CLINICAL INFORMATION

Internal carotid artery dissection after laparoscopic surgery

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Received 1 September 2016; accepted 11 January 2017
Available online 17 April 2017

KEYWORDS
Carotid dissection; Anesthesia; Laparoscopy; Complication

Abstract  Headache is a common symptom in the postoperative period and may be attributable to, dehydration, sleep deprivation, intentional or inadvertent dural puncture during a neuraxial anesthesia technique, from an inhaled anesthetic agent, or from specific surgical procedures, among other etiologies. However, more serious, uncommon and life-threatening conditions as carotid artery dissection can be associated with severe neurologic sequelae in otherwise young, healthy patients. For these reasons, clinicians involved with postoperative patients should be familiar with the presentation and management strategies for this complication.

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PALAVRAS-CHAVE
Dissecção carotídea; Anestesia; Laparoscopia; Complicação

Dissecção da artéria carótida interna após cirurgia laparoscópica

Resumo  A cefaleia é um sintoma comum no período pós-operatório e pode ser atribuída à desidratação, privação do sono, punção dural intencional ou acidental durante a administração de anestesia neuraxial, ao anestésico inalatório ou a procedimentos cirúrgicos específicos, entre outras etiologias. Entretanto, condições mais graves, incomuns e potencialmente fatais, como a dissecção da artéria carótida, podem estar associadas a sequelas neurológicas graves em pacientes jovens e, sob outros aspectos, saudáveis. Portanto, os médicos envolvidos com pacientes pós-operados devem estar familiarizados com as estratégias de apresentação e manejo dessa complicação.

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Introduction

Postoperative headache is common, can delay discharge, and can contribute to patients’ suffering and dissatisfaction with their care. It may result from dehydration, sleep deprivation, intentional or inadvertent dural puncture during a neuraxial anesthesia technique, from an inhaled anesthetic agent, or from specific surgical procedures. However, atypical features (atypical distribution, absence of postural component, unresponsiveness to analgesics), accompanying neurological symptoms, or the presence of vomiting, seizures, altered level of consciousness and focal motor and sensory deficits should lead to consideration of other causes whose consequences can be devastating if an early diagnosis and treatment are not performed. We report a case of an otherwise healthy female who presented with persistent headache and partial Horner’s syndrome after laparoscopic surgery and was ultimately found to have an Internal Carotid Artery Dissection (ICAD). The patient gave written informed consent for publication of this article.

Case report

The patient was a 39-year-old, 58 kg female without relevant medical history. She was scheduled for laparoscopic right adnexectomy due to mucinous cystadenoma. Preoperative physical examination and routine laboratory studies were unremarkable.

Following application of standard monitoring and pre-oxygenation with a facemask, general anesthesia was induced with fentanyl (2–3 μg/kg) and propofol (2 mg/kg), and muscle relaxation was achieved with rocuronium (0.6 mg/kg). Endotracheal intubation was performed using the Totaltrack™ size 3 with a 7 mm inner diameteruffed endotracheal tube. Tracheal intubation produced no significant changes in vital signs. The pressure-controlled ventilation was adjusted to maintain normocapnia using a mixture of oxygen–air (FIO 50%). Maintenance of general anesthesia was carried out using sevoflurane and remifentanil. Induction and maintenance of anesthesia throughout the surgery was uneventful. The procedure was carried out without incident, and the patient’s trachea was extubated at the end of the operation, with minimal fluctuation in arterial blood pressure and pulse rate. She was transferred to the recovery unit.

In the post-anesthesia care unit, patient developed a persistent oppressive right-sided frontotemporal headache and neck pain with swallowing. The headache was of mild intensity and did not have any “throbbing” or pulsating qualities. Acetaminophen and nonsteroidal anti-inflammatory drugs provided no substantial relief. Neurological examination showed an ipsilateral partial ptosis and reactive miosis without visual loss. There was no evidence of other neurological deficits. Computed Tomography (CT) of the brain was normal. However, a subsequent contrast angiography revealed segmental narrowing from the post bulbar segment of the right internal carotid artery extending into the petrous segment of the vessel (Fig. 1). These findings were consistent with a spontaneous ICAD. The remaining diagnostic work-up was unremarkable. Anticoagulation and antiplatelet therapy was initiated. Our patient had a favorable evolution and was discharged home without any neurologic sequelae. The patient remained asymptomatic 6 months afterwards, and a follow-up magnetic resonance imaging showed an ad integrum restitution of the artery.

Discussion

Carotid artery dissection is a rare condition, mostly occurring in previously healthy, young individuals. Its incidence is still likely underestimated, as asymptomatic patients are not captured by the majority of studies. The annual incidence of stroke due to ICAD is 1.21/100,000 per year. The pathophysiology of ICAD involves genetic, anatomic and environmental factors (Table 1). Vascular layers

Table 1 Risk factors associated with carotid artery dissection.

