Sir,
A 55-year-old woman was scheduled for laparoscopic cholecystectomy. She was diabetic and her preoperative stress echocardiography test was negative. Left ventricular ejection fraction was 58%. Anesthesia induction and tracheal intubation were done using intravenous propofol 2 mg/kg, fentanyl 2 µg/kg, and rocuronium 1 mg/kg. After few minutes, the patient had bronchospasm, high airway pressure, tachycardia (heart rate 118/min), and raised blood pressure (190/72 mmHg). Pulse oximetry saturation dropped to 82%. Anterior chest electrocardiographic leads showed ST elevation. Surgery was deferred considering perioperative myocardial ischemia. Intravenous nitroglycerin infusion was started. Nebulization with budesonide and salbutamol was administered. Intravenous hydrocortisone 100 mg was given. The patient was ventilated with 100% oxygen (FiO₂). Transthoracic echocardiography revealed hypokinetic anterior left anterior wall with left ventricular ejection fraction of 40%. The patient started improving with stable hemodynamics, oxygenation, and subsiding electrocardiographic changes. Patient was shifted to cardiac intensive care. Patient was gradually weaned off the mechanical ventilator. Coronary angiography was performed next day, and it showed no obstructive coronary artery lesions. Repeat transthoracic echocardiographic examination was suggestive of the left ventricular ejection fraction of 50% with no regional motion abnormality. In this case, differential diagnosis was takotsubo cardiomyopathy, prinzmetal variant angina, and Kounis syndrome. In takotsubo cardiomyopathy, there is left ventricular wall motion abnormalities particularly involving apical and midventricular segments (classic apical ballooning pattern) with electrocardiography evidence of ischemia after some stressful trigger. However, there is no association of any concurrent allergic reaction as per the described diagnostic criteria of stress cardiomyopathy. In prinzmetal vasospastic angina, there is transient coronary spasm with history of rest anginal symptoms. Unlike this case, coronary atherosclerosis is generally seen in 60% of such patients. Variant angina does not describe concomitant hypersensitivity reaction. Hence, takotsubo cardiomyopathy and prinzmetal variant angina were ruled out in the present case. Anterior wall myocardial ischemic changes in the presence of anaphylactic reaction (probably by rocuronium) causing bronchospasm favors the diagnosis of Kounis syndrome.

Allergic angina syndrome or Kounis syndrome is the occurrence of acute myocardial ischemia in the presence of allergic reactions. Drugs or conditions which cause mast cell degranulation with release of vasoactive inflammatory mediators (leukotrienes, histamine, and serotonin) can potentially lead to coronary vasoconstriction. Although the precise mechanisms causing vasospastic angina have not been fully understood, vasoconstrictor mediators may result in hypercontractility of vascular smooth muscle. Various drugs used during general anesthesia can cause anaphylaxis with mast cell degranulation. Commonly used drugs such as tramadol, rocuronium, and cisatracurium have also been reported with Kounis syndrome. Kounis syndrome is described in three varieties. Type I is with normal coronary artery as in the present case, Type II with coronary artery disease, and Type III is seen with coronary artery stent thrombosis.

Kounis syndrome is not uncommon but often is undiagnosed or underdiagnosed. Why do we need to be aware of Kounis syndrome? It is because it needs special attention during management. Use of morphine and aspirin in such acute coronary syndrome may be detrimental due to its histamine releasing action and leukotriene production, respectively. Steroids and antihistamines can be effective in Type I. Vasodilators such as nitroglycerin can be used for coronary vasospasm. Adrenaline use in such anaphylaxis complicated by Kounis syndrome can be hazardous due to its alpha vasoconstrictive property.

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