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Case Report

Brain abscesses following carotid blowout syndrome: a case report ☆

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ABSTRACT

We report a case of intracranial abscesses development in a patient with head and neck cancer after emergent treatment of carotid blowout syndrome with coil embolization. Our patient is a 60-year-old male who presented with hemoptysis and hematemesis, which raised concerns for impending carotid blowout syndrome. Endovascular occlusion was successfully achieved, and the patient was discharged in stable condition. Ten days later, the patient reported headaches and right facial pain, and magnetic resonance imaging revealed multiple intracranial abscesses. Broad-spectrum intravenous antibiotics were administered, leading to a variable response with some abscesses decreasing in size and others increasing. Seven weeks from discharge, the patient had no neurological deficits, and all abscesses had decreased in size.

Introduction

Carotid blowout syndrome (CBS) is a rare, but severe oncological and vascular complication that typically occurs in patients treated for widespread and/or head and neck squamous cell carcinoma [1]. The pathophysiology of CBS most commonly includes arterial wall necrosis following surgical intervention, radiation therapy, or direct tumor invasion. This results in rupture of the carotid artery or its branches, often leading to massive hemorrhage [2]. CBS can be classified into 3 stages of severity based on clinical presentation: threatened (type I), impending (type II), or acute carotid system hemorrhage (type III) [1–3]. Identification of these earlier stages is imperative as intervention leads to lower complication rates and improved survival [2,4]. Rates of CBS following head and neck surgery

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have decreased in recent years due to significant advancements in both surgical and endovascular approaches, which have resulted in high rates of technical success for bleeding cessation [2,5]. In particular, endovascular interventions with coil embolization and stent grafts have gained recent interest as standard of care treatments due to improved patient outcomes when compared to the traditional method of surgical ligation. While mortality has improved in this patient population, the rate of long-term neurological complications and severe delayed adverse events is high and may require prompt intervention [6–10]. The most common postoperative complications include ischemic stroke, hemiplegia/paresis, neck abscess, and sepsis [7,8]. As we further advance endovascular approaches in the treatment of CBS, it becomes crucial to enhance our understanding of the disease and associated treatment complications. In this case report, we present what we believe to be the third reported case of intracranial abscess following endovascular intervention for the treatment of CBS [2,5,11,12].

Case presentation

This patient was a 60-year-old male with a medical history of metastatic thyroid cancer with tracheal invasion that was initially treated with total thyroidectomy and tracheal resection in 2019. This was later complicated by infection of the tracheocutaneous fistula in 2022 and ultimately required tracheostomy and gastrostomy tube placement. He also was treated with radiation therapy to the affected area and received a total dose of 6600 cGY that was completed approximately 40 months prior to presentation.

The patient was initially admitted to the hospital following multiple episodes of hematemesis after undergoing radiation therapy earlier that day for metastasis to the thoracic spine. A preliminary evaluation with esophagogastroduodenoscopy was performed to rule out gastrointestinal bleeding, however, no active bleeding was found. The following day, the patient began experiencing episodes of hemoptysis and hematemesis, raising concerns for impending carotid blow out syndrome (CBS). Urgent diagnostic imaging with computed tomography angiography (CTA) of the neck revealed tumor progression causing 50% stenosis of the right mid-common carotid artery, which had progressively worsened compared to prior imaging. The right common carotid artery also displayed an irregular contour, suggestive of tumor invasion with pseudoaneurysm (Fig. 1). A diagnostic cerebral angiogram showed 40% stenosis of the right common carotid artery and the presence of a focal pseudoaneurysm within the proximal right common carotid artery, consistent with contained carotid blowout (Fig. 2). Endovascular occlusion was successfully achieved with the placement of 2 polydioxanone coils within the right common carotid artery and an 8 mm Amplatzer plug at the origin of the right common carotid artery (Fig. 3). Collateral circulation through a patent anterior communicating artery and right posterior communicating artery enabled complete reconstitution of the right supraclinoid internal carotid artery circulation. A repeat angiogram showed no active extravasation. Follow up CTA head and neck (Fig. 4) less than 9 hours later showed well-defined minor strokes in the right frontal lobe and insular cortex, suggesting that the timing of these

