ORIGINAL ARTICLE

# Endovascular treatment of ruptured internal carotid artery pseudoaneurysms after irradiation for nasopharyngeal carcinoma patients

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**Objectives** To evaluate the efficacy and complications of endovascular treatment for ruptured internal carotid artery pseudoaneurysms following irradiation of nasopharyngeal carcinoma.

**Design** Retrospective case series.

**Setting** Tertiary neurosurgical referral unit of a Hong Kong public hospital.

**Patients** Patients with ruptured radiation-induced internal carotid artery pseudoaneurysms that were treated endovascularly from October 1999 to October 2011 at Queen Elizabeth Hospital were reviewed. Hospital records, imaging, and angiographic data were studied.

**Results** During the study period, 15 such nasopharyngeal carcinoma patients were treated by endovascular means at Queen Elizabeth Hospital. Ten presented with epistaxis, three with otorrhagia (bleeding from the ear), and two with both. Therapeutic occlusion of the affected internal carotid artery was performed in four patients, and stenting of the artery (with or without coil obliteration of the pseudoaneurysm) was performed in 11. Immediate haemostasis was achieved in all cases. One (7%) of the 15 patients endured symptomatic recurrence of the pseudoaneurysm, and in another an asymptomatic residual pseudoaneurysm was noted in the follow-up angiogram. Three patients suffered clinically significant procedure-related complications, including cerebral infarction (n=2) and brain abscess (n=1). In the angiograms obtained after a mean post-treatment interval of 13 (range, 0.7-60) months, the stent patency rate was 67%. All three patients with occluded stents were asymptomatic.

**Conclusions** Ruptured internal carotid artery pseudoaneurysms following radiotherapy is a rare but life-threatening condition. Endovascular treatment by occlusion or reconstruction of the internal carotid artery with stents provides immediate haemostasis and obliteration of the pseudoaneurysms, with a low recurrence rate. Long-term follow-up is necessary to look out for delayed post-treatment complications.

A video of endovascular treatment of ruptured internal carotid pseudoaneurysms is available at <www.hkmj.org>.

**Key words**
Aneurysm, ruptured; Carotid artery, internal; Embolization, therapeutic; Nasopharyngeal neoplasms; Stents

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**New knowledge added by this study**

- Endovascular treatment (occlusion/reconstruction of internal carotid artery [ICA] by stents) offers immediate haemostasis (100%) and a low re-rupture rate (7%) for ruptured radiation-induced pseudoaneurysms.

**Implications for clinical practice or policy**

- Ruptured ICA pseudoaneurysms can be one of the differential diagnoses of epistaxis or otorrhagia in patients with a history of irradiation to head and neck region, for which such endovascular treatment is a reasonable option.

## Introduction

Head and neck cancers are common in Asia, and nasopharyngeal carcinoma (NPC) especially so in southern China, Hong Kong, and Taiwan.[1,2] The primary treatment for NPC is high-dose external irradiation, which achieves satisfactory local control of the disease.[3] Carotid blowout syndrome due to rupture of radiation-induced carotid pseudoaneurysm

## 鼻咽癌患者放射治療後頸內動脈假性動脈瘤破裂接受血管內治療

**目的** 評估鼻咽癌患者放射治療後頸內動脈假性動脈瘤破裂接受血管內治療的療效及併發症。

**設計** 回顧性病例系列。

**安排** 香港一所公立醫院提供第三層醫療服務的神經外科部門。

**患者** 回顧研究於1999年10月至2011年10月期間，分析於伊利沙伯醫院放射治療相關的頸內動脈假性動脈瘤破裂而接受血管內治療的患者。研究有關患者的醫院紀錄、成像和血管造影資料。

**結果** 研究期間，15名有頸內動脈假性動脈瘤破裂的鼻咽癌患者於伊利沙伯醫院接受血管內治療。10人出現鼻出血、3人出現耳出血，另2人同時有鼻和耳出血。有4人接受頸內動脈閉塞治療，另11人接受動脈支架置入術（包括使用及不使用線圈的情況下令假性動脈瘤栓塞）。所有病例均可以立即止血。15名病人中，1人（7%）仍有假性動脈瘤復發的症狀，另1人於跟進血管造影中發現一無症狀的剩餘假性動脈瘤。3人出現與手術相關的併發症，包括腦梗塞（2例）和腦膿腫（1例）。手術治療平均13個月後（介乎0.7至60個月之間），血管造影顯示支架通暢率為67%。所有栓塞支架的患者（3例）均無症狀。

