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Case Reports & Case Series

Subarachnoid hemorrhage associated with cerebral hyperperfusion syndrome after simultaneous carotid endarterectomy and coronary artery bypass grafting procedures: A case report and review of the literature

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ABSTRACT

Background: Intracranial hemorrhage associated with cerebral hyperperfusion syndrome (CHS) is a potentially devastating complication of carotid endarterectomy (CEA) or carotid artery stenting. Intracranial hemorrhage can comprise of intracerebral hemorrhage or subarachnoid hemorrhage (SAH), but SAH after CEA is rare. We report a case of SAH associated with CHS that followed simultaneous CEA and coronary artery bypass grafting (CABG).

Case description: A 78-year-old man developed left-sided hemiparesis and was admitted to our institution. A preoperative study showed severe stenosis of the right carotid artery associated with markedly reduced cerebral blood flow (CBF), and a CEA was scheduled after initiating medical treatment. However, the patient developed unstable angina requiring an emergency CABG before undergoing an elective CEA. Given the risk of stroke associated with performing CABG alone, simultaneous CEA and CABG were urgently performed. The patient received dual antiplatelet therapy preoperatively and anticoagulation intraoperatively for the CABG procedure, and the anticoagulation was continued postoperatively due to the development of atrial fibrillation. Three days after the surgery, the patient developed a headache and magnetic resonance imaging demonstrated right-sided cortical SAH. Single-photon emission computed tomography revealed a significantly increased CBF. Therefore, the SAH appears to have been associated with CHS after the CEA. The hemorrhage was managed conservatively and resolved without an associated neurological deficit.

Conclusion: SAH after CEA is rare clinical manifestation of CHS. Simultaneous CEA and CABG, or aggressive perioperative antithrombotic therapy, may increase the risk of its occurrence. Early diagnosis and careful management are important for favorable outcomes.

1. Introduction

Cerebral hyperperfusion syndrome (CHS) is a rare but potentially devastating complication of carotid revascularization by carotid endarterectomy (CEA) or carotid artery stenting (CAS), with a reported incidence of less than 3% [1]. CHS is characterized by the development of intracranial hemorrhage and/or clinical symptoms such as ipsilateral headache, seizure(s), or focal neurological deficit(s) as a result of a large

Abbreviations: ACT, activated clotting time; AF, atrial fibrillation; CABG, coronary artery bypass grafting; CAS, carotid artery stenting; CBF, cerebral blood flow; CEA, carotid endarterectomy; CHS, cerebral hyperperfusion syndrome; CT, computed tomography; DAPT, dual antiplatelet therapy; ICA, internal carotid artery; ICH, intracerebral hemorrhage; MRI, magnetic resonance imaging; NIRS, near-infrared spectroscopy; SAH, subarachnoid hemorrhage; SPECT, single-photon emission computed tomography.

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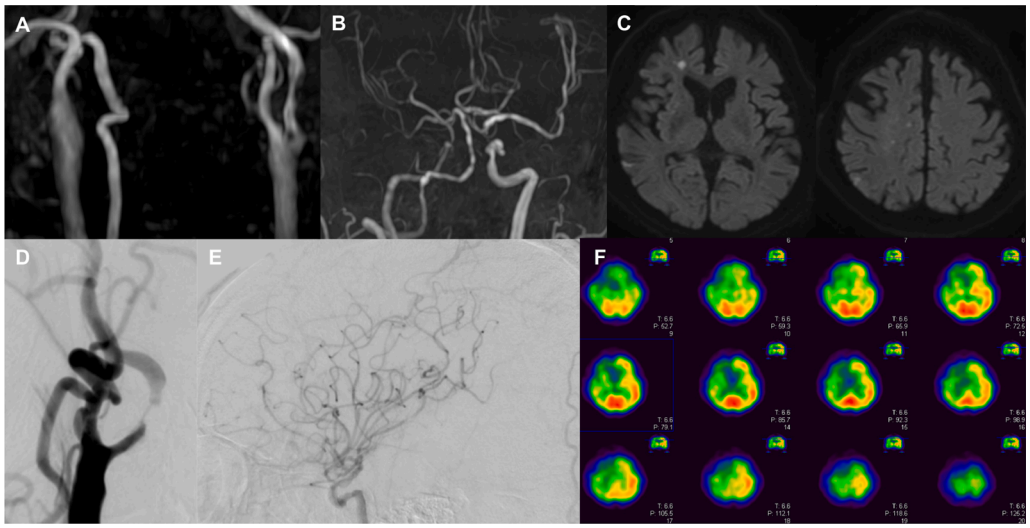

