Nonfatal Air Embolism During Shoulder Arthroscopy

Vivek Pandey, MS, Elsa Varghese, MD, Madhu Rao, MBBS, Nataraj M. Srinivasan, MD, Neethu Mathew, MBBS, Kiran K.V. Acharya, MS, and P. Sripathi Rao, MS

Abstract

An air embolism is a rare but potentially fatal complication of shoulder arthroscopy.

In this article, we report the case of a patient who developed a nonfatal air embolism during shoulder arthroscopy for an acute bony Bankart lesion and a greater tuberosity avulsion fracture. The venous air embolism occurred immediately after the joint was insufflated with air for diagnostic air arthroscopy. The diagnosis was based on a drop in end-tidal carbon dioxide and blood pressure and presence of mill wheel (waterwheel) murmur over the right heart. Supportive treatment was initiated immediately. The patient recovered fully and had no further complications of air embolism.

This patient's case emphasizes the importance of being aware that air embolisms can occur during shoulder arthroscopy performed for acute intra-articular fractures of the shoulder. Monitoring end tidal carbon dioxide can be very useful in early detection of air embolisms.

Venous air embolism (VAE), an entity with the potential for severe morbidity and mortality, tends to be predominantly an iatrogenic complication that occurs when atmospheric gas is introduced into the systemic venous system. It is a known but very rare complication of shoulder arthroscopy.[1-3] It was common in the days of air-only arthroscopy but is very rare now, as fluid is used for arthroscopy.[4,5] It can be fatal if not diagnosed immediately.[3-5] It can occur whenever there is a negative pressure gradient between the right atrium of the heart and the operative site. Air can be sucked into the open venous sinusoids of a fresh fracture or raw bone surface (acromioplasty or distal clavicle excision) and thereby enter the circulation.[6,7]

In this article, we report a case of nonfatal VAE. Air was used as the initial distending agent during shoulder arthroscopy performed for an acute bony Bankart lesion and a greater tuberosity avulsion fracture.

The patient provided written informed consent for print and electronic publication of this case report.

Case Report

After a traffic accident, a 23-year-old man was brought to our trauma center. He reported pain and swelling over the right shoulder. There was no other significant history. General and systemic examinations as well as neurovascular examination were normal. Local examination of the right shoulder revealed diffuse tenderness, flattening of the deltoid contour, and a palpable bony mass anteroinferior to the coracoid process. A clinical diagnosis of acute anterior dislocation of the right shoulder was made. Plain radiograph showed anterior dislocation of the shoulder with avulsion of the greater tuberosity, and fracture of the glenoid rim. Computed tomography showed a bony Bankart lesion with a greater tuberosity avulsion. Arthroscopic suture anchor fixation of the lesion and cancellous screw fixation of the greater tuberosity were planned. Surgery was scheduled 1 week after routine preoperative workup.

Under general anesthesia, the patient was positioned in the left lateral decubitus position with the body tilted 30° to the right. After preparation and draping, a standard posterior portal was made. A trocar with an arthroscopic cannula sheath was inserted, and the hematoma was drained. For diagnostic arthroscopy, 40 mL of air was injected into the cannula. However, visualization was inadequate because of bleeding and hematoma. We decided to fix the greater tuberosity first, using the mini-open lateral deltoid splitting technique, and perform arthroscopic fixation of bony Bankart lesion afterward. A 5-cm long skin incision was made over the lateral aspect of shoulder adjacent to the acromion border. Immediately after the incision was made, the anesthesiologist observed a significant drop in end-tidal carbon dioxide ($ETCO_2$), from 38 mm Hg to 17 mm Hg. Left-arm noninvasive blood pressure dropped suddenly from 110/74 mm Hg to 90/47 mm Hg. Oxygen saturation (SpO_2) dropped to 85%. No electrocardiographic changes were noted. Auscultation over the pulmonary area revealed the classical mill wheel (waterwheel) murmur, diagnostic of air embolism.

