

# Recurrent intraoperative silent ST depression responding to phenylephrine

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## Abstract

Intraoperative myocardial ischemia is attributed to decreased myocardial oxygen supply. We present an unusual case of recurrent, symptomless inferior wall ischemia in an apparently healthy male with no history of coronary artery disease after a spinal block. The recurring episodes were linked to tachycardia and presented with significant ST depression in Lead II with reciprocal elevation in lead aVL. The episodes responded to phenylephrine and subsided without residual sequelae.

**Keywords:** Intraoperative myocardial infarction, intraoperative ST depression, phenylephrine for intraoperative myocardial infarction

## Introduction

The perioperative cardiac events are major contributors to perioperative morbidity in general surgeries.<sup>[1]</sup> Despite immense research to find predictors of likely cardiac compromise preoperatively, the number of affected patients continues to grow.<sup>[2,3]</sup> All the predictors and set guidelines stratify the risk associated for a non-cardiac surgery with given comorbidities of the patient.<sup>[4]</sup> In patients without previous history of coronary artery disease (CAD), the incidence of perioperative myocardial infarction (PMI) amounts to 0.6% even after expert evaluation.<sup>[5]</sup> Most often the intraoperative cardiac ischemia involves the left coronary artery<sup>[6]</sup> and presents as ST segment depression in the left sided leads.<sup>[7]</sup> This oxygen demand–supply failure to heart is not restricted to left ventricle alone. It can occur to the vessels supplying the right heart, in case they have enough preoperative narrowing which cannot match the increased oxygen need, during the stress of surgery. This results in ischemic changes on the right sided or inferior

leads perioperatively. We present a similar unique case of rate-dependent reversible myocardial ischemia which responded dramatically to timely pharmacological intervention.

## Case Report

A 46-year-old, 72 kg, man was scheduled for elective right knee replacement for post traumatic osteoarthritis. The patient was a known hypertensive, well controlled on oral amlodipine 5 mg OD. He was a non-smoker with no family history of CAD. The effort tolerance was mildly restricted since last 2 years due pain associated with osteoarthritis for which patient was on occasional non-steroidal anti-inflammatory drugs. There was no history of angina, palpitation, or diaphoresis on effort. There was no significant past medical or surgical history. Pre-operative hemoglobin level was 12.3 mg/dl and the biochemical profile, including lipid profile, was normal. Chest X-ray and electrocardiogram (ECG) showed no abnormality. Being an orthopedic surgery, intermediate risk was explained to the patient and a written/informed consent was taken. The patient was advised to remain nil per oral from midnight and 2 units of packed red blood cells were arranged.

On shifting to the operating room, standard monitoring was connected which showed an ECG with normal sinus rhythm with heart rate 74/min, blood pressure 116/70 mm/Hg, and oxygen saturation 100% on room air. A 16G intravenous (IV) line was secured.

A combined spinal-epidural (CSE) block was given in right lateral position with 12.5 mg of 0.5% hyperbaric bupivacaine

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along with 25 mcg fentanyl was given intrathecally through the L3-L4 interspace on the first attempt after confirming the backflow of cerebro-spinal fluid from the spinal needle. Epidural space was obtained at 5.5 cm depth and an 18G catheter was threaded into the space and fixed with the 10 cm on the skin entry point. The patient was turned to the supine position and the sensory level of block was found to have reached around T5 after 10 minutes.

After 15 minutes, the ECG monitor on “monitoring mode” started to show down-sloping ST depression. The monitor was shifted to diagnostic mode and it recognized a ST depression of 2 mm, which in next 5 minute progressed to 3.4 mm but was limited to lead II only. The automatic ST analysis on the diagnostic monitor showed a value of +0.2 and +1.2 in lead V5 and aVL respectively and the blood pressure was 106/62 mm/Hg with heart rate 117/min [Figure 1]. The patient was asked for any chest pain or heaviness, which the patient denied. Since the ECG pattern was indicative of inferior cardiac wall ischemia and there was increasing tachycardia, we decided to use a vasopressor besides augmenting the ongoing intravenous fluid. We used phenylephrine, rather than ephedrine or mephentermine, to

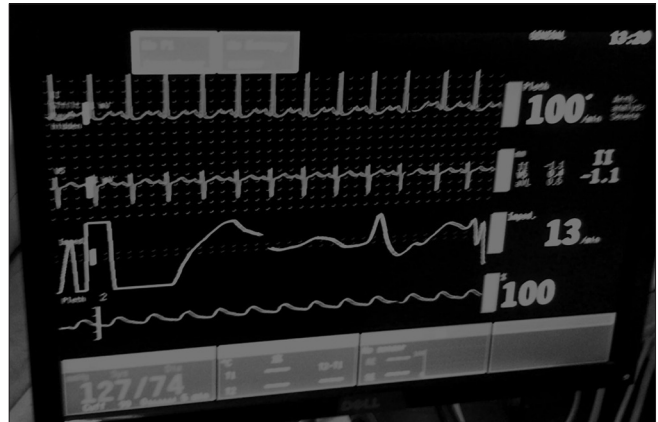
avoid more tachycardia. 75 mcg bolus of phenylephrine was given IV. The ECG pattern changed to normal with heart rate dropping to 100/min and blood pressure picking up to 127/74 in next 5 minutes [Figure 2]. The diagnosis of myocardial ischemia remained uncertain as the lowest blood pressure recording was 106/62 mm/Hg, the patient had no history of CAD and had no clinical symptoms.

