Bullet injury to the pituitary gland: a rare cause of panhypopituitarism

IBRAHIM S. SALTI, FUAD S. HADDAH, ZAHER N. AMIRI, ALI A. KHALIL, AND AHMAD A. AKAR

From the Departments of Internal Medicine and Surgery, American University Hospital, Beirut, Lebanon

SUMMARY An unusual case of head injury with a direct bullet injury to the pituitary gland is described. The hormonal profile one month after the injury showed severe panhypopituitarism which did not improve one month after surgical removal of the intrasellar bullet fragment.

Head trauma has rarely been implicated as a cause of hypopituitarism with only 17 patients reported in the literature (Altman and Pruzanski, 1961; Klacho et al., 1968; Woolf and Schalch, 1973; Paxson and Brown, 1976). In one additional case, bullet injury to the pituitary region has been implicated, although the hypopituitary state was not well documented (Frank, 1912). The recent availability of hypothalamic releasing hormones permitted a full study of hypothalamic pituitary function in only two patients (Woolf and Schalch, 1973; Paxson and Brown, 1976). In this report we describe the hormonal profile of a patient who had a bullet injury to the pituitary gland resulting in severe panhypopituitarism.

Case reports

A 32 year old single man was referred to the hospital because of a bullet injury to the head, one month before admission. He had been well, having had a normal puberty and normal secondary sex characteristics. After the bullet injury, he lost consciousness for two days, and regained it spontaneously and gradually without any residual symptoms except for a mild left temporal headache, anosmia, sexual impotence, loss of libido, and lack of energy.

Examination revealed a dry cold skin and the scar at the site of entry of the bullet in the right forehead (Fig. 1). His blood pressure was 115/80 mmHg with no postural hypotension. Pulse rate was regular at 88 per min. Movements and speech were slow but the patient was well oriented in time and place. Visual fields revealed a left homonymous hemianopsia. Both fundi were normal. On neurological examination a mild left central facial

Address for correspondence and reprint requests: Dr I. S. Salti, Department of Medicine, American University of Beirut, Beirut, Lebanon.

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Fig. 1 Photograph of patient showing site of entry of bullet in the right forehead.
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Paresis and bilateral anosmia were observed. External genitalia, including size and consistency of both testes, were normal. He had a normal male hair distribution.

Haemoglobin, haematocrit, white blood cell count, fasting serum glucose, creatinine, sodium, potassium, chloride, bicarbonate, calcium, phosphorus, and cholesterol levels were all normal. Urine analysis was normal. An overnight urine specific gravity was 1010, and the patient did not have polyuria or polydipsia.

Skull radiographs revealed a hole in the right frontal bone with in-driven bone fragments in the brain, and multiple fragments of the bullet. The largest, lodged in the sella turcica, measured $9 \times 10 \text{ mm}$ (Fig. 2a and b). A brain scan showed an area of increased uptake of radioactivity in the region of the in-driven bone fragments suggestive of a bullet fragment.

**Fig. 2** Pre-operative lateral (a) and anterior (b) skull radiographs showing in-driven bone fragments and multiple bullet fragments. A major fragment measuring $9 \times 10 \text{ mm}$ is seen within the sella turcica.
Table 1  Profile of endocrine tests

<table>
<thead>
<tr>
<th>Serum</th>
<th>TRH stimulation¹</th>
<th>Insulin hypoglycaemia²</th>
<th>LHRH stimulation³</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>T₄ (nmol/l)</td>
<td>TSH (mU/l)</td>
<td>PRL (µg/l)</td>
</tr>
<tr>
<td></td>
<td>Pre*</td>
<td>Post†</td>
<td>Pre*</td>
</tr>
<tr>
<td>Time (mins)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>10.3</td>
<td>6.4</td>
<td>5.6</td>
</tr>
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<td>15</td>
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<td>4.5</td>
<td>9.6</td>
</tr>
<tr>
<td>120</td>
<td>—</td>
<td>6.3</td>
<td>—</td>
</tr>
</tbody>
</table>

Normal ranges:
Serum T₄: 55.9–154 nmol/l.
Serum TSH: 2.7 mU/l; maximal rise after TRH: 6–25.
Serum PRL: 5–20 µg/l; maximal rise after TRH: 20–120.
Serum GH: 1–5 µg/l; after insulin hypoglycaemia >7 µg/l.
Serum cortisol (8 am): 0.15–0.68 µmol/l, peak value after insulin hypoglycaemia at least double baseline.
Serum LH: 5–20 IU/l, mean peak value after LH-RH: 86.3 ± 28.2.
¹ TRH, 200 µg given as a single intravenous dose.
² LH-RH 200 µg given as a single intravenous dose.
* Preoperative study.
† Postoperative study.
of a reaction in that location. A right carotid arteriogram was normal.

