Intraoperative Pulmonary Embolism: A Case Report Emphasizing the Utility of Electrocardiogram

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Pulmonary embolism (PE) is an important cause of perioperative morbidity and mortality. In patients with suspected PE, electrocardiogram (ECG) alone is thought to have a limited utility due to its low sensitivity and specificity. This case report describes a patient with intraoperative PE presenting with hypotension and hypoxemia for whom the ECG finding of $S_IQ_{III}T_{III}$ was key in identifying acute cor pulmonale. The ECG was paramount in our decision to acquire computed tomography angiography to confirm the diagnosis, reinforcing its invaluable role in early detection of intraoperative PE. (A&A Case Reports. 2017;9:349–52.)

Pulmonary embolism (PE) is a significant cause of morbidity and mortality, accounting for an estimated 300,000 to 600,000 cases and 100,000 deaths annually in the United States alone.[1,2] Surgery is recognized as a major risk factor predisposing patients to form deep venous thrombosis and potentially PE.[3] Surgery interferes with 3 fundamental triggers of intravascular clot formation collectively known as the Virchow triad.[4] In the perioperative context, the triad includes blood stasis secondary to immobilization during surgery, a procoagulation state caused by surgical trauma, and, in some instances, direct surgical disruption of the endovascular endothelium. Accordingly, Wells et al[3] developed a frequently used, validated scoring system to assess disease probability in patients with suspected PE (Table). Despite identifying risk factors and using the Wells score, a high level of clinical suspicion for PE is necessary in the perioperative setting.

Written consent from the patient was obtained before publication of this report per our institutional policy.

CASE DESCRIPTION

A 73-year-old male presented for robot-assisted laparoscopic left inguinal hernia repair. He denied any comorbidities and had an excellent functional status (>4 metabolic equivalents). His surgical history included a bilateral inguinal hernia repair in the distant past, which was uneventful from both an anesthesia and surgical standpoint. His medications included vitamin D3 and saw palmetto. He had never smoked, and his family history was negative for clotting disorders or thrombotic events.

The patient was evaluated 2 days before the scheduled surgery at our preoperative assessment clinic, whereby physical exam and laboratory studies were unremarkable. Electrocardiogram (ECG, Figure 1A) was also obtained and revealed sinus bradycardia at 55 beats per minute (bpm). On the day of surgery, the patient received 2 mg of midazolam for anxiolysis before transport to the operating room. Preinduction vital signs included blood pressure (BP) of 140/81 mm Hg, heart rate of 58 bpm, and oxygen saturation as measured by pulse oximetry (Spo_2) 98% on room air. Cardiac monitoring in leads II and V_5 revealed sinus bradycardia with normal QRS and T-wave morphology. The patient underwent standard induction with 100 μg of fentanyl, 100 mg of lidocaine, 150 mg propofol, and 50 mg of rocuronium, followed by easy endotracheal intubation. General anesthesia was subsequently maintained with 1.6% sevoflurane on 40% fraction of inspired oxygen (Fio_2). The patient was placed into lithotomy position, and the operating room table was rotated 90° in anticipation of docking the robot.

Fifteen minutes after anesthesia induction and 2 minutes after being placed into lithotomy position, the patient acutely desaturated to 82% and became hypotensive to a BP of 73/44 mm Hg. Noninvasive BP was immediately repeated and was undetectable despite a palpable carotid pulse. Cardiac monitoring revealed sinus bradycardia at 44 bpm. The Fio_2 was increased to 100%, and lung recruitment maneuvers were performed. Lung auscultation confirmed clear bilateral breath sounds. Hypotension and bradycardia were treated with 200 μg of phenylephrine and 0.2 mg of glycopyrrolate, respectively, to no avail. Ultimately, 10 μg of epinephrine was administered with immediate improvement of hemodynamics. Noninvasive BP improved to 157/91 mm Hg, and Spo_2 increased to 95%. Spo_2 remained at 95% despite 100% Fio_2 for the remainder of the case.

Table. Wells Scoring System for PE

Wells Score Criterion	Points
Clinically suspected DVT	3.0
Alternative diagnosis less likely than PE	3.0
Heart rate >100	1.5
Immobilization (≥3 d) or surgery in previous 4 wk	1.5
History of DVT or PE	1.5
Hemoptysis	1.0
Malignancy	1.0

For scores >4, PE is likely, and diagnostic imaging is recommended. For scores ≤4, PE is unlikely, and D-dimer can be used to rule out the diagnosis. In the reported case, the patient's Wells score was 1.5, placing him into a low-risk group, with 1.3% risk of PE.

