HYPERPERFUSION SYNDROME AFTER SUPRAAORTIC VESSEL INTERVENTIONS AND BYPASS SURGERY

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SUMMARY

Cerebral hyperperfusion and hemorrhage is a recognized complication of endarterectomy, aortocarotid bypass surgery or angioplasty and stenting of the carotid or vertebral artery. We performed 87 balloon angioplasty, stenting and bypass surgery of arteries supplying the brain over the last 15 years. We have found 6 cases of hyperperfusion syndrome in a total of 87 procedures. So the incidence of hyperperfusion was noted as 6.89%. Two cases occurred after two combined procedures in a single sitting. Hyperperfusion syndrome is a well-known complication of craniocervical revascularisation procedures. But with proper care, including strict post procedural control of blood pressure and staged procedure in case of multiples vessel stenosis, these complications can be largely avoided.

Key words: Cerebral hyperperfusion syndrome, carotid stenting, angioplasty, aorto-carotid bypass.

INTRODUCTION

Hyperperfusion syndrome is a well-known dreadful complication of endarterectomy, aortocarotid bypass surgery or angioplasty and stenting of supraaortic vessels supplying the brain. The mechanisms described for intracerebral hemorrhage after these procedures is cerebral hyperperfusion syndrome, preoperative cerebro ischemic event, cerebral infarction and the use of postoperative anticoagulation therapy [4]. Cerebral hyperperfusion syndrome may occur within hours to 3 weeks after carotid artery endarterectomy (CEA), angioplasty and stenting and is characterized by symptoms like headache, seizure, confusion and focal neurological signs due to intracerebral hemorrhage [5]. We report six cases of cerebral hyperperfusion syndrome occurring after balloon angioplasty, stenting and bypass surgery of arteries supplying the brain and try to explain the possible mechanism of hyperperfusion and discuss the measures to avoid them.

OBSERVATIONS

It is retrospective study. On reviewing medical records from 1990 to 2004, we found 6 cases of hyperperfusion syndrome that occurred after a total of 87 (47 carotid/brachiocephalic angioplasty and stenting; 40 carotid endarterectomy/aortocarotid bypass surgery) supraaortic interventions and bypass surgery. Out of six patients four were males and two females in the age group of 24 to 66 years. We strictly follow our poststenting protocol for avoiding hyperperfusion injury. We keep the patient in intensive care unit and maintain the mean blood pressure 10-20% below normal in the post procedure period. We keep the patients on routine antiplatelet agents and continue statins for those patients who were on such treatment prior to intervention. The protocol for these patients are given in table I. Detailed demographic data is given in table II. The details of the recent cases are given below.

Illustrative case reports.

Case 1: this 64-year-old female, a known hypertensive on medication and non-diabetic, presented with a one-month history of right-sided weakness, which was worsening over the last three days. She had a history of right faciobrachial monoparesis and abnormal serum lipid profile. She had right upper motor neuron type of facial palsy, nominal aphasia, right upper and lower limb spasticity along with exaggerated reflexes, grade 0 power in the right upper limb, grade 3 power in the right lower limb and extensor plantar response on the right side. Her neurological deficits were seen on left side. Her
**TABLEAU I. – Protocol for post carotid angioplasty/stenting patients.**

1. To do one vessel at a time
2. To treat the second vessel if required, 6-8 weeks after the first one.
3. To keep the mean blood pressure 10-20% below baseline for 48-72 hours
4. To keep the patient on anticoagulation for 48 hours keeping the INR 1.5
5. Continue the patient on antiplatelets
6. Continue the patients on statins if they are already on or start fresh if the cholesterol level is more than 250mg/dl
7. To keep the mean blood pressure 10-20% below baseline for 48-72 hours
8. To do MRI/CT/MRA scan of brain if the patient develops signs of focal neurological deficits
9. To repeat CT scan of brain if the patient develops headache in post procedure period

CBC was normal. She was on antihypertensive and antihyperlipidemic drugs. The remainder of her systemic examination was within normal limits. CT scan of the head showed infarcts in the left deep middle cerebral artery watershed territory (figure 1). A color Doppler ultrasound study of the neck vessels revealed concentric fibrous plaque causing about 95% area stenosis at the origins of both internal carotid arteries (ICA). There was about 50% area stenosis of the right external carotid artery (ECA) and 95% area stenosis of left ECA was also seen. Both vertebral arteries showed normal antegrade flow.