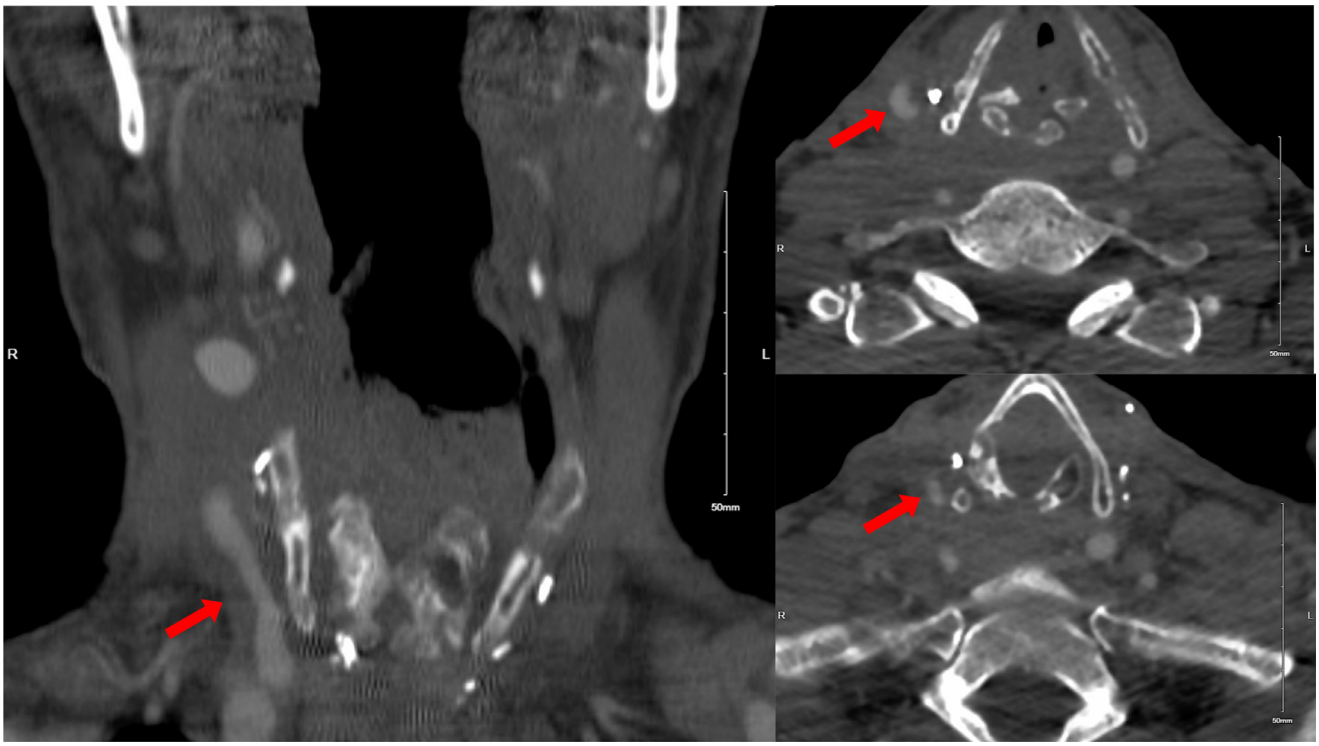

Fig. 1 – Coronal (left) and axial (right) CT scan of the neck with contrast showing right common carotid stenosis and pseudoaneurysm.

