Intrasellar aneurysm with subarachnoid hemorrhage and hypopituitarism

Case report

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A patient is described who initially presented with subarachnoid hemorrhage and later with acute hypopituitarism. She had an aneurysm situated entirely within the pituitary fossa. The usual clinical features of parasellar aneurysms are described, and the endocrine assessment of the case is discussed.

Key Words: aneurysm, subarachnoid hemorrhage, hypopituitarism, pituitary tumors, diabetes insipidus

The association of hypopituitarism with intracranial aneurysm is well recognized. Bramwell in 1887 described two patients with clinical features that in retrospect were suggestive of pituitary deficiency and who at autopsy were found to have large aneurysms of the internal carotid artery. Subsequent reports of aneurysms simulating pituitary tumors were fully reviewed by White and Ballantine in 1961. Their series numbered 35 in all, and two later reviews by Van’t Hoff, et al., and Kahana, et al. included five similar cases. A single case report of a patient with an ectatic carotid artery causing hypopituitarism was published in 1967, and further cases of aneurysms compressing the pituitary have been recorded. In all these cases the aneurysms were large; indeed, in one of Bramwell’s patients it measured 3 inches in diameter. All except one, whose neurological examination was not adequately recorded, had evidence of visual impairment or oculomotor weakness; radiological evidence of erosion of the sella turcica was almost invariable. Subarachnoid hemorrhage was uncommon. In other words, these case reports refer to aneurysms of such a size and position as to cause pressure on the hypophysis and frequently the visual pathways, resulting sometimes in hypopituitarism and often in visual failure.

We report here a patient who also had hypopituitarism resulting from an intracranial aneurysm but who differs from all previously recorded cases in that the aneurysm was entirely contained within a normal pituitary fossa. Her vision was unimpaired and she had survived a subarachnoid hemorrhage.

Case Report

First Admission

This 50-year-old woman was first admitted as an emergency on March 31, 1969. She was unable to give a coherent history and all that could be elicited from her husband was that for 3 months she had had occasional frontal headaches and that during the 3 weeks before admission she had become increasingly confused and somnolent. She had had no other specific complaints
and had been in good health previously. Menstruation had ceased 6 months earlier.

**Examination.** The patient was thin and ill-looking, with a temperature of 101.4°F. She was drowsy and confused and had neck stiffness, but Kernig's and Brudzinski's signs were negative. Cranial nerves and fundi were normal. There was slight downward drift of the outstretched arm on the left side but otherwise motor function appeared intact. However, all deep tendon reflexes were pathologically brisk and plantar responses were extensor. Pulse rate was 95/min, and blood pressure was 150/90 mm Hg. General examination was otherwise normal.

Initial routine investigations showed the hemoglobin to be 14.0 gm%, white blood cell count 14,600/mm³, erythrocyte sedimentation rate 36 mm in the first hour, and normal results for urea and electrolytes, serum calcium and phosphorus, and liver function tests. Plain skull and chest films were also normal. An electroencephalogram revealed bilateral synchronous delta wave activity more pronounced on the left side. On the day following admission bilateral carotid angiograms were performed, and the right-sided injection showed a spherical aneurysm, 1 cm in diameter, overlying the pituitary fossa (Fig. 1). The significance of its midline position as judged by the anteroposterior view was not fully appreciated at this stage. The angiogram also showed stenosis in the carotid siphon on the left side; with right carotid compression there was no flow across the midline during a left carotid injection. A lumbar puncture performed following the angiogram revealed xanthochromic fluid under normal pressure.

It was concluded that the patient had had a subarachnoid hemorrhage from the aneurysm, although there was no clear history of such an event. Direct surgical approach to the aneurysm was not considered feasible, and right carotid ligation was contraindicated because of the siphon stenosis on the left. She was therefore treated conservatively and over the next 4 weeks gradually improved. At the end of the seventh week she was discharged home. At this time her mental state was normal, she was free of symptoms, and physical examination was normal apart from persisting briskness of the deep tendon reflexes.

**Second Admission**

Eight weeks after discharge the patient was readmitted as an emergency, having rapidly developed weakness, malaise, and anorexia during a 2-week interval. She was rational though drowsy. She was dehydrated with evidence of peripheral circulatory failure including a pulse rate of 82/min and blood pressure of 90/70 mm Hg. The hemoglobin was 11.8 gm%, white blood cell count 15,200/mm³, blood sugar 82 mg%, serum sodium 110 mEq/L, potassium 3.6 mEq/L, chloride 68 mEq/L, and urea 32 mg%. Electrocardiograph showed inversion of T waves in all leads.

A diagnosis of cortisol insufficiency was made, presumably secondary to hypopituitarism. Treatment was started immediately with high doses of intravenous hydrocortisone and 0.9% saline. Within 24 hours marked improvement was noted and over the next few days she was stabilized on hydrocortisone 30 mg in the morning and 15 mg in the evening. Four days after admission she was noted to be becoming increasingly thirsty and polyuric. A 6½-hour water deprivation test confirmed that she had diabetes insipidus, and regular posterior pituitary snuff controlled the polyuria and polydipsia. Review of the previously performed angiograms convinced us that the aneurysm must lie entirely within the pituitary fossa. The patient was discharged 3 weeks later on the above therapy.

