Are the carotid kinking and coiling underestimated entities?

Da li su morfološke abnormalnosti karotidne arterije (kinking i coiling) beznačajne?

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Key words:
carotid artery, internal; carotid artery diseases; risk assessment; cerebrovascular disorders; ultrasonography; angiography; vascular surgical procedures.

Ključne reči:
a.carotis interna; aa. carotis, bolesti; rizik, procena; cerebrovaskularni poremečaji; ultrasonografija; angiografija; hirurgija, vaskularna, procedure.

Introduction

The terms “kinking” and “coiling” were first thoroughly explained and presented to the professional public in the early sixties by a group of authors – Weibel et al. 1 and Metz et al. 2 (Table 1). According to this classification coiling (loop) is defined as the elongation of the internal carotid artery (ICA), which causes the appearance of S- or C-shape angulation or the appearance of circular formations. Kinking is one of the variants of a coiling, or angulation of one or more segments of the ICA and could be associated with local or global cerebral symptoms (Figure 1).

The occurrence of carotid abnormalities (CAs), verified by arteriography varies between 10% to 43% of the cases 1-3. Kinking occurs in 5%–16% of patients 4, 5, mostly elderly men, and is associated with atherosclerotic pathology, while coiling is more common in younger women 6. Up to 75% of morphological abnormalities were localized 2–4 cm proximal to the carotid bifurcation 7. The use of ultrasound examination in the ongoing clinical practice has enabled rapid, morphological and also functional studies of CAs 7. In the study of Del Corso et al. 8 more than half of the number of patients with cerebrovascular symptoms had some forms of CAs diagnosed by ultrasound technique.

Table 1

<table>
<thead>
<tr>
<th>Type of CAs</th>
<th>Forms of morphologic abnormalities of ICA</th>
<th>Symptoms</th>
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<tr>
<td>Kinking</td>
<td>Mild – angulation of elongated ICA with an angle ≥ 60 °</td>
<td>±</td>
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<td></td>
<td>Medium – angulation of elongated ICA with angle between 30 ° – 60 °</td>
<td>±</td>
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<td></td>
<td>Severe – angulation of elongated ICA with angle &lt; 30 °</td>
<td>+</td>
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<tr>
<td>Coiling</td>
<td>Angulation of elongated ICA with the formation of S or C shape, or appearance of circular formations</td>
<td>±</td>
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CAs – carotid abnormalities

CAs are rarely observed in children and young, and could be explained by congenital etiology in these cases. During the third trimester of intrauterine development occurs the lowering of the heart in the chest (descensus cordis) and any difference in speed of growth of the skeleton and supraaortic branches can lead to the associated vascular abnormalities of blood vessels, which are usually bilateral 8, 9. These disorders of embryonic development explain the presence of CAs in newborn babies and fetuses 1. Beigelman et al. 10 in a series of 885 patients, aged 1 to 90 years, showed no difference in the prevalence of kinking or coiling among age groups, favoring the embryogenic mechanism of the development of CAs.

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Among children coiling is often the reason for reduced cognitive capacity, slow neuropsychological development as well as focal or grand mal convulsions. In contrast to congenital, acquired form of kinking and coiling is most commonly associated with aging, hypertension and traditional risk factors for atherosclerosis. It is proved that the arterial blood vessels are prone to deformation when exposed to elevated blood pressure. Hypertension causes the extension of smooth muscle cells of the media of blood vessels, which is associated with elastin loss and fragmentation of elastomers. Progression of this process eventually leads to abnormal elongation of blood vessels. Pancera et al. showed that the prevalence of hypertension in patients with kinking is significantly higher than with patients without morphological abnormalities of blood vessels. However, a clear link between vascular risk factors and kinking and coiling is still not fully understood.

Clinical features and diagnosis of carotid abnormalities

The appearance of symptoms of kinking and coiling of the carotid arteries is associated with intracranial and extracranial occlusive disease, frequent and sudden variations in blood pressure as well as the unfavorable position of the head and neck. In the chain of pathological dynamics, associated atherosclerotic stenotic changes represent the second link, but their development can transform kinking in the hemodynamically significant anomaly. It is sometimes difficult to prove the connection, but isolated CAs can lead to cerebral symptoms, without the presence of atherosclerotic changes.

Symptoms presentation most commonly appear due to transitory hypotension during sleep or sudden and extreme movement of the head and neck. In the majority of patients, symptomatology generally withdraws with the return of the head in neutral position. However, kinking without atherosclerotic plaque, although often present, rarely can be considered as the cause of stroke, as compared with carotid stenotic diseases. Ischemic cerebrovascular accident occurs in about 11%–33% of cases with CAs.

