Eye (2019) 33:1014–1019
https://doi.org/10.1038/s41433-019-0359-5

The ROYAL COLLEGE of OPHTHALMOLOGISTS

ARTICLE

Differing presenting features of idiopathic intracranial hypertension in the UK and US

R. J. Blanch[1,2,3,4] · C. Vasseneix[1] · A. Liczkowski[5] · A. Yiangou[5,6] · A. Aojula[5,6] · J. A. Micieli[1] · S. P. Mollan[4] · N. J. Newman[1,7,8] · V. Biousse[1,8] · B. B. Bruce[1,8] · A. Sinclair[5,6]

Received: 17 October 2018 / Revised: 27 November 2018 / Accepted: 5 December 2018 / Published online: 19 February 2019

Abstract

Aim Demographic factors potentially influencing the presentation and severity of idiopathic intracranial hypertension (IIH) in the US vs. UK populations include obesity and ethnicity. We aimed to compare the presenting features of IIH between populations in the UK and US tertiary referral centres, to assess what population differences exist and whether these cause different presentations and impact on visual function.

Methods Clinical data were collected on 243 consecutive UK IIH patients and 469 consecutive US IIH patients seen after 2012 in two tertiary centres. Visual function was defined as severe visual loss when Humphrey visual field mean deviation was <−15 dB, when Goldmann visual fields showed constriction or when visual acuity was <20/200.

Results US patients were more commonly of self-reported black race (58.9% vs. 7.1%) than UK patients, but had a similar mean body mass index (38.3 ± 0.63kg/m^2 UK vs. 37.7 ± 0.42kg/m^2 US; $p = 0.626$). The UK cohort had lower presenting Frisén grade (median 1 vs. 2; $p < 0.001$) and severe visual loss less frequently (15.4% vs. 5%; $p = 0.014$), but there was no difference in mean cerebrospinal fluid-opening pressure (CSF-OP) (35.8 ± 0.88cmH_2O UK vs. 36.3 ± 0.52cmH_2O US; $p = 0.582$). African Americans had poorer visual outcomes compared with US whites (19.4% vs. 10% severe visual loss; $p = 0.011$). Visual function was weakly associated with CSF-OP ($R^2 = 0.059$; $p = 0.001$), which was similar between UK and US patients.

Conclusions The UK and the US cohorts had a similar average presenting BMI. However, the worse presenting visual function in the US IIH cohort was partially attributable to differences in the black populations in the two countries.

Introduction

Idiopathic intracranial hypertension (IIH) is a rare disease where there is international acceptance on diagnosis [1], but until recently less consensus on management [2, 3]. Thus, management may vary amongst treatment centres and, in addition, the presenting phenotype may be location-specific.

Demographic factors that potentially influence the phenotype between IIH populations are body mass index (BMI) and ethnicity. IIH is known to have a marked association with those who are obese. In particular, truncal fat mass and higher BMI has been associated with more severe visual

