Br Ir Orthopt J 2004; **1:** 52–58

Paediatric idiopathic intracranial hypertension

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Abstract

***Aim*: Idiopathic intracranial hypertension usually occurs in adults. The purpose of this study was to review paediatric idiopathic intracranial hypertension and determine the specific characteristics and outcome of the paediatric condition compared with adult idiopathic intracranial hypertension.**

***Methods*: Thirteen case notes were retrospectively reviewed (11 females, 2 males). The average presenting age was 13.5 years (range 10–17 years). Diagnosis of idiopathic intracranial hypertension followed exclusion of other neurological causes. Each patient underwent full ophthalmic, orthoptic and neurological evaluation.**

***Results*: Three patients were asymptomatic whereas the remainder presented with symptoms including headaches, visual disturbances, lethargy, vomiting and nausea. Each patient had papilloedema. Initial lumbar puncture opening pressures ranged from 160 to 790 mmH_2O. Orthoptic findings included concomitant esotropia, sixth, third and fourth nerve palsies. Visual fields were impaired in 9 cases. Treatment included prednisolone, acetazolamide, lumbar peritoneal shunting and optic nerve sheath fenestration.**

***Conclusions*: Visual fields were impaired in 9 patients despite absence of visual symptoms in 3. Medical treatment was usual although 3 patients required lumbar peritoneal shunting and 1 required optic nerve sheath fenestration to arrest visual deterioration. The paediatric visual outcome of idiopathic intracranial hypertension reflects the adult condition. Paediatric patients require the same evaluation, follow-up and treatment regimes to optimise outcome.**

Key words: Idiopathic intracranial hypertension, Paediatric, Treatment, Visual field loss, Visual outcome

Introduction

Idiopathic intracranial hypertension was first described by Quincke in 1893[1] and again in 1897,[2] who termed the condition 'serous meningitis'. Today, three terms remain in common use: pseudotumor cerebri, benign intracranial hypertension and idiopathic intracranial hypertension. Idiopathic intracranial hypertension has been used in more recent studies[3–5] and is the term used here as it more accurately describes the cases of raised intracranial pressure of unknown cause where there is potential for serious visual loss.

Treatment of the condition is well documented and is indicated in the presence of progressive visual loss. In many cases, treatment is also directed at the disturbing and debilitating symptom of headache. Medical and non-invasive treatment includes the use of acetazolamide,[6] steroids[7] and diet.[8] Surgical techniques include optic nerve sheath fenestration[9,10] and shunt procedures.[11,12] Varying levels of success have been reported with these modes of treatment in idiopathic intracranial hypertension.

Visual loss, which is the only serious complication of idiopathic intracranial hypertension, may occur either early or late in the course of the disease, may be sudden or gradually progressive and is often avoidable with appropriate therapeutic intervention. The rate of vision loss is variable, symptoms may be minimal and in many patients the vision loss may be undetected until profound.[13] Wall and George[5] and Rowe and Sarkies[4] reported that visual loss is reversible with treatment and documented significant improvement in visual field loss. It is therefore of utmost importance that visual function be monitored appropriately in patients with idiopathic intracranial hypertension to detect any deterioration of vision, to treat patients promptly and to maintain their normal or optimum visual ability.

Recent literature has included studies concentrating on paediatric idiopathic intracranial hypertension addressing the epidemiology and general profile of the condition. It is the purpose of this study to review the characteristics of the paediatric condition in comparison with adults with idiopathic intracranial hypertension.

Methods

Thirteen patient case notes were reviewed in this study from two clinical centres: 11 female and 2 male. The mean age at presentation was 13.5 years (range 10–17 years). Table 1 shows the individual patient details.

