

# Clinically Suspected Systemic Arterial Gas Embolism during Robotic-Assisted Bronchoscopy: a Case Report

**Brian M. Hom**

Ronald Reagan UCLA Medical Center

**Brian Rosenberg**

Ronald Reagan UCLA Medical Center

**Nicole Y. Xu**

Ronald Reagan UCLA Medical Center

**Joseph W. Szokol**

`JSzokol@mednet.ucla.edu`

Ronald Reagan UCLA Medical Center

**Maria McCarthy**

Ronald Reagan UCLA Medical Center

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## Case Report

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# Abstract

## Background:

Systemic arterial gas embolism (SAGE) is a rare but potentially catastrophic complication of robotic-assisted bronchoscopy with lung biopsy. During general anesthesia, the earliest manifestations of SAGE may be limited to transient electrocardiographic or hemodynamic abnormalities. The absence of visible intracranial gas on subsequent imaging does not exclude the diagnosis. We report a case of clinically suspected SAGE during robotic-assisted bronchoscopy in which intraoperative sentinel signs preceded delayed emergence and focal neurologic injury.

## Case presentation:

A 78-year-old woman underwent robotic-assisted bronchoscopy under general anesthesia for biopsy of a 4.6-cm right middle lobe ground-glass opacity using transbronchial needle aspiration and cryobiopsy. Significant airway bleeding occurred after sampling. Immediately after biopsy, she developed transient tachycardia, hypertension, bradycardia, and ST-segment elevation in lead V5. Although these abnormalities resolved, she was unresponsive shortly after extubation and developed right gaze deviation, facial twitching, abnormal movements, and absent right upper-extremity withdrawal to pain. Head computed tomography showed no acute abnormality, and brain magnetic resonance imaging demonstrated left frontal and precentral cortical diffusion restriction without visible intracranial gas. Because SAGE was strongly suspected, she received 100% oxygen, levetiracetam, endotracheal reintubation, and hyperbaric oxygen therapy at 2.8 atmospheres absolute approximately 4 hours after bronchoscopy. High-sensitivity troponin I peaked at 5,116 ng/L and left ventricular ejection fraction improved from 45-50% to 60% the following day. She was extubated on post-procedure day 10 with persistent neurologic deficits. On post-procedure day 18, she experienced hypoxic cardiac arrest associated with bilateral pulmonary emboli and an acute aortic dissection and died the following day after transition to comfort-focused care.

## Conclusions:

During bronchoscopic biopsies under general anesthesia, unexplained ST-segment changes, hemodynamic instability, or delayed neurologic recovery should prompt immediate consideration of SAGE, even when initial neuroimaging is negative. Administration of 100% oxygen, airway and hemodynamic support, minimization of airway pressures, and urgent consultation for hyperbaric oxygen therapy should not be delayed while awaiting radiographic confirmation.

# INTRODUCTION

Robotic-assisted bronchoscopy (RAB) is an advanced, minimally invasive interventional procedure used for the diagnosis of various pulmonary conditions.<sup>1</sup> While RAB has expanded bronchoscopic access to challenging peripheral pulmonary lesions, there still remains some risks, as rates of pneumothorax, chest-tube placement, and significant hemorrhage are 2.3%, 1.2%, and 0.5%, respectively.<sup>2</sup> Systemic arterial gas embolism (SAGE) is a rare but potentially catastrophic complication of bronchoscopic lung

biopsy, occurring when gas gains access to the pulmonary venous circulation and embolizes to the coronary or cerebral arterial circulation. In a systematic review of 29 cases associated with bronchoscopic lung biopsy, neurologic manifestations occurred in 97%, cardiac manifestations in 28%, mortality in 28%, and residual neurologic deficits in 24%.<sup>3</sup> As such, early recognition and treatment are critical for patient survival. Though reports involving SAGE in RAB remain limited, recent cases have demonstrated presentations with ST-segment elevation, cardiac arrest, seizures, and cerebral ischemic injury.<sup>4,5</sup> We describe a case of clinically suspected SAGE during RAB in which transient intraoperative electrocardiographic and hemodynamic abnormalities preceded delayed emergence and profound focal neurologic deficits despite negative initial neuroimaging, highlighting the anesthesiologist's essential role in recognizing peri-procedural sentinel signs and initiating treatment before radiographic confirmation.