The patient was immediately moved to a head-low position, and maintained in the left lateral position. A fluid bolus of 1000

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mL of Ringer lactate was pushed using a pressurized bag, and dopamine infusion was initiated—the rationale being to increase the right heart volume and pressure. An attempt was made to introduce a central line through the right internal jugular vein to aspirate air from the right heart, but this proved technically difficult because of the patient's position. Within 15 minutes, $ETCO_2$ improved and SpO_2 returned to normal. Intraoperative transesophageal echocardiography was not attempted, as equipment was not available and the patient was rapidly stabilized. The anesthesiologist allowed us to proceed with mini-open fixation of the greater tuberosity and to abandon arthroscopic fixation of the bony Bankart lesion. The greater tuberosity was quickly fixed with two 6.5-mm cannulated cancellous screws using the deltoid splitting approach, and the wound was closed in layers. Traction was removed, and the patient was positioned supine. A right internal jugular catheter was inserted; no air or froth was aspirated. The patient continued to be stable and was extubated when awake. He was closely monitored in the intensive care unit for 12 hours after surgery, and remained stable without additional hemodynamic or ventilatory support. There were no neurologic complications. Postoperative echocardiography and chest radiograph were normal. The patient was discharged from the hospital 2 days later.

Discussion

VAE is common during neurosurgical procedures.[8] It was reported in a few cases of knee arthroscopy in the days of air arthroscopy using carbon dioxide.[4,5] However, VAE is a very rare complication of shoulder arthroscopy today, as fluid and not air is used as the definitive medium for arthroscopy. Many shoulder surgeons initially use air, to distend the joint and perform the diagnostic arthroscopy, but then use saline for the therapeutic arthroscopy. VAE can occur in any setting in which the operative site is 5 cm or higher than the right atrium. It also occurs when air is used instead of carbon dioxide, the pressure of insufflated gas exceeds that of the venous sinusoids (15-30 mm Hg), air is insufflated too fast, and the venous sinusoids of bone are exposed. Collapsible and non–collapsing fluid bags used for shoulder arthroscopy are also a potential source of air entering the venous system.[9] The typical setting of shoulder arthroscopy fulfills all the criteria. When the patient is in either the beach-chair or the lateral position, the shoulder is always at a higher level, inviting risk for VAE. Whenever the joint is entered, bubbles are found. These bubbles can enter the venous sinusoids of a fresh fracture or any raw bony surface exposed during acromioplasty, distal clavicle excision, or preparation of the tuberosity or the glenoid rim.

Zmistowski and colleagues[3] reported a case of air embolism in liquid-only shoulder arthroscopy in a patient who underwent distal clavicle excision. When the saline bags ran dry, a large amount of air suddenly entered the circulation through the subacromial space. The possible entry route was the open end of the clavicle; excision with open venous sinusoids allowed air to enter the circulation. The patient sustained severe brain damage and later died. The saline bags were found to contain some air, which could have entered the circulatory system and precipitated the embolism. Hence, Austin and colleagues[9] recommended aspirating all air from saline bags used in shoulder arthroscopy to prevent embolisms. Faure and colleagues[10] also reported a case of air embolism in shoulder arthroscopy. Air in the amount of 60 mL was injected into the joint, followed by saline. The patient was revived quickly and sustained no permanent damage to any of the vital organs. The details of the case were not clear enough for the possible route of air into the circulation to be identified. In another case, reported by Hegde and Avatgere,[11] the air embolism occurred immediately after air was injected into the joint. The patient made a complete recovery without any sequelae. Again, though, there was no demonstrable raw surface through which air could have entered the circulation. The authors postulated that air entered the circulation through a vein puncture caused by the trocar entry into the joint.

In our patient's case, air appeared to enter the circulation after the dry arthroscopy was attempted, when about 40 mL of air was injected rapidly into the joint. There was a large raw area exposing venous sinusoids over the anterior glenoid rim and the greater tuberosity, caused by an avulsion fracture. In addition, when the joint was entered, the trocar of the arthroscopy sheath may have disturbed the hematoma or punctured a vein. These factors may have contributed to the injected air being forced into the venous sinusoids and causing the VAE.

