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Carotid artery blowout producing massive hematemesis in the emergency department

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ABSTRACT

Carotid blowout syndrome (CBS) is a rare and fatal complication which arises from patients who have been treated for head and neck cancer. The incidence of CBS is rare and not commonly seen by emergency physicians. We review a case of a 68-year-old woman with a history of laryngectomy and chemo-radiation therapy presenting with massive oral bleeding and hypotension. Her course and treatments are highlighted, literature referring to CBS are described and we reintroduce the approach of managing such a patient in the emergency department.

1. Introduction

Carotid blowout syndrome (CBS) is rare and not commonly seen in the emergency department (ED). However, when it presents, a prompt recognition is paramount for the patient's survival. CBS or rupture of the carotid artery was described in 1962 by Borsany and further defined as either an acute hemorrhage or exposure of any portion of the carotid artery[1]. This syndrome occurs in 3%–4% of all head and neck cancer (HNC) patients, and it has been estimated previously that there is a 40% mortality and 60% devastating neurologic morbidity associated with CBS[1,2].

To date, the endovascular treatment of CBS has been described in case reports, case series and retrospective studies. However, there are no current cases reported in the emergency medicine literature describing this syndrome or how to manage such a patient in the ED. We aim to reintroduce and inspect a case of life-threatening hemorrhage caused by common carotid artery (CCA) blow out syndrome and discuss the ED care of such a patient.

2. Case report

A 68-year-old woman presented with a 1 h history of nausea followed by multiple episodes of vomiting blood. She arrived by the ambulance and immediately upon her evaluation, the patient was pale, hypotensive and had several continuous episodes of bright red blood coming from her mouth and nose.

She had an extensive history of laryngeal cancer treated initially with partial laryngectomy and chemo-radiation treatment 2 years prior in Venezuela. She lost contact with her physician, then moved to the United States and began feeling ill earlier in the year. She was treated for a recurrence of her laryngeal cancer at an outside institution with a total laryngectomy 4 months prior to, and finished her last chemo-radiation treatment 3 weeks prior to her presentation at our hospital.

Her initial vitals included a pulse of 94, blood pressure of 81/51 and an oxygen saturation of 99% on room air. On exam, old blood was present in her bilateral nares and oral cavity, a clear and patent trachea stoma was visualized and her neck revealed palpable bilateral carotid pulses without any open wounds. The patient proceeded to forcefully retch and vomit large amounts of bright red blood during our evaluation.

We quickly placed a tracheostomy tube into her stoma with the balloon maximally inflated, established intravenous (IV) access, and immediately ordered 2 units of O negative blood for

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transfusion. Ears, nose and throat along with gastrointestinal services were requested for endoscopy to locate this unidentifiable source of bleeding. The ED together with ears, nose and throat service proceeded to pack the patient's oral pharynx with Kerlix gauze and applied digital pressure in hopes to blindly stop the bleeding. A hemoglobin and hematocrit of 7.9/25.4 returned while the remainder of her blood count, chemistries and coagulation studies were normal.

Further history obtained from the patient's outside treating oncologist suggested a high suspicion for carotid artery involvement from her cancer; therefore, neuro-interventional radiology (NIR) was consulted with the urgent decision to take the patient to the catherization lab. Prior to catherization suite transfer, the patient's condition deteriorated quickly as the bleeding became more persistent. The blood pressure steadily dropped to 63/42, and the patient's heart rate climbed to 110 despite transfusion of 4 units of packed red blood cells.

Next during angiography, a carotid blowout of 2.4 cm proximal to the bifurcation was found, causing a large 1.8 cm pseudoaneurysm with a fistula communicating to the right hypopharynx (Figure 1).

The patient then passed a balloon occlusion test of the right CCA, and subsequently underwent successful embolization of the fistula tract, pseudoaneurysm and parent CCA using multiple coils (Figure 2). During the procedure, the patient required 16 units of packed red blood cells, 3 units of fresh frozen plasma and 2 units of platelets for additional stabilization, but suffered no further complications.