Fig. 1. (A, B) Magnetic resonance angiography performed on admission, demonstrating poor visualization of the right internal carotid artery (ICA) and moderate stenosis at the origin of the left ICA. (C) Diffusion-weighted magnetic resonance imaging performed on admission, showing multiple acute cerebral infarcts in the right cerebral hemisphere. (D, E) Preoperative cerebral angiography showing severe stenosis at the origin of the right ICA and delayed antegrade flow to the right cerebral hemisphere. (F) Preoperative single-photon emission computed tomography scan showing reduced cerebral blood flow in the right hemisphere.

increase in ipsilateral cerebral blood flow (CBF) following carotid revascularization [1–3]. Although the incidence of CHS is relatively low, it can lead to severe neurologic sequelae or life-threatening conditions, especially in patients who develop intracranial hemorrhage [1–3].

Intracranial hemorrhage associated with CHS after carotid revascularization can occur due to intracerebral hemorrhage (ICH) or subarachnoid hemorrhage (SAH) [1,4]. ICH is more common than SAH, accounting for over 80% of intracranial hemorrhages in patients following CEA or CAS [4–10]. Only eight cases of SAH after CAS have been reported in the literature [9,11–16]. SAH after CEA is rarer, and only three cases have been previously described [17–19]. Therefore, there is limited information about the occurrence of SAH as a clinical manifestation of CHS after carotid revascularization procedures, especially in patients undergoing CEA. Furthermore, its underlying mechanisms and clinical implications are unclear.

We describe a patient with severe carotid artery stenosis who developed SAH associated with CHS following simultaneous CEA and coronary artery bypass grafting (CABG).

