CASE REPORTS AND TECHNICAL NOTES

APOPLEXY IN A PITUITARY CHROMOPHOBE ADENOMA
PRODUCING THE SYNDROME OF MIDDLE CEREBRAL ARTERY THROMBOSIS

CASE REPORT

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The syndrome of pituitary apoplexy due to degenerative changes in adenomas of the pituitary body is a relatively rare condition that usually manifests itself by sudden onset of oculomotor nerve palsies, coma and bloody cerebrospinal fluid.

In a recent review of the subject Brougham, Heusner and Adams1 summarized 7 cases reported in the literature, and gave a detailed analysis of 5 additional cases from their own series. The clinical diagnosis is difficult as shown by the fact that in 10 of the 12 cases it was made only at autopsy. The treatment is likewise unsatisfactory, for as the authors pointed out, 2 of the 12 patients survived on expectant treatment and only 2 of 6 survived immediate surgery.

The following case is reported because the clinical manifestation of the pituitary apoplexy was quite unlike the picture as presented in the literature. In our case it was the typical picture of thrombosis of the middle cerebral artery in a patient with a known old pituitary chromophobe adenoma.

CASE REPORT

G. M., male, aged 65, was admitted to Flower Hospital, Oct. 10, 1949 with paralysis of the left side of sudden onset. In 1938 he had had a verified chromophobe adenoma of the pituitary operated upon by Dr. Max Peet. There had been no recent symptoms of headaches or failing vision to suggest a recurrence of the tumor. He was feeling well until October 9 when he suddenly fell to the floor at home and was found to be paralyzed on his left side and unconscious.

Examination. He was a well nourished and developed elderly man with a “pasty” appearance consistent with a hypopituitary habitus. Cooperation was only fair due to his stuporous state. Head was normal with a well healed right transfrontal craniotomy scar. The neck was moderately rigid. There was questionable anosmia. The optic discs were flat and chalky white with a sharp border indicative of primary atrophy of long standing. There was bitemporal hemianopsia by confrontation. The pupils were round and equal and reacted to light and accommodation. The extra-ocular movements were full and without diplopia or nystagmus. Corneal reflexes and facial sensation were normal. There was a left lower facial weakness. The remaining cranial nerves were normal. There was complete flaccid paralysis of the left arm and leg with edema of the left wrist and ankle. Tendon reflexes were hypactive on the left. Abdominal reflexes were absent. Babinski sign was positive on the left. Sensory examination showed hypesthesia and hypoalgesia of the entire left side. The heart was moderately enlarged with a precordial mid-diastolic rumble. B.P. was 130/80. There were a few rales at the lung bases. The abdomen was negative.

Laboratory Findings. Lumbar CSF was clear and at 120 mm. pressure. Fluid analysis showed 7 lymphocytes and total protein 50 mg. per cent. Blood count was 3.4 million; Hb. 71 per cent; WBC normal. Urine was cloudy with 10 mg. quantitative albumin.

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Skull x-rays Oct. 12, 1949 showed the old osteoplastic flap in good position. There was marked "ballooning" of the pituitary fossa, measuring $23 \times 21$ mm. The dorsum sellae was thin and the posterior clinoids were absent.

Clinical Diagnosis. On the basis of the history of onset, neurological and spinal fluid findings, a working diagnosis of thrombosis of the right middle cerebral artery was made.

Course. A right stellate ganglion block was carried out 18 hours after the initial catastrophe with 20 cc. of 1 per cent novocain. A very good Horner's syndrome was produced on the right side and within 5 minutes he was able to lift his left arm and leg off the bed. On the following morning he still had some movement of the left side. Another right stellate ganglion block was done, but without improvement. Two days later the patient became more drowsy and left hemiplegia returned. There was no change in ocular signs or other cranial nerves. He became irrational, progressed into deepening coma and expired Oct. 25, 1949, 15 days after admission to the hospital.

Autopsy. Gross findings. The body was that of a well nourished elderly white male with very little body hair and the appearance of an eunuch. There were no external deformities other than the old right frontal craniotomy scar. The dura was adherent to the right frontal lobe, which showed yellow-brown discoloration and thinning of the cortex. The pituitary body proper was transformed into a large gray, fairly soft new growth measuring 4 cm. in its widest diameter. It protruded considerably above the roof of the sella and contained a fairly recent hemorrhage, especially on the right side (Fig. 1). The optic chiasma was atrophied and stretched over the tumor. The tumor impinged upon the midbrain without any invasion of the brain or sella turcica.