On follow up, her hospital course became mildly complicated with the site of a wire protruding in her oral cavity as well as some passage of wires in her stools. These were confirmed by NIR to be a few of the coils used during her treatment to occlude the pseudoaneurysm. There was no further hemorrhaging during her hospital course, and the patient was discharged on post operative day 14 without any neurologic co-morbidities, and did well.

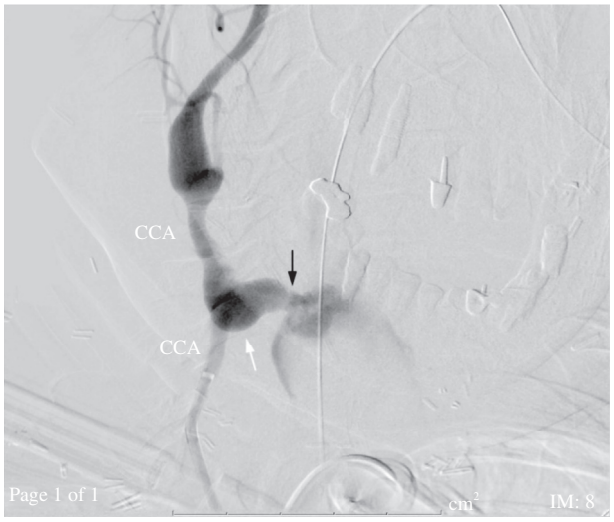

Figure 1. Digital subtraction angiography revealing a right CCA pseudoaneurysm (white arrow) with a fistula tract (black arrow) resulting in bleeding into the hypopharynx.

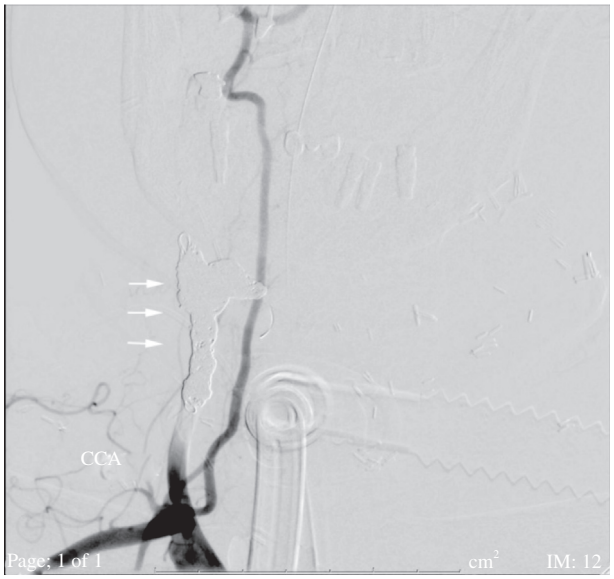

Figure 2. Digital subtraction angiography showing the right CCA after deployment of multiple coils (white arrows) producing complete occlusion of the fistula, pseudoaneurysm and CCA.

3. Discussion

Carotid artery blowout or rupture of the CCA are all used synonymously to describe a very devastating complication of HNC patients and treated with surgery or radiation therapy. It is called a syndrome as there is a clinical progression of this entity and can be further classified into three groups: threatened or type I, impending or type II and acute carotid blowout or type III[1,2].

Threatened carotid blowout signifies a breakdown of the wound from a previous neck dissection or movement of previous flap which exposes the carotid artery. Therefore, if the exposed carotid is not covered with healthy vascular tissue it will rupture[1,2]. There may also be evidence on imaging showing a non-bleeding pseudoaneurysm, or tumor invasion of the carotid artery[1]. Hemorrhage has not yet occurred in type I.

Impending carotid blowout describes patients with a short course of acute hemorrhage which resolves on its own or with surgical packing. Hemorrhages usually occur through a surgical wound or fistula. Since these hemorrhages are episodic, they are considered sentinel bleeds[1,2]. These bleeds may also be controlled by applied pressure.

The third type described in CBS is acute carotid blowout, portraying patients having profuse bleeding which does not resolve spontaneously and cannot be controlled with packing alone. The CCA has ruptured and the patient is in need of urgent intensive resuscitation and interventional care[1,2]. Our patient described above best falls into this category as bleeding continued despite packing and required necessary repair of her ruptured CCA.

