PRIMARY EMPTY SELLA SYNDROME
WITH PANHYPOPITUITARISM, DIABETES INSIPIDUS,
AND VISUAL FIELD DEFECTS

By

*Thomas R. Cupps and Paul D. Woolf*

**ABSTRACT**

A 58 year old woman with a history of hypothyroidism was evaluated for marked visual impairment and found to have the primary empty sella syndrome with multiple endocrine abnormalities. Visual field determination revealed preservation of vision only in the left inferior quadrants bilaterally. Failure of growth hormone (hGH), cortisol and prolactin to respond to insulin induced hypoglycaemia (0.1 U/kg), of luteinizing hormone (LH) and follicle stimulating hormone (FSH) to respond to gonadotrophin releasing hormone (GnRH, 100 µg) and of thyrotrophin (TSH) and prolactin to increase after thyrotrophin releasing hormone (TRH, 500 µg), confirmed the diagnosis of panhypopituitarism. Following water deprivation with a 9% loss in body weight, her urine osmolality remained at 204 mOsm./kg H₂O), indicating that she had posterior pituitary deficiency as well. During surgical exploration, which was performed in an effort to improve her markedly impaired vision, a compromised vascular supply to the left optic nerve and chronic arachnoiditis was demonstrated. This case represents one extreme of functional impairment in a syndrome which is generally considered benign and which rarely requires therapeutic intervention. Our patient is compared to 29 reported cases of the primary empty sella syndrome with visual field defects. The operative findings in eight of these cases are reviewed. The need for a multidisciplinary approach and close follow-up of patients with an empty sella and functional deficits is emphasized. Surgical intervention including lysis of adhesions and chiasmapexy has been effective in selected cases in reversing or stabilizing visual field abnormalities.
The primary empty sella syndrome has been defined as the presence of air in the sella turcica during pneumoencephalography without prior history of cranial surgery or radiation therapy (Neelon et al. 1973). Although this definition may require modification with increased utilization of computerized assisted tomography (CT), the clinical spectrum of this disease entity has been reviewed in detail (Neelon et al. 1973; Jordan et al. 1977). The majority of patients with primary empty sella syndrome are obese, multiparous women with normal visual fields and no clinically apparent endocrine abnormalities. An increased incidence of hypertension, pseudotumour cerebri, and cerebro-spinal fluid rhinorrhea is also commonly found. Most patients have an excellent prognosis and require no therapeutic intervention. The need to distinguish a primary empty sella from an expanding intrasellar mass lesion in order to avoid unnecessary surgery or radiation therapy has been emphasized in the past (Caplan & Dobben 1969; Neelon et al. 1973).

We describe a patient with primary empty sella syndrome associated with markedly abnormal visual fields, panhypopituitarism, and diabetes insipidus who underwent surgical exploration. Although visual field defects (Robertson 1967; Walsh & Hoyt 1969; Mortara & Norell 1970; Matisonn & Pimstone 1973; Wood & Dogali 1975; Buckman et al. 1976), panhypopituitarism (Mortara & Norell 1970; Neelon et al. 1973; Raiti et al. 1976; Jordan et al. 1977), and diabetes insipidus (Matisonn & Pimstone 1973; Schaison & Metzger 1976) with primary empty sella syndrome have been reported separately, this patient is the first reported case in which all deficits are present in the same individual. This case emphasizes the fact that although the majority of people with the primary empty sella syndrome require no specific therapy, there is a small subset of patients with clinically significant defects who require close follow-up and possible surgical intervention.