In many studies diagnosis of idiopathic intracranial hypertension is subject to the modified Dandy criteria (Table 2),[3–5] which relate to the signs and symptoms of raised intracranial pressure in an otherwise healthy

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Table 1. Patient details

Patient no.	Age at presentation	Gender	Symptoms	ICP (mmH_2O)	BMI (kg/m^2)	Associated factors	Visual field grade[a]		Ocular motility[b]	Treatment
							Initial	Final		
1	10	F	Nil	380	26	Nil	2 (Goldmann)	0 (Goldmann)	Normal	Acetazolamide
2	11	F	Headache, lethargy, vomiting, photophobia	395	17	Nil	3 (Goldmann)	5 (Goldmann)	VI comitant esotropia	Acetazolamide, prednisolone, lumbar shunt
3	11	F	Headache, nausea	160	20	Nil	0 (Goldmann)	0 (Goldmann)	Normal	Acetazolamide
4	11	F	Nil	350	27	Nil	1 (Humphrey)	0 (Humphrey)	Normal	Nil
5	11	F	Headache	400	23	Ear infection	2 (Humphrey)	1 (Humphrey)	VI, III	Acetazolamide
6	13	M	Headache	340	20	Head injury	0 (Goldmann)	0 (Goldmann)	Normal	Nil
7	14	F	Headache	790	37	Ear infection	2 (Humphrey)	0 (Humphrey)	VI	Acetazolamide
8	14	F	Headache	330	35	Nil	1 (Humphrey)	0 (Humphrey)	IV	Lumbar shunt, optic nerve sheath fenestration
9	16	M	Headache, TVO	175	24	Nil	0 (Goldmann)	0 (Goldmann)	Normal	Acetazolamide
10	16	F	Visual disturbance	790	32	Nil	4 (Humphrey)	3 (Humphrey)	VI	Acetazolamide
11	16	F	Headache	550	27	Flu	2 (Humphrey)	1 (Humphrey)	Normal	Lumbar shunt
12	17	F	Nil	290	43	Nil	3 (Humphrey)	3 (Humphrey)	Normal	Nil
13	17	F	Headache	250	28	Tetracyclines	0 (Humphrey)	0 (Humphrey)	Normal	Nil

ICP, intracranial pressure (normal range below 200 mmH_2O); BMI, body mass index (weight in kilograms divided by the square of height in metres; normal range 18–24); TVO, transient visual obscurations.
[a] Visual field grades: 0, normal; 1, minimal field loss; 2, mild field loss; 3, moderate field loss; 4, marked field loss; 5, blinding field loss. Goldmann/Humphrey indicates the method of visual field assessment employed.
[b] Ocular motility: VI, sixth nerve palsy; IV, fourth nerve palsy; III, third nerve palsy.

patient with no other evidence of pathology relating to the intracranial hypertension. In all but 2 cases initial diagnosis could be made according to these criteria.

Each patient underwent full ophthalmic, orthoptic and neurological examinations including a neuroimaging study. Body mass index was calculated based on weight and height measures (weight in kilograms divided by the square of height in metres); a measure greater than 25 indicates overweight and greater than 30 indicates obesity. Ophthalmic assessment included evaluation of colour vision, visual field assessment with Humphrey automated and/or Goldmann manual perimetry[4] dependent on the patient's ability at each assessment, examination of the fundus with fundus photography where indicated and assessment of ocular alignment. The 24-2 program was used on Humphrey perimetry which tested 54 points with a 6° spaced grid offset from the vertical and horizontal meridians. A modified Armaly–Drance strategy (Fig. 1) was used on Goldmann perimetry which specifically assesses the kinetic boundaries along horizontal and vertical meridia and static points within the central 30° and temporal sector of the visual field.

The criterion for reliable Humphrey visual fields was defined as achievement of 75% or greater reliability by monitoring fixation loss, positive and negative errors. Visual field results were graded according to their presence and severity using the grading scheme described by Wall and George (Table 3).[5] Change in the visual field was recorded as a change in grade of the visual field result over time and/or a change in mean deviation and global indices over time on Humphrey perimetry. The standards for visual outcome were based primarily on the visual field grading systems. An excellent outcome related to a normal visual field (grade O), a good outcome to a minimally to mildly impaired visual field (grades 1 and 2), a satisfactory outcome to a moderately impaired visual field (grade 3) and a poor outcome to a markedly impaired or blinding visual field (grades 4 and 5).