The most common etiology is squamous cell carcinoma of the head and neck along with its treatments such as radiotherapy and/or surgical neck dissection[1–3]. As Cohen and Rad[3], explained, radiation has been implicated to obliterate the vasa vasorum, causing fibrosis of the adventitia, weakening the arterial wall, producing edema of the subendothelium, and fragmenting the tunica media elastic fibers. Other risk factors for CBS include wound infection, recurrent tumor invasion,

flap necrosis, lack of supporting healthy tissue, mucocutaneous fistula and penetrating or blunt neck trauma[1–3].

The gold standard for diagnosing CBS is angiography although there is an overall 8.5% complication rate which includes stroke[4]. In patients presenting with threatened and non-hemorrhaging impending CBS, a CT angiogram of the head and neck is reasonable to evaluate the carotid circulation up to the circle of Willis[2–5]. Following diagnosis, there are a number of available treatment options. Traditional surgical ligation had been the only choice with a displeasingly high rate of neurologic morbidity and mortality. However, CBS's main treatment has shifted to endovascular techniques: either deconstructive due to occlusion/embolization of the vessel or reconstructive resulting in a repaired stented patent artery[1,5]. Should occlusion be considered as treatment by the use of deployable balloons or coils, NIR firstly performs a temporary balloon occlusion test in attempt to identify those high risk patients who may develop an acute or delayed cerebral ischemic event[1,5,6]. If the test fails, the vessel is preferably repaired by using intravascular stents[1,3,5–7]. Lesley *et al.*[7], describes a 15%–20% rate of acute or delayed cerebral ischemia following occlusion and offers as an alternative, endovascular reconstruction with stents in patients who are at high risk for cerebral ischemia such as those who have an incomplete circle of Willis, occluded contralateral CCA, or fail a balloon occlusion test to name a few.

In the HNC population with previous surgery or radiotherapy, a high index of suspicion must be maintained for CBS in patients presenting with any recent history of oral bleeding or hemorrhaging from an exposed neck wound. Once CBS is considered by the emergency physicians, and in cases of acute hemorrhage such as type II and III CBS, NIR must be contacted immediately as they advocated for endovascular management which has an 89% survival rate when compared to the more traditional surgical repair leading to a mortality rate of 40%[1–3,5,6].

At this point, ED care is crucial to bridge patients to endovascular therapy and focuses on aggressive critical care beginning with securing the airway and management of hemorrhagic shock. Our patient's total laryngectomy left a stoma with direct access to her trachea. Therefore, a tracheostomy tube with its cuff hyperinflated was placed to protect her stoma from blood and possibly tamponade bleeding from a tracheoarterial fistula if present. Endotracheal intubation may be reserved for patients without a total laryngectomy. Large bore IVs should be placed in the bilateral antecubital fossae if feasible. Central venous access is also a good alternative if peripheral IVs cannot be obtained. Hemorrhage control is the next priority either by a gloved finger to visible source or with direct packing. Our patient's source was implicated to be coming from the lower pharyngeal area resulting in the packing of the patient's oral pharynx with gauze dressing.

Finally, massive transfusion should be considered in patients who present with hemodynamic instability and uncontrollable hemorrhage. Although 4 units were typed and crossed, for our patient, she needed much more during her procedure as her condition declined. We suggest a 1:1:1 approach of packed red blood cells, fresh frozen plasma and platelets. We would also type and cross match 8 units while transfusing emergent O negative blood to females or O positive to males while waiting on matched blood to arrive[4]. Adherence to these principles can certainly allow for stabilization and acquire more time to allow patients to make it to definitive intervention.

This case demonstrates another rare emergency which can rapidly deteriorate in minutes if prompt action is not taken. Identifying CBS begins with recognition which occurs most commonly in HNC patients. Management includes airway, circulatory, and hemorrhage control in order for the patient to reach endovascular management with vessel occlusion or stenting. We conclude that with an understanding of the progression of this syndrome and firm grasp of the management described, CBS may be safely managed in the ED successfully without immediate complications.

Conflict of interest statement

The authors report no conflict of interest.

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