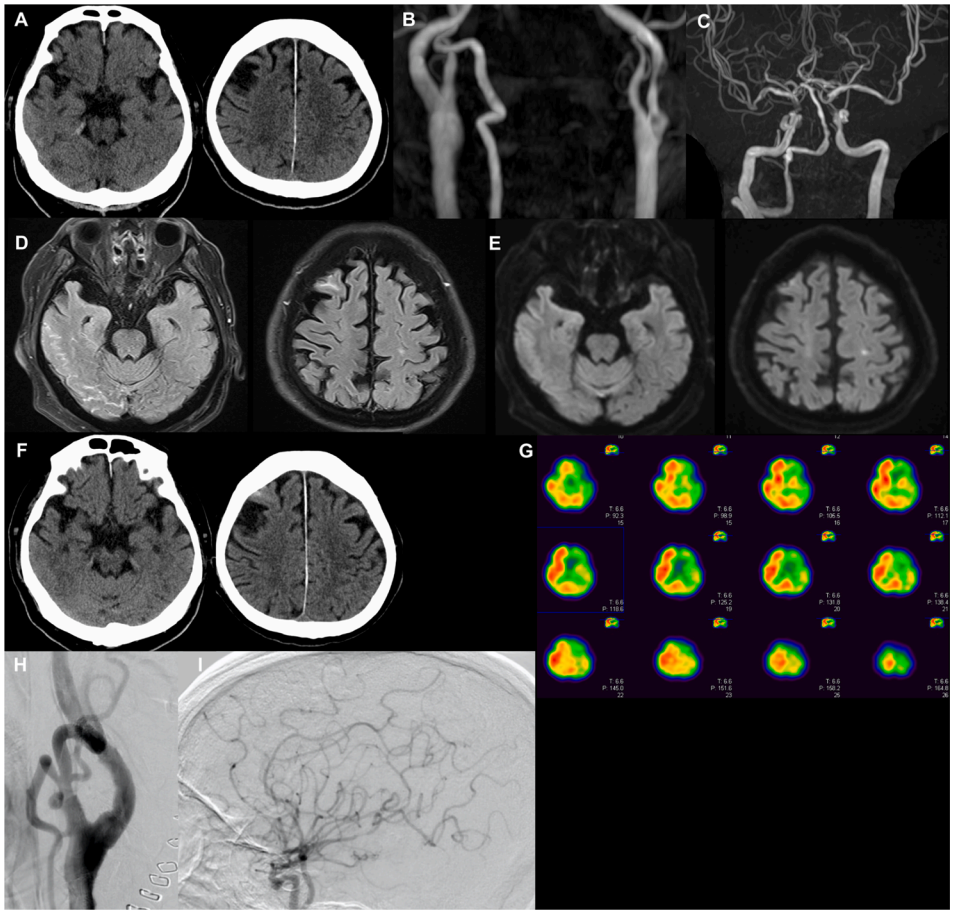

Fig. 2. (A) Postoperative computed tomography (CT) scan performed on the day after the surgery showing no evidence of hemorrhage. (B, C) Postoperative magnetic resonance angiography performed three days after the surgery showing improved opacification of the right internal carotid artery. (D) Postoperative fluid-attenuated inversion recovery magnetic resonance imaging performed three days after the surgery showing subarachnoid hemorrhage in the sulci of the right cerebral hemisphere. (E) Postoperative diffusion-weighted magnetic resonance imaging performed three days after the surgery showing small acute cerebral infarcts in the left cerebral hemisphere, but no obvious increase in acute infarcts in the right cerebral hemisphere. (F) CT scan performed three days after the surgery showing minor subarachnoid hemorrhage in the sulci of the right frontal lobe. CT also showing loss of visualization of the sulci in the right temporal and occipital lobes, which is also shown on the CT performed on the day after the surgery. (G) Postoperative single-photon emission computed tomography scan performed three days after the surgery showing a significant increase of cerebral blood flow in the right cerebral hemisphere. (H, I) Postoperative cerebral angiography showing improvement of the stenosis and intracranial flow, without any vascular abnormalities.

2. Case presentation

A 78-year-old man who developed a mild left-sided hemiparesis was admitted to our institution. He had a history of hypertension, hyperlipidemia, diabetes mellitus, and angina pectoris that had been treated with percutaneous coronary interventions 18 years ago. There was poor visualization of the right internal carotid artery (ICA) on magnetic resonance imaging (MRI), with multiple acute cerebral infarcts in the corresponding vascular territory, as well as moderate stenosis of the contralateral cervical ICA (Fig. 1A, B, C). No obvious hemorrhage was observed on T2*-weighted MRI. Angiography revealed severe stenosis (90%) at the origin of the right ICA with delayed antegrade flow to the right cerebral hemisphere (Fig. 1D, E). Single-photon emission computed tomography (SPECT) showed markedly decreased CBF in the right hemisphere (Fig. 1F). An acetazolamide challenge was not performed due to concerns of inducing ischemia in the acute stage of the hemodynamically unstable stroke. The patient was taking aspirin 100 mg/day before the current presentation. Additional treatment with cilostazol 200 mg/day was initiated and an elective CEA was scheduled for 2 weeks later. However, the patient developed unstable angina pectoris 5 days before the scheduled CEA. Coronary angiography revealed severe 3-vessel disease, which required an emergency CABG. Due to concerns of the patient developing an ischemic stroke with CABG alone, we decided to perform simultaneous CEA and CABG procedures after discussion with experienced cardiac surgeons and neurosurgeons. Written informed consent was obtained from the patient and a member of the patient's family before the procedure.

3. Surgery

The operation was performed on an urgent basis under general anesthesia with near-infrared spectroscopy (NIRS) monitoring. After performing a thoracotomy to determine the desired CABG technique, the CEA procedure was started while harvesting the saphenous vein graft. After the right common carotid, internal carotid, and external carotid arteries were exposed, the arteries were clamped in the presence of systemic heparinization (4,000 U), with the activated clotting time (ACT) maintained at approximately 200 s. A longitudinal arteriotomy was performed from the common carotid artery to the ICA, followed by insertion of a shunt tube to maintain the blood flow to the brain, in line with our routine shunt placement policy for emergency cases. After removing the plaque and closing the arteriotomy, the heparinization was reversed. The clamp times for shunt placement and arteriotomy closure after removal of the shunt were 6 min and 8 min, respectively. The ipsilateral NIRS values decreased by 15% during clamping for the shunt placement and increased after CEA to 10% more than the values obtained before clamping. Subsequently, an off-pump CABG was performed also under systemic heparinization (15,000 U) with the ACT maintained at approximately 300 s. Before closing the wounds, the heparinization was reversed with a total heparin use of 19,000 U during the seven-hour operation.