The aftereffects of air embolism depend on amount and rate of air entered, site of entrapped air, presence of a right-to-left shunt, patient position, and hemodynamic status.[12-17] Air entered into the circulation as a bolus of more than 50 mL (1 mL/kg) can cause hypotension and dysrhythmia. Air volumes of more than 300 mL (3-5 mL/kg) are invariably fatal in humans.[18] Air forced into the venous sinusoids can enter the venous circulation, reaching the right atrium and ventricle, and can block output to the lung, leading to a drop in blood pressure and poor gas exchange. This is reflected as hypotension, hypoxemia, and a drop in $ETCO_2$. It is analogous to an airlock in which a massive volume of air mechanically blocks the right atrium, ventricle, and pulmonary artery, causing decreased venous return and low cardiac output and precipitating potentially fatal cardiovascular collapse.[12,15,19,20] Pulmonary microvascular blockade leads to increased dead space. Bronchoconstriction may result from endothelial mediator release and cytokine release, which further exacerbates the dead space. VAE can be an immediate disaster in patients with a right-to-left shunt; air can rapidly enter the systemic circulation (cerebral, cardiac) without entering the pulmonary circulation and can have catastrophic consequences of cerebral infarcts and coronary artery occlusion.

The mill wheel murmur, the sound made by air churning in blood, caused by the beating of the heart,[12,21] indicates a massive air embolism and imminent cardiovascular collapse.

Drop in $ETCO_2$ is a sensitive indicator of air embolism but is not specific (sensitivity, 0.4 mL/kg/min).[22] This drop is most commonly used to detect early VAE but is not specific for VAE,

as it is also observed in low-cardiac-output hyperventilation. It indicates an increased dead space. When this drop occurs with the mill wheel murmur over the right heart, the diagnosis becomes more specific for air embolism. Transesophageal echocardiography is the most sensitive invasive tool for diagnosing air embolism; it detects air in amounts as small as 0.02 mL/kg.[7] Precordial Doppler ultrasonography is a sensitive and practical noninvasive tool for diagnosing air embolism. It can detect air in amounts as small as 0.05 mL/kg.[23] However, the urgency of the situation and the logistical difficulties in rapidly mobilizing equipment and personnel to the operating room for an urgent transesophageal echocardiography make precordial Doppler ultrasonography impractical when VAE is not expected.

Management of air embolisms is largely symptomatic and supportive. The goals are to prevent the embolus from expanding and to stop more air from entering the venous system. Compression over the jugular vein also helps in procedures performed with the patient in the sitting position.[24] As soon as air embolism is diagnosed, all anesthetic agents are discontinued, and 100% oxygen is administered. The operative site is lowered to a level below the heart by tilting the operative table, as was done in the present case. The Trendelenburg position helps in preventing air from entering the cerebral circulation. The left lateral position also helps in keeping air in the right atrium and preventing it from entering the ventricles. In the present case, the patient was already in the left lateral position; we did not reposition him supine. Unfortunately, his position made central venous access difficult, either through the internal jugular vein or the subclavian vein. Air can be aspirated with a right atrial catheter, and we attempted this when the patient was positioned supine later. Large amounts of intravenous fluid are pushed as bolus in an attempt to push the blocked airlock into the lungs, where it might be absorbed.[25] Inotropes are used to raise blood pressure. These interventions helped tide the patient over during this crisis.

Conclusion

Prompt recognition by anesthesiologists and a high index of suspicion certainly help in reducing mortality in these cases. Prevention is of pivotal importance in managing air embolisms. Patients, particularly those at risk for VAE, should be kept well hydrated. Surgeons should not perform air arthroscopy in patients with acute shoulder fractures or bony avulsions of the ligaments, and only fluid arthroscopy should be performed to avoid potential air embolism. It is recommended that air be aspirated from the plastic saline bags to prevent it from entering the circulation should the bags run dry.

Dr. Pandey is Associate Professor, Department of Orthopaedics; Dr. Varghese is Professor, Dr. M. Rao is Resident, and Dr. Srinivasan is Associate Professor, Department of Anaesthesia; and Dr. Mathew is Intern, Dr. Acharya is Associate Professor, and Dr. P. Rao is Professor, Department of Orthopaedics, Kasturba Medical College, Manipal, Karnataka, India.

Address correspondence to: Vivek Pandey, MS, Department of Orthopaedic Surgery, Kasturba Medical College, Manipal, Karnataka, 576104, India (tel, 91-820-2922754; fax, 91-820-2571934; e-mail, vivekortho@gmail.com).

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