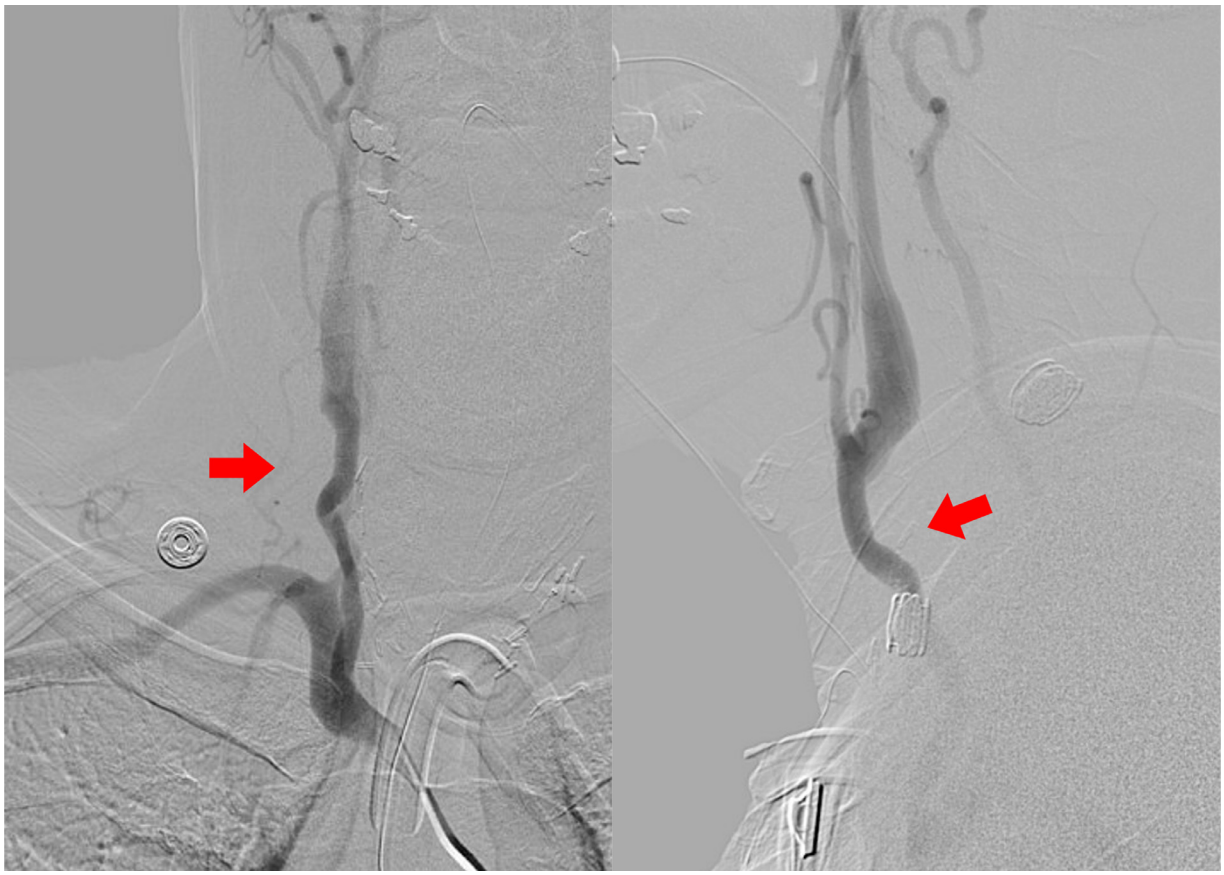

Fig. 2 – Coronal (left) and sagittal (right) diagnostic subtraction angiography of the right common carotid artery showing stenosis and pseudoaneurysm prior to embolization.

Fig. 3 – CTA of right neck showing metallic artifact as a result of the vascular plug (left) and successful coil embolization (right).

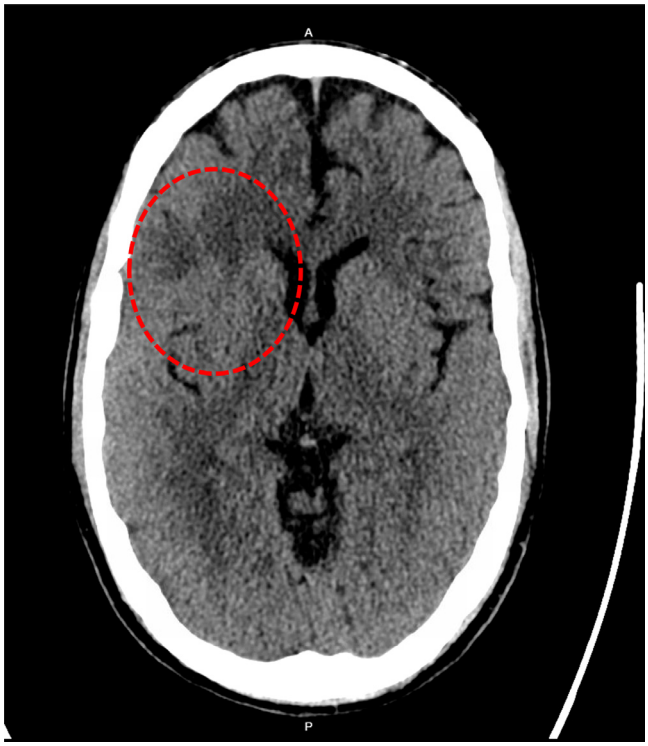

Fig. 4 – Axial noncontrast CT head with areas of hypodensities (circled).

infarcts likely occurred prior to the endovascular procedure. The patient was discharged in stable condition after 2 days with daily full dose aspirin.

