Examination. Two D. of papilledema were present bilaterally and moderate truncal ataxia was noted. Ventriculography demonstrated a block in the posterior fossa.

Operation. Immediate suboccipital craniectomy disclosed a cystic and solid tumor involving both the cerebellar vermis and the right cerebellar hemisphere. The patient was operated upon in the sitting position and unfortunately expired during the procedure.

Autopsy disclosed air embolism as the cause of death. The cerebellar tumor was a cystic hemangioblastoma. In addition, there were present cystic adenomatous of the pancreas.

The family pedigree of Cases 2 and 3 is shown in Fig. 2. In none of the 3 cases was any angiomatous involvement of the retina demonstrated. It is of interest that in each of these families, symptoms referable to the tumor appeared at a younger age in successive generations. This tendency has been commented upon in previous reports of such families.1

REFERENCE


BRAIN NECROSIS FOLLOWING X-RAY THERAPY

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Brain and nerve tissue are usually resistant to direct damage by x-ray radiations, even with high dosages.1, 4, 14 There is increasing evidence, however, that in rare instances the vascular system of the brain can be severely damaged by irradiation.2, 7, 8, 10, 12, 14, 15 We have recently studied a case that we feel demonstrates this phenomenon. The patient, originally treated for a basal cell epithelioma of the scalp with x-ray therapy, subsequently showed evidence of an underlying expanding, intracranial lesion. At operation a non-tumorous mass was removed from the temporal lobe; microscopic study of this mass revealed startling blood-vessel changes.

CASE REPORT

Past History. L.C., a 45-year-old white female, was first seen in November, 1945, when a biopsy diagnosis of a basal cell epithelioma of the right temporal scalp was made. Superficial x-ray therapy (3500 r) was given to the lesion on Nov. 12, 1945. During the next 2 years, similar lesions appeared over the neck, shoulder, chest, and abdomen. These were treated as they appeared, either by local excision or by electrofulguration. The tumor in the right temporal area reappeared in 1948. Further x-ray therapy was felt unwise because of the marked scarring, atrophy, and depilation of the skin in that area. The lesion was excised and again a diagnosis of basal cell epithelioma was made. She had no complaints suggesting intracranial disease at that time.

Present Illness. On July 30, 1948, the patient was brought to the hospital because of lethargy, confusion, incontinence, and headache of 4 weeks’ duration. The onset had been marked by disabling pain and weakness in both legs and was followed by urinary and fecal incontinence. Daily convulsive seizures, lasting about 10 minutes, started 2 weeks before admission; there were no recognized aura or localizing manifestations.

Examination. She was a well-developed, obese, white woman with marked facial hirsutism. Her pulse was 80, respiratory rate 18, and B.P. 145/85. She was apathetic, lethargic, and

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confused. Her speech was monosyllabic and explosive. There was a restless, continuous, picking motion of her hands. Slight, bilateral exophthalmos was present and the left pupil was larger than the right. Both pupils reacted sluggishly to light. There was early bilateral papilledema. Cooperation was too poor to test visual acuity or visual fields. There was left facial flattening and a minimal, but definite, weakness and hypesthesia of the left arm and leg with decreased tone and increased tendon reflexes on that side. She was right-handed. There was a dorsal Babinski response bilaterally. The patient was unable to care for herself and was incontinent of urine and feces. In the right temporal area of the scalp was a depilated area, 5 × 5 cm., of thin, atrophic, scarred skin with a central ulcer 1 cm. in diameter.

Urine was normal, Hb. was 13.5 gm. per cent, and WBC count was 7,100 with 66 per cent segmented neutrophiles. The blood Mazzini and spinal fluid Kolmer tests gave negative findings. Lumbar puncture showed an initial pressure of 240 mm. of CSF, which had a total protein of 160 mg. per cent. The spinal fluid gold curve was 111–1000. Roentgenograms of the skull showed a midline calcified falx cerebri and no apparent pineal shift. Ventriculography on Aug. 10, 1948 showed no filling of the right ventricle, minimal dilatation of the left ventricle, and a shift of the entire system, including the 3rd ventricle, to the left. The posterior 3rd ventricle was not visualized. The total protein of the left ventricular fluid was 25 mg. per cent.

Course. During the next 5 days, the patient’s status became steadily worse with an increasing left hemiparesis and slowing pulse and respirations.

1st Operation, Aug. 16, 1948. Because of the suggestion of a 3rd ventricle or right posterior parietal lesion, an exploratory craniotomy was done through a right posterior parietal bone flap. The most anterolateral portion of the cortex exposed was slightly yellow. The rest of the exploration, including examination of the 3rd ventricle through the corpus callosum, was

Fig. 1. Extreme thickening and hyalinization of small blood vessels. Infarction of brain with exudate containing many gitter cells. Changes suggesting amyloid degeneration. (van Gieson × 215)