hyper reactivity occurs, a vicious cycle happens leading to ischemia and infarction leading to patient death.

In our case there was no history of coronary disease. Probably, autonomic nervous system imbalance due to decreased parasympathetic tone might have played role in our patient due to sympathetic and parasympathetic nerve manipulations during the operations.

What could be done in our patient to prevent his death is really challenging. There is little data available in the literature regarding refractory vasospasm. The first line of treatment has been reported to be calcium channel blockers and if not responded adding TNG. However, several unconventional medications have been reported in the literature including alpha-2-agonists, corticosteroids, rho-kinase-inhibitors, statins and magnesium [8-10], but there is little data regarding their efficacy in well-established clinical trials. There is a dilemma regarding the treatment protocol for refractory vasospasm. Despite the fact, some surgical interventions with sympathetic denervation like left-stellate-ganglion denervation have been proposed by some authors with different results for refractory VSA [11, 12]. It is necessary to perform clinical trials assessing the effectiveness of such uncommon treatment options to save lives of many patients with refractory coronary vasospasm.

Ethical Considerations

Compliance with ethical guidelines

There were no ethical considerations to be considered in this article.

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Conflict of Interests

The authors have no conflict of interest to declare.

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