4. Postprocedural course

After surgery, the patient remained intubated and sedated, maintaining a systolic blood pressure below 120 mmHg. As no apparent hemorrhage was observed on the computed tomography (CT) scan performed the day after surgery, the patient's sedation and intubation were stopped (Fig. 2A). On clinical examination, the patient did not demonstrate any neurological deficits and denied the presence of a headache. The patient's systolic blood pressure was subsequently maintained below 140 mmHg. In addition to taking aspirin 100 mg/day, the patient was commenced on systemic heparinization with the ACT maintained at approximately 200 s for new-onset atrial fibrillation (AF). Three days after the surgery, the patient complained of a right-sided headache, and a cranial MRI scan demonstrated SAH in the sulcus of the right cerebral hemisphere with improved opacification of the right ICA (Fig. 2B, C, D). Small acute cerebral infarcts were observed in the left cerebral hemisphere, but there was no obvious increase in acute infarcts in the right cerebral hemisphere (Fig. 2E). A CT scan revealed a minor SAH in the sulcus of the right frontal lobe (Fig. 2F). In addition, the CT scan showed loss of visualization of the sulci of the right temporal and occipital lobes, which was retrospectively confirmed on a CT scan performed on the day after surgery. This finding was also considered as minor SAH based on the MRI scan findings. A SPECT scan performed on the same day revealed a significantly increased CBF in the right cerebral hemisphere (Fig. 2G). Angiography showed improved visualization of the vasculature but did not show any vascular abnormalities (Fig. 2H, I). Therefore, the SAH was diagnosed as an intracranial hemorrhage secondary to CHS following the CEA. The patient's aspirin prescription was stopped, but the heparinization for AF was continued with the ACT maintained at approximately 200 s. Intensive blood pressure lowering below 120 mmHg was restarted and continued until a follow-up SPECT scan that was performed on postoperative day 10 showed normalization of the CBF. After a follow-up MRI scan also performed on postoperative day 10 confirmed the resolution of the SAH, aspirin 100 mg/day and oral anticoagulant therapy (Edoxaban 60 mg/day) were commenced. The patient was discharged to a rehabilitation facility without any apparent neurological deficits. At the 3-month follow-up, the patient was independent, with no neurological symptoms.

Table 1
Incidence of intracranial hemorrhage (ICH and SAH) after CEA or CAS in previous studies.

Author, Year (Trial)	Procedure	Number of patients	Intracranial hemorrhage		
			Total	ICH	SAH
Barnett et al., 1998[5] (NASCET trial)	CEA	1108	4/1108 (0.4%)	3/1108 (0.3%)	1/1108 (0.1%)
Brott et al., 2006[6,7] (CREST trial)	CEA	1149	4/1149 (0.3%)	3/1149 (0.3%)	1/1149 (0.1%)
	CAS	1123	4/1123 (0.4%)	4/1123 (0.4%)	0/1123 (0%)
White et al., 2006[8] (BEACH trial)	CAS	747	7/747 (0.9%)	6/747 (0.8%)	1/747 (0.1%)
Ogasawara et al., 2007[4]	CEA	1596	6/1596 (0.4%)	6/1596 (0.4%)	0/1596 (0%)
	CAS	2898	21/2898 (0.7%)	19/2898 (0.7%)	2/2898 (0.1%)
Xu et al., 2009[9]	CAS	832	10/832 (1.2%)	8/832 (1.0%)	2/832 (0.2%)
Tietke et al., 2010[10]	CAS	358	5/358 (1.4%)	4/358 (1.1%)	1/358 (0.3%)

ICH, intracerebral hemorrhage; SAH, subarachnoid hemorrhage; CEA, carotid endarterectomy; CAS, carotid artery stenting.

