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Iatrogenic carotid artery injury in neurosurgery

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Abstract Iatrogenic carotid artery injury (CAI) results from various neurosurgical procedures. A review of the literature was conducted to provide an update on the management of this potentially devastating complication. Iatrogenic CAIs are categorized according to each diagnostic or therapeutic procedure responsible for the injury, i.e., anterior cervical spine surgery, central venous catheterization, chemical substances, chiropractic manipulation, diagnostic cerebral angiography, middle-ear surgery, percutaneous procedures for trigeminal neuralgia, radiation therapy, skull-base surgery, tracheostomy, and transphenoidal surgery. The incidence, mechanisms of injury, diagnostic imaging modalities, and reparative procedures are discussed for each procedure. Iatrogenic CAI may be more prevalent than had previously been thought, mostly because of a heightened awareness on the part of physicians and the earlier detection of asymptomatic patients owing to sophisticated and less-invasive imaging modalities. Prevention is the best treatment for every iatrogenic injury, and it is expected that further accumulation of experience with and knowledge of iatrogenic CAI will result in further reduction of this complication. Although some CAIs, such as radiation-induced carotid artery stenosis, may not be preventable, earlier intervention before the patient becomes symptomatic may favorably alter the prognosis. Following the rapid development of endovascular techniques in recent years, surgically inaccessible lesions can be treated in a more reliable and safe manner than before.

Keywords Carotid artery · Complication · Dissection · Iatrogenic · Incidence · Injury · Neurosurgery

Introduction

Carotid artery injury (CAI) is an uncommon but not a rare complication of various diagnostic and therapeutic procedures. The consequence of inadvertent CAI may be quite devastating, but its incidence can be reduced by understanding the mechanisms of injury, i.e., how and when it happens. Properly managing the complication can reduce a patient's mortality and morbidity. Few publications that have systematically compiled and summarized information on this issue are available for neurosurgeons. A review of the literature was conducted using PubMed, an Internet medical literature data base. Iatrogenic CAIs are categorized according to their etiology, i.e., the individual diagnostic or therapeutic procedure responsible for the injury, and the site of CAI is categorized as extracranial or intracranial, as well as the common carotid artery (CCA) or internal carotid artery (ICA). The incidence, mechanisms of injury, diagnostic imaging modalities, and reparative procedures are discussed for each procedure. Neurosurgical therapeutic procedures that directly involve the CA, such as cerebral aneurysm surgery, carotid endarterectomy, endovascular coiling or stenting, and balloon occlusion test of the cervical ICA have an inherent risk of CAI in themselves and are not the subject of this review.

Extracranial CAI

Anterior cervical spine surgery

Standard anterior cervical discectomy and fusion (ACDF) is a safe procedure with a small risk of perioperative complications. Relatively common complications include superior and/or recurrent laryngeal nerve and esophageal injuries, local hematoma, and vocal cord edema [42]. Carotid artery injury associated with ACDF is very rare, and

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its incidence is not calculable. The CA, which is retracted laterally during surgery, is located within the carotid sheath and covered with connective tissue, and it is unlikely to be damaged by a retractor blade. Prolonged retraction, however, is responsible for cervical ICA thrombosis and hemispheric stroke [120]. Intraoperative measurement of the ICA flow in patients undergoing ACDF shows that the vessel cross-sectional area is reduced significantly following retraction [96].

Anterior cervical corpectomy usually requires a longer operative time with longer vascular retraction time than does an ACDF. The patient population that undergoes corpectomy is usually older than the patient group that undergoes ACDF. Thus, it is possible that the risk of perioperative stroke may be greater in a patient undergoing a corpectomy than in a patient undergoing an ACDF. In patients suspected of having high-degree CA stenosis, preoperative Doppler ultrasonography may be useful to determine the side of surgical entry to the cervical spine [22]. If the procedure is expected to require a long operative time, such as in a multilevel corpectomy, it may be necessary to release the retractor blade intermittently to restore adequate ICA flow.

