Most carotid aneurysms are located intracranially. It is rare for aneurysmal lesions to involve common or external carotid arteries or the extracranial part of the internal carotid artery. Extracranial carotid aneurysms deserve particular attention due to their variable etiology and morphology, as well as the fact that each case that qualifies for treatment requires an individual approach.

Despite the fact that operations on carotid arteries are the most often performed vascular surgery procedure, relatively rare aneurysms in this region represent a significant problem and are challenging even for experienced vascular surgeons. In recent years more and more articles on carotid aneurysms have appeared due to the development of modern noninvasive imaging methods.

**EPIDEMIOLOGY**

About 1000 carotid aneurysms have been described in the literature since 1687 (1-5). It should be emphasized that most of these articles were case reports. In the 1940’s, a rapid growth of interest in vascular pathology occurred that could be connected with the performance of angiographies, that enabled the precise localization of aneurysms and the evaluation of their morphology and diameter (1).

It is difficult to determine the actual occurrence of carotid aneurysms. It is estimated that they comprise from 0.4 to 4% of all peripheral aneurysms, while the number of operations for this lesion amounts to only 1% of all operations performed on carotid arteries (3, 6).

According to the literature, aneurysms involving the common carotid artery or its bifurcation occur in 55% of cases, internal carotid artery occur in 45% of cases, and external carotid artery occur in only 2.2 to 5% of cases. No laterality has been found. Bilateral aneurysms occur in 3.6 to 7.8% of cases. In 1979 David C. Schechter reviewed data covering 400 years of the literature. Describing 820 patients with carotid aneurysms, he observed that in only a few cases was the aneurysm involved the entire length of the common carotid, internal carotid or external carotid artery. Most of the aneurysms were confined to a single carotid artery, and only a few affected the bifurcation and encroached on other arteries. Few of these carotid aneurysms were associated with aneurysms elsewhere. Data presented in this article were inadequate to ascertain whether or not different aneurysms were significantly associated with racial, occupational, or other demographic characteristics. In only two patients did the author obtain reliable data of blood relatives who had carotid aneurysms (1, 9).

Similar to other aneurysms, carotid aneurysms are most often detected in males. Age is not a crucial factor for their incidence. They occur in children as well as in patients over 70. More and more often they are found in middle-aged patients in connection with the occurrence of atherosclerosis (10). In young adults, posttraumatic aneurysms of the common carotid or external carotid arteries are most often diagnosed as a result of past blunt or penetrating trauma. Non-traumatic aneurysms of the internal carotid artery in children are also associated with coexisting congenital abnormalities of the vascular system or are a result of recurrent pharyngeal infections (1, 5).
ETIOLOGY AND PATHOGENESIS OF CAROTID ANEURYSMS

Similar to aneurysms in other locations, there are both true and false aneurysms. Degeneration of the artery wall is responsible for true aneurysm formation, and these are most often atherosclerotic (90% of all true aneurysms). True aneurysms most frequently involve the bifurcation of the common carotid artery; they are usually fusiform or rarely saccular (5, 6, 11). Atherosclerotic aneurysms infected with Staphylococcus, Pseudomonas, or Salmonella spp. strains comprise a distinct group. The Salmonella strain demonstrates distinguished tropism towards the artery wall damaged by the atherosclerotic process. These infections may be asymptomatic and manifest primarily with an aneurysm rupture - a very rare occurrence. In most such cases it is possible to culture microorganisms from the aneurysm wall (12, 13).

True aneurysms may coexist with congenital abnormalities and occur in Marfan’s syndrome, cystic necrosis of intima media, fibromuscular dysplasia, nodular arteritis, granuloma or giant cell arteritis, neurofibromatosis, Pseudoaxanthoma elasticum, Behçet’s and Takayasu’s syndromes, and after neck irradiation (14, 15, 16).

The very rare causes of carotid aneurysm formation mentioned above induce hypoplasia or damage of the intimal elastic lamina with the coexistence of intima media thinning and dilation of the artery. As for congenital aneurysms, an insufficient amount of elastic fibers or the damage of these fibers induces their replacement with mucopolysaccharide molecules that do not provide the artery wall with sufficient flexibility. In most cases these aneurysms involve the internal carotid artery. They are often bilateral or co-exist with intracranial aneurysms (5).