These authors contributed equally: B.B. Bruce, A. Sinclair

✉ R. J. Blanch
blanchrj@bham.ac.uk

✉ A. Sinclair
A.B.Sinclair@bham.ac.uk

1 Department of Ophthalmology, Emory University, Atlanta, GA, USA

2 Neuroscience and Ophthalmology, Institute of Inflammation and Ageing, University of Birmingham, Birmingham, UK

3 Academic Department of Military Surgery and Trauma, Royal Centre for Defence Medicine, Birmingham, UK

4 Department of Ophthalmology, University Hospital Birmingham NHS Trust, Birmingham, UK

5 Metabolic Neurology, Institute of Metabolism and Systems Research, University of Birmingham, Birmingham, UK

6 Department of Neurology, University Hospital Birmingham NHS Trust, Birmingham, UK

7 Department of Neurological Surgery, Emory University, Atlanta, GA, USA

8 Department of Neurology, Emory University, Atlanta, GA, USA

Table 1 Population and IIH rates of obesity

Country	Mean population BMI (kg/m^2)	Year	Prevalence of IIH	Incidence of IIH	Mean BMI (kg/m^2) in IIH patients
Australia [24]	27.4	2017	7.9/100,000		
France [16]	25.6	1988			33
India [25]	21.8	2007			27.7
Israel [19, 26]	27.4	2001–2016		0.94	32.2
Italy [14]	26.3	2004		0.28	
Japan [27]	22.8	2000	1/1000,000	0.03	Only two cases
Libya [14]	27.9	1993		2.2	
Portugal [19]	26.2	2016			34.8
Spain [28]	26.6	2015	1.2/100,000		73.77% obese
Sweden [29]	26.4	2017	1/100,000	0.65	34.4
Switzerland [19]	25.7	2016			36.4
Turkey [19]	27.9	2016			31.2
UK [19, 30]	27.5	1991–2011	10.9/100,000	0.51–1.57	39.7
US [14, 16, 31–33]	29.1	1998–2011	8.9/100,000	0.9	31.8–34

Population data were from the World Health Organisation [23]

IIH idiopathic intracranial hypertension, *BMI* body mass index

loss [4, 5]. Obesity affects 30.4% of British women and 38.2% of American women [6] (Table 1). Previous work has identified that those of African-American descent with IIH are more likely than white US IIH patients to have severe visual loss [7], and 2.81% of the British population identified as black on the latest census data, compared with 13.4% in the US [8, 9].

IIH incidence is rising in England and worldwide [10, 11], presumed to be related to the increasing global prevalence of obesity (Table 1) [11, 12]. The rise in cerebrospinal fluid (CSF) shunting procedures in the USA between 1998 and 2002 paralleled the rise in obesity rates over that same period [13]. The international prevalence of IIH associates with the prevalence of obesity [11, 14]. However, the proportion of obesity in IIH cohorts may vary independently of the overall population prevalence of obesity (Table 1). Given the differences between the UK and US populations, we aimed to compare two large neuro-ophthalmology IIH clinic cohorts from prospectively held databases in the two countries to assess for differences in the presenting phenotype.

Methods

The study was approved by the University Institutional Review Board at Emory and the local NHS National Research Ethics Committee (14/LO/1208) and conformed to the tenets of the Declaration of Helsinki.

We included consecutive patients over the age of 16 years with a diagnosis of IIH, seen in one US and one UK tertiary referral centres. Only patients with a diagnosis of IIH according to the modified Dandy criteria were included [1]: specifically papilledema, normal neurologic examination except cranial nerve palsies, normal neuroimaging, normal CSF constituents and elevated lumbar puncture opening pressure (OP) (>25 cm CSF). The US cohort was a retrospectively collected cohort of consecutive patients evaluated in a standardised fashion by VB, NN and BB. The UHB (University Hospitals Birmingham NHS Foundation Trust) cohort was prospectively collected in consecutive patients with a diagnosis of IIH who consented to recruitment in the IIH: Life database.

All patients were evaluated in a standardised manner by experienced neuro-ophthalmologists including complete neuro-ophthalmic history and examination with formal visual fields (VFs), fundus photography, neuroimaging, height and weight. UK data were collected and entered prospectively into the IIH: Life database. US data were entered retrospectively from the electronic patient record and written notes.

Data collected included age, race, gender, BMI, recent weight gain, presenting symptoms (headache, tinnitus, diplopia, transient visual obscurations), visual acuity (VA), VFs and CSF-OP.

US patients' fundus images taken at or close to the time of initial presentation were Frisén graded by three different neuro-ophthalmologists, all masked to clinical details. For the UK dataset, two different neuro-ophthalmologists performed Frisén grading on slit lamp examination at presentation [15]. Disagreements were settled by referral to two additional observers. VFs were graded as severe visual loss when the

Humphrey VF mean deviation (MD) was <−15 dB or when Goldmann VFs showed severe constriction.

Patients who reported use of medications that have been associated with intracranial hypertension were excluded (fluoroquinolone and tetracycline antibiotics, cyclosporin, vitamin A preparations, recent steroid discontinuation). Alternative causes for intracranial hypertension were excluded at the time of diagnosis by full blood count checking for anaemia and review of imaging, including venography.