It is known that posterior arthrodesis of the upper cervical spine carries a high risk of vertebral artery (VA) injury. It was not until recently, however, that it was realized that the cervical ICA is in proximity with the anterior cortex of the C1 lateral mass and is at risk of being injured during a C1–C2 transarticular arthrodesis [27]. Thorough understanding of surgical anatomy through meticulous preoperative evaluation of imaging studies, such as magnetic resonance imaging (MRI), is recommended to prevent this very rare but potentially lethal complication [27].

Anterior screw fixation of a fractured odontoid process has a high fusion rate and is now used although it is a technically demanding procedure. The odontoid process is a midline structure, and there is a small risk of cervical ICA laceration if either the drilling or a K-wire insertion goes too lateral. A rare case of ICA rupture by a motor drill was reported [6].

Central venous catheterization

Central venous catheterization is an essential procedure in the acute management of critically ill patients. Catheterization via an internal jugular vein (IJV) is preferred by many physicians to that via a subclavian vein because of the lesser risk of pneumothorax in the former [84]. The incidence of an inadvertent puncture of the CA, either CCA or cervical ICA, during IJV catheterization is between 0.5% and 11.4% and that of inadvertent arterial cannulation is between 0.0995% and 0.775% [106]. In most instances, patients remain asymptomatic after manual compression of the puncture site. Serious complications following arterial puncture, however, including stroke, have been reported [50, 80, 98, 121]. The incidence of stroke following arterial puncture or cannulation is unknown but may be higher than

had been previously thought. Reuber et al. reported four successive cases of stroke following inadvertent puncture of the CA within 6 months in a single institution [98]. Doppler ultrasonographic studies showed a CA dissection caused by arterial puncture coupled with a progressing thrombus formation from the site of puncture [98]. A rare case was reported in which severe compression of the CA by a retropharyngeal hematoma following arterial puncture caused a stroke [110]. Large-bore catheters, such as pulmonary artery or hemodialysis catheters, are more likely to result in more serious consequences. Shah et al. advocates surgical exploration of the artery, removal of catheter under direct vision, and surgical repair in such cases [106]. Even a case of stroke after arterial puncture with a small searching needle was reported [98]. Excessive compression of the puncture site to enable hemostasis in an atherosclerotic CA might have been responsible for the stroke in that particular case. The long-term sequelae of arterial puncture are not known. Most arterial injuries heal spontaneously, but cases of carotid–jugular fistula [28, 31] or pseudoaneurysm [26, 88] have been reported. Repair either by surgery or an endovascular procedure is necessary.

In many patients, the IJV is not lateral to the CA but, instead, the vein overlies the artery, which increases the likelihood of arterial puncture [31]. The use of Doppler ultrasonography is shown to reduce inadvertent arterial puncture and is advocated by many authors [38]. The practice of limiting head rotation less than 40° is also recommended to avoid an overlap between the CA and the IJV [31].

An unintentional withdrawal of a central venous catheter may occur even after slight motion of the neck, and the position of the catheter tip needs to be checked regularly by X-ray in patients with a long-dwelling catheter. Extravasation of noxious substances from a central venous catheter related to this catheter malposition may cause significant tissue damage around the catheter. A rare case of extravasation of potassium chloride from a dislocated central venous catheter resulting in near erosion of the CCA was reported [103].

Chiropractic manipulation

It is controversial whether chiropractic manipulation of the cervical spine is associated with an increased risk of cervical artery dissection, and there is significant disagreement between neurologists and chiropractors regarding the issue [46, 58, 118]. As for VA injury, there is accumulating evidence to suggest the causal relationship of chiropractic manipulation and VA dissection [30, 47, 100, 108, 109]. The anatomic arrangement of the VA within the transverse foramen makes it vulnerable to the stress to the artery that takes place in the course of manipulation of the neck [47]. The incidence of symptomatic VA dissection following manipulation is not clear due to the lack of prospective studies, but it is estimated at between one per 228,000 and one per 2 million manipulations [30, 47, 100, 108, 109]. In contrast, there is not enough demonstrable evidence as to