## CASE

A 78-year-old never-smoker with mild dementia, osteoporosis, hypertension, and hyperlipidemia was found to have a 4.6 cm ground-glass lesion in the right middle lobe after workup for unintentional weight loss of 40 pounds over the prior year. She was scheduled for a flexible bronchoscopy, robotic-assisted bronchoscopy, endobronchial ultrasound, and fluoroscopy-guided needle aspiration transbronchial cryobiopsies and bronchoalveolar lavage at an ambulatory surgical center (Table 1).

Table 1  
Clinical timeline

| Time                                     | Clinical event and management                                                                                                                                                                                                                                  |
|------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Pre-procedure                            | A 78-year-old woman was scheduled for robotic-assisted bronchoscopic biopsy of a 4.6-cm right middle lobe ground-glass opacity.                                                                                                                                |
| Immediately after biopsy                 | Tachycardia, hypertension, bradycardia, and transient ST-segment elevation in lead V5 occurred. Significant right middle lobe airway bleeding was treated with tranexamic acid and temporary Fogarty-balloon occlusion.                                        |
| Within 5 minutes of PACU arrival         | Unresponsiveness, right gaze deviation, facial twitching, abnormal movements, and right upper-extremity weakness prompted stroke activation, 100% oxygen, levetiracetam, and left lateral positioning.                                                         |
| Same day                                 | Head CT showed no acute abnormality. Brain MRI showed left frontal and precentral cortical diffusion restriction without visible intracranial gas. The patient was reintubated, transferred to the intensive care unit, and referred for hyperbaric treatment. |
| Approximately 4 hours after bronchoscopy | A single 142-minute HBOT session was administered at 2.8 ATA.                                                                                                                                                                                                  |
| 12 hours after the event                 | High-sensitivity troponin I peaked at 5,116 ng/L.                                                                                                                                                                                                              |
| Post-procedure day 1                     | Echocardiography showed left ventricular ejection fraction 45–50%, mildly reduced right ventricular function, and no intracardiac shunt.                                                                                                                       |
| Post-procedure day 2                     | Left ventricular ejection fraction improved to 60%.                                                                                                                                                                                                            |
| Post-procedure day 10                    | The patient was extubated with persistent focal neurologic deficits.                                                                                                                                                                                           |
| Post-procedure days 18–19                | Hypoxic cardiac arrest occurred in association with bilateral pulmonary emboli and acute aortic dissection. After transition to comfort-focused care, the patient died the following day.                                                                      |

Anesthesia was induced uneventfully using propofol, fentanyl, lidocaine, and rocuronium, and the patient was intubated with an 8.0 endotracheal tube. Consistent with published RAB ventilation protocols, the pulmonologist requested that the patient receive a positive end expiratory pressure of 10 cm H<sub>2</sub>O and tidal volumes at 10 mL/kg of ideal body weight.<sup>6,7</sup> These settings were maintained to minimize atelectasis that could obscure the target lesion during intraoperative cone-beam CT.<sup>8</sup> After induction and intubation, the Ion Endoluminal System (Intuitive Surgical, Sunnyvale, CA, USA) was navigated to the target lesion in the lateral segment of the right middle lobe. Following a breath hold performed under apnea at adjustable pressure-limiting valve setting of 20 cm H<sub>2</sub>O, a cone-beam CT was performed, and the lesion was localized. The pulmonologist used a 21-gauge needle and a 1.1 mm cryoprobe to biopsy the lesion. After biopsy, the robotic bronchoscope was removed. When the flexible bronchoscope was introduced, significant bleeding was observed from the right middle lobe bronchus. This bleeding was

controlled with aliquots of tranexamic acid (25 mg/mL) and transient balloon occlusion of the airway using a 5-French Fogarty balloon.