A digital subtraction angiogram (DSA) of the aortic arch showed normal origins of the aortic arch vessels and the left vertebral artery was seen originating directly from the arch (figure 2). There was 60% diameter stenosis of the proximal right subcla-

**TABLEAU II. – Demographic details.**

<table>
<thead>
<tr>
<th>Obs</th>
<th>Complainants/ Risk factors</th>
<th>Preprocedural CT</th>
<th>Stenosis (DSA)</th>
<th>Procedure</th>
<th>Size of Balloon/ Stent/Graft</th>
<th>Time of onset of first symptom after procedure</th>
<th>Site of bleed in Post procedural CT</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 6y/F</td>
<td>R faciobrachial monoparesis</td>
<td>Infarcts in L deep MCA territory</td>
<td>R ICA: 95% L ICA: 90% R ECA: 50% L ECA: 95% R SCA: 60%</td>
<td>1. L ICA B and St 2. R SCA St</td>
<td>1. Coronary 2.5x14mm B 8mmx21mm Wallstent Maxxum 4x20mm B 2. 6x14mm Express B expandable stent</td>
<td>3 hours (Disorientation)</td>
<td>Large left temporoparietal haematoma with intraventricular extension and hemorrhage in right anterior basal ganglia region.</td>
<td>Died 5 days after procedure</td>
</tr>
<tr>
<td>2 66y/M</td>
<td>Episodic of transient left sided weakness</td>
<td>Normal</td>
<td>R ICA: O L ICA: origin 98% stenosis</td>
<td>LICA B A and St</td>
<td>5x40mm Smash B 8x40mm Easy Wallstent</td>
<td>5 hours (Seizures)</td>
<td>NA</td>
<td>Died 13 hours after procedure</td>
</tr>
<tr>
<td>3 42y/M</td>
<td>Episodes of blurring of vision</td>
<td>Normal</td>
<td>R CCA occlusion</td>
<td>Aorto-Right Carotid bypass surgery</td>
<td>10mm Dacron graft</td>
<td>4 days (Right sided headache)</td>
<td>Right basal ganglia and intraventricular hemorrhage</td>
<td>Died 8 days after procedure</td>
</tr>
<tr>
<td>4 24y/F</td>
<td>Headache, Postural dizziness and blurring of vision</td>
<td>NA</td>
<td>L CCA: O SCA: O (vertebral steal) Innominate R CCA 50%</td>
<td>Aorto Bicarotid bypass surgery</td>
<td>14/8mm Woven Dacron bifurcated graft</td>
<td>1 day (Seizures and hyperemia of face and neck)</td>
<td>Right frontal and left insular hemorrhage with Intraventricular extension</td>
<td>Died 54 days after procedure</td>
</tr>
<tr>
<td>5 47y/M</td>
<td>Headache, R upper limb claudication</td>
<td>NA</td>
<td>Innominate artery-90%</td>
<td>Innominate artery B A</td>
<td>10mm balloon</td>
<td>1day (Severe pulsatile right temporal headache)</td>
<td>NA</td>
<td>Totally recovered</td>
</tr>
<tr>
<td>6 38y/M</td>
<td>Dizziness bilateral upper limb claudication</td>
<td>NA</td>
<td>L CCA 80% R CCA 60% Innominate artery and R SCA 50% L SCA O with vertebral steal</td>
<td>1. L CCA 2. Innominate CCA B A</td>
<td>5x40mm balloon</td>
<td>1 day after the first procedure (Intense throbbing left temporal headache)</td>
<td>NA</td>
<td>Totally recovered</td>
</tr>
</tbody>
</table>

Fig. 1. – Axial CT images show infarcts in the left deep middle cerebral artery watershed territory.