whether CA dissection is causally related to chiropractic manipulation [47] despite several case reports of CA dissection following the procedure [53, 56, 87, 91, 92, 94, 95], and the incidence of chiropractic-related CA dissection is not known. Most patients with CA dissection were diagnosed by magnetic resonance angiography (MRA), and the petrous ICA was the most frequent site of dissection. The type of manipulation maneuver responsible for developing the dissection is not identified in the literature although hyperextension of the neck seemed responsible [53, 56, 87, 91, 92, 94, 95]. Time interval between manipulation and development of ischemic symptoms resulting from dissection was variable, and delayed presentation was not uncommon. In some patients, the presence of connective tissue disorders might have predisposed them to arterial dissection [94, 95]. Patients presented with neurological signs of hemispheric cerebral ischemia or neck pain, but some remained asymptomatic. Treatment consisted of systemic anticoagulation with heparin followed by warfarin. A concerted effort of physicians and chiropractors to study the complication may end this controversy.

Diagnostic cerebral angiography

Complications of diagnostic transfemoral cerebral angiography with a standard Seldinger technique include a puncture-site hematoma, allergic reaction to iodine, renal dysfunction, and cerebral ischemia [10, 23, 32, 48, 57, 75, 116]. The incidence of temporary cerebral ischemia ranges from 0.4% to 1.9% and that of stroke from 0.4% to 1.0%. Thromboembolism from catheters or guide wires is the most common cause of cerebral ischemia [116], and disruption of an atherosclerotic plaque by a catheter or guide wire is another common cause of ischemia [116]. Continual flushing of the catheter with heparinized saline using a three-way stopcock and the use of air filters is advocated to reduce such thromboembolic events [10, 116].

Little is described in the literature regarding the incidence of arterial injury complicating diagnostic cerebral angiography. Iatrogenic arterial dissection is shown to occur rarely, with an incidence of 0.07–0.3% in all patients who underwent diagnostic cerebral angiography [23, 116]. The vertebral artery was more prone to dissection than the ICA [23]. Subintimal injection of contrast material is mostly responsible for dissection, and location of the dissection depends on the initial position of the catheter tip [23]. Causation or enlargement of dissection by injection of contrast material can be minimized by careful attention to vessel appearance and catheter position during the test injection.

Most patients with angiography-related iatrogenic CA dissection remain asymptomatic following systematic anticoagulation with heparin for 24–48 h, and follow-up angiogram shows improvement in most cases [23]. In a rare case of progressive dissection with low cerebral perfusion

despite anticoagulation with heparin, reconstruction surgery of the CA with use of radial artery graft was performed [101].

Radiation therapy

Cervical CA stenosis is a late complication of radiation therapy in survivors of head and neck malignancies. Radiation causes intimal damage, periadventitial fibrosis, and obliteration of the vasa vasorum, which in turn leads to carotid wall thickness and/or premature atherosclerosis [1]. Radiation-induced CA stenosis is more diffuse and involves the vessel in a longitudinally longer fashion than does atherosclerotic CA stenosis, and bilateral lesions are common in the former [45]. In some patients with head and neck malignancies, such as nasopharyngeal carcinoma, not only the extracranial CA but also the intracranial ICA is affected [7].

The prevalence of significant CA stenosis (>50%) in survivors of head and neck malignancies who have undergone radiation therapy ranges from 11.7% to 78.9% [1], and the mean interval between irradiation and development of CA stenosis ranges from 5 to 15 years [1, 7, 21, 29, 45, 72]. Doppler ultrasonography is the imaging modality used most commonly in the evaluation of patients. It is likely that the longer the follow-up period, the higher the prevalence of CA stenosis. Arterial thickening develops much earlier than overt CA stenosis—as early as within 12 months of radiation therapy [86].

