19 Partially Empty Sella

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INTRODUCTION

The empty sella turcica was first described in 1949 as a condition where the sella turcica is only partially filled by the pituitary gland, which appears flattened against the sellar floor (Fig. 19.1).[1] Autopsy studies confirm the high disease prevalence reported to be 5.5% to 20% of the general population.[2] Not surprisingly, many patients who undergo brain imaging will have a partially empty sella. Recent studies report an overall incidence on imaging of 12%.[3] Most patients with an empty sella on imaging are asymptomatic. However, some may develop a constellation of symptoms including hypopituitarism, inferior displacement of the optic tracts with associated visual disturbance, rhinorrhea, and other symptoms pertaining to elevated intracranial pressure (ICP). This has been referred to as the empty sella syndrome (Figs. 19.2–19.4). Other patients may develop an empty sella secondarily in response to pituitary surgery or radiotherapy for adenomas (Figs. 19.5 and 19.6), medical therapy for macroadenomas, spontaneous pituitary apoplexy, trauma, infection, autoimmune disease, and Sheehan syndrome.[4]

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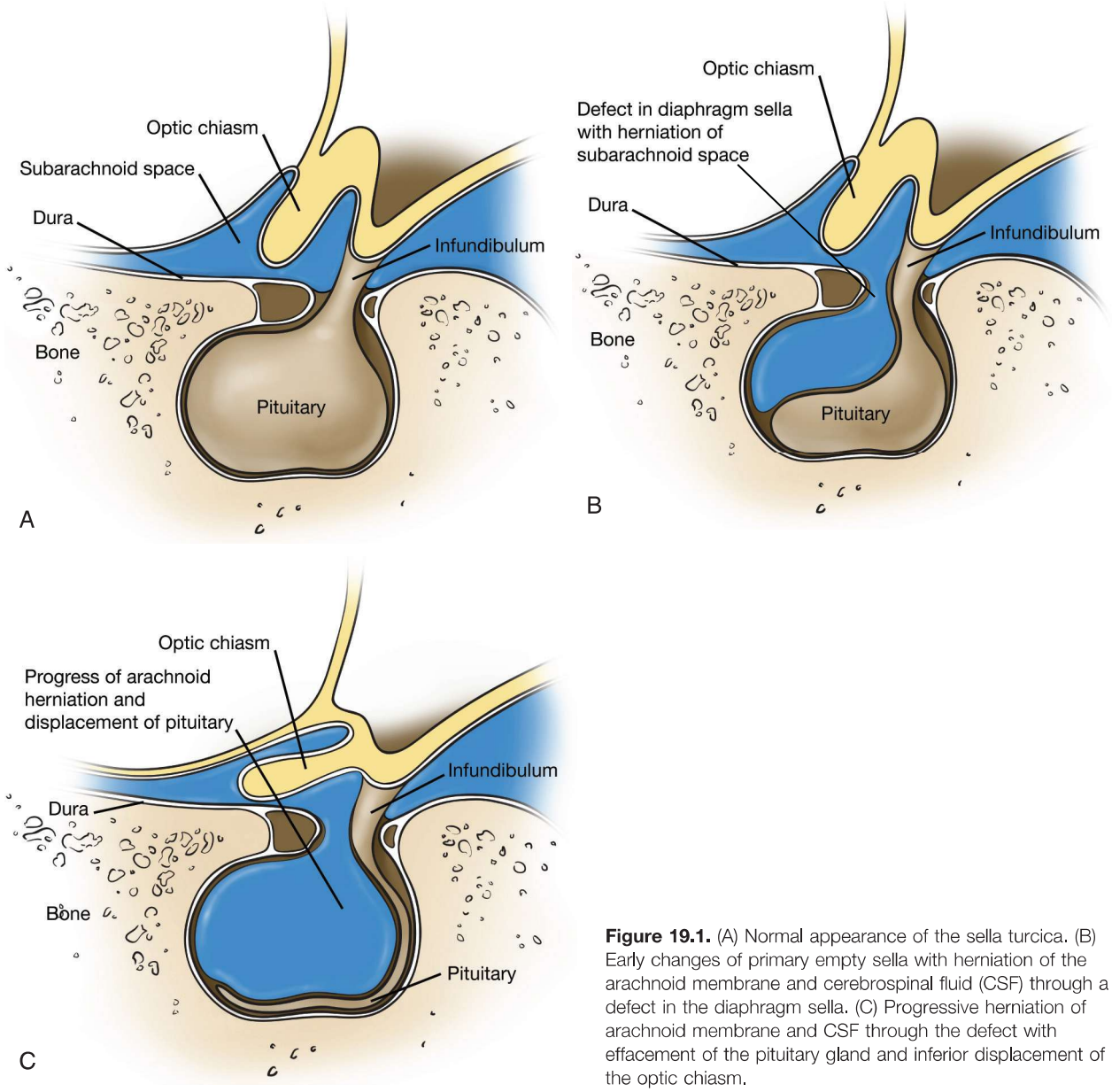