**結論** 放射治療後頸內動脈假性動脈瘤破裂是一種罕見但可致命的情況。血管內動脈閉塞治療或者重建動脈支架置入術可即時止血及閉塞假性動脈瘤，且復發率低。患者必須接受長期跟進以觀察延遲出現的治療併發症。


is an uncommon yet serious life-threatening complication, causing catastrophic bleeding.[4] Endovascular treatment is gaining popularity due to unstable patient haemodynamics after acute rupture. Studies suggest that parent artery occlusion should be the standard treatment, so long as collaterals are shown to be adequate based on the balloon occlusion test.[5,6] In the literature, however, there are only a few reports on the efficacy and choices of endovascular management of radiation-induced carotid pseudoaneurysms. We therefore report our 12-year experience with this procedure for ruptured internal carotid artery (ICA) pseudoaneurysms after radiation therapy, including endovascular arterial occlusion and reconstruction with stents.

## Methods

From October 1999 to October 2011, 15 cases of carotid blowout syndromes in 15 NPC patients were treated endovascularly at Queen Elizabeth Hospital, Hong Kong. One patient was transferred from another regional neurosurgical unit after stabilisation. Their hospital records were retrospectively reviewed. The patients included 12 men and 3 women, with a mean age of 60 (standard deviation [SD], 13; range, 35-78) years. All of them had received prior radiotherapy to the head and neck region; the mean elapsed intervals before presentation of their carotid blowouts was 11 (SD, 6; range, 1-20) years.

The patients presented with epistaxis (n=10), otorrhagia (n=3), or both epistaxis and otorrhagia (n=2). The eight patients who presented with active oronasal bleedings were initially managed by oronasal packing, intranasal Foley balloons, and blood transfusion (to maintain vital signs as necessary). Three patients were intubated for airway protection, and one underwent a crash tracheostomy before transferral to the angiography suite.

Under local (n=5) or general (n=10) anaesthesia, digital subtraction angiography (DSA) was performed via femoral artery catheterization. Angiograms of both carotid and vertebrobasilar arteries were assessed for pseudoaneurysms as well as the intracranial haemodynamics of the relevant circulation. Angiography confirmed the presence of ICA pseudoaneurysm in all 15 patients; seven were on the right and eight on the left, and located at the cervical (n=2), petrous (n=11), and lacerum (n=2) segments. In all the patients, angiography revealed no significant external carotid artery pathology (Table 1).

Therapeutic occlusion of the ICA and pseudoaneurysm was performed in four patients. (Fig 1, Table 1), three of whom presented with acute torrential oronasal haemorrhage and failure of simple oronasal packing. In these patients, the endovascular occlusion achieved immediate haemostasis. The other patient presented with on-and-off bleeding and underwent occlusion after passing the balloon occlusion test. In all four patients, haemostasis was achieved by deployment of coils to the ICA segment proximally and at the level of the pseudoaneurysms. Crossflow from the contralateral circulation through anterior communicating artery or posterior circulation through posterior communicating artery was observed after contrast injection into the contralateral ICA and ipsilateral vertebral artery. Before proceeding to ICA occlusion, the balloon occlusion test was performed for patient No. 8, and the digital compression test was applied to patient No. 15. The balloon occlusion test was performed under local anaesthesia, deploying a balloon catheter coaxially after four-vessel angiography. Occlusion was confirmed by angiography and the patient was monitored clinically for new neurological deficits. After 10 minutes, intravenous antihypertensive medication was given to further lower the systolic blood pressure for 10 to 20 minutes. The balloon was deflated immediately if there was any neurological deterioration. Angiographic venous drainage delay within 2 seconds was accepted as adequate proof of collaterals during balloon occlusion.