After 10 minutes, the ST depression, with similar lead involvement, began to reappear. A ST depression of 3.2 mm in lead II with a reciprocal ST elevation in aVL of +1.1 mm was seen [Figure 3]. The blood pressure dropped to 101/59 mm/Hg with a heart rate 110/min. The patient had received 1l of crystalloid and 500 ml of colloid. Another phenylephrine bolus of 75 mcg was given IV and the rhythm again returned to normal in 5 minutes.

A similar episode reoccurred over next 20 minutes and a phenylephrine infusion was started @ 50 mcg/min. The ST segment values returned to normal for all leads with heart rate returning back to 84/min [Figure 4]. The surgeon was asked to withhold the surgery after 2<sup>nd</sup> episode as blood loss could precipitate MI. The surgery was deferred.



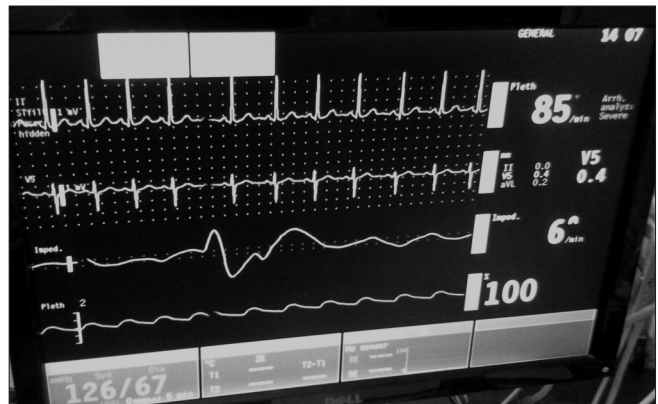
**Figure 1:** Image just after 1st dose of phenylephrine



**Figure 2:** Image after normalization of ST segment



**Figure 3:** Reappearing ST depression



**Figure 4:** Renormalization after phenylephrine

The patient was monitored in recovery and quantitative assay for troponins were sent immediately and a qualitative troponin after 4 hours both of which turned out to be negative for myocardial ischemia. Cardiology evaluation later revealed a 70% occlusion of right coronary artery. An elective coronary stenting was done subsequently.

## Discussion

Anesthesiologists are often challenged with unusual presentations of diseases in the operating room. The literature is replete with examples where an ST depression is known to be docile.<sup>[8]</sup> Up sloping ST segment depression is non-specific and may not be a marker of ischemia. Most often this nonspecific ST depression is lower than 1.5–2 mm.<sup>[9]</sup> In this case, there was down sloping ST depression amounting up to 3.4 mm. There was no history of CAD, post spinal block the fall in blood pressure was not significant enough to cause cardiac hypo-perfusion of such a degree, patient remained asymptomatic and the most sensitive lead in predicting intraoperative ischemia V5<sup>[10]</sup> continued to show normal ST segment. A non-specific ST depression is often isolated and is not associated with reciprocal ST elevation.<sup>[11]</sup> ST segment analysis in lead aVL showed a value of +1.2 with ST depression in lead II which pointed toward inferior wall ischemia.<sup>[12]</sup> In case an index of suspicion for ischemia was not there in this case, it would have probably resulted into fatal MI.

Silent myocardial ischemia/infarction is often seen in patients with autonomic neuropathy.<sup>[13]</sup> Our patient did not have any feature of or predisposing factor for autonomic neuropathy. It has been reported that normal patients perioperatively have higher incidence of silent myocardial ischemia.<sup>[14,15]</sup> The spinal block given to the patient may be responsible for obscuring the manifestations of ischemic pain. The highest sensory block noted was up to T5; the autonomic block may have been higher due to differential blockade<sup>[16]</sup> and involving the cardiac sympathetic plexus [T1–T4].

Frank *et al.* described a similar case where a patient had recurrent intraoperative painless ST depression with no hypotension and the only sign attributed to it was associated tachycardia.<sup>[17]</sup> The most reliable predictors of intraoperative MI also has emerged to be tachycardia.<sup>[18]</sup> Short acting beta-blockers are recommended to control this tachycardia but we did not administer it as the blood pressure was falling. Ephedrine and mephentermine are indirectly acting vasopressors known to cause tachycardia.<sup>[19]</sup> Phenylephrine is a directly acting pure alpha 1 agonist which not only increases the blood pressure but also lowers the heart rate and thus was the drug of our choice in the given situation.<sup>[20]</sup> Phenylephrine preferentially acts on arterial alpha-receptors as compared to

venous. This is a potential disadvantage for a diseased heart as it would increase afterload and increase cardiac oxygen demand. However, it is often associated with hypotension that lowers cardiac perfusion pressure for a normal heart.

The pathophysiology of intraoperative MI is emerging to be different than commonly seen ST depression MI where plaque instability is the cause of subendocardial ischemia. Intraoperative MI is more of a demand–supply failure and hence the treatment lines are different as well.<sup>[21,22]</sup> Administering nitrates in this case may have aggravated the tachycardia and increased myocardial workload.<sup>[23]</sup>

Our patient did not ultimately show any raised troponin levels as infarction was prevented by a timely action. A prolonged ST depression of >20–30 min or a cumulative duration 1–2 h can lead to MI.<sup>[24,25]</sup> Our patient showed three episodes of significant ST depression but the duration of each was limited to less than 10 minutes and hence did not lead to a MI.

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