Figure 19.1. (A) Normal appearance of the sella turcica. (B) Early changes of primary empty sella with herniation of the arachnoid membrane and cerebrospinal fluid (CSF) through a defect in the diaphragm sella. (C) Progressive herniation of arachnoid membrane and CSF through the defect with effacement of the pituitary gland and inferior displacement of the optic chiasm.

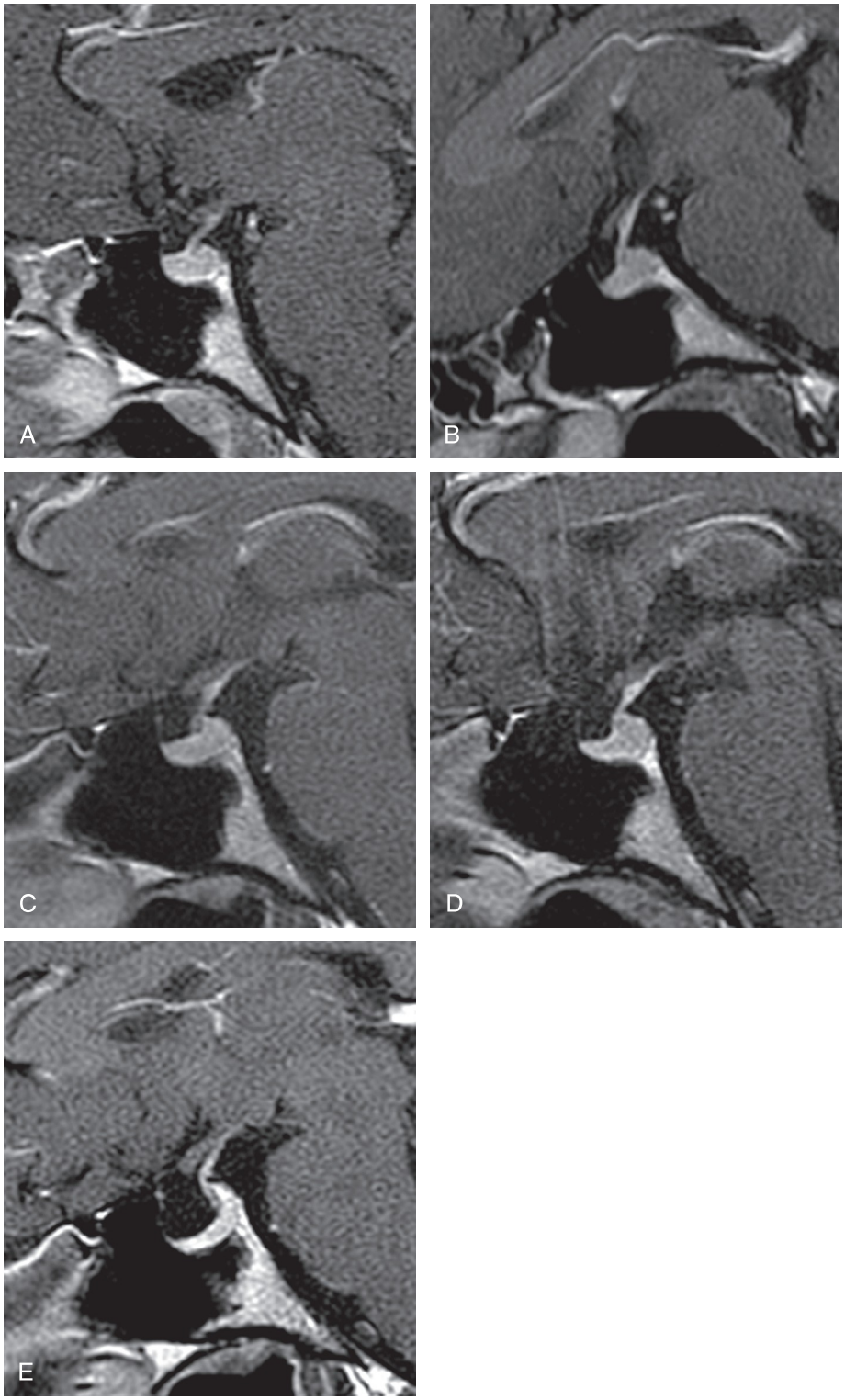

Figure 19.2. (A–E) Primary empty sella syndrome: Multiple sagittal T1-weighted postcontrast images demonstrating slow, gradual progressive effacement and downward displacement of the pituitary gland with mild expansion of the