A LETHAL DRIPPING FROM THE NECK: A CASE REPORT AND MINI REVIEW

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ABSTRACT

Post-actinic arteriopathy is one of the most devastating complications associated with therapy for head-and-neck cancers. We report a case of 67-year-old man presented to local emergency ward with left side neck blood dripping who died in a few hours before any diagnoses and treatment could be realized. Autoptic dissection found a 0,4cm length interruption in left internal carotid artery wall, within a fibrotic scar, which comprise the carotid sinus and its bifurcation. An injection and pumping proof with the water to simulate blood flow was conductedand demonstrated water jar from the left wall carotid artery injury. A diagnosis of acute carotid blowout syndrome was posed as cause of death.

Keywords*: carotid blowout syndrome, head-and-neck cancers, post-actinic arteriopathy, autopsy.*

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Introduction

Post-actinic arteriopathyis one of the most devastating complications associated with therapy for head-and-neck cancers and may manifest itself as a wide spectrum of alteration: occlusion, subocclusive sclerotic or atheromatous plaque, localized mural thrombus, aneurysm, or spontaneous rupture[1]. In the head and neck region, stenosis of the carotid is the most common manifestation of the post-actinic arteriopathy. less commonly,rupture of the carotid artery associated to hemorrhage and/or exposure of a segment of the carotid artery in a patient who had underwent aggressive management for head and neck cancer is called carotid blowout syndrome[2-9]. Symptoms usually occur from 1 to 10 years after successful radiation therapy but sometimes as much as 2-3 decades later[1, 10-16]. The morbidity and mortality rates, complicating this pathology, are respectively 40% and 60%[9].

Case report

We report a case of 67-year-old man, who came to sanitary observation with a wound at the left side of the neck from which massive blood loss occurred. In a short time and without the healthcare staff could post any treatment, the patient died.

At the external examination, in correspondence of left mastoid region, below the helix, a nodular formation, oval-shaped, of elastic-hard consistency, about 3x1,6cm, covered by bluish skin was found. In the context of this bump, there was a wound, round-shaped. Another similar lesion was present as a crateriform wound of 0,2cm diameter. (fig. 1-a) Other four similar lesions were valuable on the left-side lateral surface of the neck skin, each one of different morphology and dimension. (fig. 1-b) Always at mastoid level originated a surgical scar, linear, 11-cm-long, finishing in the sovraclavearhomolateral region.

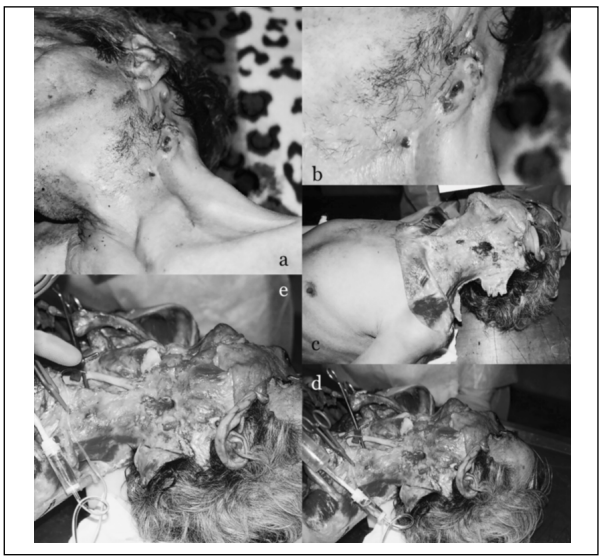

Fig. 1: **a-Externalexamination**: a crateriform wound of 0,2cm diameter at the left side of the neck from which massive blood loss had occurred;
b-Externalexamination: other four crateriform lesions were valuable on the left-side lateral surface of the neck skin, each one of different morphology and dimension; **c-Autopticalfinding**: presence of small hemorrhagic areas at the left anteroposterior laterocervical region, limited to the sole fascial layer with a wound, crater-shaped, through which the wall of the left carotid artery was seen; **d-Injection and pumpingproof**: after the solation of the arterial and venous tributary vessels of the left side was performed, the common left carotid artery was canulated, at the marginal margin, with a butterfly needle attached to a syringe; **e-Injection and pumpingproof**: In order to simulate the blood flow, water was injected and pumped in the vessel was demonstrating a water jar from the left wall carotid artery injury.