Immediately after biopsy, the patient developed approximately 2 minutes of tachycardia (heart rate approximately 110 beats/min) and 8 minutes of hypertension (systolic/diastolic blood pressure 180/110 mm Hg), treated with esmolol and fentanyl. After esmolol administration, she developed 5 minutes of bradycardia (heart rate 41 beats/min), for which phenylephrine and ephedrine were administered. ST-segment elevation in lead V5 persisted for approximately 10 minutes (Fig. 1). Following these interventions, the abnormalities resolved, and the electrocardiogram returned to sinus rhythm near baseline for the remainder of the procedure. No corresponding change in end-tidal carbon dioxide (37–41 mm Hg) or oxygen saturation (100% by pulse oximetry) occurred. No further intraoperative abnormalities were observed. Neuromuscular blockade was reversed with sugammadex and train-of-four stimulation showed four equal twitches. The patient opened her eyes to verbal stimulation, followed commands, and was extubated before transfer to the post-anesthesia care unit.

In the PACU, the patient remained unresponsive and did not recover as expected. After 5 minutes, a code stroke was called. On examination, she was found to have right gaze deviation, right facial twitching, left-hand pill rolling, and jaw movements concerning for possible seizure activity. She was noted to not move her right upper extremity in response to pain. The patient was promptly started on high-flow oxygen via a nonrebreather mask, loaded with levetiracetam, and placed in the left lateral decubitus position. An emergent brain CT with and without contrast demonstrated no abnormalities. A subsequent MRI demonstrated reduced diffusivity involving the left superior frontal, left middle frontal, and left precentral gyri, representing edematous changes from postictal swelling of the cortex with no associated hemorrhage or fluid-attenuated inversion recovery signal changes. No intracranial air was visualized on either exam. However, given high suspicion for SAGE, hyperbaric medicine was consulted for emergent therapy.

The patient was reintubated for airway protection and transferred to the main hospital intensive care unit. Approximately 4 hours after bronchoscopy, she underwent a single 142-minute hyperbaric oxygen therapy session at 2.8 atmospheres absolute, breathing 100% oxygen for 120 minutes with three air breaks. High-sensitivity troponin I peaked at 5,116 ng/L 12 hours after the event. A transthoracic echocardiogram on post-procedure day 1 showed a left ventricular ejection fraction of 45–50%, mildly reduced right ventricular systolic function, and no intracardiac shunt on agitated-saline study. Repeat echocardiography the following day showed improvement of left ventricular ejection fraction to 60%. On post-procedure day 3, histopathologic examination of the biopsy specimen demonstrated lung adenocarcinoma positive for *EGFR* mutation L858R.

The patient was extubated on post-procedure day #10. After extubation, she intermittently looked toward the examiner, followed occasional commands, and moved the left-sided extremities spontaneously with minimal right-sided movement. On post-procedure day 18, while awaiting upper endoscopy for melena, she developed acute hypotension and altered mental status followed by hypoxic cardiac arrest.

Following initiation of cardiopulmonary resuscitation was initiated and reintubation, return of spontaneous circulation was achieved. CT chest with contrast revealed extensive bilateral pulmonary emboli extending from the right and left pulmonary arteries as well as an aneurysmal ascending thoracic aorta and type B aortic dissection. After extensive discussion with family, the decision was made to compassionately extubate and provide comfort care to the patient, who passed away the following day.