sella turcica. Note the initial slight expansion of the anterosuperior sella and subtle gradual effacement of the anterosuperior aspect of the pituitary gland 2 (B) and 4 (C) years after initial imaging. Effacement of the gland is more prominent 6 years later (D) and expansion with marked effacement is clearly evident 11 years later (E).

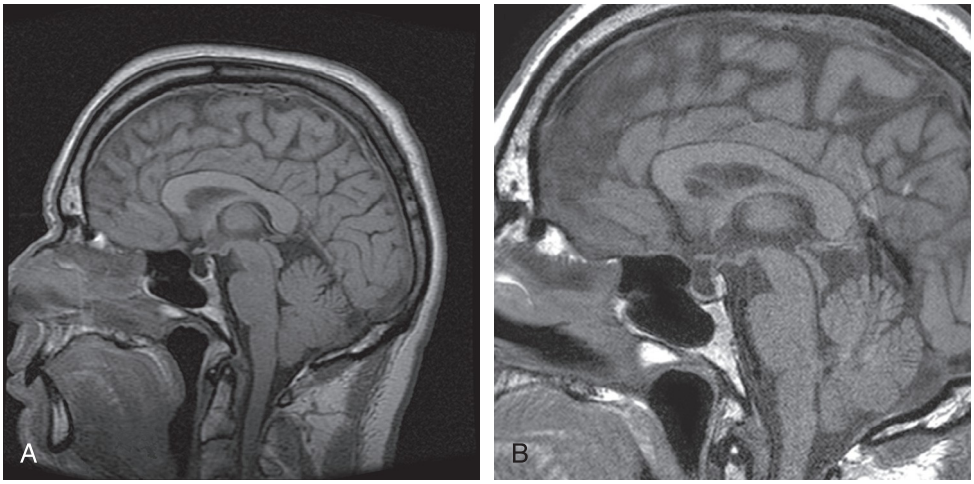

Figure 19.3. (A and B) Stable primary empty sella. Sagittal T1-weighted images demonstrating a partially empty sella. Note that there has been no progressive displacement of the pituitary gland or bony expansion of the sella turcica over a period of 9 years (B).

