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Empty Sella Syndrome with Intrasellar Herniation of the Optic Chiasm

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Many examples of the so-called "empty sella" syndrome have been reported in recent years, especially after the advent of computed tomography (CT) with the use of metrizamide [1-4]. This is a distinct radiologic entity that may or may not be symptomatic [5]. An unusual case is reported in which the optic chiasm was herniated into the sella.

Case Report

A 48-year-old woman was admitted to New York Hospital-Cornell Medical Center with visual loss and a medical history of primary amenorrhea. She had experienced severe bifrontal headaches in her 20s, which subsided by age 30 years. She noticed the onset of gradually progressive visual loss 1 year later. Neurologic evaluation

3 years later revealed a visual acuity of less than 20/800 in the right eye and 20/70 in the left, with bitemporal field defects. A sellar mass was diagnosed by arteriography and the sella was treated with 4,500 rad (45 Gy). There was partial improvement in her visual acuity. She was without further complaint until 14 years later when recurrence of diminished peripheral vision was noted. Examination revealed the visual acuity to be 20/200 in the right eye, 20/800 in the left. Goldman perimetry defined bitemporal visual field defects associated with a confluent superior nasal defect in the left eye only. Both optic disks were pale. A CT scan suggested an enlarged sella with a hypodense sellar mass. A metrizamide CT scan showed an empty sella, and a reformatted image showed the chiasm to be herniated down into the sella and vertically elongated (figs. 1A and 1B). The anterior cerebral arteries were clearly seen (fig. 1C). A right frontal craniotomy confirmed the prolapse of the

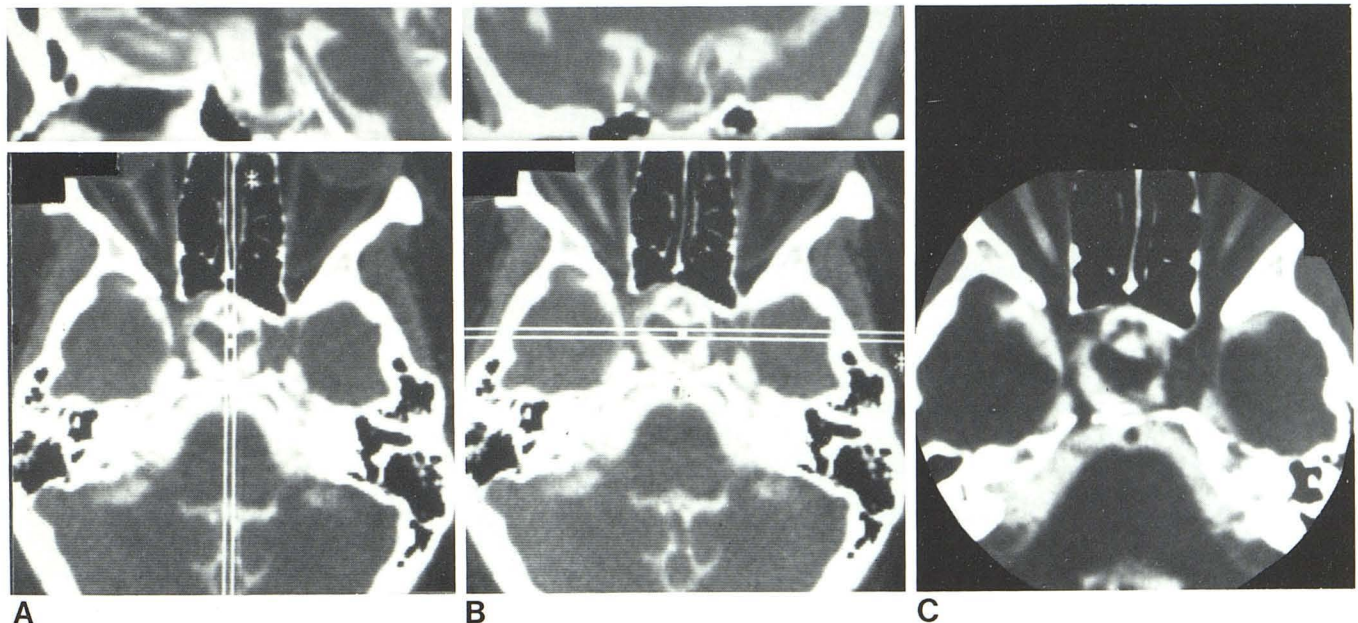


Fig. 1.—A, Sagittal reconstruction through sella. Elongated chiasm herniated into sella. B, Coronal reconstruction with similar findings. C, Anterior cerebral arteries just in front of the chiasm (arrows).

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chiasm and showed that the precommunicating portion of the anterior cerebral arteries also extended into the sella. The vasculature exhibited marked atherosclerotic changes and the arteries were closely adherent to the optic nerves due to fibrosis. Microsurgical decompression of the right optic nerve and chiasm was performed but it was impossible to relocate the elongated atherosclerotic anterior cerebral arteries out of the sella. The visual fields expanded immediately after this operation, but 2 days later her vision failed again.

Discussion

The empty sella syndrome was initially described in cases of scarred pituitary gland after postpartum pituitary necrosis [6]. The syndrome is the result of a diaphragma sellae deficiency. In the primary form, it is unusually asymptomatic and the diagnosis is made radiologically [7]. In the secondary form it may be due to spontaneous or postirradiation ischemic necrosis of a large pituitary tumor, or infrequently is seen in the presence of diabetes mellitus, granulomatous meningitis (e.g., sarcoid), or septic shock. The atrophic, shrunken pituitary gland leaves an empty space which is taken up by the expanded suprasellar cistern. The CT scan suggests the diagnosis by showing the presence of cerebrospinal fluid density in the sella turcica [8–11]; however, a necrotic intrasellar pituitary tumor may give a similar CT picture [11, 12]. The diagnosis is confirmed by the demonstration of contrast material, either air or, as in our case, metrizamide, within the sella turcica [13–17]. Our case is unusual in that we were able to demonstrate the optic chiasm and both precommunal anterior cerebral arteries within the sella turcica. The CT findings, confirmed at operation, are best explained on the basis of postirradiation tumor necrosis and adhesive arachnoiditis [18, 19] drawing the anterior cerebral arteries, optic chiasm, and nerves down into the sella along with the shrunken tumor capsule [20–23].

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