## DISCUSSION

This case describes clinically suspected SAGE during RAB, in which significant airway bleeding, transient ST-segment elevations, and hemodynamic abnormalities were followed by delayed emergence, seizure-like activity, and focal neurologic deficits. Although intravascular gas was not directly visualized, the close temporal relationship to transbronchial needle aspiration and cryobiopsy, cortical diffusion abnormalities on magnetic resonance imaging (MRI), and objective myocardial injury supported emergent treatment for SAGE. The principal educational feature is that, during general anesthesia, transient cardiovascular abnormalities may be the only observable warning before cerebral injury becomes clinically apparent.

While SAGE has been estimated to occur in fewer than 0.02% of flexible bronchoscopies, its true incidence during RAB is unclear.<sup>9,10</sup> In characterizing SAGE, Jain et al. reported airway bleeding, ST-segment elevation, cardiac arrest, visible gas in the left ventricular apex, and subsequent cerebral injury.<sup>5</sup> Likewise, one five-patient conference series on SAGE described abrupt ST-segment elevation, bradycardia or hypotension, myocardial injury, and nonobstructive coronary anatomy after RAB biopsy.<sup>11</sup> This case aligns well with the literature in that a transient, self-resolving cardiovascular phenotype may precede substantial neurologic injury despite initial CT imaging remaining negative.<sup>4</sup> Accordingly, unexplained peri-biopsy electrocardiographic changes should be interpreted as possible sentinel events rather than assumed to be isolated anesthetic phenomena.

The most plausible mechanism of SAGE requires both disruption of a pulmonary vein and a pressure gradient that drives gas from an airway or alveolus into the pulmonary venous circulation.<sup>3,12–14</sup> Needle or cryoprobe sampling may create a transient bronchial-pulmonary venous or alveolar-pulmonary venous fistula. The airway bleeding in this case supports local vascular injury, although it does not prove that such a fistula formed.<sup>3</sup> Once gas enters a pulmonary vein, it can pass through the left atrium and ventricle to the coronary and cerebral arterial circulations without requiring an intracardiac shunt.<sup>3,14</sup>

The ventilation strategy used in this case (positive end-expiratory pressure of 10 cm H<sub>2</sub>O, tidal volume of 10mL/kg ideal body weight, and an inspiratory breath hold) was intended to prevent dependent atelectasis and improve target localization during cone-beam CT, consistent with advanced guided-bronchoscopy protocols.<sup>4,7,8</sup> After vascular injury, however, elevated airway pressure may increase the airway-to-pulmonary-vein pressure gradient and facilitate gas entry. Positive-pressure ventilation has been associated with systemic air embolism in the percutaneous lung biopsy literature, but

bronchoscopic SAGE has also occurred during spontaneous or negative-pressure ventilation.<sup>3,12</sup> Thus, positive pressure should be regarded as a plausible contributor rather than a sufficient cause, and no bronchoscopic airway-pressure threshold has been shown to eliminate risk. The practical implication is to reassess the ventilation strategy immediately after biopsy-related bleeding or unexplained cardiovascular changes and to use the lowest airway pressures compatible with oxygenation and procedural safety.

The diagnosis of SAGE was difficult because each early finding had a more common alternative explanation. Transient bradycardia and blood-pressure changes may accompany apnea, airway manipulation, anesthetic depth changes, or vasoactive medication administration.<sup>15,16</sup> Plaque rupture, coronary vasospasm, demand ischemia, and stress cardiomyopathy could also explain the troponin elevation and transient ventricular dysfunction because coronary angiography was not performed. Additionally, residual anesthetic effect could have contributed to delayed emergence, although it would not adequately explain the gaze deviation, focal motor activity, and asymmetric motor response. Conventional thromboembolic stroke and a primary seizure disorder also remained in the differential diagnosis. Seizures are common in cerebral arterial gas embolism and may result from cortical ischemia, endothelial injury, blood-brain barrier disruption, and secondary inflammation.<sup>17,18</sup> In a contemporary series, CT identified free intracranial gas in only 4 of 13 patients, whereas focal deficits, impaired consciousness, seizures, and multifocal diffusion restriction were more frequent.<sup>17</sup> Evidently, gas may redistribute or dissolve before imaging, and current hyperbaric guidance specifically cautions that the absence of radiographically visible gas should not exclude arterial gas embolism.<sup>14,19</sup>