TABLE 1. Patient characteristics, presentations, pseudoaneurysm features, endovascular interventions*

| Patient No. | Sex | Age (years) | NPC before (years) | Presentation | | Anaesthesia | ICA segment | Methods | Materials |
|---|---|---|---|---|---|---|---|---|---|
| 1 | M | 53 | 7 | Epistaxis | Acute | GA | Petrous | Occlusion | 6 Coils |
| 2 | M | 35 | 1 | Epistaxis and otorrhagia | On-and-off | GA | Petrous | Stent | 1 Coronary stent |
| 3 | M | 62 | 14 | Epistaxis | On-and-off | GA | Petrous | Stent + coil | 1 Coronary stent, 3 coils |
| 4 | M | 43 | 3 | Epistaxis | Acute | GA | Petrous | Stent + coil | 1 Coronary stent, 4 coils |
| 5 | M | 73 | 10 | Epistaxis | On-and-off | GA | Petrous | Stent + coil | 1 Coronary stent, 1 coil |
| 6 | M | 69 | 20 | Epistaxis | Acute | GA | Petrous | Occlusion | 6 Coils |
| 7 | M | 67 | 9 | Otorrhagia | On-and-off | LA | Petrous | Stent | 1 Covered stent |
| 8 | F | 50 | 13 | Epistaxis and otorrhagia | On-and-off | LA | Petrous | Occlusion | 3 Coils |
| 9 | M | 61 | 6 | Epistaxis | On-and-off | LA | Petrous | Stent + coil | 1 Wingspan stent (Boston Scientific, Natick [MA] US), 1 Wallstent (for concomitant carotid stenosis), 3 coils |
| 10 | M | 78 | 17 | Otorrhagia | Acute | LA | Cervical | Stent | 4 Pipeline embolisation device |
| 11 | F | 48 | 14 | Epistaxis | Acute | GA | Lacerum | Stent | 2 Pipeline embolisation device |
| 12 | M | 71 | 19 | Epistaxis | Acute | GA | Petrous | Stent | 2 Pipeline embolisation device |
| 13 | M | 57 | 8 | Epistaxis | Acute | GA | Lacerum | Stent | 2 Pipeline embolisation device |
| 14 | F | 75 | 13 | Otorrhagia | On-and-off | LA | Petrous | Stent | 2 Pipeline embolisation device |
| 15 | M | 61 | 5 | Epistaxis | Acute | GA | Cervical | Occlusion | 2 Coils |

* NPC denotes nasopharyngeal carcinoma, ICA internal carotid artery, GA general anaesthesia, and LA local anaesthesia

In all, 11 patients underwent stenting for reconstruction of the affected ICA (Fig 2), four of whom underwent stent-assisted coiling of the pseudoaneurysm. Antiplatelet agents were given before or on the day of stenting to nine (82%) of the patients, and included aspirin only (18%), clopidogrel only (36%), or both (27%). Antiplatelet agents were given to all the patients after stenting, and included aspirin only (27%), clopidogrel only (27%), or both (45%). A simplified algorithm of management is outlined in Figure 3.

## Results

Results and follow-up findings are shown in Table 2. Immediate cessation of haemorrhage was achieved by endovascular means in all patients who failed haemostasis by simple packing. All pseudoaneurysms were either occluded or significant contrast stasis was demonstrated within the aneurysm sac at the end of procedure. There was one patient with concomitant carotid stenosis at the upper cervical portion of the ICA, for which a Carotid Wallstent (Boston Scientific, Natick [MA], US) was deployed. Patients were last followed up after embolisation at a mean of 22.4 (range, 0.4-134) months. After embolisation, DSA was performed in 60% (n=9) of the cases at a mean of 13 (range, 1-60) months.