Fig. 1. – Scanner cérébral, coupe axiale. Infarctus du territoire profond de l’artère cérébrale moyenne gauche.

Fig. 2. – Arch aortogram shows normal origins of arch vessels and the direct origin of left vertebral artery from the arch.

Fig. 2. – Aortographie. Artère vertébrale gauche née de l’aorte.

Fig. 3. – (a) Brachiocephalic artery injection show 60% diameter stenosis of proximal subclavian artery with post stenotic dilation and a normal vertebral artery origin. (b) Right Common Carotid artery injection shows 95% stenosis of right internal carotid artery and 50% stenosis of external carotid artery origins.

Fig. 3. – Tronc artériel brachio-céphalique (a) : sténose à 60 % de l’artère sub-clavière proximale avec dilatation post-sténotique. Opacification de l’artère carotide commune droite (b) : sténose à 95 % de l’artère carotide interne droite et à 50 % de l’origine de l’artère carotide externe.

Fig. 4. – (a) Left Common carotid artery injection shows 90% diameter stenosis of left internal carotid artery and 95% diameter stenosis of left external carotid artery origins. (b) Left common carotid artery injection shows atheromatous narrowing of cavernous and supraclinoid segment of left internal carotid artery and A1 segment of left anterior cerebral artery.

Fig. 4. – Artère carotide commune gauche (a) : sténose à 90 % de l’artère carotide interne gauche et à 95 % de l’artère carotide externe gauche. Artère carotide commune gauche (b) : rétrécissement athéromateux des portions cavernueuse et suprACLinoïdienne de l’artère carotide interne et du segment A1 de l’artère cérébrale antérieure gauches.
vian artery with post-stenotic dilatation with normal right vertebral artery origin (figure 3a). Both common carotid arteries (CCA) were normal. The right ICA origin showed 95% stenosis and there was 50% stenosis of the right ECA origin (figure 3b). The left ICA also showed around 90% stenosis and there was 95% stenosis of the ECA (figure 4a). There was atheromatous narrowing of the A1 segment of the left ACA (figure 4b) and of both cavernous and supraclinoid ICAs. The right anterior cerebral artery (ACA) was not visualized (figure 5a). The right ACA territory was filling from the posterior circulation via the splenial and pericallosal arteries presumably from vessel occlusion (figure 5b). The posterior circulation was normal. The venous phase of the intracranial circulation was normal.

It was decided to stent the left ICA first as it was symptomatic. Indications of treatment were to treat the symptomatic vessel first. Under distal filter protection (figure 6a) the left ICA stenosis was first angioplastied using a monorail coronary 2.5mm×14mm balloon (Cordis Corporation, Miami, Fl). Then a Wallstent of 8mm×21mm size (Boston Scientific Corporation, USA) was deployed across the stenosis and postdilated with a Maxxum 4mm×20mm balloon (Cor-

FIG. 5. – (a) Right Common Carotid artery injection shows atheromatous narrowing of cavernous and supraclinoid segment of right internal Carotid artery and no opacification of the right anterior cerebral artery. (b) Left vertebral artery injection shows the filling of right anterior cerebral artery territory via splenial and pericallosal branches.

FIG. 5. – Artère carotide commune droite (a) : rétrécissement athéroscléreux des segments caverneux et supraclinoidien de l’artère carotide interne et aplasie de l’artère cérébrale antérieure droite. Artère vertébrale gauche (b) : opacification du territoire de l’artère cérébrale antérieure droite par l’intermédiaire d’anastomoses péricalleuses.

FIG. 6. – (a) Filter Wire EZ is seen in left cervical internal Carotid artery above the stenosis. (b) Wall stent in situ in left internal carotid artery and (c) post stenting left common carotid angiogram shows patent internal carotid artery.

FIG. 6. – Filtre Wire EZ dans l’artère carotide interne gauche au-dessus de la sténose (a). Wall stent (b) dans l’artère carotide interne gauche et aspect après stenting (c).