A database was designed to record patient details and a computerised statistical program (SPSS version 9) was used to analyse the results with the following statistical tests: comparison of normally distributed quantitative data was with the sign test and comparison of normally distributed quantitative data for two or more variables was with the independent *t*-test. Investigation of patients was according to the guidelines of the Declaration of Helsinki.

Table 2. Modified Dandy criteria[3–5]

- Signs and symptoms of increased intracranial pressure
- An awake and alert patient
- Normal neuro-imaging studies except for small ventricles or empty sella
- Documented increased pressure (>200 mmH_2O in non-obese patients and >250 mmH_2O in obese patients) with normal cerebrospinal fluid composition
- No other cause of intracranial hypertension present

Results

At presentation 10 patients complained of symptoms of headaches, lethargy, nausea, vomiting, photophobia and visual disturbance. The remaining 3 patients were asymptomatic and had been referred as papilloedema had been noted by chance at routine optometry assessment. Each patient had papilloedema documented at initial assessment. Differential diagnoses included pseudopapilloedema and optic disc drusen.

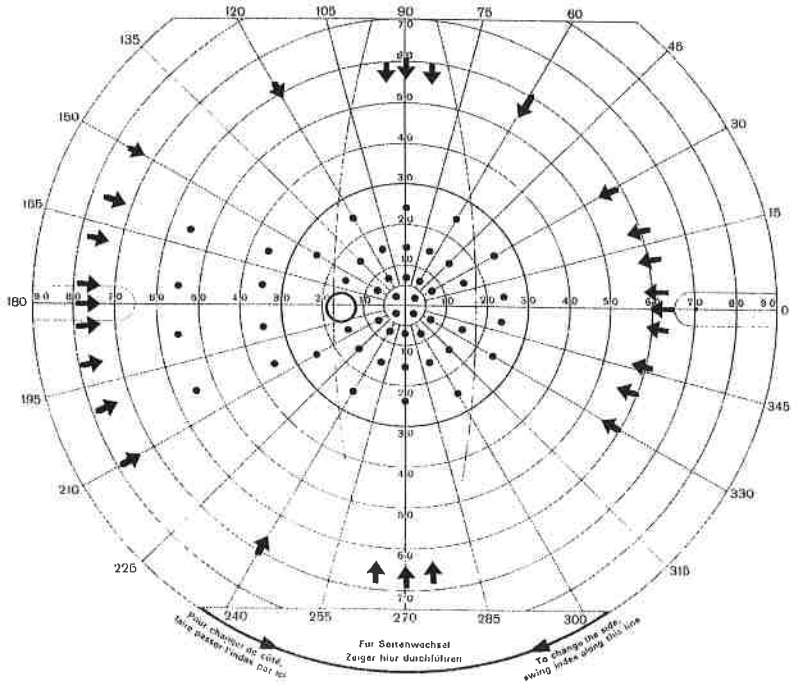

Fig. 1. Modified Armaly–Drance strategy. Arrows indicate the direction from which the kinetic target is moved from the periphery of the visual field. Points indicate the position of the static target presented in the central visual field.

Initial lumbar puncture opening pressure ranged from 160 to 790 mmH_2O with a mean pressure of 400 mmH_2O (normal range is below 200 mmH_2O). Only 2 patients had a pressure measurement below 200 mmH_2O but with subsequent measurements of raised intracranial pressure. Further neurological assessment was normal with no apparent abnormalities on CT and/or MRI brain scans.

Associated factors were noted in 5 patients including ear infection, 'flu, minor head injury and use of tetracyclines. However, there was no evidence of secondary intracranial hypertension due to venous sinus thrombosis, inflammation, blood clotting disorder or other pathology in any case. Five children had normal weight for height, 4 were classed as overweight and 4 were classed as obese. Five children under the age of 12 years had a mean BMI of 22.6 kg/m^2. Eight children aged 13 years or above had a mean BMI index of 30.75 kg/m^2.