MRI may provide supportive evidence after the gas itself is no longer visible. Recent imaging studies describe predominantly gyriform, multifocal, and cortical border zone patterns of diffusion restriction that differ from the typical wedge-shaped pattern of cardioembolic stroke.<sup>20,21</sup> In this patient, however, the unilateral frontal and precentral diffusion abnormalities were interpreted as possible postictal cortical edema and were not pathognomonic for SAGE. The imaging therefore increased concern for acute cortical injury but could not independently distinguish gas embolism from ischemic stroke or seizure-related change. This limitation reinforces that SAGE is fundamentally a clinical diagnosis in the appropriate procedural context.

When SAGE is suspected, further sampling should stop, the suspected source of gas entry should be controlled, and 100% oxygen should be administered immediately. High inspired oxygen accelerates nitrogen elimination from the bubble and supports oxygen delivery to ischemic tissue. Furthermore, airway protection, seizure treatment, and hemodynamic stabilization should proceed immediately.<sup>13,14,19,22,23</sup> The 2025 American Heart Association guideline recommends 100% oxygen for life-threatening gas embolism (Class 1, level of evidence C-EO), considers hyperbaric oxygen therapy (HBOT) reasonable for adults (Class 2a, level of evidence B-NR), and considers supine or recovery positioning on a level surface reasonable (Class 2a, level of evidence C-EO).<sup>23</sup> Older literature described left lateral head-down positioning primarily for venous gas embolism, but current guidance does not

recommend routine Trendelenburg positioning because bubble redistribution data are inconsistent and head-down tilt may aggravate cerebral edema.<sup>13,14,23</sup> In this case, left lateral positioning without head-down tilt was reasonable as a recovery position while mental status was impaired. Additionally, focused echocardiography may identify intracardiac gas, new regional wall-motion abnormalities, or acute ventricular dysfunction during active instability.<sup>13</sup> However, routine transesophageal echocardiography or precordial Doppler monitoring for every RAB procedure is unlikely to be practical, despite immediate access to point-of-care cardiac imaging potentially improving recognition in selected high-risk or unstable cases.

Urgent HBOT consultation was appropriate despite negative CT imaging. HBOT reduces bubble volume through increased ambient pressure, accelerates inert-gas elimination, increases oxygen delivery beyond an obstructed vessel, and may attenuate secondary endothelial and inflammatory injury.<sup>13,14,19,23</sup> Blanc et al. reported better overall outcomes when treatment began within 6 hours in a retrospective cohort of 86 patients.<sup>24</sup> In addition, Fakkert et al. found that favorable outcomes became progressively less likely as time to HBOT increased, even after adjustment for manifestation severity.<sup>25</sup> This data supports treatment as early as feasible, but they do not establish a rigid time threshold beyond which consultation or treatment is futile.

Restoration of intravascular volume is appropriate when hypovolemia or shock is present to maintain coronary and cerebral perfusion. Increasing pulmonary venous filling pressure could theoretically narrow the airway-to-vein pressure gradient and limit further gas entrainment; however, support for this concept comes indirectly from venous and paradoxical air-embolism literature rather than from bronchoscopic SAGE studies.<sup>26,27</sup> Routine prophylactic fluid loading solely to prevent SAGE should therefore not be presented as an established intervention. Hemodynamic resuscitation should be individualized to perfusion, bleeding, and cardiac function.

This report has several limitations. SAGE was not confirmed by direct visualization of intravascular gas, and no bronchial-pulmonary venous or alveolar-pulmonary venous fistula was demonstrated. Coronary imaging was not performed, so the transient ST-segment change, troponin elevation, and ventricular dysfunction cannot be attributed definitively to coronary gas embolism. The MRI abnormalities were nonspecific, the exact airway pressure at the presumed moment of embolization is unknown, and only one hyperbaric oxygen therapy session was administered. Therefore, the incremental value of repeat treatment cannot be assessed. The later bilateral pulmonary emboli and acute aortic dissection provide separate explanations for the terminal deterioration and substantially limited assessment of long-term neurologic recovery. These later events should not be attributed to the suspected SAGE.