On section of the brain the right hemisphere was found to contain an extensive area of encephalomalacia, particularly the anterior portion. Grossly, the encephalomalacia was demarcated anteriorly at a level of the tip of the anterior horn of the right lateral ventricle and extended posteriorly to a vertical plane passed through the vestibulum of the right lateral ventricle. In depth it extended from the midline to the lobus insularis laterally.

On opening the arteries that comprise the circle of Willis, there was noted slight arteriosclerotic thickening, but no obstruction of the lumen anywhere. There was, however, a
marked compression of the right middle cerebral artery and, to a lesser extent, the right anterior cerebral artery by the pituitary tumor which was expanded by the hematoma within its capsule. The encephalomalacia in the right hemisphere was in the area of blood supply of the right middle cerebral artery and seems therefore to have been caused by the compression of the arteries.

Microscopic findings. Brain: Sections from the area of encephalomalacia, taken from the olfactory area and internal and external capsule of the right hemisphere, show extensive necrosis of brain tissue. Sections from the right frontal lobe show atrophy and scarring of the cortex. No significant changes were found in brain tissue distant from the lesions.

Pituitary: The tumor (Fig. 2) is composed of uniform-appearing cells arranged in cords separated by sinusoidal spaces (sinusoidal type). There are numerous thin-walled and rather wide blood vessels between the cords of cells, indicating considerable vascularity. A portion of the section includes the edge of recent and extensive hemorrhage with great extravasation of red cells into the tumor. The connective tissue is quite variable. In some areas there are heavy septa which form an interlacing framework, while in other areas the interstitial connective-tissue element is hardly discernible. The cytoplasm took a neutrophil stain and is rather pale and free from granules. The nuclei show slight variations in size and shape and are vesicular, normal or oval. Mitotic figures are infrequent and not atypical. Degenerative changes are slight, with some vacuolization but no necrobiosis.

Pathological Diagnosis. (1) Pituitary adenoma, chromophobe sinusoidal type, with recent hemorrhage into tumor. (2) Compression of the right middle cerebral artery by the hemorrhage into the pituitary tumor. (3) Extensive infarct with encephalomalacia in the right cerebral hemisphere. (4) Status postcraniotomy (old) with atrophy of right frontal lobe and dural adhesions to the brain. (5) Rheumatic heart disease—chronic; mitral stenosis and insufficiency with cardiac dilatation and chronic passive congestion of the lungs.

DISCUSSION

A number of cases of acute hemorrhage into pituitary tumors were found in isolated reports in the literature. The majority of the patients apparently harbored
unsuspected pituitary adenomas, mostly of the acidophile type and less commonly the chromophobe type. In these cases the diagnoses were made only at autopsy. There were only a few instances of an acute fulminating hemorrhage into a verified adenoma treated previously (Cairns, Jefferson, Cushing and Sosman). Heroic measures of sellar decompression were life-saving procedures and might have saved our patient had it been instituted.

The symptoms of coma, diplopia and ophthalmoplegia were given as the usual clinical picture in pituitary apoplexy. Our case presented transient coma, left hemiplegia and no ocular symptoms whatever. Hence in this individual, aged 65, with sudden coma and a left hemiplegia with clear cerebrospinal fluid, our attention was directed toward the treatment of a thrombosis of the right middle cerebral artery rather than to a pituitary apoplexy in the absence of pituitary neighborhood signs even though we knew he harbored an old chromophobe adenoma.

In a study of the pathological anatomy of chromophobe adenomas of the pituitary body it was found that they can be divided into two fairly distinct groups—the "diffuse" and the "sinusoidal" type. Not only was the cell type the same, but the same arrangement of cells was found throughout a given tumor. In none of the previous reports has any mention been made as to the cell pattern of either of the above two types occurring in cases of pituitary apoplexy. Theoretically one might expect hemorrhage from the delicate capillaries around a cyst, but again there apparently was no cyst in any of the reported cases. Our patient had a solid tumor of sinusoidal type without any evidence of cyst formation.

Had we re-explored the known tumor rather than resorting to stellate ganglion block with novocain on the basis of the clinical picture presented, the outcome would still be conjectural, as pointed out by Jefferson—that "operation on old standing and massive tumors is not often profitable for the patient."

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REFERENCES