Statistical analysis was performed in SPSS 21 (IBM Corp., Armonk, NY, USA) and used *t* test for continuous numeric data and χ^2 for categorical data except for VFs and VA. Because VFs (MD) and VA had two measurements per patient, they were analysed using generalised estimating equations. To allow model fit, groups with few patients (e.g. transgender, South Asian race) were collapsed and combined. In particular, missing race data were combined with white race, because most patients with missing race data were from the UK cohort. To assess systemically the effect of these missing data, the data were also replaced with multiple imputation and pooled analyses are reported. To minimise the risk of type 1 error, we analysed Frisén grading and severe VF loss analysis, which are non-numeric data, for the worse eye only using χ^2 tests. Means are reported as mean ± standard error of the mean unless otherwise specified.

Results

Presenting demographics

Consecutive cohorts of 243 UK patients and 469 US patients presenting for evaluation of IIH after 2012 in two tertiary centres were included. One patient in the UK cohort was not included because she did not consent to inclusion in IIH: Life.

US patients were more commonly of self-reported black race (58.9% vs. 7.1%; Table 2) and UK patients were more commonly of South Asian descent (8.8% vs. 1.0%), reflecting the ethnicity of the local populations surrounding the treatment centres.

There was no evidence that the UK and US patients differed in BMI (38.3 ± 0.63 kg/m^2 UK vs. 37.7 ± 0.42 kg/m^2 US; $p = 0.626$; 95% confidence interval (CI) for the difference −0.8 to 2.1) or in the proportion of obese patients (84.4% UK vs. 79.7% US; $p = 0.147$).

The gender proportions were similar between UK and US patients (6% males US vs. 4.1% UK; $p = 0.284$).

Visual function

Compared with US patients (Table 2), the UK cohort had better presenting VA (logMAR 0.09 ± 0.02 vs. 0.15 ± 0.02; $p < 0.001$) and MD (−4.74 ± 0.40 vs. −6.52 ± 0.35dB; $p < 0.001$). Frisén grade was also lower in UK patients (median 1 vs. 2; $p < 0.001$). Because of the potential for systematic differences in how VA and VFs are assessed, we also looked at the proportion with severe visual loss, defined as diffusely constricted Goldmann VFs or an MD <−15 dB, as previously described [16]. The US patients were more likely to have severe VF loss at presentation (15.4% vs. 5%; $p = 0.014$).

There was no evidence of a difference in mean CSF-OP between UK and US patients (35.8 ± 0.73 cmH_2O UK vs. 36.3 ± 0.46 cmH_2O US; $p = 0.582$).

Table 2 Summary of presenting features

Parameter	UK	US	*P* value
Proportion of black race (%)	7.10	58.90	N/A
Proportion with severe visual loss (%)	5	15.4	0.014
HVF mean deviation (dB)	−4.74 ± 0.40	−6.52 ± 0.35	<0.001
CSF opening pressure (cmH_2O)	35.8 ± 0.73	36.3 ± 0.46	0.582
Visual acuity (logMAR)	0.085 ± 0.02	0.152 ± 0.01	<0.001
BMI (kg/m^2)	38.3 ± 0.59	37.7 ± 0.41	0.626
Proportion female (%)	95.9 ± 1.8	94.0 ± 1.27	0.284
Frisen grade	1 (IQR 1–2)	2 (IQR 1–3)	<0.001
Age (years)	31.7 ± 0.51	32.8 ± 0.58	0.17

HVF Humphrey visual field, *CSF* cerebrospinal fluid, *BMI* body mass index, *IQR* interquartile range, *N/A* not applicable

History

Among symptomatic US patients, the mean reported duration of symptoms was 10.0 ± 0.64 weeks; equivalent data on symptom duration were not available in the UK cohort. The prevalence of headache as a presenting symptom was higher in UK patients than in US patients (Table 3; 85% vs. 65%; $p < 0.001$). Incidental finding of papilledema on routine examination was also more common in UK patients than in US patients (Table 3; 48% vs. 30%; $p < 0.001$). About half of the patients reported recent weight gain (54% UK vs. 46% US; $p = 0.236$).