One (7%) of the patients had symptomatic recurrence of the pseudoaneurysm and one had an asymptomatic residual pseudoaneurysm revealed in the follow-up DSA. Patient No. 5 had a history


FIG 1. Patient No. 15: (a) Anteroposterior (AP) view of the left internal carotid angiogram showing a pseudoaneurysm at the distal cervical portion. (b) On right internal carotid artery (ICA) injection with compression of the left common carotid artery, cross-flow from the right ICA through the anterior communicating artery and bilateral patent A1 is observed. (c) Non-subtracted film, AP view, showing the column of coils in the left ICA. (d) Immediate post-coiling left internal carotid angiogram, AP view, showing no opacification of the distal left ICA, including the pseudoaneurysm

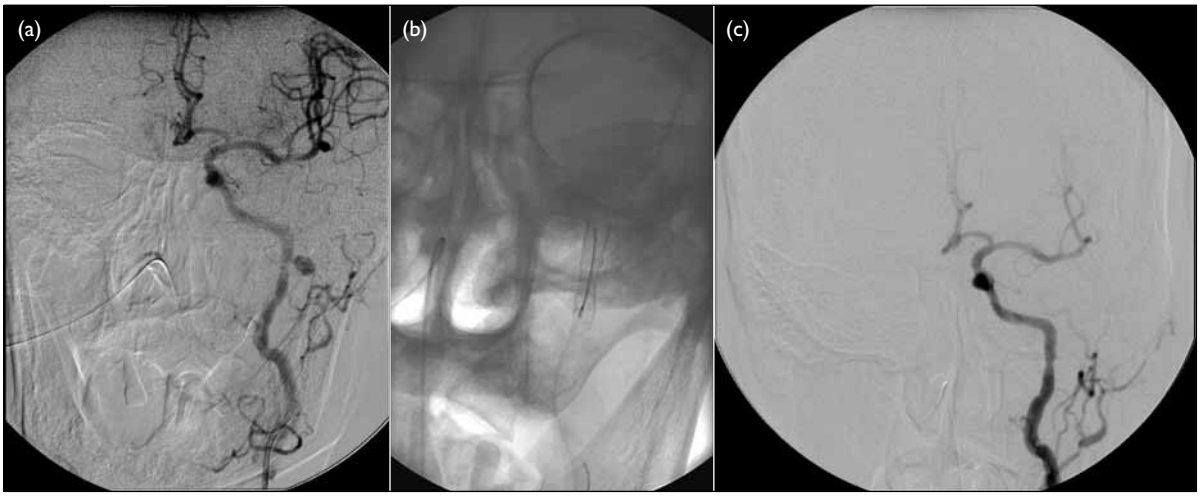


FIG 2. Patient No. 10: Anteroposterior view of (a) left internal carotid angiogram showing a pseudoaneurysm at the cervical portion, with three pipeline embolisation devices deployed across the aneurysm, (b) non-subtracted film showing the position of stents in the left internal carotid artery (ICA), and (c) post-stenting 1-month left internal carotid angiogram showing complete obliteration of aneurysm and patent ICA