The natural history of radiation-induced CA stenosis is relatively unknown, and the risk of developing stroke in previously asymptomatic patients needs to be studied to define treatment strategy. It has been shown that patients who receive radiation therapy have a more accelerated progression of CA stenosis than patients with a matched degree of stenosis but who do not received radiation therapy [21]. No measures to prevent the occurrence or to arrest the progression of the stenosis are known. Both carotid endarterectomy and carotid stenting are employed for symptomatic patients. Because of the high risk of surgery in patients with radiation-induced CA stenosis, carotid stenting is preferred [19, 112].

Spontaneous rupture of cervical CA, also known as “carotid blowout syndrome,” is a rare but potentially fatal complication following radiation therapy [18, 20, 79, 89]. Pan-necrosis of the vessel wall or rupture of a pseudoaneurysm is thought to be responsible for this complication [89]. Prior surgery is a risk factor for this condition. Surgical dissection of the carotid adventitia and vasa vasorum, depriving the artery of vascular supply and weakening the vessel walls, leads to ischemic damage and renders the artery to be prone to rupture [79]. Patients present with a sudden, massive hemorrhage and resultant hemodynamic compromise, such as hypotension or airway obstruction. Emergency endovascular occlusion of the ruptured CA has been a life-saving procedure in several cases [18, 79].

Tracheostomy (tracheotomy)

Bleeding is one of the most common intraoperative complications of tracheostomy, with a reported incidence of 4–80% [34, 40]. Most intraoperative bleeding is of venous origin, such as from the anterior jugular or facial vein, and can usually be managed without much difficulty [40]. Fortunately, CAI during the procedure is extremely rare, and no incidence rate of the complication is reported in the literature. An emergency setting and a pediatric population may put patients at greater risk for such injuries [37]. Percutaneous dilatational tracheostomy has a smaller risk of perioperative vascular injury than does conventional surgical tracheostomy [34] and can be a good alternative to surgical tracheostomy in neurosurgical patients, particularly in emergency settings [13].

Delayed arterial bleeding is a more frequent and troublesome complication of tracheostomy. Damage to the tracheal mucosa from prolonged or excessive pressure by a tracheostomy tube balloon or the tip of the tube may produce erosion of an overlying vessel, which may progress to a true fistula between the trachea and the vessel [14, 113]. Fistula formation is suspected if tube pulsation is noted or if a sentinel bleed is observed. Patients usually present with massive arterial bleeding from a tracheostomy tube. The incidence of the tracheoarterial fistula is estimated to be 0.3–0.7% of all tracheostomies [14].

The innominate artery is the most common vessel involved in fistula formation, followed by the right CCA [113]. Without treatment, the condition is usually fatal, and surgical repair is the only and definite treatment. Cuff overinflation and digital compression of the arterial site can be used as temporizing maneuvers while surgery is being arranged [33, 83]. A preventive measure for this complication includes the correct placement of the tracheostomy stoma through the second and third tracheal rings rather than lower in the trachea and by avoidance of overinflation of the tracheostomy tube cuff [14, 83].

Intracranial CAI

Chemical substances

In cerebral aneurysm surgery, “coating” or “wrapping” techniques are often used to reinforce a friable arterial defect, particularly when an aneurysm or part of an aneurysm is not clippable. The aneurysmal remnant is coated or wrapped with cottonoids or muscle fragments and is reinforced with fibrin glue or cyanoacrylate glue, which is sold as Biobond. Occlusion of the ICA and middle cerebral artery following the use of Biobond has been reported, and it occurs 4–5 weeks after surgery [63]. The incidence of this chemical vasculitis is not clear. Steroids are not effective. The vascular toxicity of cyanoacrylate glue via an elicitation of vasculitis is also shown in a rat carotid artery by the same authors [52, 59]. Biobond was recently withdrawn from market in Japan.

Chemical meningitis following spontaneous rupture of a craniopharyngioma that caused significant ICA stenosis has been reported [107]. A high concentration of cholesterol crystals in the cerebrospinal fluid might have been responsible for the stenosis. Although spill of the irritant from a tumor cyst was spontaneous and occurred before surgery, it suggests that utmost attention is necessary to prevent the spill of the cyst content during craniopharyngioma surgery.