Variation in visual function at presentation

When the US and UK datasets were analysed together, Frisén grade and CSF-OP were weakly associated ($R^2 = 0.109$, $p < 0.001$). CSF-OP was available on 539/712 patients (76%) and initial Frisén grade on 288/712 patients (40%). When Frisén grade, CSF-OP, race, country, BMI and duration of symptoms were analysed together, CSF-OP and the interaction between race and country were independently associated with VF at presentation (Table 4), assessed as MD ($R^2 = 0.042$, $p < 0.001$).

The binary measure of VF "severe visual loss in either eye" associated with race ($p = 0.02$) and CSF-OP ($p < 0.001$), but a model could not be fitted for the interaction term ($p < 0.001$; generalised estimation equation binomial logit).

To assess the effect of missing data, multiple imputation of the missing values with pooled analysis of the 10 imputed datasets yielded results consistent with the primary analysis: every 1 cmH_2O increase in CSF-OP was associated with a 0.168 dB reduction in MD ($p < 0.001$) and VF was worse in African-American than white US patients by an average of 1.60 dB ($p = 0.018$), whereas UK African Caribbean visual function was, on average, 3.15 dB better than in US white patients ($p = 0.039$).

Race

Within the US cohort, African-American patients had a higher proportion of severe visual loss at presentation (19.4% vs. 10%; $p = 0.011$) and a worse MD on VF testing (-7.38 ± 0.52 vs. -5.58 ± 0.49dB; $p = 0.003$). There was weak evidence of a difference in CSF-OP, which was higher in African-American patients ($37.69 \pm 0.720 cmH_2O$ vs. $34.95 \pm 0.794 cmH_2O$; $p = 0.055$), though minimal evidence of a difference in Frisén grade (median 3 African American vs. 2 white; $p = 0.205$). There was no difference in presenting VA (logMAR 0.14 ± 0.03 white vs. logMAR 0.17 ± 0.03 African American; $p = 0.857$). On average, white patients had a longer duration of symptoms before presentation (11.5 ± 1.13 weeks vs. 9.02 ± 0.75 weeks; $p = 0.042$), but no difference in the proportion of patients with incidentally discovered papilloedema (24.1% African American vs. 26.3% white; $p = 0.624$).

There were eight African Caribbean patients in the UK dataset, who had lower CSF-OPs (33.4 ± 1.81 vs. $39.6 \pm 2.11 cmH_2O$; $p = 0.037$) and better MDs on Humphrey VF testing (-2.02 dB ± 0.63 vs. -6.02 dB ± 0.85; $p = 0.001$).

Table 3 Presenting symptoms in UK and US IIH patients

Symptoms	UK proportion (%)	US proportion (%)	*P* value (χ^2)
Incidental papilledema	48.1	30.0	<0.001
Headache	85.4	65.0	<0.001
Diplopia	14.6	11.0	0.207
Nausea or vomiting	17.3	7.90	<0.001
Neck or back pain	4.86	2.86	0.208
Pulsatile tinnitus	43.2	17.2	<0.001
Transient visual obscurations	28.1	21.1	0.058
Other visual symptoms	40.0	35.0	0.236

IIH idiopathic intracranial hypertension

Discussion

This collaborative study compared two large neuro-ophthalmology IIH clinic cohorts from prospectively held databases in the UK and the USA and assessed for differences in the presenting phenotype between the two centres. US patients with IIH presented with significantly worse visual function, being more likely to have severe visual loss at presentation. African-American patients in the US cohort had worse visual function than white patients, who had similar baseline features in both the US and UK cohorts.