Clinical history of the man revealed that, 4 years earlier, he was hospitalized for the presence of a left laterocervical tumor that progressively increases in volume during past months. During the hospitalization, the healthcare providers performed a CT examination of the neck region which detected the presence of a partially liquid neoformation that embedded the nerve-vascular bundle of the neck. He was subjected to left-cervical surgery with removal of neoformation, as well as enrolled in subsequent radiotherapy.

During autoptic dissection, the area of the left anteroposterior laterocervical region was bared, showing the presence of small hemorrhagic areas at the lesions described in the external examination, limited to the sole fascial layer. (fig. 1-c) There was, furthermore, a wound, crater-shaped, which let us see the wall of the left carotid artery. Then, isolation of the arterial and venous tributary vessels of the left side was completed and the inculcation of the common left carotid artery was performed, at the marginal margin, by a butterfly needle attached to a syringe, suitably filled with water, in order to simulate the blood flow. At the injection and pumping of the liquid in the vessel was observed water jar from the left wall carotid artery injury. (fig. 1-d-e)

The left carotid-heart block was removedand fixed in formalin. Once examined showed that left internal carotid artery, at 11 cm from its aortic arch root, at level of the glomus, was enveloped by fibrotic scar, comprising the carotid sinus and its bifurcation, over which, on its lateral wall, there was a 0,4cm length interruption, with facing-out margins.

Functional examination, made by means of syringe cannulation of the vessel filled with methylene blue, confirmed the autoptic finding, showing a spurt throughout vessel laceration.

At the end, histopathology confirmed the presence of a cystic degenerated node that can be attributed to a squamous cells carcinoma metastasis.

In this case hemorrhagic shock, due to spontaneous rupture of the left internal carotid artery, was the cause of death, in a patient affected by squamous cell carcinoma of the neck with node metastasis.

Discussion

Radiotherapy represents, nowadays, the primary treatment choice for nasopharynx cancer, with excellent long-term results achieved in most patients without metastatic diseases, thanks to the advancement of radiation techniques, even combined with chemotherapy[17-21]. Simultaneously, late treatment toxicity has become a relevant healthcare concern among survivors[22-23]. Indeed, during the last years a wide number of reports have documented the relationship between irradiation and post-actinic damage to the carotid vessels, usually bilaterally, in patients with head and neck cancer, such as squamous cell carcinoma or lymphoma, both treated with radiotherapy or not[24-31]. However, these prooves only came from retrospective studies being deficient of information about pretreatment carotid artery wall status.

Though Gleysteenet al.[32] stated the etiologies of carotid blow-out syndrome in inclusion into the context of head and neck cancer and subsequent ulceration of the vessel wall, tissue necrosis after surgery or radiotherapy, blunt trauma or penetrating wound of a thickening vessel wall, and iatro-

genic injury during operation.Moreover, radiotherapy of the neck is believed to accelerate atherosclerosis. Indeed, Gianicolo et al.[33] demonstrated that the common carotid artery becomes thick and thick simultaneously to the increasing doses of radiation to the neck and that irradiated young patients showed higher wall damage compared to the non-irradiated patients.Further the thickening of artery walls, post-actinic damage seems to be lead to atherosclerotic plaque formation, decreased flow, and coronary artery stenosis[34-40]. Other complications reported are thrombosis, carotid rupture, and progressive stenosis[2-4, 41-42].

Despite a large number of cases, the exact pathophysiology of post-actinic damage to the vascular wall is still unknown. A possible scenario is subsequent described: a primary lesion affecting the vasa vasorum and peri-adventitial tissues, which causes ischemic lesions of the arterial wall which, in turn, lead to the formation of dense pre-adventitial fibrosis[43-48].

However, this whole atherosclerotic process seems to be accelerated in the irradiated patient compared to the normal process of thickening 21 times quicker than that seen normally per year. From a clinical point of view, instead, carotid blowout syndrome could be classified into three pathological entities: such as threatened, impending, and acute carotid blowout[49-51].Threatened carotid blowout refers to a patient with an exposed carotid artery in the neck, following surgery or tumor, but no history of bleeding[52-54]. Impending carotid blowout refers to a patient with the same physical findings but who have also experienced a self-limited bleeding event from the carotid artery system Acute carotid blowout refers to a patient who presents with active carotid bleed or carotid rupture[55-57]. Within this group belong our case report. In the threatened and impending groups, diagnosis can be difficultly achieved due to the paucity of symptoms claimed or signs showed. In case of the acute carotid blowout, the challenge is represented by the promptness of active management.

In order to provide the most adequate approach to all these conditions, further studies are needed to define the exact mechanism of post-actinic arteriopathy and the relationship between radiation dose and vessel injury.

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