Several chemical substances are known to cause arterial dissection after being ingested, injected, or inhaled. In most instances, these substances are illegal, such as cocaine or amphetamines [41], but even prescription drugs may cause arterial dissection if taken excessively. A case of ICA dissection after the overdose of ergotamine prescribed for migraine is reported [3].

Middle-ear/craniofacial surgery

The petrous ICA has a close anatomical association with the middle ear. The bony covering over the tympanic segment of the ICA in the transition zone between the vertical and horizontal segment is 0.5 mm or less in thickness, and approximately 1% of the population has a congenital dehiscence of the bony covering at this segment [49, 115]. The ICA may be injured in seemingly benign otological procedures, particularly in myringotomies. Between 50 and 60 cases of ICA laceration associated with myringotomy is reported in the literature [15, 51, 54]. Most patients had an aberrant lateral ICA, and massive bleeding occurred following incision of the eardrum. Preoperative high-resolution computed tomography (CT) with coronal reconstruction has been shown to be useful to detect the aberrant anatomy preoperatively. Myringotomy is a very common procedure, however, and the incidence of CAI during the procedure is not calculable. It is also controversial as to whether all surgical candidates of myringotomy need to undergo a screening CT scan in view of the medical cost. Once the bleeding occurs, hemostasis is achieved by packing with Surgicel and cottonoids [15, 51, 54, 115]. Myringotomy needs to be terminated, and angiography or MRA is necessary postoperatively to evaluate the patency of the ICA. Six cases of pseudoaneurysm resulting from myringotomy have been reported in the literature [4, 15, 51, 54]. Endovascular procedures are the preferred treatment for this surgically inaccessible lesion [4, 51]. Stenting, in which flow of the ICA can be preserved, may be preferable to coil occlusion of pseudoaneurysm in which the vessel wall is fragile, and occlusion of the ICA together with the pseudoaneurysm is usually necessary.

Although injury of the external CA branches are reported to occur often during various craniofacial/oral surgical procedures [119], iatrogenic injury of the intracranial ICA is actually rare. Cases of ICA laceration following septorhinoplasty [66] or endoscopic sinus surgery [55] have been reported. Dissection of the petrous ICA after molar extraction has been reported in which hyperexten-

sion of the neck during the procedure is thought to be responsible for the injury [17, 97].

Percutaneous procedures for trigeminal neuralgia

Many treatment options are available for pain relief of trigeminal neuralgia. Percutaneous techniques for trigeminal neuralgia are favored by many physicians as a primary treatment for patients who failed medical treatment, and they include radiofrequency rhizotomy, glycerol rhizotomy, and balloon compression [12, 62, 111]. Dysesthesia, corneal anesthesia and keratitis, and trigeminal motor dysfunction are common complications of the percutaneous procedures. Because of the proximity of the petrous ICA in the foramen lacerum to the trigeminal ganglion in the Meckel's cave or to the V3 division in the foramen ovale [99, 104], there is a danger of possible arterial injury when percutaneous techniques are performed. In up to 4% of cases, there was a fusion of the foramen ovale and foramen lacerum, the primitive foramen lacerum medius, which might predispose to the arterial injury [99, 104]. Extension of a thermal lesion may also cause arterial damage without arterial penetration in radiofrequency rhizotomy [104]. The incidence of inadvertent puncture of the ICA ranges from 0.8% to 6% [12, 104]. In such situations, termination of the procedure may be necessary. Some authors stress the importance of avoiding placing the needle too medially into the foramen lacerum [104]. Permanent complications following arterial puncture are rare. Most arterial punctures occur within the cavernous sinus, and carotid cavernous fistula (CCF) is a resulting complication [39, 62, 70, 97, 99, 104, 111]. The incidence of the CCF from two large studies is 0.01–0.06% [62, 111]. Endovascular transarterial balloon [70] or coil embolization [44, 70, 81] is the mainstay of treatment of CCF.

