Accidental Transtumoral Microparticle Embolization of Eloquent Brain Areas in a Case of Large Temporofrontal Meningioma

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Summary

We report a case of transtumoral diapedesis of microparticles during preoperative embolization of a large symptomatic temporofrontal meningioma, resulting in severe neurological deficit. In retrospective analysis of angiographic images, the pattern of supply to the tumour was found to be predictive for wide anastomotic channels connecting the dural and pial supply, thereby increasing the risk of the procedure. Recognising this pattern can prevent this rare cause of complication in a still controversial procedure.

Introduction

Preoperative embolization of meningiomas is routinely performed in many hospitals depending on the personal preferences of the neurosurgeon involved. Despite its widespread use, data on the benefit of the procedure are limited. Therefore embolization of meningiomas must not add to the risks of a scheduled operation and should be free of complications.

Despite all efforts serious neurological complications do occur, as well documented in the literature. A rare cause of complication is transtumoral embolization of microparticles which, as we suspect in our case, could have been prevented by thorough reading of angiographic images, as shown below.

Case Report

We describe the case of a 51-year-old woman who presented with a four month history of severe headache, amnesic aphasia, intention tremor of both hands and coarse tremor of the head. Primary seizure led to admission in our hospital, where a 6*4*5 cm sized meningioma of the right temporofrontal convexity was diagnosed in CT.

Three days prior to scheduled surgical removal, the patient was sent to our department for routine particle embolization. Due to an allergic reaction to contrast agents the patient received an infusion containing 60 mg diphenhydraminhydrochlorid, 1g prednisolone and 40mg pantozol before the procedure, which was performed in regional anesthesia.

As a first step complete angiographic assessment of the vascular anatomy of the head was performed. Angiography of the right internal carotid artery (ICA) showed a highly vascularized tumour displacing the M2 to M4 segments of the right middle cerebral artery (MCA) medially and cranially. The tumour had rich pial supply from the MCA branches, supplying the entire medial half of the tumour, as well as a large superiorly located compartment, filling both the medial and lateral half of the tumour (Figure 1A). Other ICA branches supplying the tumour were not identified.

Right external carotid artery (ECA) an-

giogram (Figure 1B) showed an enlarged middle meningeal artery (MMA), with a hypertrophied frontal branch as the main contributor of vascular supply to the tumour. Four additional small tumour supplying branches were seen coming off the parietal branch of the MMA and no connections to the ipsilateral ophthalmic artery were found. There was no visible supply from any other vessels from the ipsi- or contralateral side.

It was decided to primarily embolize the frontal MMA branch, being the main supply to the tumour. After superselective microcatheterization of this artery, angiography again showed no connection to the ophthalmic system and a strong tumour blush. The microcatheter was assumed to be well distal to a possible cavernous branch of the MMA (Figure 1C) and therefore embolization was performed from this position with 125-250 μ m bead-block (preformed, deformable microspheres consisting of a biocompatible, acrylamide polyvinyl al-

cohol macromer) microparticles (Biocompatibles, Chapman House, Farnham Business Park, Weydon Lane, Farnham, Surrey, GU9 8QL, UK) in a free flow technique with no visible reflux, until almost complete stasis was achieved in this vessel. Despite minor headaches this was very well tolerated by the patient. Subsequently the microcatheter was placed in the parietal branch of the MMA to occlude the remaining small feeders, where embolization was performed with the same technique.

At this point the patient began to complain of nausea and dizziness, and her speech was slightly slurred. Control angiography of the right ECA showed complete devascularisation of the tumour, in rICA angiogram a dramatic reduction of pial supply was seen with only small compartments remaining posteriorly and inferiorly (Figure 2). After control angiography the patient deteriorated rapidly with British Medical Research Council (BMRC) grade 3 paresis of the left upper extremity, progressively slurred speech,

increasing nausea and headache.

A bolus injection of hydrocortisone was administered in the angiosuite and the patient was immediately transferred to magnetic resonance imaging (MRI) with stable symptoms.

MRI showed cortical swelling and diffusion restriction in the right middle frontal gyrus (in the premotor area), small DWI positive lesions in the centrum semiovale and small cortical and subcortical DWI positive lesions in the parieto-occipital area on the right, corresponding to acute ischemic infarctions (figure 3). There was no marked swelling of the tumour compared to preinterventional CT.

In the intensive care unit (ICU) the patient developed facial nerve paresis on the left side, left neglect and the left arm paresis deteriorated to BMRC grade 4 despite high cortisone levels. High glucocorticoid levels were maintained for her entire stay in ICU. Facial nerve paresis improved rapidly and was gone on the fourth postinterventional day. Paresis improved

over the first eight days to BM-RC grade 3, the neglect did not improve. On the ninth postinterventional day surgical resection of the meningioma was performed and well tolerated by the patient.