**Third Admission**

After a lapse of 4 months the patient was readmitted for full endocrine assessment. She was then asymptomatic; she had stopped the snuff and had had no return of polyuria or polydipsia; the electrocardiogram was normal. The results of all pituitary function tests carried out on this and the previous admission are recorded in Table 1. These showed a normal response to adrenal stimulation by Synacthen but an impaired response of the pituitary-adrenal axis to insulin-induced hypoglycemia. She was discharged taking hydrocortisone 20 mg in the morning and 10 mg in the evening, and L-thyroxine 0.2 mg daily. When subsequently examined at 6 months and 1 year later, she remained well.

Throughout all of her illness, serial visual

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field charting had revealed no abnormality and skull radiographs remained normal.

Discussion

The fact that a subarachnoid hemorrhage from an aneurysm had caused the initial hospitalization was not diagnosed until after angiography; the history at that stage was more suggestive of a cerebral tumor with increasing intracranial pressure. However, the patient recovered with virtually no sequelae until the later presentation with hypoadrenal shock secondary to hypopituitarism. The fact that her blood pressure remained normal at the time of her first admission despite the stress of a subarachnoid hemorrhage strongly suggests that pituitary-adrenal function at this stage was adequate.

The endocrine tests (Table 1) confirm the diagnosis of hypopituitarism. The plasma 11-hydroxycorticosteroid ("cortisol") of 6.3 μg/100 mL when the patient was shocked must be interpreted as low and suggests impaired "cortisol" secretion. The normal response to Synacthen (250 μg intramuscularly) indicates normal adrenal function, and the poor rise in plasma cortisol during the insulin sensitivity test (0.1 μ/kg body weight intravenously) despite adequate hypoglycemia confirms abnormal function of the hy-
Intrasellar aneurysm, SAH, and hypopituitarism

pithalamic-pituitary axis in the production of adrenocorticotrophin (ACTH). The im-
paired rise in growth hormone (GH) likewise 
confirms pituitary dysfunction. Although the 
protein-bound iodine (PBI) is at the lower 
end of the normal range, the 24-hour I^{131} up-
take (normal greater than 20%) which be-
comes normal after thyrotrophin (TSH) in-
jections confirms impaired TSH secretion 
from the pituitary.

The development of diabetes insipidus 
following initial treatment for hypopituitar-
ism with parenteral fluids and hydrocorti-
sone is well recognized, and the results of the 
blood and urine osmolality measurements af-
fter 6½ hours of water deprivation confirm 
that there was also temporary impairment of 
antidiuretic hormone (ADH) secretion. 
Normally after this period of water depriva-
tion, urine osmolality will rise to at least 
twice that of the blood. We can thus con-
clude that the patient had deficiency of 
ACTH, TSH, GH, and ADH due to pitui-
tary compression by the aneurysm, and the 
rapid deterioration before the second admis-
sion must have been due to sudden failure of 
the remnants which hitherto had been just 
adequate.

We report this case because of the unique 
form and position of the aneurysm. It is not 
surprising that large carotid aneurysms as 
they expand occasionally cause compression 
and destruction of the pituitary fossa, occa-
sionally accompanied by damage to the optic 
pathways. These aneurysms seldom bleed 
and diagnostically they may be difficult to 
distinguish from parasellar tumors of other 
orts. However, in this case the sac of an an-
eurysm came to lie within the fossa itself and 
and to replace the substance of the pituitary 
gland without having caused bone erosion or 
optic nerve compression. The sudden dev-
lopment of hypopituitarism was clearly a 
consequence of the subarachnoid hemor-
rhage, although the precise mechanics must 
remain a matter for speculation.

References
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of polyuria. JAMA 185:699-703, 1963

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
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<tbody>
<tr>
<td>protein-bound iodine</td>
<td>4.0 μg%</td>
</tr>
<tr>
<td>24-hr I^{131} uptake:</td>
<td></td>
</tr>
<tr>
<td>before thyrotrophin</td>
<td>4%</td>
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<tr>
<td>after thyrotrophin</td>
<td>39%</td>
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<tr>
<td>plasma cortisol on admission</td>
<td>6.3 μg%</td>
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<tr>
<td>Synacthen:</td>
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<tr>
<td>basal cortisol</td>
<td>7.8 μg%</td>
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<tr>
<td>30-min cortisol</td>
<td>20.2 μg%</td>
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<tr>
<td>insulin sensitivity:*</td>
<td></td>
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<tr>
<td>basal blood glucose</td>
<td></td>
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<tr>
<td>½ hr</td>
<td>74 mg%</td>
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<td>37 mg%</td>
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<td>37 mg%</td>
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<td>2 hr</td>
<td>41 mg%</td>
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<tr>
<td>2 hr</td>
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<td>basal plasma cortisol</td>
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<td>½ hr</td>
<td>1 μg%</td>
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<tr>
<td>1 hr</td>
<td>1 μg%</td>
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<tr>
<td>1½ hr</td>
<td>1 μg%</td>
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<tr>
<td>2 hr</td>
<td>1 μg%</td>
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<tr>
<td>basal serum growth hormone</td>
<td></td>
</tr>
<tr>
<td>½ hr</td>
<td>1.2 μg/mL</td>
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<tr>
<td>1 hr</td>
<td>1.8 μg/mL</td>
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<tr>
<td>1½ hr</td>
<td>1.1 μg/mL</td>
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<td>1.1 μg/mL</td>
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<td>Dose:</td>
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<tr>
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<td>after 63 hr water deprivation</td>
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<td>297 mO/kg H₂O</td>
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<tr>
<td>basal urine osmolality</td>
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<tr>
<td>after 63 hr water deprivation</td>
<td>122 mO/kg H₂O</td>
</tr>
<tr>
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<td>365 mO/kg H₂O</td>
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* Insulin sensitivity after 5 units of soluble insulin injected intravenously.

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