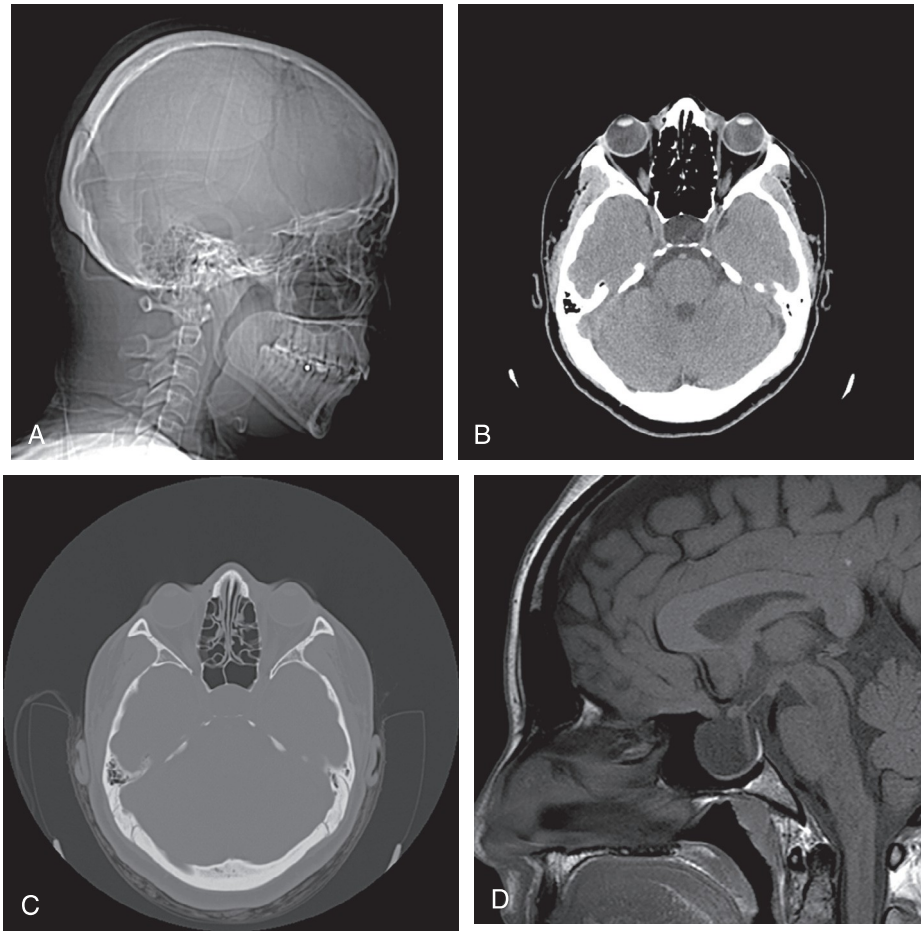

Figure 19.4. Primary empty sella. Lateral scout radiograph (A) and axial noncontrast computed tomography (B and C) brain images with soft tissue and bone windows demonstrating cerebrospinal fluid density within an expanded sella turcica. Pre- and postcontrast coronal and sagittal magnetic resonance imaging (D–H) in the same patient revealing posterior and inferior displacement of the pituitary gland with marked expansion of the sella, consistent with primary empty sella.

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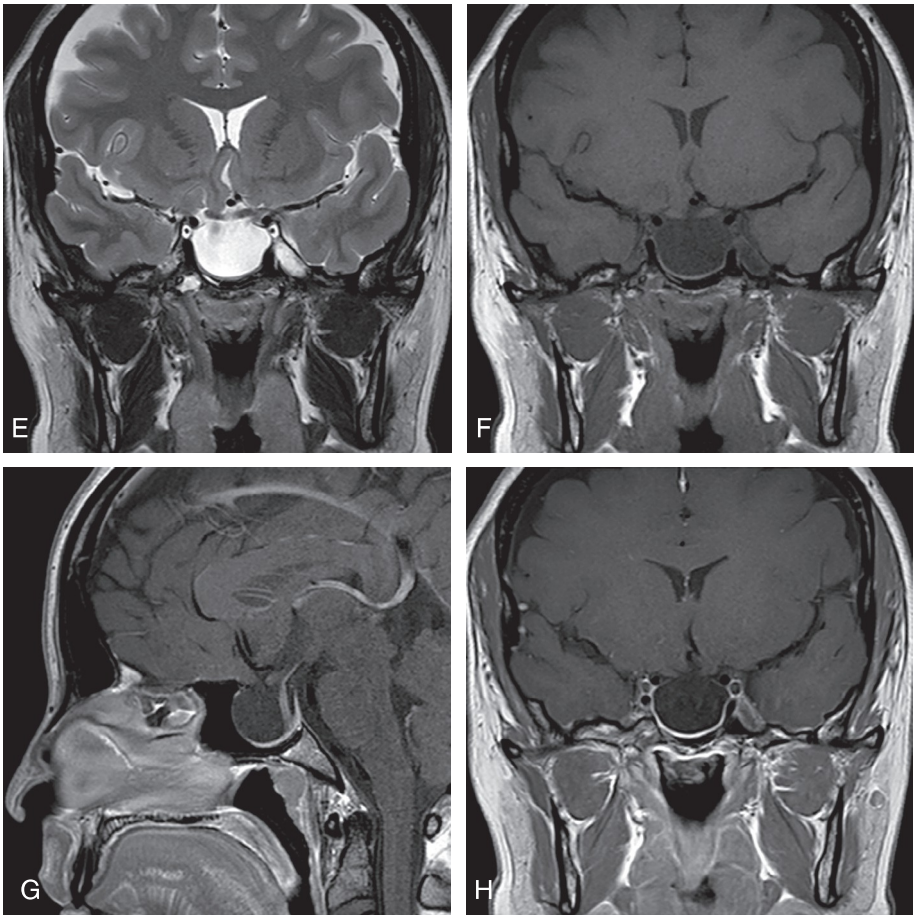