CASE REPORT

A 58-year old Caucasian female (SMH No. 97-09-62) was admitted to the Strong Memorial Hospital Neurology service for evaluation of vision abnormalities in September, 1977. The patient had been in excellent general health the majority of her life. Four years prior to admission, she was started on thyroid replacement with 2 grains of desiccated thyroid per day for reasons that are not known. This same year an erythematous facial rash which had been present for approximately 3 years was biopsied. The diagnosis of sarcoidosis was made based on the finding of a granuloma in the skin biopsy and possible bilateral adenopathy on chest X-ray. No Kveim test was placed. The patient received 20 mg of prednisone per day with rapid resolution of the rash, but it recurred following a decrease to 5 mg on alternate days. Several years prior to admission, she was forced to tilt her head back to see the mailboxes on the upper rows while working as a mail sorter. By March, 1977, she stopped driving because of difficulty maintaining her car in the correct lane. She also noted difficulty
doing fine work with her hands due to decreased visual acuity. Optic atrophy and a non-correctible visual acuity of 20/400 were found by an optometrist and a referral was made. The patient also noted increased thirst and frequency of urination 6 to 8 h after taking the prednisone on her alternate-day schedule. There was no history of previous cranial surgery or radiation therapy, head trauma, CNS infection, seizure disorder, or joint problems. The patient had 4 successful pregnancies and had undergone menopause at age 44. On physical examination, she was a mildly obese, elderly woman in no distress. Vital signs included a pulse of 80, a blood pressure of 112/60 mmHg in supine position without significant orthostatic changes, a temperature of 36.7°C orally, a respiratory rate of 18, and a weight of 64 kg. On neurological evaluation, the patient was alert and oriented with a normal mental status. The pupils were equal, round and light reactive. Disc pallor was noted bilaterally, and gross visual field defects were detected by confrontation. The remainder of the cranial nerves were normal except for mild bilateral decrease in the corneal reflexes. Deep tendon reflexes were brisk and equal with a normal relaxation phase except for the ankle jerks which were absent. Pathological reflexes and frontal release signs were absent. Motor, sensory, cerebellar testing, and gait were normal. Facial telangiectasia and an erythematous blanching rash on the nose and left cheek were present. The skin was dry, somewhat thin with no abnormal pigmentation. There was decreased axillary and pubic hair. The remainder of the general examination was unremarkable.

Initial laboratory data included a normal chest X-ray, a normal urinalysis with a specific gravity of 1.003, normal serum albumin, total protein, inorganic phosphorous, cholesterol, urea nitrogen, creatinine, uric acid, bilirubin, alkaline phosphatase, glutamic oxalacetic transaminase, lactic dehydrogenase and electrolytes, a fasting serum glucose

![Sagittal tomogram during pneumoencephalography demonstrating air within the sella turcica.](image)

Fig. 1.
Auto-plot tangent peripheral fields with 6 mm (●——●) and 15 mm (——) white test objects revealing preservation of vision limited to the left homonymous inferior quadrants.

Eye examination. – The best corrected vision was O. D. 20/40, O. S. 20/60. Auto-plot tangent peripheral fields were performed with 3, 6 and 15 mm white test objects and revealed preservation of vision only in the left inferior quadrants bilaterally (Fig. 2). Microstrabismus O. D. with eccentric fixation O. S. explained why the field in this eye crossed the vertical meridian. The vision present in the left inferior quadrant was also peripherally constricted.

Endocrine evaluation. – The patient had normal thyroid function studies on hormone replacement. (T₄ 8.4, normal 5–13.5 µg/100 ml; T₃ 55; normal 50–200 ng/100 ml TSH < 1, normal 1–10 µU/ml). Her gonadotrophins were quite low for a post-menopausal female (LH 2.1, normal 35–200 mIU/ml, FSH 2.1, normal 40–200 mIU/ml). Baseline serum cortisol of 1.8 µg/100 ml at 09.00 a.m. (a.m. normal 9–20) rose to
5.8 µg/100 ml 1 h after an iv infusion of 0.25 mg of synthetic ACTH. Plasma cortisol, growth hormone and prolactin levels were measured in response to insulin-induced hypoglycaemia (0.1 U/kg) 24 h after the last dose of hydrocortisone (40 mg). The fasting blood glucose of 72 mg/100 ml dropped to 40.5 mg/100 ml at 20 min and to 20.5 mg/100 ml at 30 min. There were inadequate or absent responses of the anterior pituitary hormones to the hypoglycaemia stress (Fig. 3 A). Following the simultaneous intravenous administration of thyrotrophin releasing hormone (TRH, 500 µg Abbott Laboratories) and gonadotrophin releasing hormone (GnRH, 100 µg Parke Davis & Company), there was no change in TSH, prolactin, LH or FSH (Fig. 3 B and C).

A water-deprivation test was performed while the patient was on hydrocortisone (40 mg) and thyroid (2 grains) replacement. The baseline serum osmolality was 292 mOsm. while the urine was 253 mOsm./kg H₂O. After 6 h of water deprivation, the urine output was 4600 ml and a 9% loss of body weight had occurred. Serum osmolality had increased to 297 with a simultaneous urine osmolality of only 204 mOsm./kg H₂O. One hour after a subcutaneous injection of 5 units of vasopressin, her urine osmolality had increased to 358 mOsm./kg H₂O. These results are compatible with the diagnosis of centrally mediated diabetes insipidus.

