Is "benign intracranial hypertension" really benign?

S G Barber and N Garvan

From the Department of Medicine, General Hospital, Birmingham, and the Department of Neurosurgery, Midland Centre for Neurology and Neurosurgery, Smethwick

SUMMARY Hypothalamic-hypophyseal insufficiency has been found in seven of eight patients with so-called benign intracranial hypertension, of whom four showed an inadequate adrenal response to stress. The syndrome of benign intracranial hypertension cannot therefore be considered entirely benign and patients should receive full endocrinological assessment and follow up.

The development of sensitive and specific tests of hypothalamic and pituitary function has enabled the mechanisms of pituitary infarction to be re-examined. The ability to monitor intracranial pressures led to the detection of bursts of intracranial hypertension in "normal pressure" hydrocephalus which have been implicated as the cause of hypothalamic hypophysyal damage in such patients. Since the intracranial pressure in patients with the syndrome of benign intracranial hypertension may be severe enough to cause blindness in addition to papilloedema, we decided to assess the endocrine status of such patients.

Patients, Materials and Methods

Nine patients aged 24–55 years (mean 41·9 years) were diagnosed as having benign intracranial hypertension on the basis of history, physical examination, and the absence of demonstrable pathology to explain the raised intracranial pressure. One patient was male and the others female. Anterior pituitary function was assessed in response to insulin-induced hypoglycaemia, thyrotrophin-releasing hormone (TRH), and gonadotrophin-releasing hormone (GnRH) after an overnight fast. Basal samples were taken for routine blood count, biochemical profile, thyroxine, T3 uptake test, and either oestradiol or total androgen (depending on the sex of the patient). All hormone assays were performed subject to the quality control criteria of the supraregional assay service, using methods described elsewhere. Blood sugar estimations were performed by the ferricyanide method of Technicon. All blood samples were taken via an indwelling forearm venous cannula. In the absence of suggestive symptoms, posterior pituitary function was not tested.

Results

Basal thyroid function (thyroxine and T3 uptake) tests were normal in all patients. The results of other endocrine tests are shown in the table. Only two patients (cases 2 and 6) had received steroids at any time and in the former they had not been given for six months before testing. The latter patient was on dexamethasone at the time of testing and this probably explains the blunted, sustained TSH response to TRH.

The cortisol response to insulin-induced hypoglycaemia was abnormal in five patients (cases 1, 4, 7, 8, and 9) and was not measured in the patient receiving dexamethasone. Although the basal resting cortisol levels were within normal limits, the response to stress was inadequate. All patients experienced definite symptoms of hypoglycaemia (see table). Growth hormone responses to the same hypoglycaemic stimulus were abnormal in five patients (cases 2, 4, 6, 7, and 8) although a rise from undetectable levels to 18 mU/l in case 6 probably represented "normal" function for a patient receiving steroids.

The luteinising hormone (LH) response to GnRH was abnormally sustained in three patients.
Discussion

Benign intracranial hypertension presents with signs and symptoms of obstructive hydrocephalus, including proptosis, exophthalmos, and papilloedema. Our findings suggest that hypothalamic damage, as evidenced by a decrease in hypothalamic-pituitary axis function, plays a role in the pathogenesis of this condition. The decrease in FSH levels observed in our patients may be related to hypothalamic damage, as FSH levels are known to be under hypothalamic control. The decrease in prolactin levels, on the other hand, may be due to a primary pituitary defect, as prolactin levels are under direct hypothalamic control.

In conclusion, our findings suggest that hypothalamic damage plays a role in the pathogenesis of benign intracranial hypertension. Further research is needed to clarify the role of hypothalamic damage in the pathogenesis of this condition and to better understand the mechanisms underlying the observed hormonal changes.

References

cranial hypertension may be caused by hypothalamic damage leading to inadequate production of prolactin-release-inhibiting factor.

The difficulties in interpreting the results in patients receiving steroids are exemplified in case 6, where the TSH and growth hormone responses may have been due either to the steroids or to local damage, and the ACTH-adrenal response to stress could not be tested. In the light of the other results, patients such as these need reassessing after cautious withdrawal of steroids, and replacement therapy with steroids should be given for trauma or illness pending such reassessment.

So-called benign intracranial hypertension may not be completely benign and patients with this diagnosis should be assessed and followed up endocrinologically. Assessment should be performed after cessation of steroid therapy if this has been given, and if patients have an inadequate cortisol response to stress, replacement therapy should be started, as for other patients with adrenal insufficiency.

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References


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