



RADIATION CAROTID BLOWOUT SYNDROME IN NASOPHARYNGEAL CARCINOMA

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Learning objectives:

- Understanding of the pathophysiology and recognition of the clinical manifestations of radiation carotid blowout syndrome (RCBS) in irradiated nasopharyngeal carcinoma (NPC)
- To familiarize with the imaging features of RCBS
- Knowledge of the available treatment options

Background:

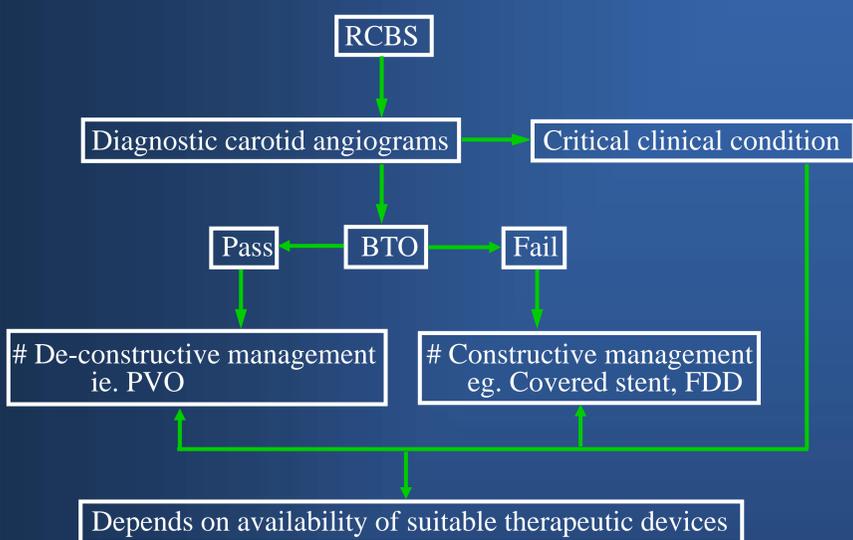
NPC is a common neoplasm in east Asia and high dose radiation remains the primary treatment offering promising results. However, potential radiation complications include xerostomia, otitis media, sinusitis, temporal lobe necrosis, panhypopituitarism, cranial nerve palsy, palatal perforation, brainstem damage and radiation arteritis with carotid stenosis. RCBS occurs as a result of damage to the protective adventitial layer. Wide variety of radiation induced morphological alterations include fibrosis, inflammation, premature atherosclerosis, stenosis and vascular occlusion. RCBS usually presents with sudden catastrophic bleeding in the form of epistaxis which could be a consequence of residual or recurrent disease, neovascularization, pseudoaneurysm, osteonecrosis and soft tissue inflammation.

Imaging findings:

- irregular segmental stenosis with subtle aneurysm
- arterial ballooning
- pseudoaneurysm formation
- free rupture of the carotid artery

Management:

Surgical management is technically difficult as direct exploration and repair of the previously irradiated field is complicated. Treatment paradigm of RCBS is basically determined by the sufficiency of cerebral collateral circulation and therefore balloon test occlusion (BTO) has to be included in the angiographic assessment since upto 15-20% of patients treated with permanent vessel occlusion (PVO) can have immediate or delayed cerebral ischaemia. Off-label use of covered stent and flow-diverting device (FDD) can produce satisfactory results should BTO fail, but long-term safety, stent patency and permanency of haemostasis appear unfavorable with high incidence of delayed complications in particular rebleeding, thrombosis and occlusion. Therefore PVO remains the gold standard of treatment with favorable short- and long-term outcomes.



Algorithm for endovascular management of RCBS

Depending on clinical response, may be treated by bypass in the long run.

Conclusion:

Understanding of the evolution, distribution and pattern of arterial involvement in RCBS along with knowledge of the treatment options can greatly help in the management of this devastating complication.

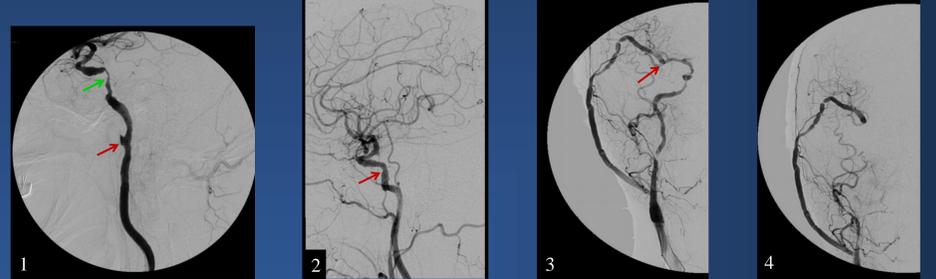
References:

- 1.Luo CB, Teng MMH, Chang FC, et al. Radiation carotid blowout syndrome in nasopharyngeal carcinoma: Angiographic features and endovascular management. Otolaryngol Head Neck Surg 2008; 138: 86-91.
- 2.Wong GKC, Chan KK, Yu SCH, et al. Treatment of profuse epistaxis in patients irradiated for nasopharyngeal carcinoma. ANZ J. Surg. 2007; 77: 270-274.
- 3.Wan WS, Lai V, Lau HY, et al. Endovascular treatment paradigm of carotid blowout syndrome: Review of 8-years experience. Eur J radiol 2011; Feb 8.

Spectrum of imaging findings:

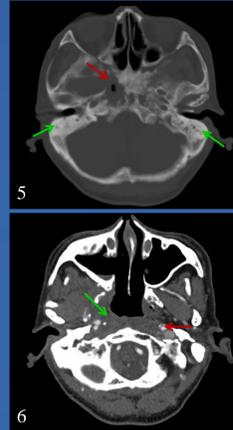
Case 1:

54 year old male with known case of NPC treated with radiation and admitted for sentinel bleeding.



4-vessel cerebral angiogram (Fig 1,2,3 & 4):

Small pseudoaneurysm arising from the anterior wall of the petrous segment (Fig 1 red arrow) with severe irregular narrowing of cavernous segment (Fig 1 green arrow) of the right ICA. Long segment irregularity and stenosis in left ICA secondary to previous radiation therapy (Fig 2 red arrow). Attempts to conduct BTO could not be accomplished as patient complained of severe headache and increased blood pressure as soon as the balloon was inflated. Finally a decision was made to conduct a EC-IC bypass (Fig 3 red arrow) prior to occlusion of right ICA pseudoaneurysm using platinum coils and occlusion of parent artery using Amplatzer occlusion device (Fig 4).

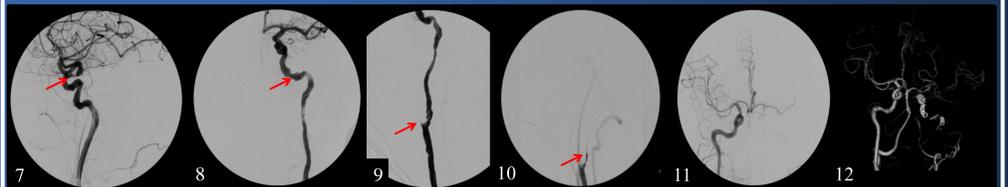


CT scan of the same patient (Fig 5 & 6):

Destruction of the right foramen ovale, foramen spinosum, sphenoid bone and anterior part of the carotid canal (Fig 5 red arrow) with under-aeration and sclerosis of both mastoid air cells (Fig 5 green arrows) secondary to radiation. Arterial wall thickening with calcified plaques (Fig 6 red arrow) in left ICA resulting in irregular luminal narrowing as demonstrated in the 4-vessel cerebral angiogram (Fig 2 red arrow). Soft tissue thickening in the nasopharynx due to NPC (Fig 6 green arrow).

Case 2:

52 year old male with known case of NPC post radiotherapy who presented to the emergency with epistaxis and collapsed.



4-vessel cerebral angiogram (Fig 7,8,9,10 & 11) & CTA (Fig 12):

Dysplastic changes in both ICAs (Fig 7 & 8) with 50% stenosis in right supraclinoid ICA (Fig 7 red arrow), possible pseudoaneurysm in left petrous ICA (Fig 8 red arrow) and occluded left ECA (Fig 9 red arrow). Post embolisation of left ICA (Fig 10 red arrow) there is good cross filling of left MCA and ACA across midline from the right ICA via the Acom (Fig 11 & 12).

Case 3:

55 year old male with known case of NPC post radiotherapy who presented to the emergency with epistaxis and collapsed.



CTA & 4-vessel cerebral angiogram (Fig 13,14 & 15):

Rupture of right ICA at C1 level (Fig 13 & 14 red arrows). 5F Boston balloon was used to obtain proximal occlusion and control of bleeding. Through this catheter an Excelsior 1018 microcatheter was placed past the bleeding point and multiple coils and liquid histoacryl embolic agents were used to obtain occlusion of the right ICA (Fig 15). A second microcatheter (Prowler select) was required to deposit a few final coils to complete occlusion after the residual flow was still present even after the glue injection.