Ten days after the endovascular embolization of the pseudoaneurysm, the patient reported increasing severity of headaches with right facial pain. Outpatient magnetic resonance imaging (MRI) of the brain showed multiple rim-enhancing masses with surrounding vasogenic edema diffusely scattered throughout the right cerebral hemisphere concerning for intracranial abscess (Fig. 5). A stereotactic biopsy was performed, and the cultures grew *Viridans streptococci* and *Eikenella corrodens*, both of which are typical of esophageal flora. Treatment was started with broad-spectrum intravenous antibiotics. During the immediate postoperative hospitalization, the patient was afebrile and had an uncomplicated course. The patient was discharged 3 days later with intravenous ceftriaxone.

Two weeks after discharge, a follow-up MRI of the brain revealed a mixed response to the treatment, with some abscesses exhibiting a reduction in size and others demonstrating an increase. At the patient's most recent follow up, which occurred approximately 7 weeks from discharge, there were no observed neurological deficits. However, the patient did report experiencing a persistent headache that was responsive to daily dexamethasone. MRI of the brain at this 7-week follow-up visit showed a decrease in size of all brain abscesses. The patient was closely monitored and was scheduled for routine MRIs of the brain every 3 weeks to monitor treatment response. However, approximately 11 weeks after the embolization procedure, the patient tragically experienced sudden death while sleeping. The exact cause of death remains unknown, but it is suspected to be secondary to airway compromise.

Discussion

While the endovascular repair of CBS is inherently a high-risk procedure with known serious postoperative complications, the development of intracerebral abscess has been reported in the literature in only 2 previous cases [11,12]. In the prior cases, the patients presented at 3- and 4-months post-op. This case highlights not only that intracranial abscess can form after carotid embolization with coils, but also that the abscess can develop as quickly as 10 days post treatment. The prompt discovery of the intracranial abscesses has proven to be crucial as duration of symptoms is related to the overall mortality [13].

The underlying etiology of intracranial abscess development following endovascular repair for CBS remains unclear. Previous case studies suggest that radiation necrosis creates an intrinsically contaminated environment, which promotes local infections and increases the risk of septic emboli [11,12]. Additionally, there is evidence to suggest that intracranial abscess formation can occur after endovascular intervention in the setting of both hemorrhagic and ischemic strokes. The infarcted brain tissue may have a weakened ability to detect and eliminate bacterial pathogens, contributing to the development of abscesses [14–16].

In the case of our patient, the primary cause of abscess formation is likely attributed to the presence of an esophageal-carotid fistula, which established a direct communication between the esophagus and the carotid artery prior to the embolization procedure. This connection provided a pathway for esophageal bacteria to directly enter the bloodstream, serving as a mode of contamination. The occurrence of hematemesis experienced by the patient further supports the formation of the fistula. The growth of *Viridans streptococci* and *Eikenella corrodens* in culture biopsy samples obtained from the abscesses also provides evidence of infection from the esophagus. The clinical course and symptomology strongly indicate that the esophageal-carotid fistula is the primary cause of abscess formation. However, we must also consider the potential contribution of the endovascular intervention to the pathogenesis. It is plausible that the introduction of coils into a contaminated environment, due to the esophageal-carotid fistula, may have accelerated the development of cranial abscesses, which sets this case apart from the 2 previous cases. The fact that both the abscesses and endovascular intervention were on the right side further supports the suggestion that the abscesses were a sequalae of contaminated devices. The origin of these abscesses is likely multifactorial as the patient had significant radiation exposure, newly developed ischemic strokes, tumor invasion around the esophagus, and underwent a procedure that puts the patient at risk for propagation of infection.

CBS is a recognized, albeit rare, postoperative complication following major head and neck surgeries, affecting 3% to 4.5% of all patients [2]. However, in patients who undergo radiation therapy, the incidence of CBS significantly rises, ranging from