In summary, the patient demonstrated absent anterior pituitary function and a partial deficiency of antidiuretic hormone.

Surgery. – Because of the marked visual impairment, surgical exploration of the optic chiasm was performed through a right frontal craniotomy. Thickened arachnoid membranes covered both optic nerves and chiasm. The right optic nerve was yellow in color and reduced in size by 30%. The left optic nerve was white and of normal size. Neither optic nerve appeared under tension or had prolapsed into the sella turcica. The vascular supply to the left optic nerve was intact but it was compromised on the right side. A thin grey piece of tissue extended from the anterior orbital surface
of the frontal lobe across to the proximal end of the right optic nerve at the level of chiasm. Partial kinking of the nerve was noted. The tissue was removed restoring neural anatomy. A small arachnoid cyst was noted in the sella, but no definite pituitary tissue could be identified. The arachnoid adhesions were removed, and the sella was packed with temporalis muscle. Histology of the tissue showed fragments of cerebral cortex and a focal leptomeningeal lymphocytic infiltrate (Fig. 4). No granuloma or necrosis was present. Cultures for bacteria, fungi, or acid-fast organisms were negative. Four months post-operatively, the visual fields were essentially unchanged.

Fig. 3 B.
TSH and prolactin response to TRH infusion (500 µg).

Fig. 3 C.
FSH and LH response to GnRH infusion (100 µg).
Fig. 4.
Photomicrograph of intraoperative biopsy showing superficial cerebral cortex with focal leptomeningeal lymphocytic infiltrate (100×).

DISCUSSION

The multiple proposed mechanisms for the primary empty sella syndrome have been discussed in detail (Kaufman 1968; Neelon et al. 1973; Jordan et al. 1977). Kaufman (1968) suggested that cerebrospinal fluid pressure acting through an incomplete diaphragm causes expansion of the sella turcica and compression of pituitary tissue. An association between the empty sella syndrome and diseases with increased intracranial pressure such as pseudotumour cerebri, extrasellar neoplasms, congestive heart failure, hydrocephalus and Pickwickian syndrome has been noted (Kaufman 1968; Neelon et al. 1973). Other suggested mechanisms include pituitary infarction, pituitary hypertrophy and subsequent abiotrophy, cyst rupture, subarachnoid adhesions, and inflammatory processes (i.e. gumma or giant cell granuloma) (Neelon et al. 1973; Jordan et al. 1977).

The endocrine function in the primary empty sella syndrome has also been extensively studied (Brisman et al. 1972; Faglia et al. 1973; Neelon et al. 1973; Weisberg et al. 1976; Jordan et al. 1977). When the growth hormone (hGH) response to insulin-induced hypoglycaemia was evaluated (Brisman et al. 1972; Faglia et al. 1973; Weisberg et al. 1976), an abnormality was present in approximately half of the patients tested. The clinical significance of this finding
is unclear as blunted hGH responses to hypoglycaemia may be related to the obesity which is usually present, rather than to the empty sella syndrome itself (Jordan et al. 1977). Indeed, a normal hGH response to arginine infusion but not to hypoglycaemia in patients with empty sella syndrome has been reported (Buckman et al. 1976). The response of pituitary hormones to the hypothalamic releasing factors, TRH and GnRH has been reviewed and most patients respond normally (Jordan et al. 1977). A wide variety of endocrine syndromes have been associated with empty sella, including Cushing's syndrome (Ganguly et al. 1976), amenorrhoea (Schaison & Metzger 1976), galactorrhoea (Schaison & Metzger 1976) and isolated hyperprolactinaemia (Weisberg et al. 1976). The aetiology of the hypersecretory states is unclear but might be due to residual secretion from an infarcted hormonally active pituitary adenoma. Alternatively, in the case of hyperprolactinaemia, disruption of the integrity of the hypothalamic-pituitary axis would result in removal of tonic prolactin inhibition by prolactin inhibiting factor (PIF) or by dopamine, resulting in enhanced prolactin secretion (Martin et al. 1977).