False aneurysms of carotid arteries form as a result of trauma, postoperative complications, iatrogenic lesions, or as a consequence of recurrent bacterial infections.

In the past when slashing and stabbing weapons were used, the mortality from carotid artery injury was very high. Few of the wounded lived long enough for an aneurysm to form. After the introduction of firearms, the number of posttraumatic carotid aneurysms increased due to injuries of the vessel wall with shrapnel. Direct injuries of the carotid artery with bullets are usually fatal (1).

Most traumatic carotid aneurysms are the result of penetrating wounds. There are also some reports in the literature describing the particular circumstances of carotid artery injuries, such as swallowing a foreign body: a fishbone, a needle, a safety pin, or even a pipe stem (1). The pervasion of drug addiction more and more often results in injuries of the carotid artery due to narcotic injections (12). Aneurysms forming as a result of penetrating injuries predominantly involve the common carotid artery.

Blunt trauma due to blows received while practicing combat sports, accidental pressure, and strangling may produce an aneurysm. Other injuries that cause aneurysm formation include acute neck hyperextension or rotation and smashing injury during a vehicular accident. These injuries all cause a compressing of the carotid artery to the transverse processes of the atlas. Another particular mechanism of blunt carotid artery trauma is the artery’s compression between the second vertebral body and the transverse processes of the third cervical vertebra (single extensive or repeated microinjuries). This type of trauma may cause intimal dissection, leading to aneurysm formation or aneurysm presence without any signs of vascular wall dissection. Extreme strain while coughing or playing wind instruments when the arterial wall is weakened may result in its damage. These aneurysms are most often located at the level of the styloid process (1, 17-22).

Pseudoaneurysms may form at the top of the kinking of an elongated internal carotid artery as a result of its weakness and damage to its wall caused by atheromatous plaques (2).
Infections are the main etiological risk factor for non-traumatic pseudoaneurysms. The carotid artery’s location in the vicinity of the pharynx causes suppurative tonsillitis, recurrent mastoiditis, or untreated dental caries with pyorrhea alveolaris spread along tissue that continually infiltrates the vascular wall. However, mycotic common carotid artery aneurysms are associated more frequently with non-pyogenic bacteria. Suppurative infections strike the common carotid artery not across adjacent tissues, but from lymph nodes. These infections may also strike the common carotid artery through hematogenous metastasis, where the source of infection may be bacterial arteritis, endocarditis, cholecystitis, or other infected organs. The most common contaminants of non-traumatic suppurative aneurysms are haemolytic streptococci, diplococci, and pneumococci, but rarely Haemophilus influenza or Escherichia coli (3, 12, 13, 24, 25).

Antibiotics and chemotherapy have almost completely eliminated syphilis and tuberculosis as etiological factors causing aneurysm formation (1, 17).

**SYMPTOMS**

The symptoms produced by aneurysms depend on their location and etiology. In most cases the diagnosis of a carotid aneurysm is not difficult. False aneurysms, in particular those caused by an injury, grow quickly in contrast to true aneurysms of atherosclerotic etiology. Patients often visit a doctor after becoming concerned about bulge or tumour appearance on their neck. Pulsation is not a permanent sign of aneurysm formation. This sign depends on the size of the aneurysm, thrombus inside the sac, blood pressure, and neck position during examination. In 20-25% of cases patients complain of pulsation in their ear or head.

Aneurysms of the common carotid artery and the proximal segment of the internal carotid artery are located below the mandibular angle. These aneurysms situated above the angle almost always involve the distal segment of the internal carotid artery, and they may not be visible on the neck. Patients with aneurysms located high at the base of the skull may complain of only a sensation of throat swelling and difficulty in swallowing. They often come to a laryngologist because of these symptoms (26). Cases have been described where carotid aneurysms have been misidentified as peritonsillar abscesses (1, 5, 24, 27, 28). Although it happens very rarely, the incision of the peritonsillar abscess should always be preceded with an ultrasound examination.