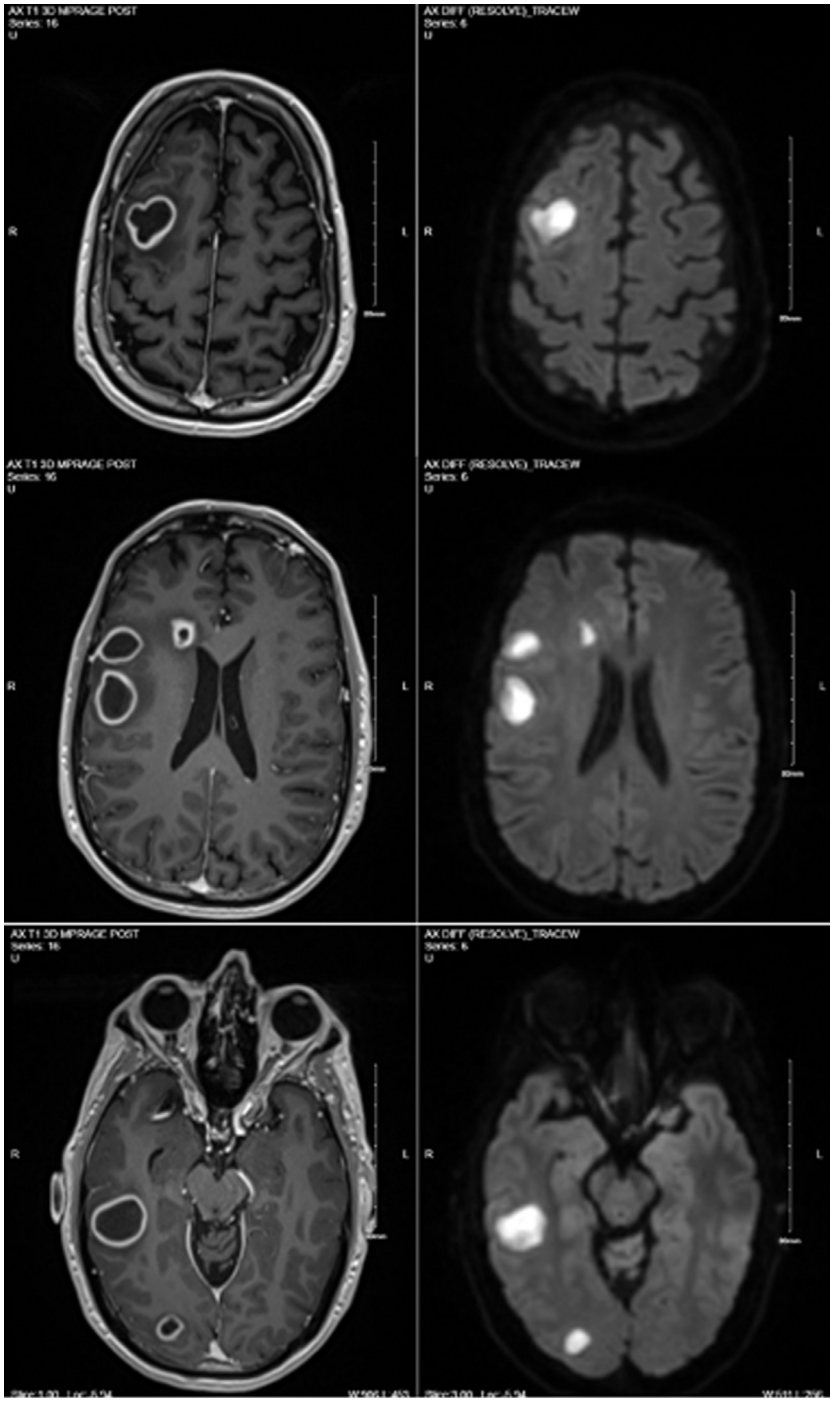

Fig. 5 – Axial MRI T1-weighted images with contrast (left) and diffusion-weighted images (right) showing multiple intracranial abscesses in the right cerebral hemisphere.

4.5% to 21.1%. It is reported that these patients have a 7 times higher risk of developing CBS [2,17]. Despite improved outcomes with the advent of endovascular repair, 1 study showed that there is still an in-hospital mortality rate of 8.2% with coil embolization showing better outcomes than carotid stenting [7]. In this same study by Brinjikji and Cloft [7], outcomes of endovascular blowout repair for 1218 patients were examined. The most common severe postoperative complications were acute ischemic stroke (2.4%), hemiplegia/paresis (1.7%), and hemorrhage (2.0%). To avoid the serious complications associated with CBS, there is a need for more case reports to compile the experiences of various institutions and provide optimal care for future patients.

The initial management of an impending CBS requires prompt intervention with interdisciplinary management. Endovascular repair of a threatened CBS (type II) is necessary as development to acute carotid system hemorrhage (type III) is rapidly fatal. Long-term prophylaxis antibiotics is typically not

recommended in vascular and interventional radiology procedures but rather only in cases that are deemed high risk [18,19]. In a retrospective review of patients undergoing intervention for CBS with underlying head and neck cancer, it was found that only 5% of patients received long-term antibiotic therapy [5]. The use of long-term broad-spectrum prophylactic antibiotics should be considered for patients undergoing CBS repair.

Patient consent

The patient in this report provided informed consent for describing and publishing their clinical details.

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