Orthoptic assessment revealed normal ocular motility and ocular alignment in 8 patients. Three patients had acquired sixth nerve palsy, 2 of whom recovered and 1 of whom developed a comitant left esotropia. One patient had an acquired sixth nerve and partial third nerve palsy which recovered during follow-up. Recovery periods ranged from 6 weeks to 3 months. One patient had a congenital fourth nerve palsy with consistent repeated measurements over her period of follow-up.

Visual field assessment at initial presentation showed normal results in 4 patients. The remaining 9 patients had field defects ranging from grade 1 to 4, which included enlarged blind spots, arcuate defects, paracentral scotomas, nasal step defects and global constriction of the field. Consideration of the differences in the visual field grades at initial and last assessment using a discrete version of the sign test indicated that the improvements in field deficit scores were significant ($p \leqslant 0.035$) at the 95% confidence limit. Their follow-up period was a mean of 2.8 years (range 1.5–8 years) and at last assessment their fields ranged from grade 0 to 5. Figs. 2 and 3 show visual field results at initial and last assessment for patients 2 and 7. The patient with a grade of 5 was the only child to develop secondary optic atrophy. A higher intracranial pressure measurement (mean 475 mmH_2O) was obtained for those patients with documented visual field defects compared with those with normal visual fields (mean 231 mmH_2O), which was significant ($p = 0.036$, independent t-test).

Active treatment was not implemented in 4 patients. Six patients received acetazolamide only, 1 had a lumbar peritoneal shunt and 2 patients had combined treatment modalities including optic nerve sheath fenestration, acetazolamide, prednisolone and lumbar peritoneal shunt. Children requiring intervention had a higher mean intracranial pressure of 441 mmH_2O compared with those not requiring treatment (mean 307 mmH_2O), but this was not significant ($p = 0.286$, independent t-test).

Table 3. Grading scheme for visual field assessment[5]

Grade 0	Normal visual field
Grade 1	Minimal visual loss – unlikely to be noticed by the patient
Grade 2	Mild visual field loss – may be noticed by the patient and usually compromises function
Grade 3	Moderate visual field loss – nearly always noticed by the patient and interferes with function
Grade 4	Marked visual field loss
Grade 5	Blinding visual loss