Baseline serum thyroxine (T₄), growth hormone (GH), prolactin (PRL), glucose, cortisol and follicle stimulating hormone (FSH), and luteinising hormone (LH) levels are shown at the top of Table 1. There was no response of serum TSH and PRL to administration of thyrotropin release hormone (TRH) (Table 1), nor was there a response of serum GH and cortisol to insulin-induced hypoglycaemia. On the other hand, there was a sluggish, yet definite response of serum LH to administration of luteinising hormone release hormone (LHRH), while serum FSH showed no response. Baseline urinary 17-ketosteroid and 17-hydroxysteroid outputs were low and showed no rise after metapyrone administration (Table 2). Baseline serum testosterone level was 0.027 nmol/l (normal range, 1.04–3.47 nmol/l) and rose to 0.29 nmol/l on the fourth day of treatment with human chorionic gonadotropin (HCG), 5000 units intramuscularly per day.

A right frontal craniotomy was performed with excision of granulation tissue and in-driven bone fragments from the right frontal lobe. The right subfrontal area was explored through an extension of the previous incision in the forehead. The arachnoid mater was extremely thickened and had to be cut with a sharp instrument. The optic nerves were normal, but a cavity filled with granulation tissue was found between the optic nerves. The bullet fragment was found within the granulation tissue, inferior and slightly medial to the right optic nerve. It was removed with no difficulty. The postoperative course was satisfactory.

Pituitary functions were re-evaluated four weeks after the operation and were found unchanged (Tables 1 and 2). The visual fields and anosmia were unchanged.

The patient was discharged on replacement therapy with prednisone 7.5 mg/day, levothyroxine sodium 0.2 mg/day and HCG, 5000 units intramuscularly every two days. One month later, he showed a definite improvement in his energy and potency. He continued to have no polyuria or polydipsia and normal urinary specific gravity.

Hormonal determinations were performed according to methods previously reported (Arafah and Salti, 1978).

**Discussion**

The results of endocrine studies on this patient indicate that one month after the injury, he had almost total anterior pituitary insufficiency resulting in hypogonadism, adrenocortical insufficiency, and absence of prolactin and growth hormone secretions. The deficiencies improved on a second evaluation a month after surgical removal of the bullet fragment. Although the patient did not have overt evidence of diabetes insipidus, a partial deficiency of antidiuretic hormone could have been masked by the coexistent adrenal and thyroid deficiencies.

The lack of response of serum PRL and TSH to TRH stimulation indicate deficiencies of the thyrotropic and lactotropic hormones in the pituitary gland. The absence of response of serum GH and cortisol to insulin-induced hypoglycaemia could be the result of either pituitary or hypothalamic injury or both. The definite, albeit small response of serum LH after LHRH administration suggests the presence in the pituitary of some residual luteotrophic hormones. Thus, the low baseline serum LH and testosterone could be, at least in part, the result of a coexistent hypothalamic or infundibular injury resulting in failure of release of LHRH.

Endocrine evaluation of the previously described patients revealed pituitary insufficiency of varying degrees. In one of the two patients who underwent investigation with hypothalamic releasing hormones, the pituitary failure was at-

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Table 2  Results of metapyrone test

<table>
<thead>
<tr>
<th>Day</th>
<th>Treatment</th>
<th>17-Hydroxy-corticosteroids mg/24hr</th>
<th>17-Ketosteroids mg/24hr</th>
<th>Creatinine mmol/l Volume ml/24hr</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre*</td>
<td>Post†</td>
<td>Pre*</td>
<td>Post†</td>
</tr>
<tr>
<td>1</td>
<td>none</td>
<td>2.1</td>
<td>0</td>
<td>1.8</td>
</tr>
<tr>
<td>2</td>
<td>metapyrone‡</td>
<td>2.5</td>
<td>1.1</td>
<td>5.7</td>
</tr>
<tr>
<td>3</td>
<td></td>
<td>2.6</td>
<td>0.8</td>
<td>2.5</td>
</tr>
</tbody>
</table>

Normal:

| 1 | Urinary 17-hydroxycorticosteroids: 2–10 mg/24hr maximal rise after metapyrone at least double baseline. |
| 2 | Urinary 17-ketosteroids: 10–25 mg/24hr. |
| * | Preoperative. |
| † | Postoperative. |
| ‡ | Dosage 750mg every 4 hours for 6 doses. |
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tributed to a hypothalamic lesion (Woolf and Schalch, 1973). While hypopituitarism is apparently rare in patients who survive head trauma (Altman and Pruzanski, 1961), pituitary necrosis was observed in as many as 22% of cases of fatal head injury (Kornblum and Fisher, 1969). It has been suggested that undetected, and hence untreated hypopituitarism, may be a contributory factor to the fatal outcome of severe head trauma (Klacho et al., 1968).

It is possible that in some patients who survive head injury, hypopituitarism may develop but may go undetected indefinitely or may improve with time. In the majority of the reported cases of post-traumatic hypopituitarism, the diagnosis was made several months or years after the trauma (Altman and Pruzanski, 1961; Klacho et al., 1968). The detection of the hypopituitary state in our patient as early as one month after the injury was a reflection of the fact that there was direct evidence of pituitary destruction which may account for the failure to detect any improvement in pituitary function one month after surgical removal of the bullet. In head trauma, recovery of pituitary function presumably involves regeneration of pituitary portal vessels and anterior pituitary cells (Daniel et al., 1959). It is possible that, in our patient, one month was too short a period for this regenerative process to be of any functional significance or that the hypopituitarism was a permanent state.

References