Abbreviations: DVT, deep venous thrombosis; PE, pulmonary embolism.

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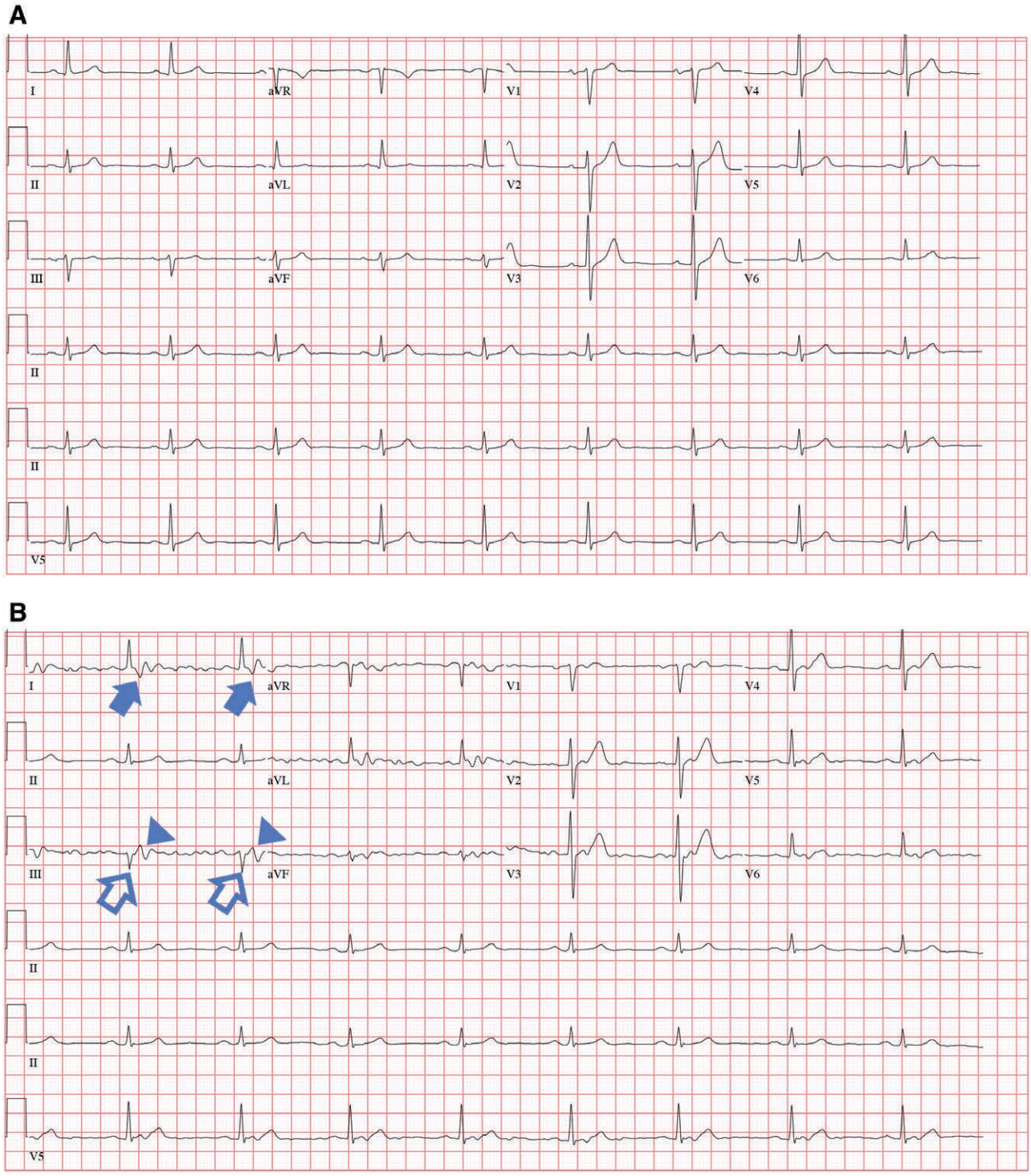

Figure 1. 12-lead ECG obtained 2 weeks before surgery (A) showed sinus bradycardia of 55 beats per minute with normal QRS complex and T-wave morphology. Postoperative ECG (B) revealed S deflections (full arrows) in lead I and Q waves (empty arrows) with T-wave inversions (arrowheads) in lead III, consistent with the $S_IQ_{III}T_{III}$ pattern of acute cor pulmonale. ECG indicates electrocardiogram.