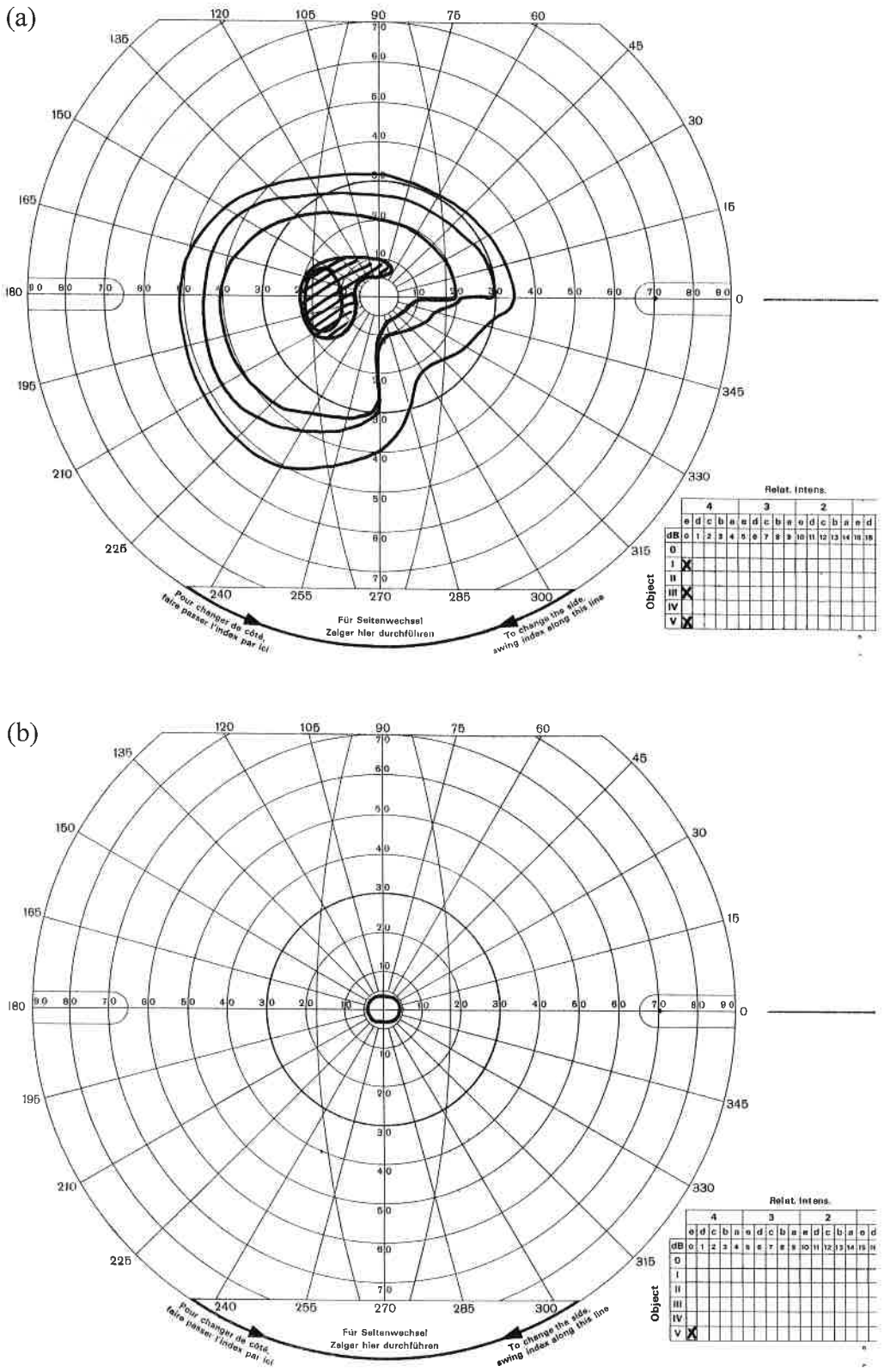

Fig. 2. Goldmann visual field assessment at initial (*a*; grade 3) and final (*b*; grade 5) assessment for patient 2.