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After the procedure, the patient was admitted to the intensive care unit with heparinisation (750 IU/hour infusion) and under strict blood pressure control. She was conscious and oriented. There was no fresh neurological deficit. Three hours after the procedure the patient suddenly became disoriented with rapid deterioration to a semi comatose state with irregular respiration. She was immediately intubated. An emergency CT scan of the head was done which showed a large left temporoparietal hematoma with intraventricular extension on the left side and hemorrhage in the right anterior basal ganglia region (figure 8). She was started on aggressive antiedema measures and continued on conservative management, but died after five days.

RESULTS

We have found 6 cases of hyperperfusion syndrome from a total of 87 procedures. So the incidence of hyperperfusion was noted as 6.89%. Two cases occurred after two combined procedures in a single sitting. The details of the events are given in table II.

DISCUSSION

Cerebral hyperperfusion syndrome is a well-known complication causing intracerebral hemorrhage after cerebral endarterectomy (CEA), aortocarotid bypass surgery, carotid and vertebral angioplasty and stenting [12]. The other mechanisms described for intracerebral hemorrhage after these procedures are perioperative cerebral ischemic event, cerebral infarction and use of postoperative anticoagulation therapy [4]. The symptoms of cerebral hyperperfusion syndrome can range from headache, seizures, confusion and focal neurological signs to intracerebral hemorrhage [5]. One of our patients became disoriented three hours after left ICA stenting with rapid deterio-
oration to a semicomatose state and CT scan showed bilateral intracranial hemorrhage with intra ventricular extension on the left side. Phatouros et al. have described intracerebral hematoma associated with subarachnoid, subdural or intraventricular bleeding in hyperperfusion syndrome after carotid angioplasty and stenting [13]. Bonaldi et al. found two strokes consistent with hemorrhagic hyperperfusion syndrome out of 53 patients following angioplasty and stenting of the cervical carotid bifurcation under filter protection occurring a few hours after the stenting procedure [3]. Mori et al. described an isolated left intraventricular hemorrhage in a patient who developed hyperperfusion syndrome after left internal carotid artery stenting [10]. In their case preprocedural single photon emission computed tomography had revealed severely reduced vasoreactivity in the affected territory after acetazolamide challenge.

Hyperperfusion phenomenon after percutaneous transluminal angioplasty for atherosclerotic stenosis of the intracranial vertebral artery has been reported by Bando et al. [1]. Mandalam et al. had reported three cases of hyperperfusion syndrome in the form of unilateral headache following balloon angioplasty of brachiocephalic arteries in two patients and massive cerebral hemorrhage after aortocarotid bypass surgery in one patient [8]. In our present case there was bilateral intracerebral bleed after stenting of symptomatic left internal carotid artery and right subclavian artery stenoses. The right anterior cerebral artery was not visualized on angiography and was filling from the posterior circulation via splenial and pericallosal collaterals. The right anterior cerebral circulation was filling via collaterals presumably from proximal vessel occlusion and not because of any congenital atresia. The right anterior basal ganglia region bleed can thus be explained as being caused by hyperperfusion due to right subclavian artery stenting. The possibility of hemorrhagic transformation of infarct was not likely as the haemorrhage developed in areas that showed no infarct on the preprocedural CT Scan. Heparinisation was only at a dose of 750 IU/hour and was just adequate to prevent stent thrombosis. Only three hours had passed post procedure when the patient bled and there was no significant blood pressure rise or fluctuation was recorded during that period.

The other patient who underwent carotid stenting developed seizure five hours after the procedure. A CT scan could not be done as the patient suddenly collapsed and was ventilated. The patient died thirteen hours after the procedure. The case of aorta to right carotid artery bypass developed right-sided headache four days after the procedure and blood pressure raised up to 270/130mmHg. CT scan showed right basal ganglia and intraventricular hemorrhage. Later the patient became unconscious and developed cardiac arrest and died on the eighth postoperative day. As such, sudden blood pressure rise along with hyperperfusion caused bleeding in this patient. The case of aorto-bicarotid bypass surgery developed seizures and hyperemia of face and neck one day after bypass surgery. CT scan showed bilateral intracerebral bleed with intraventricular extension. This patient remained vegetative and died from respiratory infection. The other two patients developed ipsilateral headache after balloon angioplasty of innominate artery and left common carotid artery respectively that resolved with analgesics. Therefore, the last two patients showed the clinical features of hyperperfusion without any ominous consequences.