Involvement of the posterior pituitary in the primary empty sella syndrome is quite rare with only three previously reported cases (Matisonn & Pimstone 1973; Schaison & Metzger 1976). Matisonn & Pimstone (1973) reported a 43-year old woman with diabetes insipidus, visual field defects, a 3-year history of post-partum galactorrhoea, and treated pulmonary tuberculosis. Schaison & Metzger (1976) reported 2 female patients (ages 23 and 35 years), out of a series of 12 with diabetes insipidus, amenorrhoea, galactorrhoea, and normal serum prolactin levels. It is of interest, that our patient only noted increased thirst and urinary frequency 6 to 8 h after taking 5 mg of prednisone on her alternate-day schedule. As the permissive effect of anterior pituitary hormones on the free water clearance in the kidney is well established (Leaf et al. 1952), inadequate pituitary function is the most likely explanation for this observation.

VISUAL FIELDS

The association of visual field abnormalities1 with the primary empty sella syndrome is well known (Robertson 1967; Walsh & Hoyt 1969; Mortara & Norell 1970; Matisonn & Pimstone 1973; Dahlstrom & Acers 1975; Buckman et al. 1976; Schaison & Metzger 1976; Martin et al. 1977; Jordan et al. 1977). There is, however, marked variation in reported incidence of associated visual field defects. Neelon et al. (1973), in a review of 121 patients, found two cases for a frequency of 1.6%. In a more recent report, however, Foley & Posner

1) Neither enlargement of physiological blind spot nor papilloedema was included in this summary.

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(1975) noted a 16\% incidence in a review of 130 patients, a frequency confirmed by Schaison & Metzger (1976) and Jordan et al. (1977).

In 20 cases, the specific nature of visual field defect was stated and is summarized as follows: Bitemporal 11/20, concentric narrowing 5/20, binasal (included 2 patients with bitemporal defects) 3/20, homonymous 2/20, and unilateral altitudinal hemianopsia 1/20. The severity of the defects ranged from a bilateral quadrantanopsia demonstrated with 1 mm object and normal acuity (Raiti et al. 1976) to bilateral involvement of 3 visual quadrants and an acuity of 20/400 (Mortara & Norell 1970). Concentric narrowing of the peripheral fields may be caused in some cases by pseudotumour cerebri (Foley & Posner 1975). This defect, however, has also been described in patients with primary empty sella syndrome and normal cerebrospinal fluid pressures (Jordan et al. 1977).

Clinical parameters including age, sex, obesity, and pituitary hormone abnormalities are quite similar in patients with or without visual field abnormalities. Data from the 29 reported cases with visual field defects are summarized

Table 1.
Summary of clinical parameters of patients with primary empty sella syndrome and visual field defects. Comparative data from two large series irrespective of visual field abnormalities are provided.

<table>
<thead>
<tr>
<th>Patients with primary empty sella and visual field abnormalities</th>
<th>Neelon’s 1) series</th>
<th>Jordan’s series</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (years)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>38 (14)</td>
<td>48</td>
</tr>
<tr>
<td>Range</td>
<td>5–58</td>
<td>27–72</td>
</tr>
<tr>
<td>% Female</td>
<td>75 (16)</td>
<td>87</td>
</tr>
<tr>
<td>% Obese</td>
<td>75 (16)</td>
<td>–</td>
</tr>
<tr>
<td>% Pituitary endocrine abnormality</td>
<td>50 (12)</td>
<td>41</td>
</tr>
<tr>
<td>a) % Abnormal hGH response only</td>
<td>17</td>
<td>11</td>
</tr>
<tr>
<td>b) % Other</td>
<td>33</td>
<td>30</td>
</tr>
</tbody>
</table>

Numbers in brackets represent the compilation of cases for which adequate information is provided.
1) Based on patients reported in the series of Neelon et al. (1973) exclusive of literature review.
2) Based on 12 patients in the series of Jordan et al. (1977) exclusive of literature review.
3) Based on review of 245 cases in the literature.
4) Based on review of 162 cases in the literature.

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Table 2.
Summary of 8 case reports of patients with primary empty sella syndrome who have undergone intracranial surgery.