The more severe disease in African-American patients has been previously reported [7], and does not seem to be explained by different access to care in our cohort, because duration of symptoms and incidentally discovered papilledema was not different between white and African-American ethnicities. Although duration of symptoms was not available in the UK cohort, there was a higher rate of incidental papilledema compared with the US cohort. The higher prevalence of incidental papilloedema in the UK cohort is in contrast to the higher reported rate of headache in the UK and could be explained by access to care, as greater access to eye examinations may be expected to associate with greater incidental detection of papilloedema.

Visual function was similar in white US and white UK patients (0.76 dB worse in the USA, $p = 0.192$), in contrast to a previous comparison between white US and white French patients with IIH [16], which found that white US patients had a longer pre-diagnosis duration of symptoms and were more likely to have VF constriction and poor VA at presentation.

Table 4 Model output for the comparison of race, nationality and CSF opening pressure

Modelled comparison	Effect	Effect size (95% CI)	*P* value
US white vs. US black	Worse visual function in African-American than white US patients	1.55dB (0.27–2.83)	0.018
US white vs. UK white	Non-significant difference with worse visual function in US white	0.76dB (−0.38–1.89)	0.192
CSF opening pressure	Higher CSF pressure associated with worse visual function	0.123dB/cmH_2O (0.05–0.20)	0.001
Race ×country interaction	UK African Caribbean visual function is better than US white	3.05dB (0.82–5.29)	0.007

CSF cerebrospinal fluid, *CI* confidence interval

Table 1 shows a weak relationship between population obesity in the general population and IIH patients. A recent English paper reports not only an increase in the incidence of IIH between 2002 and 2016, but the association with obesity over this time [10]. In Iowa in 1988, the mean weight in an IIH population was 38% above ideal weight for height (BMI 34.5) and 67% were obese [17]. At that time, 17.5% of the US population was obese. Comparison with the recent IIH cohorts suggests that the average weight of IIH patients has increased over time in concert with the increased prevalence of obesity in the population. In the Idiopathic Intracranial Hypertension Treatment Trial (IIHTT), the mean initial BMI was much higher, at 39.9, and in this trial recruitment was restricted to mild VF defects with MDs <7dB, although the study did not report the characteristics of patients declining to participate or failing screening [18].

The USA has higher prevalences of both overweight and obesity than the UK (UK 68.6% male and 58.9% female overweight, 26.9% male and 28.6% female obese; US 72.7% male and 63.2% female overweight, 35.5% male and 37% female obese). The similar weights and proportions of obesity between US and UK IIH patients probably reflects the fact that only obese patients suffer from IIH and we do not have data on the average BMI of obese patients in the UK and USA. The equivalent average BMI in our US and UK IIH cohorts excludes the degree of obesity as an explanatory factor in the more severe presentation of US patients.

Similar to previous studies, most IIH patients were female [19, 20]. In contrast to weight and gender, the racial mix of patients reflects the population local to the treatment centres, suggesting that whilst being African American confers a worse prognosis, it does not affect the risk of disease.

The relationship between Frisén grade and CSF-OP has been previously reported in the IIHTT [21], although there was no relationship between CSF-OP and baseline visual function in the IIHTT, which may be related to the exclusion of patients with severe visual loss from that population. The association between high CSF-OP and visual loss has not been previously reported, except that cases series of patients with fulminant disease have reported high CSF-OP [22]. CSF-OP may affect visual function secondary to the association between Frisén grade and visual function, but does not appear to explain the observed UK–US differences and, with $R^2 < 0.1$, has a modest effect.

Conclusions

Visual loss at presentation was more severe in the US cohort, despite similar BMIs and similar LP pressures. The population differences in presenting visual function may relate to the higher proportion of patients of black race in the US population.

Summary

What was known before

- Demographic factors influencing the presentation and severity of IIH include obesity and ethnicity.

What this study adds

- Visual function at presentation was worse in US patients than in UK patients with IIH. UK and US patients with IIH have similar BMI. Worse visual function in the US cohort may be explained by the higher proportion of African-American patients compared to the UK cohort.