Transcranial Doppler (TCD) and dynamic CT Scan had demonstrated remarkable increase in blood flow velocity and peak height respectively in cases with intracranial hemorrhage in cases reported by Masuo et al. [9]. Similarly significant increase in blood flow velocity in middle cerebral arteries has been reported after carotid endarterectomy on TCD evaluation [15]. The hyperperfusion was more pronounced in patients with preoperative hypoperfusion, who also suffered more neurological complications [9]. In our cases, we could not do TCD routinely in all patients to see velocity increase in the respective intracranial vessels.

From the existing literature and our experience in this field, we strictly maintain following protocols in treating these patients. We treat only one vessel at a time in case of multiple vessel critical stenoses in the anterior circulation supplying the brain. The second vessel should be dealt at least 6 to 8 weeks after the first one, so that the brain adjusts with the hemodynamic alteration. In cases of well-formed collateral circulation in a brain without any clinical signs of cerebral ischaemia, the revascularisation procedure can be delayed till the patient becomes symptomatic and then graded revascularisation can be attempted. In the present case, we retrospectively think that subclavian stenting should have been done in a second sitting and that it might have been the precipitating factor for hyperperfusion in this case. We prefer to use nitinol stents for these purposes because these stents dilate over time after deployment and no abrupt post procedure dilation is required. Post deployment stent dilation might precipitate sudden hypervascularisation in intracerebral territory with consequent hyperperfusion syndrome and that should be avoided. Preprocedure MRI perfusion studies can give a clue for the extent of hypoperfusion in the affected territory. Post procedure blood pressure should be strictly controlled with antihypertensive medication. Blood pressure should be strictly controlled if patient complains of headache. Headache is a most important clinical indicator for predicting hyperperfusion problems. The level of anticoagulation in postprocedure state should be balanced to avoid stent thrombosis and at the same time avoid hemorrhagic complications. We keep INR values between 1.5-2 and never above 2.

Hyperperfusion syndrome after carotid and vertebral reperfusion procedures is not a rare entity and can occur especially in patients who have the features of preprocedural cerebral hypoperfusion and critical vessel stenosis. All preventive measures should be taken in the postprocedure period to avoid this complication. The revascularization procedures in multiple arch vessel stenoses should not be carried out in the same sitting. Regardless of the nature of the procedure, surgical or interventional, diligent control of blood pressure is essential [2]. Headache on the same side as the procedure may be a warning sign for more serious sequelae and anticoagulation...
and even platelet inhibitors should be discontinued in such patients [8]. Our experience is consistent with previous authors [11]. Three of our patients were on statins for reducing their blood cholesterol levels and low blood cholesterol levels are associated with increased risk of intracranial hemorrhage [6, 14]. Therefore, we are not sure whether statins can play a role in the risk of bleeding in post stenting patients. Other interesting factors are the age and the degree of stenosis in these patients. Although we have seen this complication in elderly patients, younger people are not immune as one of our patients was in the third decade of her life. The degree of stenosis correlates with the rate of this complication since critical stenosis predisposes the hemispheric vessels to maximally dilate for maintaining circulation predisposing for hyperperfusion syndrome. Kaku et al. in their study found that significant predictors of hyperperfusion included patient age, pretreatment CVR, and pretreatment asymmetry index ([ipsilateral resting CBF/contralateral resting CBF] × 100). Variables determined not to be significant risk factors included pretreatment resting CBF value, degree of carotid stenosis, and interval from the onset of ischemic symptoms [7].

Hyperperfusion syndrome is a well-known complication of craniocervical revascularisation procedures. With proper care and technique these complications can be largely avoided.

REFERENCES