Importantly, there were no changes to capnography (end-tidal CO_2 between 35 and 40 mm Hg) and/or airway pressures during the event. The case was canceled given the elective nature of the surgery. Arterial blood gas on 100% FIO_2 was obtained before extubation and proved to be reassuring, with pH 7.4, $PaCO_2$ 40 mm Hg, PaO_2 465 mm Hg, HCO_3 28.6 mmol/L, and base excess 4. Emergence from anesthesia was uneventful except for hypertension of up to 186/100 mm Hg, which decreased to 116/64 mm Hg after 15 mg of hydralazine. The patient remained in sinus bradycardia at 44 bpm.

On arrival to the postanesthesia care unit, the patient was fully awake and oriented. He denied any chest pain or shortness of breath. Vital signs and physical exam remained

within normal limits. Laboratory studies consisting of basic metabolic panel, complete blood count, cardiac enzymes, and coagulation screen were all unremarkable. Portable chest X-ray was also normal. In contrast, a repeat 12-lead ECG (Figure 1B) showed sinus bradycardia at 52 bpm associated with a questionable S deflection in lead I and Q waves with T-wave inversions in lead III, consistent with the $S_IQ_{III}T_{III}$ right heart strain pattern. However, bedside transthoracic echocardiogram did not show signs of right heart failure or strain. In light of these findings, the patient was admitted to the surgical service for suspected acute cor pulmonale likely due to PE for further diagnostic workup and management.

Computed tomography of the chest with intravenous contrast obtained on the same day revealed filling defects within the branches of the right lower lobe pulmonary artery, compatible with acute pulmonary emboli (Figure 2). Ultrasound of lower extremities failed to demonstrate evidence of deep venous thrombosis. A full coagulation panel was normal, with a borderline protein C activity of 71%. Oral anticoagulation therapy with apixaban was initiated for acute PE. The patient remained asymptomatic and was discharged home on postoperative day 2.

DISCUSSION

The reported case underlines the essential role of ECG in evaluating patients with suspected PE. The $S_IQ_{III}T_{III}$ pattern observed here was first described by Ginn and White[5] in 1935, when they presented 7 patients with acute cor pulmonale secondary to PE. $S_IQ_{III}T_{III}$ is an indicator of acute volume and pressure overload of the right heart, whereby S wave in lead I is a manifestation of incomplete right bundle branch block (RBBB), and Q wave, along with T-wave inversion in lead III, suggests repolarization abnormalities within the right ventricle.[6] $S_IQ_{III}T_{III}$ is a nonspecific sign of acute cor pulmonale and cannot differentiate cor pulmonale secondary to PE versus other causes. Despite this, $S_IQ_{III}T_{III}$ was reported to be found in 11% to 52% cases of confirmed acute PE but is usually associated with persistent hemodynamic instability.[7,8]

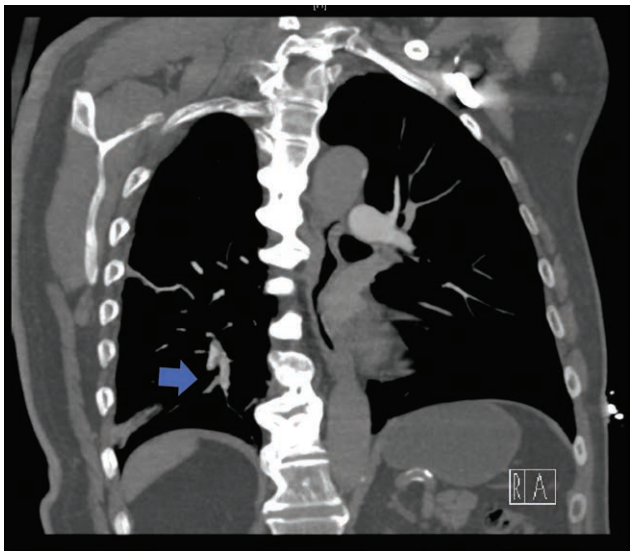

Figure 2. Computed tomography angiogram demonstrated filling defects within the branches of the right lower lobe pulmonary artery (arrows), consistent with acute pulmonary emboli.