Radiation therapy

Occlusion of large and medium-sized intracranial arteries, including the ICA, and the development of moyamoya vessels is reported in pediatric brain tumor patients who underwent whole-brain radiation therapy [11, 43, 64, 90]. The primary diseases were optic glioma, followed by hypothalamic glioma, craniopharyngioma, and leukemia, in order of frequency [8, 11, 43, 64, 68, 90, 93]. The majority of patients were younger than 7 years of age and developed the vasculopathies within 5 years after radiation therapy. The total dose of radiation therapy exceeded 50 Gy in most cases. The incidence of radiation-induced moyamoya syndrome is not clear. Kestle et al. [64] and Grill et al. [43] independently report that the moyamoya syndrome developed in five out of 28 (18%) and 13 out of 69 (19%) children with optic glioma treated with radiation therapy. Patients with neurofibromatosis (NF)-1 are more likely to develop vasculopathy than those without NF-1 [43, 64]. Radiation therapy should not be the first line of treatment in these settings [8, 69].

Patients presented with transient ischemic attacks, seizures, or strokes [8, 11, 43, 64, 68, 90, 93]. Conventional angiography is the “gold standard” imaging study, but MRA is also effective. Treatment of radiation-induced moyamoya syndrome is nearly the same as that of idiopathic moyamoya disease. Aspirin is the first line of treatment, and revascularization surgery is performed for those refractory to medical treatment. The outcomes seem poorer in patients with radiation-induced moyamoya syndrome, however.

In adults, radiation-induced cerebral vasculopathy takes a different form from that in children [5]. Stenosis and obliteration of major cerebral arteries without moyamoya vessels are the major findings in adults. The incidence and risk factors in developing vasculopathy following whole-brain radiation therapy in adults is yet unknown. Patients of younger age at the time of radiation therapy (<30 years) and those with pathology in and around the optic chiasm or basal ganglia seem more likely to develop vasculopathy [5].

Cerebral aneurysm formation is another sequela of whole-brain radiation therapy. This complication is rare, with less than 20 reported cases in the literature, and is seen in a wide age group [82, 85]. Both pseudo- and true aneurysms are reported. The presence of a concomitant occlusive vasculopathy in some patients suggest that altered hemodynamic stress might be responsible for a true aneurysm formation [82]. The interval between the radiation therapy and clinical presentation is variable, as is the location of the aneurysm. More than half of these patients presented with a subarachnoid hemorrhage (SAH) and had a poor prognosis. Endovascular coil occlusion of the aneurysm is successful in some patients [82, 85].

Skull-base surgery

In skull-base surgery, surgical exposure and mobilization of the ICA is occasionally necessary for securing proximal control of the vessel or for performing high-flow bypass. The exposure can be performed safely owing to the accumulated knowledge of skull-base surgical anatomy [25, 74, 77, 117]. Occurrence of CA laceration during the exposure, particularly during drilling, has been reported very rarely [105]. In skull-base tumor surgery, however, there is an inherent risk of arterial injury during dissection of the ICA from a tumor, particularly during surgery for a tumor encasing the ICA, such as occurs with a large sphenoparietal or petroclival meningioma [2, 65, 71, 74]. The incidence of the CA laceration in those circumstances may vary significantly depending on the tumor's pathology, invasiveness, and extension, as well as a surgeon's treatment strategy. Tumor invasion of the vessel wall makes it vulnerable to mechanical stress during dissection, and bleeding from avulsion of one of its small branches may occur commonly. Although CA laceration is usually amenable to primary suture, such cases need to be followed postoperatively with serial, noninvasive, vascular imaging studies

because of the risk of development of a pseudoaneurysm [65, 71].

Delayed occlusion of the ICA following its exposure and dissection from a tumor occurred in one (5%) out of 19 patients in a series reported by Leonetti et al. [76] and in one (3%) out of 30 patients in a series by Sekhar et al. [105]. These patients in both series had large tumors that encased the petrous ICA. Endothelial injury aggravated by manipulation and mobilization of the ICA is thought to be responsible for its delayed thrombosis. In another patient in the series of Leonetti et al., hemispheric edema without infarction occurred [105]. The reason for the edema is not described.