<table>
<thead>
<tr>
<th>Genetic factors</th>
<th>Acquired and anatomic factors</th>
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<tr>
<td>Marfan syndrome</td>
<td>Cervical trauma</td>
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<tr>
<td>Ehlers–Danlos syndrome type IV</td>
<td>Iatrogenic after surgical interventions</td>
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<tr>
<td>Osteogenesis imperfecta type I</td>
<td>Medical procedures</td>
</tr>
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<td>Fibromuscular dysplasia</td>
<td>Oropharyngeal infections</td>
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<tr>
<td>Hyperhomocysteinemia</td>
<td>Arterial hypertension</td>
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<tr>
<td>Hemochromatosis</td>
<td>Vascular tortuosity</td>
</tr>
<tr>
<td>α1-Antitrypsin deficiency</td>
<td>Aorta coarctation</td>
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<tr>
<td>Turner syndrome</td>
<td>Illicit drug abuse</td>
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<td>Down syndrome</td>
<td>Chiropractic cervical manipulation</td>
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<td>Williams syndrome</td>
<td>Severe exercise</td>
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<tr>
<td>Menkes disease</td>
<td>Vomiting</td>
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<tr>
<td>Moyamoya disease</td>
<td>Violent coughing or sneezing</td>
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<tr>
<td>Polycystic kidney disease</td>
<td>Migraine</td>
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<tr>
<td>Pseudoxanthoma elasticum</td>
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separate, creating a false lumen, which may lead to steno-
sis, occlusion or formation of an intramural hematoma and pseudo-anurysm.2 Thrombus formation may lead to life-threatening emboli by fragmentation.1 Its etiology is either spontaneous or traumatic, although simple manip-
ulation of the neck can evoke a dissection.4 Thus, there
have been reports of ICAD related to medical procedures
such as bronchoscopy, rigid esophagoscopy, endotracheal
intubation, catheterization of the jugular vein, hyperex-
tension of the head during mask ventilation and tracheal
intubation or tonsillectomy.6-6 In our case, several factors
could have contributed to formation of ICAD. The combina-
tion of pneumoperitoneum along with the steep Trendelenburg position during laparoscopy affect cere-
brovascular and hemodynamic homeostasis increasing the
risk of arterial dissections.7 Pneumoperitoneum may cause
intraoperative hypertension and vascular instability. It is
thought to be either through the activation of a neuro-
humoral response or as a result of pain from stretching of
the peritoneum. Increasing intrathoracic pressure during
laparoscopy induces high shear stresses on the arterial
wall of the brachiocephalic vessels due to pressure changes.7
It might produce intimal injury. The Trendelenburg position
also cause increased intracranial pressure and increase car-
diac output that may intensify hemodynamic forces applied
to vessel walls. Cervical extension and rotation or sudden
lateroverension neck movements can compress the cervical
carotid artery against any of the transverse processes, cer-
vical vertebrae or the styloid process of the mastoid bone,
leading to dissection. Likewise, neck hyperextension with
axial rotation of the head during endotracheal intubation
could have played a role,6 however the use of TotaltrackTM
device allowed an orotracheal intubation with the head in
a neutral position.8

Diagnosis of ICAD can be delayed because of a low index
of suspicion or symptoms may be absent or may seem
unimportant.1-3 The classic triad of unilateral pain in the
head, face or neck, partial Horner’s syndrome and cerebral
ischemia is found in fewer than 30% of cases.1 However,
the 60%-90% of patients present with headache, neck, or
headache and neck pain, which typically precede neurologic
symptoms by hours to days. Horner’s syndrome results from
disruption of the ocular and facial sympathetic pathways
that ascend within the carotid sheath.9 Anhidrosis may be
absent because sympathetic fibers that innervate the facial
sweat glands follow the external carotid artery.10 Cranial
nerve palsies and pulsatile tinnitus have also been reported.

Because diagnosis of carotid injury is rarely suspected,
the first diagnostic test performed is usually CT of the brain.
However, the gold standard for diagnosis of ICAD is con-
trast angiography, showing a string sign, a double lumen,
or intimal flaps.11 Magnetic Resonance Imaging (MRI) and/or
magnetic resonance angiography are reliable methods for
diagnosis and follow-up.10

Treatment of carotid artery dissection consists of avoid-
ing or limiting neurological deficit through the prevention
of thrombus formation or associated embolism. Therefore,
anticoagulation, antiplatelet therapy, or antiocoagulation
and antiplatelet therapy are the mainstay of the treatment,
although the optimal antithrombotic strategy has been
controversial.1 Endovascular therapy or surgery is the
treatment of choice in specific situations,2 including the
following: dissections with flow limiting stenosis; “isolated
hemisphere” dependence on the dissected artery for per-
fusion; failure of medical therapy with worsening hemody-
namic dependent neurologic symptoms or imaging findings;
multiple bilateral arterial dissections that may progress
to cause hemodynamic compromise or expanding aneurysm.

Long-term prognosis appears to be favorable if diagnosis
and treatment are prompt, with complete recovery in at
least 50% of cases. The risk of recurrence is highest within
the first month and remains about 1% per year after the
first year, so it requires follow-up imaging studies.2

Carotid artery dissection should be considered in the dif-
erential diagnosis of postoperative headache. The presence
of ipsilateral headache, facial pain or neck pain, oculo-
sympathetic paresis or transient ischemic attack should lead
to a high index of suspicion. Early recognition is important
so that treatment may be initiated before complete vessel
occlusion or embolic sequelae to avoid serious and poten-
tially life-threatening neurologic deficits.

Conflicts of interest

The authors declare no conflicts of interest.

References