5. Discussion

We described a case of SAH that developed after simultaneous CEA and CABG procedures. This case highlights a rare presentation of SAH as an intracranial hemorrhage associated with CHS following CEA. Simultaneous CEA and CABG procedures or aggressive antithrombotic therapy in the perioperative period can increase the risk of SAH. The prognosis of SAH associated with CHS after CEA could be favorable with early diagnosis and careful management.

Among the two types of intracranial hemorrhage that can occur after carotid revascularization (CEA or CAS), previous studies have identified ICH much more commonly than SAH (Table 1) [4–10]. Furthermore, SAH is less frequent following CEA than following CAS, with a reported incidence of 0–0.1% and 0.1–0.3%, respectively [4–10]. Eight cases of

Table 2
Summary of cases of SAH following CEA or CAS in the literature.

	Author, Year	Age, Sex	Timing of SAH onset	Symptom of SAH onset	Location of SAH	Postoperative CBF study	Management	Outcome	Potential risk factors for SAH
CEA									
1	Dalton, 1992[17]	62, F	5 days	Headache	Diffuse	Not performed	Conservative (not described in detail)	No neurological sequelae	Severe stenosis (90%) Contralateral stenosis
2	Bodenant et al., 2010[18]	74, M	9 days	Headache Seizure	Ipsilateral frontal sulci	Not performed	Antihypertensive Anticonvulsant	No neurological sequelae	Severe stenosis (90%)
3	Thanabalasundaram et al., 2013[19]	66, M	6 days	Headache Seizure	Ipsilateral frontal and temporal sulci	MR perfusion	Antihypertensive Anticonvulsant	Mildly impaired cognitive function	Severe stenosis (99%) Contralateral stenosis
4	Our case, 2021	78, M	3 days	Headache	Ipsilateral frontal, temporal, and occipital sulci	SPECT	Antihypertensive	No neurological sequelae	Severe stenosis (90%) Contralateral stenosis DAPT Heparinization Simultaneous CABG
CAS									
1	Al-Mubarak et al., 2001[11]	61, M	2 h	Headache Nausea	Diffuse	TCD	Antihypertensive	Death	Severe stenosis (90%) Contralateral occlusion DAPT Heparinization
2	Hartmann et al., 2004[12]	77, F	5 h	Loss of consciousness	Diffuse	Not performed	Conservative (not described in detail)	Death	Severe stenosis (95%) Contralateral occlusion DAPT Heparinization
3	Przewlocki et al., 2007[13]	66, M	2 h	Headache Nausea	Diffuse	TCD	Antihypertensive Diuretic	No neurological sequelae	Severe stenosis (95%) Contralateral occlusion DAPT
4	Xu et al., 2009[9]	43, F	0.5 h	Loss of consciousness	Not described	Not performed	Ventricle drainage	Death	Severe stenosis (99%) DAPT Heparinization
5		52, M	7 h	Headache Nausea	Not described	Not performed	Ventricle drainage	No neurological sequelae	Severe stenosis (90%) DAPT Heparinization
6	Okamura et al., 2016[14]	78, F	Just after CAS	Headache Nausea	Ipsilateral Sylvian fissure	SPECT	Antihypertensive	No neurological sequelae	Severe stenosis (85%) DAPT Heparinization
7	Isozaki et al., 2016[15]	64, M	20 h	Attention disorder	Ipsilateral frontal sulci	SPECT	Antihypertensive	No neurological sequelae	Severe stenosis (80%) TAPT Heparinization
8	Nii et al., 2020[16]	81, M	Just after CAS	None	Ipsilateral parietal sulci	SPECT	Antihypertensive	No neurological sequelae	Severe stenosis (95%) DAPT Heparinization Warfarin

SAH, subarachnoid hemorrhage; CHS, cerebral hyperperfusion syndrome; CEA, carotid endarterectomy; CAS, carotid artery stenting; CBF, cerebral blood flow; MR, magnetic resonance; SPECT, single-photon emission computed tomography; TCD, transcranial Doppler; DAPT, dual antiplatelet therapy; CABG, coronary artery bypass grafting; TAPT, triple antiplatelet therapy.