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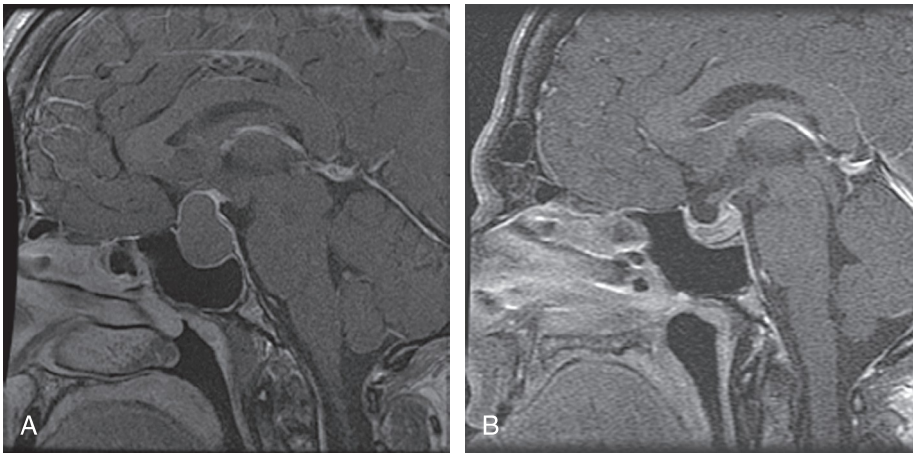
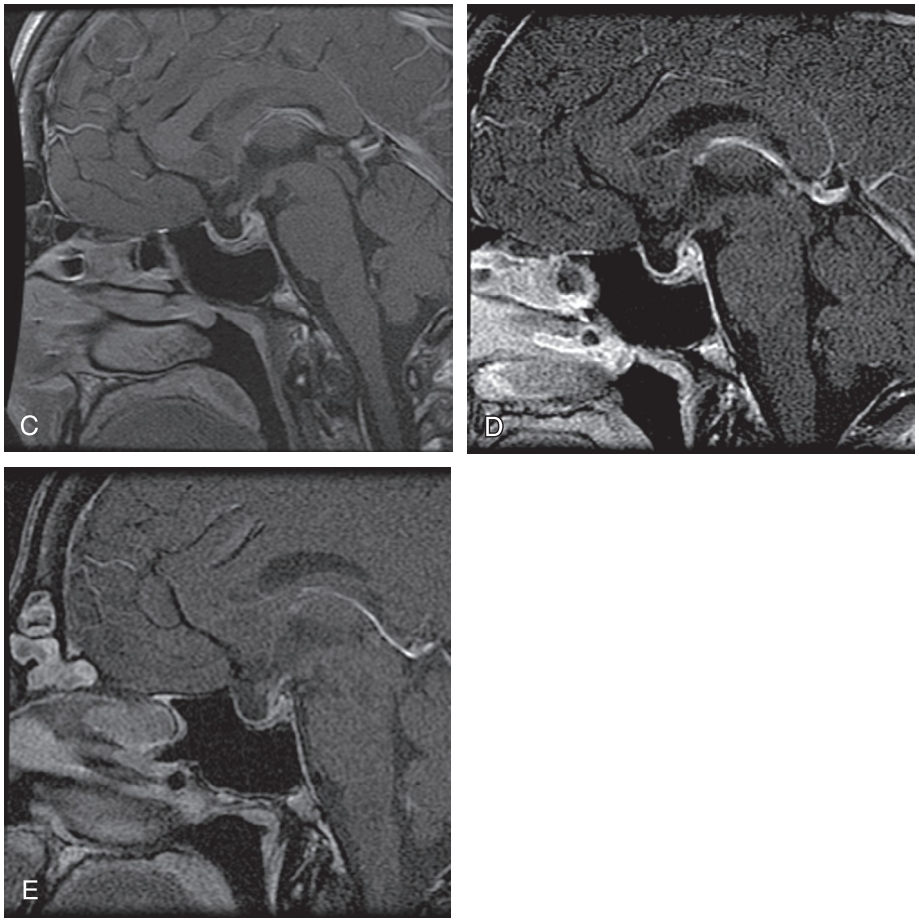


Figure 19.5. Secondary empty sella. (A–E) Sagittal postcontrast imaging at time 0, 3 months, 7 months, 1 year, and 2 years after medical treatment for a pituitary macroadenoma demonstrating progressive volume loss of the pituitary gland and replacement with cerebrospinal fluid, reflecting secondary empty sella.



