ABSTRACT
Postoperative hypoxemia can be a challenging diagnostic and management dilemma for the clinician. We present here a case of postoperative hypoxemia following laparoscopic gastric bypass surgery secondary to presumed pulmonary embolism complicated with a patent foramen ovale. The diagnostic pitfalls associated with a negative spiral computed tomography scan and the impact of coexisting medical conditions aggravating the pulmonary dysfunction are reviewed.

Key Words: Pulmonary embolism, Hypoxemia, Atrial septal defect, Bariatric surgery.

INTRODUCTION
Hypoxemia in obese patients is a well-documented occurrence following surgery.1-3 The postoperative hypoxemia seen in obese patients is often secondary to respiratory dysfunction with an increase in the ventilation-perfusion mismatch.1-4 In addition, other causes of postoperative hypoxemia, such as pulmonary embolism with a reported incidence of 600,000 per year in the United States, are also common in patients undergoing laparoscopic gastric bypass surgery.5 Comorbid conditions can aggravate the high mortality rates associated with massive pulmonary embolism. A probe-patent foramen ovale, seen in 20% to 25% of the population, can manifest flow under conditions that increase right atrial pressure.6-7 Elevated right atrial pressures, seen with pulmonary embolism, adult respiratory distress syndrome (ARDS), and positive-pressure ventilation, increase flow through a patent foramen ovale, which can lead to a greater amount of intracardiac shunt thereby worsening the level of hypoxemia.6,8-10 Several cases of pulmonary embolus complicating a previously undetected patent foramen ovale have been reported as the cause of profound postoperative hypoxemia.5,11-14

CASE REPORT
Patient J.M. was a 42-year-old morbidly obese (BMI > 40) female who underwent elective bariatric surgery. Her past medical history was significant for depression, anxiety, gastro-esophageal reflux disease, hiatal hernia, cholelithiasis, and degenerative joint disease. Her past surgical history included laparoscopic cholecystectomy and bilateral knee surgeries. She underwent an uneventful laparoscopic gastric bypass surgery, with preoperative prophylaxis against pulmonary embolism, including sequential compression devices and low molecular weight heparin.

Our patient’s postoperative course was significant for sudden onset hypoxemia. Four hours after the surgery, she was noted to be hypoxic with an oxygen saturation of 88%. Administration of 100% nonrebreather did not correct her hypoxemia (Figure 1). She was also found to be hypotensive with a blood pressure of 76/41 and tachycardic up to 138 beats per minute. A helical com-
Computed tomography (CT) scan was performed under urgent conditions because of the suspicion of a massive pulmonary embolism as the cause of hypoxemia. The CT scan showed no evidence of pulmonary embolism with minimal dependent atelectasis of the lung bases. An abdominal CT scan performed concurrently did not reveal any significant fluid collection in the abdominal cavity. A pulmonary angiogram was attempted the following day for a definitive diagnosis of pulmonary embolism, but the procedure was abandoned after multiple episodes of ventricular tachycardia during cannulation of the pulmonary artery. Interestingly, a swift enhancement of the aortic arch was noted each time during contrast injection of the right atrium. An upper gastrointestinal (UGI) Gastrografin study on the same day showed a patent gastric anastomotic site with no evidence of leakage. Despite a negative CT scan, a vena caval filter was inserted and intravenous heparin was started empirically to prevent the progression of a pulmonary embolus. Additional studies including Doppler ultrasonography of the distal extremities and perfusion scan of the lungs did not reveal evidence suggestive of venous thromboembolism. A transesophageal echocardiography was performed to search for a shunt as a possible cause of the postoperative hypoxemia. A secundum type atrial septal defect with a bidirectional shunt was found during the echocardiography. The size of the shunt was deemed small enough to be treated conservatively. Our patient was kept on her anticoagulation for empiric treatment of pulmonary embolism with subsequent improvements in her respiratory status. She was discharged following 2 weeks of hospitalization for management of her respiratory status and postoperative ileus.

