SHORT REPORT

Computed tomographic evidence of cerebral swelling in benign intracranial hypertension

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Abstract

Computed tomography of 30 patients presenting acutely with benign intracranial hypertension was compared with that of 30 normal controls matched for age and sex. Qualitative and quantitative assessments showed smaller cranial CSF spaces in the cases of benign intracranial hypertension, suggesting that cerebral swelling is involved in the pathogenesis of benign intracranial hypertension.

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The pathogenesis of benign intracranial hypertension is unknown. There have been conflicting reports of cerebral oedema,¹ cerebral hyperaemia,² and increased resistance to CSF drainage,³ but it remains unclear which element of the cerebral contents is increased in volume. The aim of this study was to examine the size of the cranial CSF spaces on CT of patients with benign intracranial hypertension compared with age and sex matched normal controls to determine whether raised intracranial pressure in benign intracranial hypertension is associated with an increase in volume of the cerebral tissues or the CSF.

Methods

Thirty consecutive patients with newly diagnosed benign intracranial hypertension who presented to the Departments of Medical and Surgical Neurology in Edinburgh between 1985 and 1992 were studied. Inclusion criteria comprised papilloedema; a CSF pressure greater than 25 cm of water; normal CSF constituents; and no evidence of intracranial

mass lesion or hydrocephalus. Computed tomography was performed on a General Electric 8800 scanner at the initial presentation with suspected benign intracranial hypertension. The 30 control CT scans were derived from scans of a cohort of 62 patients with peripheral sensory symptoms, in whom detailed inpatient investigation showed no pathology of the CNS. Cases were paired with controls according to sex and nearest date of birth. No other criteria were used and matching was blind to the CT appearance.

On two occasions, three months apart, the 60 scans were shown independently to two neuroradiologists (RJG and RJS) who were blind to the clinical diagnosis. They were asked to grade the lateral ventricles as small, normal, or large, the third ventricle as normal, slit, or absent, and the cerebral sulci as normal or effaced. Each scan from a patient with benign intracranial hypertension was then presented with an age and sex matched control scan and the neuroradiologist was asked to indicate which scan suggested the lower cranial CSF volume. The size of the third and lateral ventricles, the sulci, and the basal cisterns were each taken into account in this qualitative assessment.

Three previously validated quantitative indices of ventricular size—the Huckman Index,⁴ the inverse cella media index,⁵ and the third ventricular diameter⁶—were also measured on the 60 scans by a single observer (PMR) who was blind to the clinical details.

Results

Only abnormal CT scan findings on which both radiologists agreed are reported. Small lateral ventricles, a slit or absent third ventricle, and effaced cerebral sulci were each significantly more frequent in cases of benign intracranial hypertension than in controls (table 1). The radiologists agreed on which scan showed the smaller cranial CSF spaces in 28 of the 30 case and control pairs (93%), choosing the benign intracranial hypertension scan in 24 (86%). The between observer and within observer agreements of the radiologists for each of the CT assessments were high (table 2). The mean values of each of the three quantitative indices of ventricular size were significantly lower in cases of benign intracranial hypertension than in controls

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Table 1 Qualitative assessments on CT of 30 cases of benign intracranial hypertension and 30 controls about which both radiologists agreed

	Small lateral ventricles	Slit/absent 3rd ventricle	Effaced Sulci	Smaller cranial CSF spaces
CT:				
Cases (n = 30)	9	15	13	24
Controls (n = 30)	1	2	1	4
Odds ratio (95% CI)	12.4 (1.5-105)	14 (3.0-70)	22 (2.7-185)	6 (1.8-20)

Table 2 The reproducibility (kappa statistic (SD)) of the qualitative assessments of the two neuroradiologists on 60 CT scans

Assessment	Between observer agreement	Within observer agreement	
		A	B
Size of third ventricle	0.7 (0.09)	0.85 (0.06)	0.74 (0.07)
Size of lateral ventricles	0.56 (0.11)	0.60 (0.11)	0.54 (0.12)
Effacement of sulci	0.68 (0.1)	0.91 (0.06)	0.78 (0.12)
Smaller cranial CSF spaces	0.76 (0.08)	0.82 (0.07)	0.80 (0.07)

Table 3 Mean (SD) values of three quantitative measures of ventricular size in 60 CT brain scans

	Third ventricle diameter (mm)	Huckman index	Inverse cella media index
CT:			
BIH cases (n = 30)	0.70 (0.46)	49 (21)	0.074 (0.02)
Controls (n = 30)	1.44 (0.68)	73 (27)	0.124 (0.07)

BIH = benign intracranial hypertension.

