ABSTRACT
A 59-year-old woman presented with advanced, symptomatic carotid artery stenosis in the setting of severe medical comorbidities including coronary artery disease, congestive heart failure with recent admission for exacerbation, and diabetes mellitus. She underwent awake transcarotid artery revascularization because of her medically high-risk status. Postoperatively, she was noted to have developed pneumothorax, pneumomediastinum, and dysphonia, thought to be secondary to entrained air during the course of low neck dissection for carotid artery exposure in the setting of partial airway obstruction and high negative intrathoracic pressures during the procedure. After conservative treatment, she ultimately enjoyed complete clinical resolution. This case demonstrates an unusual complication of awake transcarotid artery revascularization. (J Vasc Surg Cases and Innovative Techniques 2020;6:133-5.)

Keywords: Carotid artery stenosis; Carotid artery stenting; Transcarotid arterial sheath was connected to the femoral venous portion of the system, and reversal of carotid artery-common carotid artery velocity ratio of 7.1. Dual antiplatelet therapy was initiated with aspirin 81 mg daily and clopidogrel 75 mg daily, and she was referred for carotid revascularization. Her risk of periprocedural cardiac complications was clinically estimated to be high, especially in the setting of recent admission for decompensated congestive heart failure; therefore, TCAR was planned. Preoperative P2Y_12 assay yielded 64 platelet reactivity units.

Under anesthesia-monitored sedation with propofol, a skin incision was planned at the base of the right side of the neck overlying the common carotid artery. Local anesthesia was administered, the skin was incised, and the platysma was divided. The common carotid artery was dissected free and encircled. The patient was sedated to a level of 5 on the modified Ramsay scoring system, resulting in some partial airway obstruction during inspiration. This led to the generation of negative intrathoracic pressures with accessory muscle use during respiration to overcome the partial airway obstruction. During dissection, there were large excursions of the chest wall. We were aware that there was some air that became trapped deep within the wound because of bubbling that would occur with expiratory effort. This began when we had exposed but had not yet completely dissected the common carotid artery circumferentially. Oxygen saturations during the entirety of the procedure remained at 97% or higher. The venous sheath was placed into the left femoral vein under ultrasound guidance. After systemic heparinization, access to the carotid artery was undertaken, and the 8F arterial sheath of the ENROUTE (Silk Road Medical, Sunnyvale, Calif) system was placed in the usual fashion according to the manufacturer’s instructions for use. Sedation was lightened to allow neurologic monitoring, which appeared to coincide with a decrease in wound bubbling. The arterial sheath was connected to the femoral venous portion of the system, and reversal of carotid flow was confirmed. Deployment of the internal carotid stent under reversal of flow.
conditions was performed as planned and without any change in neurologic status. The arterial 5-0 mattress suture was secured, and hemostasis was obtained throughout the wound. A total of 30 mg of protamine reversal were given after closure of the arteriotomy. The bubbling in the wound had stopped, and a 10-mm flat closed suction drain was left in the subplatysmal space to facilitate evacuation of any trapped air. The platysma and skin were closed with absorbable suture. An upright chest radiograph in the recovery area demonstrated a moderate pneumothorax (Fig 1). The patient demonstrated no focal neurologic deficits, normal vital signs, and normal phonation.

The following morning, repeated chest radiography revealed improvement in the size of the pneumothorax and no change in the neurologic examination findings. Just before anticipated discharge, approximately 30 hours after the conclusion of surgery, she developed rapidly progressive hoarseness of voice without other symptoms. Physical examination revealed no stridor, respiratory distress, or obvious neck hematoma. A computed tomography angiogram was obtained. This revealed the absence of hematoma and normal-appearing vasculature. In addition, moderate-volume pneumothorax, pneumomediastinum, and subcutaneous emphysema were apparent (Fig 2). Bedside flexible fiberoptic laryngoscopy was undertaken by an otolaryngology consultant to evaluate for other causes of dysphonia: this demonstrated the presence of surface cysts consistent with emphysema on the vocal cords without evidence of vocal cord paralysis or cranial nerve injury.

