Sir,

Localized progressive intravascular accumulation of atheromatous deposits represents the leading cause of stenosis or occlusion of the brain supplying arteries in adults. These atherosclerotic changes may be found extra- and intracranially within the anterior and the posterior circulation with varying extent.\(^1\)\(^,\)\(^2\) Rarely, the spontaneous development of an extensive extracranial-intracranial collateral network that resembles the late stages of Moyamoya disease (MMD), i.e., a Moyamoya-phenomenon, is observed.

A 36–year-old woman presented with a five-year history of intermittent episodes of blurred vision and vertigo. Cranial CT showed mild supratentorial atrophy and borderzone infarctions with right hemispheric accentuation. Diagnostic cerebral panangiography demonstrated a left-sided internal carotid artery (ICA) occlusion. The left external carotid artery (ECA) led to a significant intracranial blood supply using physiological extracranial-intracranial bypasses via the vessels of the mucous membrane of the ethmoidal sinus and the nasal meatus. Transdural anastomoses were observed involving the middle meningeal and the occipital arteries (Figures 1, 2). Intracranially, extensive leptomeningeal collaterals were seen between the middle cerebral artery (MCA) and anterior cerebral artery (ACA) vascular territory on the left side. Right-sided injection showed an occlusion of the ICA and revealed multiple extracranial-intracranial anastomoses involving the pharyngeal, meningeal and the occipital branches of the ECA (Figure 2). This pattern of blood supply led to an atypical and delayed intracranial opacification with leptomeningeal collateralization between the MCA and the ACA vascular territory (Figure 1). Neither right nor left-sided injection revealed any parts of the circle of Willis, thus being indicative for complete obliteration of the proximal segments of the basal cerebral vasculature. Angiography demonstrated a distal basilar artery (BA) occlusion and a collateral network arising from the right posterior inferior cerebellar artery (PICA) and the left vertebral artery. The paired functional blood flow (rCBF) study using stable Xenon-CT showed a symmetrical rCBF in all vascular territories and a preserved cerebral perfusion reserve.

The stenosis and occlusion of cervicocerebral arteries represents one of the major manifestations of atherosclerosis in adulthood. It affects the extracranial segments of the brain supplying arteries with preference for the carotid fork, leading to stenosis or occlusion of either the ICA or the common carotid artery.\(^2\) The extent and the progression of the atherosclerotic changes within the affected vessels trigger the development of collateral pathways as a physiological response, in order to avoid cerebral ischemia. The type of collaterals recruited depends on the topology, the extent of the atherosclerotic lesion, its hemodynamic relevance and the temporal course of the disease progression.\(^3\)\(^,\)\(^4\)

The complete functional reconstruction of the ICA through skull base collaterals from the ECA, however, as observed in the present case, represents a rare condition. The functional recruitment of the extracranial circulation as collateral pathway is a well recognized phenomenon in patients with MMD.\(^5\) Moyamoya disease, a disease of unknown etiology that mainly affects children and...
young adults shows the progressive narrowing of the terminal portions of the internal carotid arteries and the development of a network of pathological collaterals in the vicinity of the steno-occlusive lesions. In addition spontaneous extracranial-intracranial collaterals develop using the ECA and vessels of the mucous membrane of the ethmoidal sinus and the nasal meatus.[5] The development of the spontaneous collaterals is assumed to be triggered by the chronic hypoperfusion in these patients. Although the etiology of the vascular changes in the present case is known, the angiographic pattern detected here resembles the late stages of MMD rather than the classical collateralization pattern due to atherosclerosis. Therefore, the capability of complex collateral pathway formation as observed here suggests shared mechanisms for the compensation of chronic ischemia in atherosclerotic cerebrovascular disease and MMD.

Peter Horn, Johann Scharf*, Peter Schmiedek

Departments of Neurosurgery and *Neuroradiology, Universitätsklinikum Mannheim, University of Heidelberg, Germany

References


Accepted on 12-02-2007

Scalp and intracranial metastasis from pleomorphic adenocarcinoma of the parotid gland

Sir,

Adenocarcinoma of the salivary gland is an aggressive tumor with the tendency to metastasize locally or through lymphatics. Intracranial metastasis through hematogenous route from salivary gland malignant tumor is uncommon and most of the reported cases are from the adenocystic carcinoma of the parotid gland.[1] We are reporting a case of scalp and supratentorial cerebral metastasis from pleomorphic adenocarcinoma of the parotid gland, which has never been reported in the English literature.

A 30-year-old man presented with painless swelling scalp in the midline of vertex for two months, progressively increasing weakness of right half of the body, headache and vomiting for one month. He was an operated case of pleomorphic adenocarcinoma of the left parotid gland. Parotidectomy with Type 2 neck dissection and removal of left facial nerve was done two months ago followed by radiotherapy. Neurological examination revealed papilloedema, facial nerve palsy and right side spastic hemiparesis with intact higher mental functions. There was a non-tender, firm immobile swelling in the scalp of about 1.5x1.5 cm size in the midline. The MRI brain revealed a hetrointense lesion in mid-parasagittal region enhancing brightly on contrast with dural tail, perilesional edema and mass effect. There was an additional mass, approximately 1.2x1.2 cm in size which was also enhancing on gadolinium contrast, arising from the scalp just above the intracranial lesion without any continuity with underlying bone or cerebrum [Figure 1]. Other hematological and biochemical examination was normal.

Left parietal craniotomy and total excision of intracranial tumor and excision of scalp mass was done. Both the scalp and intracranial tumors were highly vascular. The intracranial tumor had no continuity with the scalp lesion. Intracranial tumor was extra-axial, attached to falx, grayish, firm with well-defined plane of cleavage. Postoperative CT head revealed no residual tumor. The histopathological examination of the scalp and the intracranial mass was consistent with metastasis from the malignant pleomorphic adenoma. Patient was lost in the follow-up.

Adenocarcinoma of the salivary gland is a very aggressive tumor and metastasizes to the lung, bones, liver, choroid and brain.[1,2] The involvement of brain and scalp is rare and only 11 cases of intracranial metastasis from parotid carcinoma have been reported...