SAH associated with CHS following CAS have been reported, but only three cases after CEA (Table 2) [9,11–19]. These three cases of SAH following CEA and our case are almost similar in terms of the timing of SAH onset, its clinical manifestations, and anatomical location. However, CBF studies were not performed in two of the three previously reported cases and postoperative CBF was evaluated in only one case, using MR perfusion. In our case, a postoperative SPECT scan performed on the day of the SAH symptom onset confirmed a significantly increased CBF in the area that corresponded to the location of the SAH, indicating a strong association between the SAH and CHS. Accordingly, our case suggests that SAH following CEA is a potential clinical manifestation of intracranial hemorrhage associated with CHS following carotid revascularization, although other etiologies and factors may be involved in the development of SAH.

The most accepted pathophysiological mechanism contributing to the development of CHS following carotid revascularization is the

impairment of cerebral autoregulation [1–3]. An acute increase in cerebral perfusion pressure following surgery to the chronically hypoperfused brain tissue is thought to lead to ICH [3]. When reviewing the 11 reported patients with SAH after CEA or CAS and our case, it is apparent that the timing of the SAH onset following CAS tended to be much earlier than that following CEA. This difference in timing of SAH occurrence between CAS and CEA procedures is also observed with ICH-associated CHS [4–10]. Although SAH may have a substantial association with CHS after carotid revascularization as well as ICH, little is known about the underlying mechanisms of the occurrence of SAH.

Potential risk factors for developing SAH in the patients that underwent carotid revascularization are shown in Table 2. All patients had high-grade stenosis preoperatively with or without contralateral lesions, which is consistent with the known risk of CHS in that this could impair cerebral hemodynamic reserve [20,21]. Patients developing SAH after CAS received dual antiplatelet therapy (DAPT) and heparinization as the common antithrombotic therapy for CAS procedures. In our case, DAPT and heparinization were also administered because we performed simultaneous CEA and CABG emergently, instead of the scheduled CEA alone. Previous studies have shown that dual antithrombotic therapy (DAPT or anticoagulation plus antiplatelet therapy) increases the long-term risk of intracranial hemorrhage [22,23]. Aggressive antithrombotic therapy after carotid revascularization, albeit for a short period, may increase the risk of SAH associated with CHS, as was seen in our case.

There is no evidence that performing simultaneous CEA and CABG increases the risk of postoperative intracranial hemorrhage [24–26]. However, as observed in our case, new-onset AF is a postoperative complication of CABG, with a reported incidence of 15–40% after CABG [27,28]. In our case, anticoagulation was administered intraoperatively for CABG and postoperatively for new-onset AF. Therefore, simultaneous CEA and CABG could indirectly increase the risk of SAH due to the postoperative and intraoperative use of heparin. In addition, improvements in cardiac function after CABG might also have influenced the increase in postoperative CBF. In our case, loss of visualization of the sulci was identified on CT imaging even on the day after surgery, which was considered to result from the SAH detected on the MRI scan that was performed later. This finding suggests that minor hemorrhage might have occurred relatively early after the surgery. Minor SAH can be underdiagnosed on CT imaging, but may become more apparent following aggressive antithrombotic therapy. This may explain why aggressive antithrombotic therapy after carotid revascularization can increase the risk of SAH. MRI can be useful for detecting minor SAH, as shown in our case.

Among the previously reported cases of SAH following CEA or CAS, three patients died, but the prognosis was favorable in the other cases. The three patients who died had developed a considerably massive SAH after CAS, within a few hours after treatment. Although the underlying mechanisms are not known, SAH associated with CHS may be more aggressive after CAS than CEA, based on the postoperative course of these reported cases. On the other hands, after CEA, serious complications may be avoided through early diagnosis and careful management, including blood pressure control. SAH after carotid revascularization is rare, but should be considered as a potential presentation of intracranial hemorrhage associated with CHS following carotid revascularization. Further studies are required to clarify the underlying mechanisms of SAH following carotid revascularization and to attempt to prevent its occurrence.

6. Conclusion

We reported a case of SAH associated with CHS following simultaneous CEA and CABG procedures. SAH after CEA is a rare clinical manifestation of intracranial hemorrhage associated with CHS. Simultaneous CEA and CABG procedures and perioperative aggressive antithrombotic therapy may increase the risk of its occurrence. With early diagnosis and careful management, the prognosis of SAH in this setting should be favorable.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Informed consent

Informed consent has been obtained from the patient for publication of the case report and accompanying images.

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