It is pathognomonic that typical (stereotypical) movements of the head and neck can induce ischemic symptoms, with ipsilateral rotation of the head usually leading to the biggest reduction of the carotid flow. Extreme flexion or extension of the neck can manifest a pre-existing kinking leading to almost complete occlusion of viable arteries. Hemodynamic mechanism of brain ischemia involves episodic or permanently reduction in perfusion through the anomalous part of the artery. If baroreceptor activity is preserved, due to specific rotation of the head and neck, the reduction of lumen of kinking or coiling occurs and the increased pressure level proximal to the compression may appear. As a result, a decrease in perfusion pressure occurs with symptoms of a global ischemia of the brain. It has been proven that a reduction regional cerebral flow in extracranial elongation of carotid segments appears with 30–35 mL per min (normal range 50–60 mL per min per 100 g. brain tissues). Importance of kinking becomes significantly increased if there is associated stenosis of carotid bifurcation.

In the diagnostic process of CAs, duplex ultrasonography as an initial diagnostic tool is most commonly used. However, for these patients who have an ultrasound verified kinking or coiling with the angulated segment speeds over 100 cm/s, as well as ones with cerebrovascular symptoms, additional diagnostics procedures are required. Angiography [multidetector computed tomography angiography and magnetic resonance imaging (MRI)] allows visualization of the intracranial and extracranial part of carotid vessels, while selective angiography is reserved for CAs of non-atherosclerotic origin, because of their frequent association with anomalies in other arterial segments. Hemodynamically significant kinking is the one with angulation less than 60°, and with the flow rate within a limited segment of over 200 cm/s, while the coiling is significant if there is a full cir-

cle segment formed (360°). Computed tomography (CT) and MRI of the brain provide information about ischemic changes in brain structure and serve as a differential diagnostic tool, which can exclude similar symptoms, but of another etiology.

**Treatment of carotid abnormalities: indications and surgical techniques**

Choosing the right method for treatment of CAs is still a subject of numerous debates. A large number of studies 1, 2, 3, 5, 9, 11, 21, 24, 27–30, 31, 32 have shown the efficiency and safety of surgical correction of kinking and coiling. However, the first randomized, prospective study that to some extent clarified this dilemma was published in 2005 by Ballotta et al. 31. They concluded that the surgical correction of symptomatic CAs is better in preventing stroke compared with best medical therapy, as well as it completely eliminates the symptoms in patients with non-hemispheric symptoms.

Similar with these findings, Radak et al. 32 underlined that operative management is best therapeutic option for patients with near total occlusion of ICA. Surgical correction of symptomatic kinking and/or coiling makes up about 5% of all reconstructive procedures for cerebrovascular insufficiency. 24, 33

Surgical correction of CAs is indicated for: patients with transient ischemic attacks (focal, hemispheric ischemic symptoms); patients in whom angiography demonstrated hemodinamically significant kinking and/or coiling; if CT and MRI examination excludes other significant ischemic brain injury; and in symptomatic and asymptomatic patients with one-sided CAs and occlusion of contralateral carotid artery; bilateral kinking and/or coiling, with the correction of one side first, if the symptoms do not disappear, then the other side as well, and patients with simultaneous lesions of vertebral artery 6, 31, 34, 35. Kinking and/or coiling are predisposing factor for ICA dissection (Figure 2). Even associated with dramatic ultrasound finding, treatment of these lesions remains medical in the majority of cases, with anticoagulants and antiplatelet drugs. 36 However, bilateral high kinking with distal dissection of ICA is indicated for surgical repair but only in cases with confirmed cerebral symptoms. This type of intervention is associated with frequent peripheral nerve lesions and high mortality rate. 36

The surgical technique of resection of tortuous segment, with dilatation and reimplantation has become the method of choice in treating carotid kinking and coiling. If there are atherosclerotic changes associated with CAs in the same procedure, eversion carotid endarterectomy can be done. 21, 31, 35. Correction of kinking and coiling is performed with peroperative mortality rate below 1% and low postoperative morbidity 11, 21, 26, 27. Similarly, Radak et al. 37 reported low total mortality rate after endarterectomy procedures of carotid artery. 37 Neurological symptoms of cerebral ischemia retreat even in 85% of patients. The lowest percentage of improvement occurs in patients with global (nonspecific) neurological symptomatology, as well as in the case of severe preoperative neurological deficit. 23, 38, 39. Recurrences of kinking and coiling are rare and can appear in patients with uncontrolled hypertension 6, 19, 40, 41.

**Fig. 2 – Kinking with dissection of internal carotid artery**

**Conclusion**

Morphological CAs, kinking and coiling, are common in the general population, with still unexplained etiopathogenesis and the course of the disease.

Despite the great success of surgical reconstructions, because of the lack of multicentric, randomized, prospective studies, appropriate therapeutic treatment is still subject of numerous debates.

The purpose of this work was systematization of current data for preparation our multicentre, randomized, prospective trail.

**Acknowledgments**

This manuscript was partly funded, grant number 41002, by the Ministry of Science and Technological Development of the Republic of Serbia.

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