Neurological defects are found in 50-80% of patients with internal carotid artery aneurysms. The most common defects are transient ischemic attacks – TIA (21%) – or amaurosis fugax (33%) caused by microemboli from the aneurysm sac. These symptoms may also be caused by a growing aneurysm pressuring the artery, thereby limiting blood flow to the central nervous system. Paresis, hemiplegia, or massive ischemic stroke are rarely the first neurological symptoms. Other symptoms often involve ipsilateral hemikrania radiating to the auricle and the occiput, dizziness, dysphagia, tongue deviation, trismus, hoarseness, hypacusia on one side, and neuralgia of the V and VII cranial nerves supply area. Carotid artery aneurysms may be accompanied by Horner’s syndrome (2, 23, 29-32).

About 30% of patients diagnosed with a carotid aneurysm are asymptomatic. This concerns small aneurysms of the internal carotid artery. These aneurysms are occasionally revealed by ultrasonographic or other imaging examinations(33).

Contrary to aneurysms in other locations, rupture is very rarely the first sign of a carotid artery aneurysm. Hemorrhages from the throat, ear, and nose have also been described. Death or irreversible neurological effects are caused by late diagnosis or complications connected with the treatment (1, 34, 35).

**DIAGNOSIS AND DIAGNOSTIC IMAGING**

In most cases a well-documented history and physical examination allow the physician to perform an accurate diagnosis.

A slightly mobile tumor situated alongside the angle of the jaw that pulsates in all directions is often found on examination. For infected aneurysms, the tumor may be painful upon palpation, and the skin may be tight and irritated.

The bulge may be easily compressed and refills promptly after the fingers are released. It should be highlighted that because an aneurysm may occur in this region, particular cau-
performed to exclude other causes of neurological defects. It allows the visualization of ischemic foci in different brain structures in about 80% of patients, including asymptomatic patients. It also excludes brain tumor, intracranial hematoma, or brain abscess from the diagnosis, and also differentiates the aneurysm from other neck tumors (2, 37, 38).

EEG examination is necessary in patients with a history of epileptic attacks.

Arteriography still remains irreplaceable and is a conclusive preoperative examination. Some authors define it as a gold standard for diagnostic imaging of carotid aneurysms (39). It allows the precise description of the size, location, and morphology of the aneurysm and the adjacent arteries. The evaluation of the proximal part of the internal carotid artery is very important because of the possibility of coexisting atherosclerotic stenosis. In aneurysms coexisting with arterial dissection a string sign is found. This indicates a major grade stenosis of the arterial lumen. True aneurysms are fusiform in most cases, but this is not a rule.

It is surprising that although many different imaging methods are available, some carotid aneurysms are found accidentally during operations.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis of carotid aneurysms include: kinking or looping of the internal carotid, common carotid, or subclavian artery aneurysm, lymphadenopathy (metastases, lymphoma, Hodgkin’s disease, inflammatory process), peritonsillar abscess, parotid tumor, parotitis, hygroma, chemodectoma, hemangioma, and malignances (1, 29, 31).

TREATMENT

The diagnosis of common or internal carotid artery aneurysm should always be an indication for surgical treatment due to the risk of ischemic stroke. The main goal of the operation is not to reduce the risk of aneurysm rupture – a very rare event – but to prevent central nervous system ischemia (3, 5, 37, 39).

Surgical access to a carotid aneurysm is achieved with an incision from the clavicle to the auricle alongside the sternocleidomastoid muscle. For high aneurysms, extension of the inci-
Extracranial carotid aneurysms

The loss in the arterial wall may be supplemented with a venous or prosthetic patch. R. El-Sabrout and D. A. Cooley consider that avoidance of extensive resection of the posterior wall of the aneurysm reduces the risk of injury to the vagus, recurrent laryngeal, and glossopharyngeal nerves (33). However, P. Moreau et al. are of the opinion that a pathologically changed arterial wall should be removed because it may cause the formation of new aneurysms (46). For aneurysms of the proximal segment of the common carotid artery, carotid-carotid or carotid-subclavian bypass should be considered for the restoration of blood flow. Similarly, bypasses from the aortic arch to the common carotid or internal carotid artery can be performed, and require sternotomy or thoracotomy (5, 17, 31, 33, 36, 45, 51).