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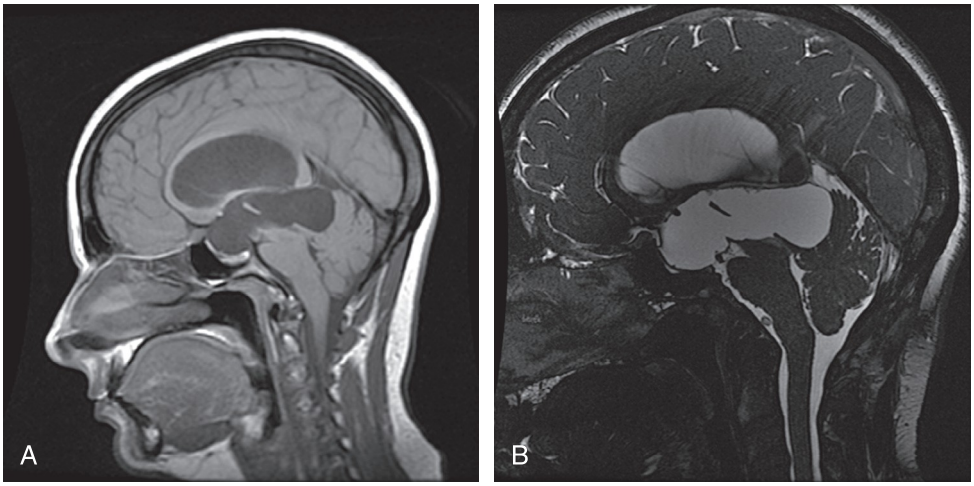

Figure 19.6. Secondary empty sella. Sagittal T1 (A) and sagittal three-dimensional constructive interference in steady state (B) sequence demonstrating severe enlargement of the third and lateral ventricles with normal appearance of the fourth ventricle in a patient with aqueductal stenosis. Marked dilation of the anterior third ventricle led to secondary chronic severe expansion of the sella with effacement of the pituitary gland.

EVOLUTION: OVERVIEW

The exact etiology of the primary form of the empty sella is unknown; however, it is hypothesized to develop from a congenital defect or enlarged diaphragm sella.[4] It is through this prominent diaphragm sella that arachnoid membrane may herniate into the sella. Chronically transmitted cerebrospinal fluid (CSF) pulsations enlarge the arachnoid herniation with eventual flattening of the pituitary gland and bony remodeling with expansion of the sella turcica.

EVOLUTION: IN GREATER DEPTH

Not all patients with elevated ICP will develop an empty sella, nor will all those patients with an empty sella have elevated ICP. It has been hypothesized that normal CSF pulsations may be sufficient in the presence of a diaphragm sella defect to form an empty sella. Nonetheless there appears to be a strong relationship between idiopathic intracranial hypertension and empty sella syndrome.[4] Additional risk factors for primary empty sella syndrome include headaches, pregnancy, and hypertension. It is unclear how long it takes for the empty sella to develop, and this will likely depend on whether it is a primary or secondary empty sella.

In addition to ICP, multiple other conditions are associated with an empty sella, including posteriorly displaced optic chiasm, reduction of pituitary gland volume due to menopause, multiparity, pituitary gland infarction, diabetes, or bromocriptine treatment for pituitary adenoma.[5]

MIMICS AND DIFFERENTIAL DIAGNOSIS

Any disease process causing elevated ICP may result in an empty sella, especially in the presence of a preexisting diaphragm sella defect.

SUGGESTED READING

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2. Bergland RM, Ray BS, Torack RM. Anatomical variations in the pituitary gland and adjacent structures in 225 human autopsy cases. *J Neurosurg*. 1968;28:93–99.
3. Sage MR, Blumbergs PC. Primary empty sella turcica: a radiological-anatomical correlation. *Australas Radiol*. 2000;44:341–348.
4. Oh M, Laws ER, et al, eds. Empty sella syndrome. In: *Pituitary Disorders: Diagnosis and Management*. 1st ed. John Wiley and Sons, Ltd.; 2013:77–86.
5. Kaufman B. The "empty" sella turcica – a manifestation of the intrasellar subarachnoid space. *Radiology*. 1968;90:931–941.