Future multicenter registries should standardize collection of lesion-vessel anatomy, biopsy tools, airway pressures, bleeding severity, electrocardiographic and end-tidal carbon dioxide changes, point-of-care ultrasound findings, time to oxygen and HBOT, and neurologic outcomes. RAB programs, particularly those based in ambulatory centers, should also establish explicit emergency transfer and hyperbaric consultation pathways for this rare but time-sensitive complication.

# CONCLUSION

During RAB, abrupt ST-segment or hemodynamic changes after transbronchial sampling, particularly when accompanied by airway bleeding, should prompt consideration of SAGE. Because intravascular gas may no longer be visible when imaging is obtained, 100% oxygen, airway and hemodynamic support, and early hyperbaric consultation should proceed on clinical suspicion. Ambulatory RAB programs should maintain explicit emergency-transfer pathways for this rare, time-sensitive complication.

## Abbreviations

ATA  
atmospheres absolute  
CT  
computed tomography  
ECG  
electrocardiogram  
FLAIR  
fluid-attenuated inversion recovery  
HBOT  
hyperbaric oxygen therapy  
MRI  
magnetic resonance imaging  
PACU  
post-anesthesia care unit  
PEEP  
positive end-expiratory pressure  
RAB  
robotic-assisted bronchoscopy  
SAGE  
systemic arterial gas embolism.

## Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication:

Written informed consent was obtained for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Availability of data and materials:

All data supporting the conclusions of this case report are included within the article. Additional patient-level data are not publicly available because of privacy restrictions.

Competing interests:

The authors declare that they have no competing interests.

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Authors' contributions:

BMH prepared the initial manuscript draft and literature review. BR, NYX, JWS, and MM contributed to interpretation of the case and critically revised the manuscript for important intellectual content. All authors read and approved the final manuscript and agree to be accountable for the work.

