Cranial nerve III palsy

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The onset of cranial nerve (CN) III palsy in a patient with an unruptured posterior communicating artery (PCoA) aneurysm is indicative of aneurysm expansion requiring urgent treatment. Although both surgical clipping and endovascular coil embolization are associated with the recovery of CN III function, surgical treatment seems to provide a greater chance of improvement. Whether or not decompression of the nerve itself following aneurysm exclusion improves the chance of recovery is a matter of controversy. In this issue of the Journal of Neurosurgery, Güresir and coworkers1 present a systematic review of published studies as well as 6 cases of their own and conclude that CN III decompression after clipping does not provide any additional benefit. In their multivariate analysis, only the degree of CN III function before surgery (complete vs partial paralysis) was a predictor of nerve outcome, with patients with incomplete palsy experiencing higher recovery rates than those with preexisting complete palsy.

Proposed mechanisms by which an aneurysm can cause CN III palsy include direct mechanical compression and aneurysm pulsation against the nerve. However, other important factors at play are growth rate of the aneurysm, compromise of CN III vascularity, and accompanying inflammatory reaction,2 rather than mechanical compression alone. As noted by Professor Yaşargil in his monumental monograph,3 it is not unusual to see severe compression of CN III from a PCoA aneurysm without any compromise of function, while in other cases simple contact between the aneurysm and an otherwise intact nerve is associated with severe palsy.

The study by Güresir et al. is limited by the variable quality of the data analyzed. Nevertheless, their conclusions support the notion that decompression of the nerve after aneurysm clipping may not improve the rate of recovery and should probably be avoided in the presence of significant adhesions of the sac to CN III because of the risks of added mechanical trauma.3 As the authors correctly point out, recovery of CN III function is maximal at 1 year and unlikely to progress after this time interval.

Disclosure

The author reports no conflict of interest.

References


Response

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We thank Dr. Lanzino for his thoughtful comments regarding our paper. Posterior communicating artery aneurysm–related oculomotor nerve palsy (ONP) has mainly been attributed to the direct compressive effect of
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the aneurysm sac or aneurysm pulsation.\textsuperscript{2,4} Several other possible mechanisms of injury, for example, local inflammatory reaction, blast effect from aneurysm rupture, and effect of subarachnoid blood in ruptured aneurysms, have been discussed previously.\textsuperscript{3} Posterior communicating artery aneurysm–related ONP improves after surgical clipping and endovascular coil embolization; however, surgical clipping seems to result in a greater chance of improvement.\textsuperscript{1}

Surgical treatment resolves the mass effect of the aneurysm but may expose the oculomotor nerve to additional injury caused by manipulation. In our systematic review, we found that additional decompression of the oculomotor nerve after clipping the PCoA aneurysm had no additional benefit on the resolution of ONP.

Selecting the treatment modality for an aneurysm is a complex and individualized decision that should be based on patient- and lesion-specific factors. In patients with PCoA aneurysm–related ONP, surgical treatment leads to better recovery and supports clipping, all else being equal, as the first-choice treatment in this setting. Additional oculomotor nerve decompression after clipping does not provide any additional benefit and should therefore be avoided to reduce the risk of mechanical injury due to manipulation.

References


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