ECG may provide critical insight into the cardiac function in patients with PE, although the prevalence of ECG signs without symptoms is low. For example, Ferrari et al[9] examined 80 patients hospitalized for acute PE and described the 5 most common ECG findings to be T-wave inversion in anterior leads (present in 68% of patients), $S_IQ_{III}T_{III}$ (50%), peripheral low voltage (29%), sinus tachycardia (26%), and complete or incomplete RBBB (22%). Rodger et al[10] compared the ECG in patients with confirmed acute PE to those presenting with suspected PE (for whom an alternative diagnosis was made) and found sinus tachycardia and incomplete RBBB to be more frequent in the patients with confirmed PE ($P = .08$ and .002, respectively).

While these studies may suggest that ECG alone has a limited utility to make the diagnosis of PE, the presented case illustrates its pivotal role in promptly narrowing the differential diagnosis in patients with acute hemodynamic compromise. In this case, the $S_IQ_{III}T_{III}$ pattern was the only finding pointing to acute cor pulmonale as the cause of intraoperative hypoxemia and hypotension. Notably, other changes associated with acute PE, such as sinus tachycardia or decline in the end-tidal CO_2, were absent. In addition, the patient had no risk factors predisposing him to acute PE. His Wells score was 1.5 (for ongoing surgery), placing him into the low-risk group with a 1.3% chance of PE.[11] The ECG was thereby paramount in the decision to obtain computed tomography angiography, which confirmed the diagnosis of acute PE and enabled the patient to receive therapeutic anticoagulation. ■

DISCLOSURES

Name: Josef Pleticha, MD.
Contribution: This author helped care for the patient, review the literature, and write the manuscript.
Name: Evan M. Sutton, MD.
Contribution: This author helped care for the patient, review the literature, and write the manuscript.
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REFERENCES

1. Beckman MG, Hooper WC, Critchley SE, Ortel TL. Venous thromboembolism: a public health concern. *Am J Prev Med*. 2010;38:S495–S501.
2. Horlander KT, Mannino DM, Leeper KV. Pulmonary embolism mortality in the United States, 1979-1998: an analysis using multiple-cause mortality data. *Arch Intern Med*. 2003;163:1711–1717.
3. Wells PS, Anderson DR, Rodger M, et al. Excluding pulmonary embolism at the bedside without diagnostic imaging: management of patients with suspected pulmonary embolism presenting to the emergency department by using a simple clinical model and d-dimer. *Ann Intern Med*. 2001;135:98–107.
4. Bagot CN, Arya R. Virchow and his triad: a question of attribution. *Br J Haematol*. 2008;143:180–190.
5. Ginn S, White P. Acute cor pulmonale resulting from pulmonary embolism: its clinical recognition. *JAMA*. 1935;104:1473.
6. Chan TC, Vilke GM, Pollack M, Brady WJ. Electrocardiographic manifestations: pulmonary embolism. *J Emerg Med*. 2001;21:263–270.
7. Digby GC, Kukla P, Zhan ZQ, et al. The value of electrocardiographic abnormalities in the prognosis of pulmonary embolism: a consensus paper. *Ann Noninvasive Electrocardiol*. 2015;20:207–223.
8. Gallotta G, Palmieri V, Piedimonte V, et al. Increased troponin I predicts in-hospital occurrence of hemodynamic instability in

patients with sub-massive or non-massive pulmonary embolism independent to clinical, echocardiographic and laboratory information. *Int J Cardiol*. 2008;124:351–357.
9. Ferrari E, Imbert A, Chevalier T, Mihoubi A, Morand P, Baudouy M. The ECG in pulmonary embolism. Predictive value of negative T waves in precordial leads–80 case reports. *Chest*. 1997;111:537–543.
10. Rodger M, Makropoulos D, Turek M, et al. Diagnostic value of the electrocardiogram in suspected pulmonary embolism. *Am J Cardiol*. 2000;86:807–809.
11. Wolf SJ, McCubbin TR, Feldhaus KM, Faragher JP, Adcock DM. Prospective validation of Wells criteria in the evaluation of patients with suspected pulmonary embolism. *Ann Emerg Med*. 2004;44:503–510.