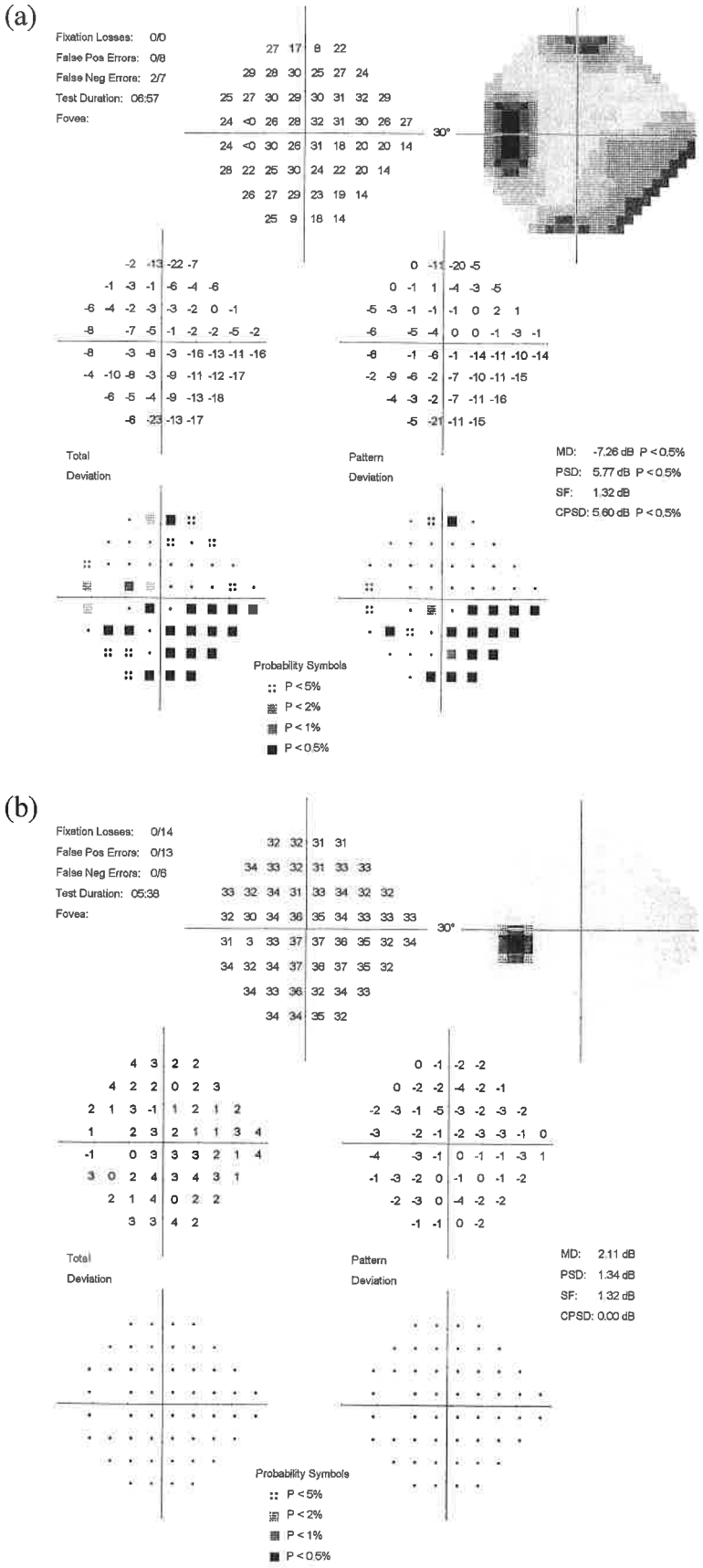

Fig. 3. Humphrey visual field assessment at initial (*a*; grade 2) and final (*b*; grade 0) assessment for patient 7.

Discussion

The majority of studies on idiopathic intracranial hypertension report the condition in either adults or children or both, but predominantly the former.[3–5,14–17] In recent years there have been more specific studies of the paediatric population of idiopathic intracranial hypertension looking at epidemiology and general profile.[18–20] This study specifically reviews a paediatric population and the characteristics will be discussed in relation to those of an adult population who have undergone similar medical evaluation.[4] The diagnosis of idiopathic intracranial hypertension is important and therefore it is imperative that those clinicians who routinely assess children are aware of the specific characteristics of the condition in the childhood population.

The diagnosis of idiopathic intracranial hypertension should relate to the signs and symptoms of raised intracranial pressure but should also be a diagnosis of exclusion to ensure no other existing neurological pathology is missed. Documentation of elevated intracranial pressure is important but such measures can be difficult to obtain in children,[21–23] as occurred in two cases in this study. Definitive diagnosis could be made at follow-up with subsequent measures of elevated intracranial pressure.

The most common presenting symptoms in children have been reported as headaches, blurred vision, stiff neck and diplopia.[24–26] The most common symptom in this study was headache, and this and the other symptoms reported in this study were typical of those experienced by adults with idiopathic intracranial hypertension.[27,28] The finding of asymptomatic idiopathic intracranial hypertension in 3 of the children in this study was not unusual as asymptomatic cases have been reported in the adult condition.[27,29]

The prevalence of overweight and obesity in this childhood population has been addressed in a separate study due to the specific importance of this issue in idiopathic intracranial hypertension.[30] Of note is the age-related differences in the prevalence of overweight and obesity. Suspicion of this diagnosis should occur in the adolescent population with evidence of weight increase, whereas younger pre-adolescent children are more likely to have normal weight-for-height measures and as such differ from the adolescent and adult condition in this respect.

