Pituitary apoplexy can be a fatal condition. It rarely causes cerebral ischemia through compression or vasospasm. We report a case of pituitary apoplexy causing compression of bilateral anterior cerebral arteries and leading to bilateral caudate infarcts.

Report of a Case

An 81-year-old woman known to have hypertension and a pituitary macroadenoma compressing the prechiasmatic optic nerves and causing significant bilateral blindness presented with stupor and generalized weakness. On the day of presentation, she had significant weakness and was not able to stand. She was brought to the emergency department and was found to be stuporous, hypotensive, and in shock, with the lowest recorded systolic blood pressure of 76 mm Hg. She was subsequently resuscitated with intravenous fluids, supported with 4 intravenous pressors, and endotracheally intubated. Neurological examination revealed a stuporous woman, not opening her eyes or following commands but with intact brainstem reflexes. She had an asymmetric quadriplegia with left hemibody weakness greater than right, left-sided spasticity and hyperreflexia, and upturning toes bilaterally. Brain magnetic resonance imaging showed a sella-based mass with intrinsic hemorrhage (Figure, A) and infarcts in the anterior cerebral artery (ACA)-middle cerebral artery (MCA) and MCA-posterior cerebral artery (PCA) watershed zones (Figure, B).Brain magnetic resonance angiography showed anterior displacement of bilateral ACAs and anterior communicating artery with patency of the distal ACAs (Figure, C). Diffusion-weighted imaging additionally showed bilateral caudate infarcts (Figure, D). During her hospitalization, her condition did not improve and her family decided to transition her to comfort care measures.

Discussion

Pituitary tumor apoplexy is an acute clinical syndrome caused by either hemorrhage or infarction of the pituitary gland, leading to mass effect on surrounding structures causing altered mental status, headache, disturbed vision, and pituitary dysfunction. Only a few case reports in the literature describe cerebral ischemia due to pituitary apoplexy. Two mechanisms have been postulated for pituitary apoplexy-related cerebral ischemia: cerebral vasospasm and mechanical compression of the circle of Willis by the expanding mass. Anterior circulation hypoperfusion and ischemia could be secondary to chronic compression of the ACAs from the encasing pituitary tumor in the setting of sustained systemic hypertension from acute secondary adrenal insufficiency. In addition, pituitary apoplexy can also cause a sudden increase in the pituitary mass leading to vasospasm of the ACAs and cerebral ischemia.
in local pituitary pressure to a median of 47 mm Hg, which in our case could have further potentiated the mass effect from this tumor on the ACAs. Sudden hemorrhage within the pituitary tumor led to pituitary apoplexy, which caused systemic hypotension and in turn led to watershed infarcts in the ACA-MCA and MCA-PCA zones. With respect to the bilateral caudate nucleus infarcts, the blood supply to the head of the caudate is variable. Most reports show that the caudate nuclei receive dual blood supply from the MCA and ACA; however, they can also be solely supplied by the ACA or the MCA. Our patient may have had compression of bilateral Heubner arteries, which are most commonly branches of the ACAs that run along the A1 segments and supply the heads of the caudate nuclei. Compression of the MCAs is unlikely to have been the culprit given that there is no evidence of other MCA territory infarcts. Internal carotid artery compression, which has frequently been reported in the literature in association with pituitary apoplexy, should also have caused more extensive infarcts. Another possible mechanism of bilateral caudate nucleus infarcts in our patient is compression of the first branch of the ACA (A1 segment) by the mass on one side and congenital absence of the A1 segment on the other side, a scenario that could not be ruled out on magnetic resonance angiography. In summary, to our knowledge, this is the first case report in the literature that describes pituitary apoplexy leading to bilateral caudate infarcts.

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REFERENCES