<table>
<thead>
<tr>
<th>Clinical</th>
<th>Eye findings</th>
<th>Endocrine</th>
<th>Surgical findings</th>
<th>Tissue pathology</th>
<th>Outcome</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Male with ↓ libido, beard growth, and dry wrinkled skin</td>
<td>Bitemporal field defects</td>
<td>–</td>
<td>Arachnoid membrane present; optic nerve stretched backward with chiasm thinned. Thin rim of pituitary tissue found; median side of the anterior clinoid incised</td>
<td>–</td>
<td>–</td>
<td>Caused by possible cyst or infarcted adenoma (Robertson 1967)</td>
</tr>
<tr>
<td>2. 35 year old white male with impotence</td>
<td>Bitemporal field defects; temporal disc pallor</td>
<td>–</td>
<td>Few strands of filamentous tissue around optic nerve</td>
<td>–</td>
<td>–</td>
<td>Caused by possible cyst rupture (Walsh &amp; Hoyt 1969)</td>
</tr>
<tr>
<td>3. 44 year old female with hypertension and seizures</td>
<td>Tubular vision</td>
<td>Normal</td>
<td>Absent sellar diaphragm, normal appearing pituitary in anterior-inferior aspect of enlarged sella</td>
<td>–</td>
<td>Normal endocrine and visual function</td>
<td>Visual field abnormalities caused by: 1) thinning of chiasm and floor third ventricle, resulting from intra-ventricular pulsations</td>
</tr>
</tbody>
</table>
2) pushing down and kinking of optic nerve and chiasm
3) pressure on optochiasmatic track by anterior cortical artery displaced by the third ventricle (Mortara & Norell 1970)

<p>| Case | 58 year old male with weakness, fatigue and loss of appetite; PEG showed dilated ventricles and air in sella turcica | Incomplete bitemporal hemianopsia | Panhypo pituitarism | Absent sellar diaphragm, optic nerves and chiasm dipped into sella. Enlarged sella with small pituitary gland in the base. The anterior cerebral artery intimated dorsal aspect of chiasm. Lamina terminalis was opened | – | – | See case 3 |</p>
<table>
<thead>
<tr>
<th>Clinical</th>
<th>Eye findings</th>
<th>Endocrine</th>
<th>Surgical findings</th>
<th>Tissue pathology</th>
<th>Outcome</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>5. 43 year old female with headache, visual loss</td>
<td>Incomplete bitemporal and binasal hemianopsia; O. D. 20/400, O. S. hand perception</td>
<td>–</td>
<td>Optic nerves normal. Coarse but thinned optic chiasm posteriorly displaced. Absent sellar diaphragm. Strands of arachnoid tissue from chiasmatic cistern down into sella turcica</td>
<td>Normal adenohypophysis</td>
<td></td>
<td>See case 3</td>
</tr>
<tr>
<td>6. 30 year old obese female with painless ↓ vision, remaineder of examination normal</td>
<td>O. S. inferior altitudinal hemianopsia, with constricted field; O. D. no light perception</td>
<td>Normal</td>
<td>Incomplete diaphragm sellae, atrophic optic nerves stretched between optic foramina and anterior edge of pituitary fossa; tension on optic chiasm relieved by filling of the sella turcica</td>
<td>–</td>
<td>O. D. markedly improved O. S. improved light perception</td>
<td>(Wood &amp; Dogali 1975)</td>
</tr>
<tr>
<td>7. No clinical history</td>
<td>Optic atrophy; visual field abnormalities</td>
<td>Entrapment of optic chiasm into superior portion of sella turcica by arachnoid tissue adhesions; excision of adhesions</td>
<td>Complete recovery</td>
<td>Role for surgical intervention in presence of visual field abnormalities emphasized (Martin et al. 1977)</td>
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<tr>
<td>8. 5 year old male with loss of vision</td>
<td>O. D. 20/60–10/200, O. S. 14/200 with constricted fields</td>
<td>Thyroid function normal; 24-h urinary steroids low</td>
<td>Empty sella with prechiasmatic adhesions which were lysed</td>
<td>Non-specific inflammation</td>
<td>Vision stabilized O. D. 20/40 O. S. 19/200</td>
<td>Visual loss secondary to interruption of vascular supply by adhesions, possibly secondary to a ruptured cyst (Dahlstrom &amp; Acers 1975)</td>
</tr>
</tbody>
</table>
in Table 1. Many of the case reports are incomplete, and therefore some of the numbers are based on a small number of data points. Selected statistics from series of all patients with the empty sella syndrome are provided for comparison (Neelon et al. 1973; Jordan et al. 1977). Endocrine abnormalities including abnormal response of hGH to stress testing (Matisson & Pimstone 1973; Foley & Posner 1975; Buckman et al. 1976), galactorrhoea (Matisson & Pimstone 1973), secondary hypothyroidism (Jordan et al. 1977), low 24-h urine ketosteroids (Dahlstrom & Acers 1975), panhypopituitarism (Mortara & Norell 1970), and diabetes (Matisson & Pimstone 1973) have been reported.