DISCUSSION

Postoperative hypoxemia can be a source of significant diagnostic challenge for the junior clinician. The reasons for new onset hypoxemia can be quite variable depending on the patient’s medical comorbidities and the type of recent surgery (Figure 2). Surgical manipulation interferes with lung function and oxygen exchange, which can worsen preexisting pulmonary dysfunction. The majority of postoperative hypoxemia is from pulmonary dysfunction, which includes causes from shunted and nonshunted physiology. In the immediate postoperative period, other common causes of hypoxemia are bleeding and hypovolemia. Cardiac-induced pulmonary dysfunction can also lead to significant hypoxemia. Finally, neurogenic and anesthesia-related causes of hypoxemia in the immediate postoperative period are often overlooked.

The case report presented here illustrates 2 causes of new-onset postoperative hypoxemia in the presence of reduced pulmonary reserve, secondary to morbid obesity. In obese patients, Vaughan et al. have repeatedly shown that arterial oxygenation is diminished both intraoperatively and postoperatively. The causes for the hypoxemia in the obese patient may be due to a ventilation-perfusion abnormality and a reduction in expiratory reserve volume. In addition, gastric bypass surgery in the morbidly obese has been shown to lead to clinically significant hypoxemia when compared with preoperative oxygenation measurements.

Pulmonary embolism, though radiographically absent, was suspected in this patient because of the profound new-onset hypoxemia. Clinical suspicions were paramount in guiding the therapy for this patient. The patient developed sudden onset hypoxemia uncorrectable with 100% oxygen, indicating the presence of a significant shunt physiology, either pulmonary or cardiac in origin. Our patient underwent a helical CT scan, which was read as negative for clinically significant pulmonary embolism. Although helical CT scans were initially met with enthusiasm by the medical community as a possible replace-
ment for pulmonary angiogram, the validity of a negative CT scan and its implications for treatment were questioned recently. In their extensive review of the literature, Rathbun et al pointed out the fallacy of the studies on the effectiveness of helical CT for the diagnosis of pulmonary embolism. As a testament to his findings, Drucker et al found that helical CT scans only had sensitivities of 53% to 60% when compared with angiograms. Furthermore, Velmahos et al demonstrated that helical CT scans had a sensitivity of 45% in critically ill surgical patients. Based on their findings, pulmonary angiography remains the gold standard in the diagnosis of pulmonary embolism. Because the mortality of untreated pulmonary embolism approaches 30%, we elected to anticoagulate this patient empirically despite a negative CT scan and an inconclusive angiogram.

Concurrent discovery of a patent foramen ovale in the presence of pulmonary embolism causing right-to-left shunt has been reported in the literature. The majority of reports indicate that right-to-left intracardiac shunt aggravates the baseline hypoxemia seen with massive pulmonary embolus. Elevations in the right atrial pressure stemming from pulmonary embolus, ARDS, and positive-pressure ventilation can lead to an increase in the interatrial right-to-left shunt. The pulmonary embolus may be the inciting event in this case that led to an increase in the right-to-left shunt, through pulmonary vascular outflow obstruction and an increase in right ventricular dysfunction.

The presence of an atrial septal defect is associated with an increase in mortality and a shorter life span. In the presence of hemodynamically significant pulmonary embolism, several authors advocate aggressive management strategies because of the significant mortality rates associated with both conditions. The treatment of a patent foramen ovale includes traditional surgical closure and newer percutaneous techniques. Estagnasié et al have also introduced the use of nitric oxide as an alternative to promote closure of a patent foramen ovale in the event of pulmonary embolism. Nitrous oxide is
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thought to promote closure of a foramen ovale through a decrease in pulmonary vascular resistance, which in turn, leads to a decrease in right-to-left atrial pressure gradient. Our patient was treated conservatively initially because of the size of the shunt, although plans are being made for correction in the near future.

Patent foramen ovale and concurrent pulmonary embolism can cause profound hypoxemia, as described in this case. Frequently, pitfalls in the diagnostic workup, such as a negative CT scan, can cloud the judgment of the clinician. The best diagnostic strategy for both of these conditions remains a high degree of clinical suspicion.

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