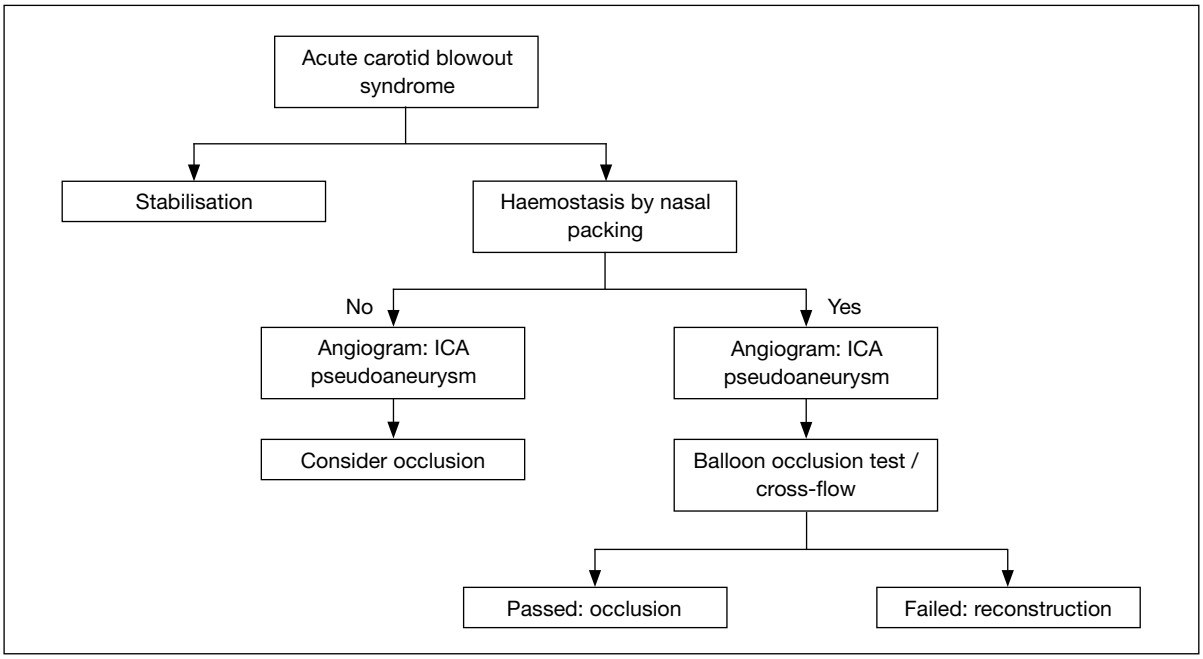


FIG 3. Algorithm for the management of patients with acute carotid blowout
ICA denotes internal carotid artery

of external radiotherapy for an NPC in 1992, which was followed by brachytherapy for local tumour recurrence in 2000. He presented with on-and-off epistaxis, and DSA in June 2002 showed a 3 x 8 mm aneurysm at the left ICA petrous segment. Clopidogrel 75 mg daily was given 3 days before embolisation, and the aneurysm was obliterated with a 4 mm x 10 cm Guglielmi detachable coil–18, assisted by a 4 mm x 16 mm Express Stent (Boston Scientific, Natick [MA], US) for reconstruction of the ICA. Immediate postoperative DSA showed no filling of the pseudoaneurysm. One month later, he was admitted for torrential epistaxis controlled by nasal packing; DSA showed coil compaction and contrast filling of the pseudoaneurysm, which was packed with additional coils. Nine months after initial embolisation, patient No. 2 was shown to have a small residual pseudoaneurysm at the petrous ICA but remained stable and asymptomatic.

The procedure-related complication rate was 40% (n=6). Two patients with obliterated ICAs endured cerebral infarcts with poor neurological recovery. Patient No. 6 underwent occlusion of the ICA without a balloon occlusion test as he was haemodynamically

TABLE 2. Follow-up data and complications*

| Patient No. | FU (months) | Cause of death | Clinical recurrence | Complications | FU angiogram (months) and relapse on imaging | | Stent patency |
|---|---|---|---|---|---|---|---|
| 1 | 5.9† | Sepsis | Nil | Nil | N/A | - | - |
| 2 | 11.0† | Meningitis | Nil | Nil | 8.7 | Small residual | Yes |
| 3 | 133.8 | - | Nil | Nil | 59.8 | Nil | Yes |
| 4 | 3.3† | Tumour progression | Nil | Nil | N/A | - | N/A |
| 5 | 1.8† | Meningitis | Epistaxis 1 month later | Nil | 1.0 | Coil compaction | Yes |
| 6 | 7.1† | Pneumonia | Nil | Right ICA territory infarct | N/A | - | - |
| 7 | 3.8† | Pneumonia | Nil | Left temporal abscess | 2.8 | Nil | Yes |
| 8 | 97.6 | - | Nil | Nil | N/A | - | - |
| 9 | 11.3† | Myocardial infarction | Nil | Nil | 11.1 | Nil | No (ICA occluded, asymptomatic) |
| 10 | 2.4† | Pneumonia | Nil | Nil | 0.7 | Nil | Yes |
| 11 | 35.6 | - | Nil | Nil | 25.6 | Nil | No (ICA occluded, asymptomatic) |
| 12 | 2.3† | Pneumonia | Nil | Nil | N/A | - | N/A |
| 13 | 11.8 | - | Nil | Nil | 3.3 | Nil | No (ICA occluded, asymptomatic) |
| 14 | 7.8 | - | Nil | Nil | 3.8 | Nil | Yes |
| 15 | 0.4† | Myocardial infarction | Nil | Left MCA territory infarct | N/A | - | - |