Funding AS is funded by an NIHR Clinician Scientist Fellowship (NIHR-CS-011-028) and by the Medical Research Council, UK (MR/K015184/1). IIH:Life database is funded by the Healthcare Quality Improvement Partnership (HQIP) and IIHUK, (registered patient charity in England and Wales: 1143522). The Emory cohort study was supported in part by an unrestricted departmental grant (Department of Ophthalmology) from Research to Prevent Blindness, Inc., New York, and by NIH/NEI core grant P30-EY006360 (Department of Ophthalmology). VB received research support from NIH/PHS (UL1-RR025008). NJN is a recipient of the Research to Prevent Blindness Lew R. Wasserman Merit Award. CV is the recipient of the Philippe Foundation, Inc. grant.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

References

1. Friedman DI, Liu GT, Digre KB. Revised diagnostic criteria for the pseudotumor cerebri syndrome in adults and children. Neurology. 2013;81:1159–65.
2. Markey KA, Mollan SP, Jensen RH, Sinclair AJ. Understanding idiopathic intracranial hypertension: mechanisms, management, and future directions. Lancet Neurol. 2016;15:78–91.
3. Mollan SP, Davies B, Silver NC, Shaw S, Mallucci CL, Wakerley BR, et al. Idiopathic intracranial hypertension: consensus guidelines on management. J Neurol Neurosurg Psychiatry. 2018;89:1088–1100.
4. Hornby C, Botfield H, O'Reilly MW, Westgate C, Mitchell J, Mollan SP, et al. Evaluating the fat distribution in idiopathic intracranial hypertension using dual-energy X-ray absorptiometry scanning. Neuroophthalmology. 2018;42:99–104.
5. Szewka AJ, Bruce BB, Newman NJ, Biousse V. Idiopathic intracranial hypertension: relation between obesity and visual outcomes. J Neuroophthalmol. 2013;33:4–8.