The patient was observed in an inpatient setting for any evidence of airway compromise or progression of pneumothorax and was discharged home on postoperative day 3 with slowly improving phonation. A chest radiograph obtained at the time of discharge revealed persistent but improving right apical pneumothorax and neck soft tissue emphysema. Outpatient follow-up on postoperative day 5 yielded complete resolution of the pneumothorax and near-normal phonation. Three weeks after surgery, her voice was noted to have returned completely to her normal baseline and she remained without neurologic symptoms. Duplex ultrasound examination revealed carotid stent patency with peak systolic velocity of 108 cm/s.

**DISCUSSION**

To our knowledge, this is the first report in the literature of pneumothorax, pneumomediastinum, and vocal changes after TCAR. The remarkable chest excursions and clinical evidence of high intrathoracic pressures combined with the development of bubbling in the wound on expiration early in the case suggested to us that air was being entrained into the subcutaneous tissues. Several reports have identified cases of large accumulations of pneumothorax and pneumomediastinum without direct pleural violation, including air trapping after distal extremity barotrauma, colonoscopic perforation, and perineal dissection.\(^1\)\(^3\) Hamman syndrome, a spontaneous event that has similar findings to those in our patient, includes pneumomediastinum, subcutaneous emphysema, and occasionally dysphonia that can occur in association with high intrathoracic pressures, such as Valsalva during labor.\(^4\) One mechanism postulated by Parmaksizoglu et al\(^5\) is the creation of a one-way valve phenomenon that encourages air to entrain along natural tissue planes, which is then unable to escape. In this case, large negative intrathoracic pressures generated by a heavily sedated patient with partial airway obstruction appear to have created such an effect.
Another explanation for the clinical findings may be direct pleural injury. Pneumothorax after dissection low in the neck for vascular procedures is not uncommon and occurs frequently during operations for thoracic outlet syndrome using a supraclavicular incision. In contradistinction to this case, rib resection-related pneumothorax is most often caused by direct pleural violation, which is often visualized during the course of the procedure. Other procedures involving dissection in the low neck similar to TCAR include carotid-subclavian bypass and subclavian transposition performed for a variety of indications. Most recent reports of outcomes after these operations do not mention pneumothorax as a frequent complication. One early report from Salam et al referenced the occurrence of 1 pneumothorax in 41 patients in their series (2%). These findings highlight the low likelihood of direct pleural violation when operations are performed in this anatomic space and lend credence to alternative explanations.

CONCLUSIONS
Procedures to correct thoracic outlet syndrome and to bypass the subclavian artery are almost universally performed under general anesthesia with intubation and positive pressure ventilation, which prevents the development of negative intrathoracic pressures during surgical dissection. TCAR has been advanced as an option for medically fragile patients in part because of its minimally invasive nature and the option to perform the procedure under local anesthesia. Dissection low in the neck near the chest while the patient maintains spontaneous respiratory effort, generating sometimes large negative intrathoracic pressure, creates a relatively unique opportunity for air to be entrained into the pleural space, mediastinum, and possibly other extrapleural spaces. Avoidance of partial airway obstruction and exacerbation of air trapping by this mechanism could be mitigated by the administration of a relatively light level of sedation and making more liberal use of infiltrated local anesthetic during most TCAR procedures.

If pleural injury or air entrapment is entertained during the TCAR procedure, consider taking measures to decompress the pleural or subcutaneous space through the wound before complete wound closure. Options include temporary intrapleural placement of a red rubber catheter for aspiration of accumulated air after airtight wound closure has been completed. As in this case, a subplatysmal closed suction drain may also be considered. It has not been established whether these adjuncts can provide substantial reduction in symptoms. If pleural entry is clearly evident during any low-neck procedure and clinical concern for a large pneumothorax is recognized, a small-tube thoracostomy may also be used for definitive pleural decompression postoperatively.

Dysphonia presenting after neck dissection always requires primary vigilance for neck hematoma and nerve injury. As with most cases of spontaneous pneumomediastinum in the absence of hollow viscus injury, no additional procedures are necessary in the postoperative period, with expectation of resolution of symptoms over time.

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