The findings of the orthoptic assessment were normal in 8 cases. In the remaining 5, diagnoses of sixth nerve palsy, combined sixth and partial third nerve palsy and congenital fourth nerve palsy were made.[31,32] A wide variety of ocular motility disturbances have been reported in both the adult and childhood condition and include the typical false localising sign of sixth nerve palsy, third and fourth nerve palsy, sensory exotropia and comitant esotropia.[31–34] One possible outcome of sixth nerve palsy with onset in childhood is the development of concomitant esotropia rather than recovery to normal ocular motility. This has been reported previously in paediatric idiopathic intracranial hypertension[33,34] and also occurred in 1 patient in this study.[35]

Visual function was assessed routinely in all cases. The most important evaluation was visual field assessment, done either by automated Humphrey perimetry or manual Goldmann perimetry, or both. Visual field assessment in children can be perceived as difficult. However, children are currently so computer- and playstation-literate that visual field assessment, using automated perimetry in particular, is not that difficult to achieve and should certainly be attempted even in younger children.

Nine children had visual field loss documented at presentation that ranged from mild deficit which was not noted by the patient (grade 1) to marked visual field loss (grade 4). Previous studies have documented visual field impairment in up to 96% of patients.[4,5,13,15] The visual field defects documented were typical of those reported in the adult condition,[4,14,36] including the typical enlarged blind spots and field defects of nasal steps, arcuate defects and paracentral scotomas. Global constriction was associated with more extensive grades of visual field loss.

The prognosis for visual outcome in idiopathic intracranial hypertension is generally good. Where treatment is required prompt intervention results in a favourable outcome.[37] Generally indications for the treatment of idiopathic intracranial hypertension include recent visual field loss, progressive visual field loss in a pre-existing visual field defect, a reduction in visual acuity not due to macular oedema and persistent diplopia. Where the patient is asymptomatic with no visual deficit, treatment is not indicated. Four patients in this study did not receive any treatment but were observed over their follow-up period with no deterioration of their condition. Acetazolamide was sufficient as a sole treatment agent in 6 cases for alleviating the symptoms and signs of idiopathic intracranial hypertension. However, surgery or combined surgery and medical treatment was required in 3 patients.

There was a significant improvement in the level of visual function over time, with 10 patients having normal visual fields or only minimal visual deficit of grade 1 at last assessment. This improvement was similar to that reported in previous studies.[4,5] Two patients had moderate visual field deficit and 1 patient had marked visual deterioration to grade 5 with development of optic atrophy. Risk factors for poor visual outcome have been addressed in the literature, and are poor visual function documented at presentation which is unlikely to improve,[4,36,37] the degree of obesity[38] and high-grade papilloedema.[39,40] That visual function is affected in paediatric idiopathic intracranial hypertension is an important consideration in the evaluation of these children. It is encouraging that such improvement is noted in visual function following appropriate and timely intervention in the treatment of idiopathic intracranial hypertension. However, as was well illustrated by one child in this study and in other studies,[19,26] optic atrophy can develop with significant consequences to the visual function of the child, their generally development and progress in everyday activities.

Idiopathic intracranial hypertension is an uncommon condition in adults but even more so in paediatric

patients. The diagnosis of paediatric idiopathic intracranial hypertension follows the same criteria as for adults but may be made more difficult by the problems encountered in obtaining accurate and reliable intracranial pressure measurements. Visual field assessment reveals visual impairment in many paediatric cases which can be substantial and is similar to that of the adult population. However, it is evident that for the majority of these children, prompt and appropriate treatment to reduce the raised intracranial pressure leads to partial if not complete resolution of the visual field loss.

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