* FU denotes follow-up, ICA internal carotid artery, MCA middle cerebral artery, and N/A follow-up angiogram not done
† Death at last follow-up

TABLE 3. Reports of destructive treatment for ruptured common carotid artery or internal carotid artery pseudoaneurysms

| Study | Total No. of patients | No. of patients underwent destructive procedure | Immediate haemostasis | Rebleed | Procedure-related mortality | Procedure-related complications |
|---|---|---|---|---|---|---|
| Chaloupka et al,[8] 1996 | 6 | 6 | 100% | 0 | 0 | 1 (17%) |
| Luo et al,[16] 2006 | 16 | 16 | 100% | 0 | 0 | 3 (19%) |
| Luo et al,[17] 2008 | 11 | 11 | 100% | 0 | 0 | 3 (27%) |
| Chang et al,[7] 2008 | 24 | 13 | 100% | 3 (23%) | 0 | 2 (15%) |
| Patsalides et al,[18] 2010 | 7 | 3 | 100% | 0 | 0 | 1 (33%) |
| Powitzky et al,[4] 2010 | 6 | 4 | 100% | 0 | 0 | 0 |
| Wan et al,[5] 2013 | 14 | 10 | 100% | 1 (10%) | 0 | 4 (40%) |
| Zussman et al,[6] 2012 | 8 | 8 | 100% | 1 (13%) | 1 (12.5%) | 2 (25%) |
| Wong et al,[19] 2010 | 11 | 11 | 100% | N/A | 0 | 0 |
| Present study | 15 | 4 | 100% | 0 | 0 | 2 (50%) |

TABLE 4. Reports of reconstructive treatment for ruptured common carotid artery or internal carotid artery pseudoaneurysms

| Study | Total No. of patients | No. of patients underwent reconstructive procedure | Immediate haemostasis | Rebleed | Procedure-related mortality | Procedure-related complications |
|---|---|---|---|---|---|---|
| Cheng et al,[20] 2001 | 2 | 1 | 100% | 0 | 0 | 0 |
| Auyeung et al,[21] 2003 | 2 | 2 | 100% | 0 | 0 | 0 |
| Chang et al,[22] 2007 | 6 | 6 | 100% | 2 (33%) | 2 (33%) | 1 (17%) |
| Chang et al,[7] 2008 | 24 | 11 | 100% | 5 (46%) | 0 | 5 (46%) |
| Ellens et al,[23] 2010 | 1 | 1 | 100% | 0 | 0 | 0 |
| Patsalides et al,[18] 2010 | 7 | 4 | 100% | 1 (25%) | 0 | 0 |
| Powitzky et al,[4] 2010 | 6 | 1 | 100% | 0 | 0 | 0 |
| Wan et al,[5] 2013 | 14 | 4 | 100% | 1 (25%) | 0 | 0 |
| Shah et al,[24] 2011 | 10 | 10 | 100% | 3 (30%) | 0 | 4 (40%) |
| Present study | 15 | 11 | 100% | 1 (9%) | 0 | 4 (36%) [3 asymptomatic] |

too unstable to tolerate the manoeuvre and his severe epistaxis was not controlled by nasal packing. Presence of crossflow from the contralateral ICA and ipsilateral vertebral artery injection was observed before the therapeutic occlusion. Patient No. 15 underwent a compression test of the ICA, which showed presence of contralateral filling of the cortical arteries. Despite the presence of a cerebral infarct, he was asymptomatic. One patient with ICA stenting developed a brain abscess of the ipsilateral temporal lobe 2 months later, and underwent ultrasound-guided drainage and treatment with systemic antibiotics. Moreover, three patients suffered from ICA occlusion of the stents (noted in follow-up angiograms), but remained asymptomatic as elaborated in the next paragraph.