6. Ng M, Fleming T, Robinson M, Thomson B, Graetz N, Margono C, et al. Global, regional, and national prevalence of overweight and obesity in children and adults during 1980–2013: a systematic analysis for the Global Burden of Disease Study 2013. Lancet. 2014;384:766–81.
7. Bruce BB, Preechawat P, Newman NJ, Lynn MJ, Biousse V. Racial differences in idiopathic intracranial hypertension. Neurology. 2008;70:861–7.
8. Statistics OfN. Census: Key statistics for Birmingham and it's constituent areas. London: Office for National Statistics; 2011. p. 2012.
9. Bureau USC Population estimates. 2017. https://www.census.gov/quickfacts/fact/table/US/PST045217. Accessed 17 Jul 2018.
10. Mollan SP, Aguiar M, Evison F, Frew E, Sinclair AJ. The expanding burden of idiopathic intracranial hypertension. Eye. 2018. https://doi.org/10.1038/s41433-018-0238-5. [Epub ahead of print].
11. McCluskey G, Doherty-Allan R, McCarron P, Loftus AM, McCarron LV, Mulholland D, et al. Meta-analysis and systematic review of population-based epidemiological studies in idiopathic intracranial hypertension. Eur J Neurol. 2018;25:1218–27.
12. Mollan SP, Ali F, Hassan-Smith G, Botfield H, Friedman DI, Sinclair AJ. Evolving evidence in adult idiopathic intracranial hypertension: pathophysiology and management. J Neurol Neurosurg Psychiatry. 2016;87:982–92.
13. Curry WT Jr, Butler WE, Barker FG 2nd. Rapidly rising incidence of cerebrospinal fluid shunting procedures for idiopathic intracranial hypertension in the United States, 1988–2002. Neurosurgery. 2005;57:97–108. discussion 197–108.
14. Chen J, Wall M. Epidemiology and risk factors for idiopathic intracranial hypertension. Int Ophthalmol Clin. 2014;54:1–11.
15. Frisen L. Swelling of the optic nerve head: a staging scheme. J Neurol Neurosurg Psychiatry. 1982;45:13–18.
16. Mrejen S, Vignal C, Bruce BB, Gineys R, Audren F, Preechawat P, et al. Idiopathic intracranial hypertension: a comparison between French and North-American white patients. Rev Neurol (Paris). 2009;165:542–8.
17. Durcan FJ, Corbett JJ, Wall M. The incidence of pseudotumor cerebri. Population studies in Iowa and Louisiana. Arch Neurol. 1988;45:875–7.
18. Wall M, Kupersmith MJ, Kieburtz KD, Corbett JJ, Feldon SE, Friedman DI, et al. The idiopathic intracranial hypertension treatment trial: clinical profile at baseline. JAMA Neurol. 2014;71:693–701.
19. Rosenblatt A, Klein A, Roemer S, Borruat FX, Meira D, Silva M, et al. Idiopathic intracranial hypertension—a comparison of clinical characteristics between 4 medical centers in different geographic regions of the world. J Neuroophthalmol. 2016;36:280–4.
20. Bruce BB, Kedar S, Van Stavern GP, Monaghan D, Acierno MD, Braswell RA, et al. Idiopathic intracranial hypertension in men. Neurology. 2009;72:304–9.
21. Kattah JC, Pula JH, Mejico LJ, McDermott MP, Kupersmith MJ, Wall M. CSF pressure, papilledema grade, and response to acetazolamide in the Idiopathic Intracranial Hypertension Treatment Trial. J Neurol. 2015;262:2271–4.
22. Thambisetty M, Lavin PJ, Newman NJ, Biousse V. Fulminant idiopathic intracranial hypertension. Neurology. 2007;68:229–32.
23. World Health Organisation. Mean body mass index trends among adults, crude (kg/m^2): estimates by country. Geneva: World Health Organisation. 2017. http://apps.who.int/gho/data/node.main.BMIMEANADULTC?lang=en. Accessed 2 Jun 2018.
24. Lichtenberg I, Blackwood E, Gordon J, Hammond S, Hawke S. The prevalence of idiopathic intracranial hypertension and associated co-morbidities in central western New South Wales (NSW). J Neurol Neurosurg Psychiatry. 2017;88:e1–e1.
25. Gafoor VA, Smita B, Jose J. Long-term response of cerebrospinal fluid pressure in patients with idiopathic intracranial hypertension—a prospective observational study. Ann Indian Acad Neurol. 2017;20:220–4.
26. Kesler A, Gadoth N. Epidemiology of idiopathic intracranial hypertension in Israel. J Neuroophthalmol. 2001;21:12–14.
27. Yabe I, Moriwaka F, Notoya A, Ohtaki M, Tashiro K. Incidence of idiopathic intracranial hypertension in Hokkaido, the northernmost island of Japan. J Neurol. 2000;247:474–5.
28. Contreras-Martin Y, Bueno-Perdomo JH. Idiopathic intracranial hypertension: descriptive analysis in our setting. Neurologia. 2015;30:106–10.
29. Sundholm A, Burkill S, Sveinsson O, Piehl F, Bahmanyar S, Nilsson Remahl AIM. Population-based incidence and clinical characteristics of idiopathic intracranial hypertension. Acta Neurol Scand. 2017;136:427–33.
30. Raoof N, Sharrack B, Pepper IM, Hickman SJ. The incidence and prevalence of idiopathic intracranial hypertension in Sheffield, UK. Eur J Neurol. 2011;18:1266–8.
31. Friesner D, Rosenman R, Lobb BM, Tanne E. Idiopathic intracranial hypertension in the USA: the role of obesity in establishing prevalence and healthcare costs. Obes Rev. 2011;12:e372–380.
32. Daniels AB, Liu GT, Volpe NJ, Galetta SL, Moster ML, Newman NJ, et al. Profiles of obesity, weight gain, and quality of life in idiopathic intracranial hypertension (pseudotumor cerebri). Am J Ophthalmol. 2007;143:635–41. e631.
33. Kilgore KP, Lee MS, Leavitt JA, Mokri B, Hodge DO, Frank RD, et al. Re-evaluating the incidence of idiopathic intracranial hypertension in an era of increasing obesity. Ophthalmology. 2017;124:697–700.