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## Figures

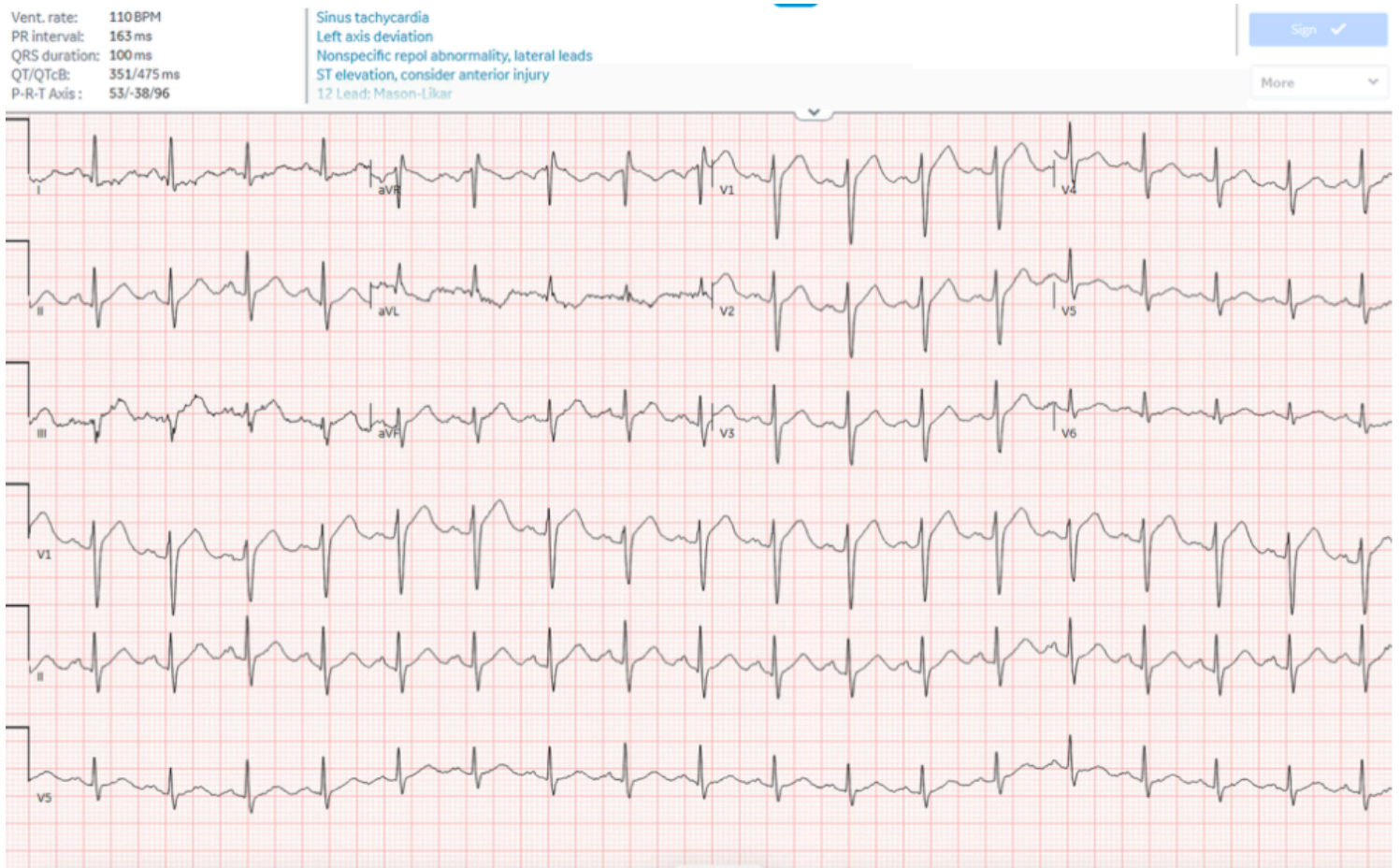


**Figure 1**

EKG displaying ST-segment abnormality in V5 during RAB

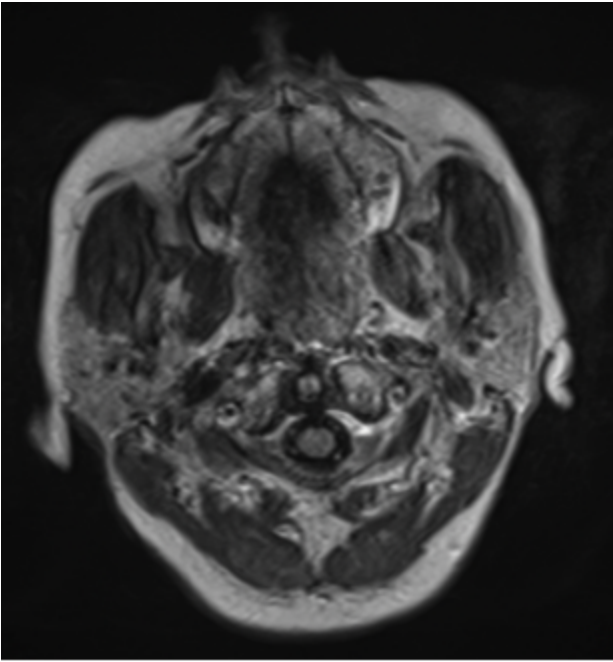


**Figure 2**

MRI imaging showing gyriform region of reduced diffusivity involving the left superior frontal, left middle frontal, and left precentral gyri suspected to represent edematous changes related to the postictal swelling of the cortex. No associated hemorrhage or FLAIR signal changes in these regions