Operations may be performed under local or regional anesthesia. Some authors propose general anesthesia with monitoring of the blood flow to the brain as a treatment of choice (4, 33, 52, 53). T. Skau et al. suggest the additional use of moderate hypothermia, mannitol, and barbiturates to decrease ischemic lesions caused by the transitional clamping of carotid arteries (38).

The use of shunts during surgery on carotid aneurysms is controversial. Their routine use was recommended in the past (17, 54). At present some authors suggest using them only in select cases that have an elevated risk of stroke, whereas others use them routinely during every operation for aneurysms (5, 53, 55-58). E. Rosset et al. and Q. Zhang et al. did not use shunts, claiming that their application elongates operating time, increases technical difficulties, and may be the cause of neurological complications (4, 37).

In the 1990’s, some reports on the use of endovascular techniques for carotid artery pathologies started appearing; few of them concerned aneurysm treatment (10, 59, 60-64). There were attempts at their embolization with coils, stents, and a combination of both methods, with good results seen in short-term follow-ups (65, 66). However, in long-term observations in some cases, recanalization of the aneurysm sac and the increase of the aneurysm’s diameter were described (67-70). In the past few years, increased interest in the use of the stent-grafts for carotid aneurysm treatment has been observed (10, 68). There are no guidelines for endovascular treatment of caro-
The use of neuroprotection during endovascular aneurysm exclusion remains subject to discussion. The presence of unstable atherosclerotic plaques in the aneurysm sac is considered a relative contraindication for endovascular procedure, or requires the use of neuroprotection during the procedure (75, 78). Maras et al. in a review article describing 20 post-traumatic internal carotid aneurysm repairs with stent-grafts found only two cases of neuroprotection use: one distal and one proximal (77, 79, 80).

The main indications for stents in carotid aneurysms are atherosclerotic stenoses in patients with multiple co-morbidities, recurrent stenoses after previous endarterectomies, or other operative repairs and difficult surgical access to high internal carotid aneurysms (81, 82, 83).

Anticoagulant prophylaxis and treatment are crucial factors in the success of endovascular methods. There is still no unanimous standpoint on the use of antiaggregation and anticoagulant drugs in the perioperative period and after stent-graft implantation. Some authors recommend using two antiplatelet drugs, while others give acetylsalicylic acid combined with heparin for one day. Some others use two antiplatelet drugs with heparin for 3 postoperative days (77). McCready et al. recommend the use of clopidogrel for 6 postoperative weeks, followed with permanent warfarin treatment (71). After the analysis of The Cleveland Carotid Stent Registry, F. M. McKevitt et al. suggest combined therapy of clopidogrel and acetylsalicylic acid for the whole prosthesis healing period – at least 4 weeks (84).

CONCLUSION

In the past few years, many articles about carotid aneurysms have appeared in the literature. These articles are connected with better detectability of this vascular pathology, thanks to the development of new diagnostic methods and the better availability of these methods. Moreover, an increased number of patients with posttraumatic aneurysms after car accidents and aneurysms as a result of extreme sports injuries can be noticed. As it was observed in the past, armed clashes and the victims of these clashes are a source for many valuable findings that may contribute to the development of new operative methods. A few
years after the Balkan War, an increased number of carotid aneurysms was reported by doctors working in this region. In 2007, Radak et al. described 91 aneurysms in 76 patients, one of the most numerous groups that may be found in the literature (45). Others have observed soldiers fighting in Iraq and Afghanistan who suffered neck trauma during military service. Upon return to the United States, all of these soldiers underwent precise diagnostic examinations of carotid vessels. Some of them had false aneurysms that developed sometime after the trauma without any neurological defects (85).

In the recent literature concerning the treatment of carotid aneurysms, a tendency for less invasive treatment may be noticed. This indicates a constant development of endovascular methods and the expansion of indications for the application of these methods.

REFERENCES

29. Amirjamshidi A, Abbassion K, Rahmat H: Traumatic aneurysms and arteriovenous fistulas of


Received: 24.09.2007 r.
Adress correspondence: 03-242 Warszawa, ul. Kondratowicza 8