Vertebral Artery Dissection after Radiation Therapy: A Case Report

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CADs are characterized by a low rate of recurrence, and familial occurrence is low. These observations suggest that short-lived trigger mechanisms may play an important role in the pathogenesis of CAD. We report for the first time a case of VAD in a man who received ipsilateral head-and-neck radiation therapy for a cancer of the base of the tongue, to illustrate that CAD should also be included among the radiation-induced vascular diseases.

Case Report

A 53-year-old man, successfully treated for a squamous cell carcinoma of the base of the tongue with interstitial radiation (32 Gy) and external radiation therapy (46 Gy), incurred 8 months later a massive stroke due to embolic occlusion of the posterior cerebral artery at origin.

The patient initially presented with acute onset, 1 day before, of ipsilateral constriction occipital headache and (simultaneously) neck pain, accompanied by nausea and vomiting. The patient was not engaged in any activity that would promote artery dissection; there was no history of antecedent trauma and no premonitory clinical history of cerebrovascular attacks. At admission to our Department, neurological examination revealed left hemiparesis, left hemihypesthesia and homonymous left hemianopsia; the blood pressure was 128/84 mm Hg.

Doppler ultrasonography showed signs of severe obstruction of the right vertebral artery in the absence of atherosclerosis; the other cervical vessels were normal.

Brain magnetic resonance imaging (MRI), in particular the diffusion-weighted images, revealed acute infarctions in the right occipital lobe, the right medial temporal lobe, the right ventral basal thalamus, and the right posterior limb of the internal capsule (probably abnormal origin of the anterior choroidal artery; fig. 1). Sagittal MRI showed a typical intramural dissecting hematoma in the vessel wall of the right vertebral artery at origin.

The patient was treated with heparin initially, and discharged on warfarin with a plan for anticoagulation for 6 months.
Artery Dissection after Radiation Therapy

Discussion

Vascular abnormalities are known to be an important cause of postirradiation complications.

We describe for the first time the occurrence of VAD in a patient who received head and neck irradiation for the treatment of a cancer of the base of the tongue. The V3 and V4 segments of the vertebral artery were unusually involved in our patient; in fact, the most frequent localization of dissection is the distal V1 and the proximal V2 segment (at the level of the C6 vertebra) [3]. This case report further confirms that the resolution of MRA/MRI now approaches that of the conventional invasive catheter angiography. The diagnosis can be established if a typical intramural vessel wall hematoma is seen on T1-weighted MRI in combination with MRA findings of irregular artery stenosis or occlusion.

Several types of radiation damage to the cervicocerebral arteries (especially to the internal carotid artery) after head and neck irradiation have already been described. The most frequent complication is the increased intima media thickness [4, 5] and the late development of an atherosclerotic-like disorder in the arteries included in the radiation fields [6, 7]; a carotid rupture, when radiotherapy was associated with radical neck dissection [8, 9], and an early arterial occlusion within months after radiotherapy [10] have also been reported.

It is possible that the apparent injury to the large arteries is due to vasa vasorum occlusion because the microvasculature is especially vulnerable to radiation damage. There is no universal agreement as to which wall is the primary site of CAD. A rupture within the connective tissue and vasa vasorum of the media is the most probable initial event in dissection [11], even if other authors consider that an intimal tear may occur, which allows blood under arterial pressure to enter the wall of the artery [12].

Radiation therapy may also be responsible for endothelial injury which results in dissection of the vertebral artery. Irradiation may contribute to endothelial dysfunction by increasing free radical formation and oxidative stress, production of reactive oxygen species or by selective impairment of the nitric oxide component of endothelium-dependent vasodilation, similar to that described in experimental studies [13, 14]. It has been demonstrated [15] that in irradiated human cervical arteries especially the nitric oxide- and prostacyclin-mediated endothelium-dependent relaxation is impaired; the impaired nitric oxide-mediated relaxation is associated with a lack of endothelium nitric oxide synthetase expression. Recently, an evidence of impaired endothelium-dependent vasodilatation in CAD patients that is not the result of stroke [16] has been described, suggesting that an underlying arteriopathy of the arterial wall layers may predispose to CAD.

Moreover, endothelial injury induces morphologic changes of the internal elastic lamina, smooth vessel proliferation, various matrix abnormalities involving one of the fibrillar components or its enzymatic regulation, arterial wall neoangiogenesis and finally artery dissection.

Fig. 1. Diffusion-weighted MRI showed hyperintense areas in the right occipital lobe, the right medial temporal lobe, the right ventral basal thalamus, and the right posterior limb of the internal capsule, indicating acute ischemic infarctions.

Fig. 2. Sagittal T1-weighted MRI showed a typical intramural dissecting hematoma in the wall of the right vertebral artery as rounded hyperintense signal; fat suppression techniques differentiate clearly the intramural hematoma from the surrounding soft tissues.

Fig. 3. MRA revealed irregular stenosis in the V3 and V4 segments of the right vertebral artery.
Therefore, the impaired endothelial function in irradiated human blood vessels may explain the development also of this vascular complication, and we believe that in our case a pathogenetic link might exist between radiation and VAD occurrence.

We demonstrated that it is important to identify patients who have received head and neck radiation therapy as being at risk also for dissection of cervicocerebral arteries. These subjects should be carefully evaluated for carotid or vertebral artery disease; screening and treatment of additional cerebrovascular risk factors which contribute to stroke development is recommended, especially in radiotherapy patients with a favorable prognosis.

References