Cerebral Perfusion Changes in Chronic Subdural Hematoma

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To the Editor:

WITH INTEREST WE HAVE READ the recent article of Slotty and colleagues. They performed computed tomography (CT) perfusion imaging in patients with chronic subdural hemorrhage to investigate global and local (hematoma-related) cortical perfusion and volume parameters and their association with neurological deficits. Mildly affected patients (median National Institutes of Health Stroke Scale of 3) before surgical decompression were included, unfortunately without reporting clinical or radiological follow-up.¹ In our opinion, their interpretation and statement that intact local brain perfusion autoregulation is present in these patients seems preliminary for several reasons. First, dimensionless “relative” values of cerebral blood flow (CBF) and volume (CBV) in presumed vascular territories were used. It is not clear what served as the reference region of interest for this calculation. Using (mirrored parts of) the compressed contralateral hemisphere is not ideal. Therefore, conclusions regarding hyperaemia, oligoemia, or autoregulation status as explanations for their group-averaged findings (and individual associations with neurological deficits) is, in our opinion, not possible.² Second, the researchers state that the brain maintains normal perfusion despite the overlying hematoma, which is contrary to their findings of a significant difference between the CBF and CBV values below the hematoma and the contralateral hemisphere. A simultaneous increase in CBF and CBV does not imply intact autoregulation, which is a mechanism of maintaining stability of CBF, and has not been previously reported to lead overshoot and hyperemia. Conversely, with hypertension (blood-pressure values were not measured in this cohort), the observed increases in CBF and CBV could represent local vasoparalysis and pressure passivity (i.e., impaired autoregulation).³ On the other hand, these findings could be a consequence of factors such as the release of vasoactive substances from the hematoma or venous congestion resulting from outflow obstruction caused by the hematoma, which were not discussed.⁴ Third, CBF and CBV were increased in the cortex below the hematoma, but global perfusion was comparable between the hemispheres. To accomplish the latter, cortical areas with reduced or elevated CBF and CBV have to be present elsewhere in the brain or the cortical hyperemia is just small and, overall, did not signifi-

cantly contribute to the whole-hemisphere values.⁴ Fourth, the interpretation of mean transit time as abnormal in both hemispheres is dangerous because, in the referred study, no patient data could be found.⁵ And finally, the exclusion of more-severe patients limits the generalizability of their observation, in particular, because deteriorating patients are expected to show more-severe autoregulation disturbances. Performing a perfusion CT in combination with the obligatory unenhanced CT brain is not expected to cause major delay before surgical decompression.² The comparison of perfusion parameters of compressed cortical tissue between mildly and severely affected patients would provide at least some support of the assumptions in the discussion and conclusion (increased local intracranial pressure, intact autoregulation, or symptomatic borderline perfusion).

References

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