Regarding the 11 stented patients, follow-up DSAs were performed for nine of them. Their stent patency rate was 67%, based on angiograms obtained at a mean of 13 (range, 0.7-60) months. Patients Nos. 9, 11, and 13 showed ICA occlusion with angiograms performed 3 weeks, 1 week, and 3 months later, respectively. Adequate crossflow from the contralateral circulation (via anterior communicating arteries) was demonstrated in all cases, and brain computed tomography showed no infarct. All patients remained asymptomatic after ICA occlusion. No factors—including age, gender, clinical presentation, treatment method, and antiplatelet regimen—correlated significantly with recurrence, complications, or stent patency.

## Discussion

Carotid blowout syndrome is an uncommon yet serious complication following the aggressive management of head and neck cancer. Contributing causes include radical surgery, presence of mucosal flaps following surgery for necrosis or infection, tumour recurrence, and irradiation.[7] All our cases only underwent radiotherapy as treatment for NPC. Thus we assumed that formation of pseudoaneurysms was due to radiation. In patients with head and neck cancers, previous irradiation increases risks of carotid blowout syndrome by 7.6-fold.[8,9] The exact mechanism of radiation-induced vascular injury is not fully understood, but possible causes include obliteration of the vasa vasorum, premature atherosclerosis, and weakening and necrosis of the arterial wall.[10,11]

Acute rupture of ICA pseudoaneurysms can lead to torrential bleeding through the ear, nose, or oropharyngeal space. Due to the deep-seated position of the bleeding source, it can cause non-stop and often life-threatening haemorrhage (from shock or airway compromise). Management should start in the emergency department or at first attendance by medical professionals, and involves stabilising vital functions, and especially airway function. Three of our patients underwent emergency intubation, one of whom was then transferred from another hospital post-stabilisation. Another underwent a crash tracheostomy due to severe oropharyngeal bleeding. Endonasal and ear packing are useful means of initial temporary haemostasis in those who present with acute bleeding.[12]

Emergency surgical ligation of the ICA or common carotid artery was the traditional treatment for carotid blowout syndrome, prior to the era of endovascular intervention, which gave rise to neurological morbidity in up to 60% of the patients and a mortality rate of about 40%.[13] Extracranial to intracranial bypass has also been reported to be successful in treating radiation-induced pseudoaneurysms, especially when recurrent,[14,15] but is technically demanding and not a feasible option for haemodynamically unstable patients seen as an emergency. Endovascular treatment provided a possible alternative with fewer procedure-related complications and better outcomes. Tables 3[4-8,16-19] and 4[4,5,7,18,20-24] summarise the experience of both destructive and reconstructive treatment of acutely ruptured ICA pseudoaneurysms reported in the literature. Immediate haemostasis was achieved in all cases.

Endovascular trapping of the ICA and pseudoaneurysm using coils, glue, or detachable balloons have been reported with high success rates for immediate haemostasis,[6,17,18] yet are associated with a 15 to 20% risk of immediate or delayed cerebral ischaemia.[8,25] The balloon occlusion test is used to assess the tolerance of brain to permanent ICA occlusion.[7] Cross-compression angiographic studies of the carotid artery during a contralateral ICA injection or vertebral artery injection are useful adjuncts, especially in emergencies where adequacy of collateral blood flow can only be determined angiographically.[20] However, test occlusions may miss identifying patient subsets who develop delayed haemodynamic ischaemia following permanent ICA occlusion.[8,9,13] We experienced two patients with cerebral ischaemia after endovascular occlusion, despite adequate crossflow during an occlusion test and angiographic blood flow. Our disappointing experience can be partly explained by the poor clinical status of these patients due to continuing profuse haemorrhage and shock. Assessing clinical tolerance by subjecting such patients to further induced hypotension for up to 30 minutes is a challenge. Caution is therefore necessary when interpreting the above results against those of the standard balloon occlusion tests reported in the literature, which is used as a stratification tool for endovascular trapping.[19] Besides, the contralateral ICA or vertebral arteries may also be included in the radiotherapy field and result in intimal changes and

atherosclerosis. Thus crossflow may not be adequate when one ICA has been occluded. Deployment of coils could stretch and disturb the atherosclerotic vessel lumen, leading to thromboembolic events.