(table 3), indicating a smaller ventricular size in patients with benign intracranial hypertension.

Discussion

Small lateral ventricles, a slit or absent third ventricle, and effaced cerebral sulci were often seen on CT in cases of benign intracranial hypertension, but rarely in controls. When age and sex matched case and control scans were compared directly, the benign intracranial hypertension scans showed consistently smaller cranial CSF spaces than controls. The high between and within observer agreements for these qualitative assessments support their validity. Indeed, such highly reproducible qualitative measures are likely to be more valid than arbitrary and technically difficult semiquantitative measures.⁴

Five previous studies have looked at cerebral ventricular size by CT of patients with benign intracranial hypertension.⁷⁻¹¹ Only one found small ventricles, but CT of patients with multiple sclerosis and cerebrovascular disease, which may be associated with cerebral atrophy and ventricular enlargement, were used as controls.¹¹ Of the remaining studies, three had no controls⁷⁻⁹ and all were limited by the small number of cases. The sizes of the other cranial CSF spaces, such as the basal cisterns and cerebral sulci have not been studied.

Small or slit lateral ventricles, a slit or absent third ventricle, and effaced basal cisterns and sulci are seen on CT after acute head injury and correlate with the severity of raised intracranial pressure and the degree of cerebral swelling at postmortem.^{12,13} The identical CT findings in this study raise the possibility that cerebral swelling is important in the pathogenesis of raised intracranial pressure in benign intracranial hypertension. This is supported by the finding that ventricular size on CT,¹⁴ and cranial CSF volume, measured with MRI,¹⁵ increase with resolution of benign intracranial hypertension.

It is possible that small cranial CSF spaces in patients with benign intracranial hyperten-

sion are constitutional and might predispose the patient to develop raised intracranial pressure. The Monro-Kellie doctrine states that the cranial contents are incompressible and the total intracranial volume must remain constant. Raised intracranial pressure due to cerebral swelling, caused by oedema or hyperaemia, can only be compensated for by a reduction in cranial CSF volume. In patients with constitutionally small cranial CSF spaces, this compensatory capacity would be limited, thereby increasing the risk of developing raised intracranial pressure in response to minor degrees of cerebral swelling.

The reduction in the size of the cranial CSF spaces relative to the cerebral volume in cases of benign intracranial hypertension, compared with controls, does not support the popular hypothesis that raised intracranial pressure in patients with benign intracranial hypertension is caused by an increased resistance to CSF reabsorption.³ There is disagreement as to whether increased resistance to reabsorption of CSF would necessarily lead to hydrocephalus, but even if it is accepted that hydrocephalus would not ensue, it is difficult to explain the smaller than expected cranial CSF spaces. Increased intracranial pressure leads to reflex cerebral vasodilation, which maintains cerebral blood flow.¹⁶ Raised intracranial pressure due to resistance to CSF drainage could in theory cause cerebral swelling due to this reflex increase in cerebral blood volume. Animal experiments, however, suggest that the increase in cerebral blood volume expected at the levels of intracranial pressure found in benign intracranial hypertension is small, equivalent to less than 1% of cranial volume.¹⁷ There is evidence of increased cerebral blood volume in patients with benign intracranial hypertension, but the increase is much greater than would be expected from autoregulation alone,^{16,18} and there is no fall in cerebral blood flow after restoration of intracranial pressure to normal by lumbar puncture.¹⁷ Moreover, the largest and most detailed study of CSF haemodynamics in benign intracranial hypertension found that in most cases, resistance to CSF reabsorption was normal or only moderately increased, and concluded that such changes were probably secondary to compression of the venous sinuses and lateral lacunae due to cerebral swelling.¹⁹ This is consistent with the finding of increased resistance to CSF reabsorption in association with cerebral tumours and other conditions associated with raised intracranial pressure.^{20,21} The findings of our study support the hypothesis that cerebral swelling is the primary pathophysiological event in benign intracranial hypertension. Further research is required to determine whether this is due to oedema or hyperaemia.

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