Vasospasm of intracranial arteries, including the ICA, is another ischemic complication of skull-base tumor surgery [9, 78]. In a series of Bejjani et al., vasospasm occurred in nine (1.9%) out of 470 patients with skull-base tumors treated surgically [9]. Vasospasm in this series manifested clinically 1–30 days postoperatively, with findings of altered mental status and/or hemiparesis. Blood spillage into the basal cisterns and manipulation of the major vessels were considered to be responsible. Preoperative embolization, tumor size, vessel encasement/narrowing by tumor, and total operative time were associated with the development of vasospasm. Three (33%) out of nine patients were left with permanent neurological deficits [9].

Following the popularized use of stereotactic radiosurgery in the management of skull-base tumors in recent years, the treatment strategy for these tumors may have shifted toward a less aggressive one, i.e., from radical excision of the tumor with extirpation and reconstruction of the ICA to subtotal removal of the tumor and anatomical preservation of the ICA followed by stereotactic radiosurgery [2, 74]. In cases in which the sacrifice of the ICA is deemed to be unavoidable or to outweigh the potential risk of cerebral ischemia, the deliberate surgical or endovascular occlusion of the ICA has often been attempted before the tumor surgery, with or without bypass surgery [102]. Discussion on the indications, risks, and benefits of the deliberate occlusion of the ICA in skull-base surgery exceeds the scope of this review.

Transsphenoidal surgery

Transsphenoidal surgery is the preferred approach for an intrasellar tumor, particularly for a pituitary adenoma [73]. Laceration or perforation of the ICA is a rare and dreaded complication of pituitary surgery. The incidence of CAI in transsphenoidal surgery from two large studies was 0.3–0.4% [35, 73]. The cavernous ICA is most likely to be injured during aggressive lateral dissection of macroadenomas [36]. Previous transsphenoidal surgery, cavernous sinus invasion by the tumor, and a small sella predisposed to CAI [16]. The cavernous ICA may be quite tortuous, and it may be located just behind the dura where it is vulnerable to injury during the opening of the sellar dura and exposure of the pituitary lesion [73]. Preoperative confirmation of the course of the cavernous ICA with MRI

may prevent this complication, and intraoperative image guidance may also be helpful. Less frequently, the ICA may be injured at the bony canal that lies on each side of the sphenoid sinus [73]. Bleeding should initially be treated by packing with cottonoids, Gelform, or muscle fragments. Direct suture of the laceration is accomplished in some cases [73]. Fukushima and Maroon successfully used Teflon mesh and methyl acrylate to reinforce the injured arterial wall [35]. The long-term outcomes are unknown, however.

The sequelae of ICA laceration include CCF, pseudoaneurysm, and thrombosis [16, 24, 35, 36, 60, 61, 67, 73, 114]. Conventional angiography or MRA is mandatory for assessment of patients with CAI. Symptoms vary according to the underlying pathology. In patients with pseudoaneurysm, SAH or massive epistaxis are the common manifestations. Endovascular treatment is preferred to open surgery for iatrogenic cavernous ICA lesions. Balloon or coil occlusion for CCF and coil occlusion or stenting for pseudoaneurysm have recently been used successfully [24, 61, 67, 114].

Conclusion

Regarding iatrogenic CAI, this review shows that we have a better understanding of its etiology and incidence and better preventive and reparative measures than before. Prevention is the best treatment for every iatrogenic injury, and it is expected that accumulation of experience and knowledge will result in the further reduction of complications. Although some injuries may not be preventable, such as radiation-induced CA stenosis, earlier intervention before the patient becomes symptomatic may favorably alter the prognosis. Following the rapid development of endovascular techniques, surgically inaccessible lesions, such as the petrous or cavernous ICA pseudoaneurysm, can now be treated more reliably and safely than before.

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