Of the 29 summarized cases, 8 patients (Robertson 1967; Walsh & Hoyt 1969; Mortara & Norell 1970; Wood & Dogali 1975; Dahlstrom & Acers 1975; Martin et al. 1977) underwent surgical exploration of the optic chiasm and pituitary fossa. The clinical, surgical and pathological findings of these cases are summarized in Table 2. The presence of arachnoid adhesions involving the optic nerve or chiasm was found in 5 patients (cases 1, 2, 5, 7, 8). Although optochiasmatic arachnoiditis has been associated with infection (Craig & Lillie 1931; Coyle 1969; Dahlstrom & Acers 1975), trauma Vail 1938; Dahlstrom & Acers 1975), and spinal anaesthesia (Mandel & Steinmetz 1962), the aetiology is often not apparent (Craig & Lillie 1931; Dahlstrom & Acers 1975). Stretching of the optic nerves or posterior displacement of the chiasm was noted in cases 1, 5, and 6. Prolapse of the optic nerve and chiasm through an incomplete diaphragm sella was present in cases 4 and 7. Pressure on the visual pathways by the anterior cerebral artery (case 4) was also described.

Specific detail of the surgical procedure is included in 4 of the cases. Lysis of adhesions was performed in patients 7 and 8. Chiasmapexy which involved packing the sella with crushed autologous muscle was performed in case 6. Opening of the lamina terminalis to decrease the pressure effect of cerebrospinal fluid was done in case 4. The outcome of the surgery was commented on in 5 cases. Visual field abnormalities and acuity improved following chiasmapexy in case 6. Return to normal visual fields was noted after lysis of arachnoid adhesions in case 7. Progressive decline in visual acuity and constriction of visual fields was stabilized in case 8 following lysis of adhesions. No change was noted in cases 3 and 4 despite the opening of the lamina terminalis in case 4. In a series by Vail (1938), 50% of patients with optochiasmatic arachnoiditis (not associated with empty sella syndrome) noted improvement of visual abnormalities following lysis of adhesions.

The pathophysiology of visual field abnormalities in the primary empty sella syndrome appears to be multifactorial. Interruption of the vascular supply to the optochiasmatic regions secondary to adhesions was the proposed mechanism in case 6. The finding of a comprised circulation to the right optic nerve in our case suggests that vascular interruption is important in the pathogenesis of the visual abnormalities. In addition, the suggested mechanism of visual
field defects in a patient with chiasmal arachnoiditis and a normal sella turcica was a comprised vascular supply (Coyle 1969). Anatomical abnormalities including backward displacement of optic nerve, posterior displacement of the chiasm, stretching of the nerve between the optic foramen and anterior portion of the pituitary fossa, and indentation of optic nerve by the anterior cerebral artery may also be important. Partial prolapse of the optic tracts into the sella is a third possible mechanism. This finding was present in cases 4 and 7. Case 7 also involved arachnoid adhesions. This mechanism may also be important in visual field abnormalities associated with empty sellas secondary to surgery radiation therapy (Lee & Adams 1968). The aetiology of the arachnoiditis is unclear, but cyst rupture or infarct of a pituitary adenoma has been proposed (cases 1, 2, 8).

In summary, there are several points about the primary empty sella syndrome which this case report and literature review emphasize. 1. Although the majority of people with this syndrome have a benign course, a significant number of patients will have functionally important deficits. 2. Visual field defects in association with this syndrome are probably more frequent than previously appreciated. 3. Multidisciplinary examination including endocrinology, neurology, and neurosurgery is indicated when deficits are present. 4. The pathophysiology of visual field deficits in this syndrome appears to be multifactorial. 5. Lysis of adhesions and chiasmapexy have effectively reversed visual deficits in selected cases of the primary empty sella syndrome. 6. Surgical exploration of the optic chiasm seems indicated in the presence of marked visual field abnormalities or progression of field deficits on serial evaluations.

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