Alternatively, to reconstitute the ICA, pseudoaneurysms can be treated by endosaccular occlusion with coils,[16] sometimes together with stents.[26] However irradiation can cause osteonecrosis of the petrous bone and tissue surrounding the aneurysm becomes ischaemic and friable. The coils can extrude into the nasal cavity or auditory canal with the risk of infection.[27,28] To decrease exposure to foreign materials, stents alone were used to exclude the aneurysm from the circulation. Our initial experience with coronary stents was satisfactory, thus we continued the reconstruction of ICAs with covered stents when they became available. Case reports have suggested that covered stents,[21-23,29,30] and even multiple overlapping stents are feasible.[31] Flow-diverting stents have recently gained popularity for treating ICA aneurysms.[32] Pipeline embolisation devices, used as flow-diverting stents, were primarily indicated for the endovascular treatment of wide-necked intracranial aneurysms of the ICA. Gradual occlusion of intracranial aneurysms by thrombosis was demonstrated, with an initial obliteration rate of 8% to over 90% complete obliteration rate after 6 months noted on follow-up angiography.[33] We extended the indication of flow-diverting devices for the treatment of ruptured ICA aneurysms induced by irradiation as an off-label use, and showed its technical feasibility. Pipeline stents have a 30 to 35% metal surface area when fully deployed,[34] which may not be secure enough to immediately seal off the pseudoaneurysm to secure haemostasis. We therefore deployed them in tandem, ranging from two to four stents, to increase coverage of the vessel surface area for haemostasis purposes. The number of stents used during each procedure was determined by the surgeon and depended on significant stasis of contrast noted within the aneurysm sac. Immediate haemostasis was achieved in all five cases involving Pipeline stents (EV3; Irvine [CA], US). Moreover, we did not experience any recurrence. However, Wan et al[5] showed that flow-diverting stents failed to attain good haemostasis and resulted in delayed in-stent thrombosis leading to massive cerebral infarction. This may have been due to the fact that initially only one Pipeline stent was deployed, as well as differences in the antiplatelet/anticoagulation regimens that were used. Longer follow-up may be necessary to assess development of delayed complications.

Several clinical reports describe the use of stents with or without secondary coiling in the treatment of cerebral aneurysms,[20,30] and their complications include brain abscesses, stent and ICA thrombosis, and embolic events.[35-38] Among our 11 cases, one patient developed a brain abscess, and three endured inadvertent asymptomatic ICA occlusion. However, the development of brain abscess may be due to skull base osteonecrosis rather than a direct complication of stenting. All three patients with occluded ICAs had adequate crossflow and none had neurological deficits. Two of them (67%) did not receive clopidogrel before stenting, whereas 33% of the patent stents did not receive clopidogrel before DSA. Although statistically not significant (P=0.524; possibly due to the small sample size), further studies on the effect of clopidogrel on stenting for radiation-induced pseudoaneurysms appear necessary, as there is no current consensus about use of antiplatelet regimens following such stent deployment. As of now, the current usual practice of giving double antiplatelet agents is adopted from the coronary literature, as it is believed that symptomatic post-procedure thromboembolism in the cerebral circulation is usually associated with inadequate antiplatelet preloading.[7]

Our study was limited by its retrospective nature and the heterogeneous clinical status of our patients, 40% of whom did not have follow-up angiography. The balloon occlusion tests were not performed optimally in our unstable patients, thus degrading the reliability of the results. The numbers and types of stents in use differed across the study period, which prevented meaningful analysis. The small number of patients rendered generalisation of our findings difficult. Moreover, due to retrospective nature of our study, our antiplatelet regimen differed slightly from that reported in the literature.

## Conclusion

Ruptured ICA pseudoaneurysm following radiotherapy of NPC is a rare but life-threatening condition presenting with epistaxis and/or otorrhagia. After initial haemodynamic stabilisation, endovascular treatment by occlusion or reconstruction of the ICA with stents provides immediate haemostasis and obliteration of pseudoaneurysms with a low recurrence rate (7%). Long-term follow-up is needed to look out for delayed complications after reconstruction treatment with stents.

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