Intensive physical therapy in the postoperative period improved the patient's condition and she was discharged with second to third degree paresis of the left arm and unchanged neglect.

Discussion

Literature data on the benefit of preoperative embolization of meningiomas compared to surgery alone are limited. Retrospective studies show a beneficial effect in terms of estimated blood loss at surgery^{1,2}. One prospective study found differences in intraoperative blood loss only in completely devascularized meningiomas, whereas other parameters did not significantly differ³. Data on the neurologic risk of embolization are also sparse and controversial. In small, unselected studies no complications could be found⁴ or occurred at rates of 12 to 16%⁵. In a large series of selected skull base meningiomas 21.6% of patients had neurologic deficits while 9% had permanent deficits⁶. Bendzus retrospectively found in his series of 185 patients with unselected meningiomas an overall rate of neurologic complications of 6.5% with persistent deficit in 2.2% and death in 0.5%. These were either caused by ischaemia (thromboembolism in most of the cases, but also one case of leptomeningeal anastomoses) or hemorrhagic events (extremely rare in literature with nine cases found by us⁶⁻¹³). Bendzus stated that all his ischemic events were unpredictable.

In our case correct interpreta-

Figure 1 A) In arterial (above) and venous (below) phase ICA angiogram, pial supply to the meningioma is seen. Note the compartmental supply to the medial half of the tumour including a small superior compartment (arrows). B) In arterial (above) and venous (below) phase ECA angiogram, supply to the lateral half of the meningioma is seen. Note the triangular filling defect in a superiorly located tumour compartment (arrows), which is supplied via pial branches of the MCA (A). C) Superselective angiography of the frontal branch of the MMA (left) shows filling of the primarily ICA supplied tumour compartment (arrow). Note that in the ECA angiogram (right) this compartment did not fill.

Figure 2 Lateral view postembolization angiography of MMA (left) and ICA (right) show subtotal devascularization of the tumour with small compartments remaining posteriorly and inferiorly. Note the radial subtraction artefact due to intratumoral stases of contrast agent and the delayed perfusion of embolized vessels in MMA angiography.

tion of angiographic images could almost certainly have avoided the complication, as evaluation of angiographic images showed us retrospectively.

Figure 1A shows supply of the meningioma from pial branches of the middle cerebral artery. The MCA supplies the medial third of the tumour and a large superior compartment reaching the lateral border of the tumour.

As expected, angiography of the external

carotid artery shows no supply to this compartment, but when the microcatheter was placed in the frontal branch of the middle meningeal artery with the intention to embolize it, angiography showed blush of parts of the very compartment previously seen by ICA -injection only. So superselective angiography of the frontal MMA- branch shows a contribution to the superior, predominantly MCA supplied compartment. Our proposed explanation is the vascu-

Figure 3 FLAIR (left) and DWI (right) sequences showing the meningioma (*) as well as cortical oedema and DWI restricted areas (arrows), confirming ischaemia.

larity of the tumour, in particular a wide anastomotic network between the tumour supplying MMA and MCA branches. Rich pial and capillary anastomoses in meningiomas are well known in the literature¹⁴. We assume that this arterial mesh is permeable for contrast material and microparticles of certain size, leading to transtumoral microparticle embolization.

Sindou and Alaywan¹³ noted that subpial dissection of meningiomas may correlate with surgical morbidity. They reported that surgical cleavage was often subpial when the tumour had a cortical-pial vascular supply, and that there was positive correlation between the cortical-pial blood supply and the tumour size. Nakasu et al¹⁴ showed tumours attached directly to the pia mater or invasive to the brain or the tumour capsule fused with the pia mater, where in both instances, the subarachnoid vessels were entrapped in the tumour or tumour capsule, and the tumour was receiving a pial blood supply as noted on angiography.

Recognizing the above pattern of supply

and, as we propose here, deducing the presence of anastomoses, should lead either to an approach via a different vessel, or to interrupting the procedure. Surprising changes in compartmental supply are a strong clue for the presence of a wide anastomotic network in the tumour with a high risk of transtumoral embolization of particles, leading to neurologic deficits.

Conclusions

When analyzing the diagnostic angiographic workup prior to tumour embolization, attention should be paid not only to anastomoses between ECA and ICA branches but also to intratumoral anastomoses, which, we suggest, were missed in this case. Dynamic changes in supply to a compartment are a strong clue for a wide anastomotic network and special care should be paid to this to prevent ischemic complications.

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