ORIGINAL ARTICLE



Evaluation of the signs and symptoms of pseudotumor cerebri syndrome in *pediatric* population

G. Sager¹ · A. T. Kaplan² · S. Ö. Yalçin² · E. Çalişkan³ · Y. Akın⁴

Received: 20 March 2021 / Accepted: 29 June 2021 / Published online: 14 July 2021 © The Author(s), under exclusive licence to Springer-Verlag GmbH Germany, part of Springer Nature 2021

Abstract

Purpose Pseudotumor cerebri syndrome (PTC) is characterized by increased intracranial pressure without a space-occupying lesion and a normal cerebrospinal fluid (CSF) composition without evidence of CSF infection. In this study, we aimed to compare the symptoms, signs, and clinical characteristics of patients presenting with a preliminary diagnosis of pseudotumor cerebri syndrome (PTC) who were diagnosed and not diagnosed with PTC.

Method We conducted a retrospective study of patients who were referred to our clinic with signs and symptoms of PTC. We compared the patients' symptoms, signs, and clinical characteristics who were diagnosed with PTC with those who were not diagnosed with PTC using modified Dandy criteria.

Results Ninety-four patients with the pre-diagnosis of PTC were included in the study. LP procedure was done in all patients. After LP, 75.3% of the patients were diagnosed with PTC, but 24.7% did not meet the criteria for PTC. A statistically significant relationship was found between the increase in headache complaints when leaning forward, headache that keeps the child from playing, and the CSF pressure level (p = 0.014, p = 0.019; p < 0.05). There was no statistically significant correlation between papilledema and CSF pressure level (p > 0.05). A statistically significant relationship was found between papilledema grade and CSF pressure level (p = 0.038; p < 0.05), and the rate of high CSF pressure in the groups with Grades 2–3 and Grade 4 papilledema was higher than that in the group with Grade 1 papilledema. Cranial nerve 6 palsy (CN6) (p = 0.048) and flattening of the posterior aspect of the globe (FPS) are found independent risk factors ($p = 0.004 \ p < 0.05$). **Conclusions** PTC signs and symptoms show variability among pediatric population.

Keywords Pseudotumor cerebri syndrome · Papilledema · Intracranial hypertension · Lumbar puncture

Introduction

Pseudotumor cerebri syndrome (PTC) is characterized by increased intracranial pressure without a space-occupying lesion and a normal cerebrospinal fluid (CSF) composition without evidence of CSF infection [1]. Recent studies

G. Sager sgunessenturk@gmail.com

- ¹ Department of Pediatric Neurology, Kartal Dr. Lutfi Kirdar City Hospital, Semsi Denizer Avenue, Cevizli, 34890, Kartal, Istanbul, Turkey
- ² Department of Ophthalmology, Kartal Dr. Lutfi Kirdar City Hospital, Istanbul, Turkey
- ³ Department of Radiology, Kartal Dr. Lutfi Kirdar City Hospital, Istanbul, Turkey
- ⁴ Department of Pediatrics, Kartal Dr. Lutfi Kirdar City Hospital, Istanbul, Turkey

estimate the PTC incidence as 0.9/100.000 in the general population [2]. A complication of PTC is irreversible optic nerve injury [3]. Modified Dandy criteria by Friedman [4] are used for the diagnosis of PTC. Headache, nausea and vomiting, double vision, and blurred vision are the symptoms. Furthermore, papilledema and cranial sixth nerve (CN6) palsy are the signs of PTC [5]. However, not all the patients have all of the symptoms and signs listed above. Spinal fluid examination and measuring the opening pressure of CSF with a lumbar puncture (LP) are critical for the diagnosis of PTC.

Materials and methods

In this retrospective study, we aimed to compare the symptoms, signs, and clinical characteristics of patients presenting with a preliminary diagnosis of pseudotumor cerebri syndrome (PTC) who were diagnosed and not diagnosed with PTC.

Approval was obtained from the local ethics committee of Lutfi Kirdar City Hospital with the number 314–194-1 before the analysis of data. The study was conducted in accordance with the principles of the Declaration of Helsinki. The informed parental consent was not obtained because this is a retrospective study and we had only used patients' electronic data without ID information.

The modified Dandy criteria revised by Friedman [4] in 2014 were used to define the probable and definite diagnosis of pseudotumor cerebri syndrome(PTC).

All the patients between the age of 6 and 18 years who undergone lumbar puncture (LP) procedure with the suspicion of PTC were included in the study. Patients with the secondary etiologies of increased intracranial pressure (meningitis, sinus venous thrombosis, demyelinating disease, or space-occupying lesions of the central nervous system (CNS)) were not included. Also, patients with missing information necessary for classification were excluded.

Ophthalmological examination: fundoscopic examination was performed by a neuroophthalmologist and papilledema was graded according to the Frisen Grading Scale. For the diagnosis of drusen syndrome, all the patients were evaluated with optical coherence tomography (OTC) and USG. Patients with a definite diagnosis of the drusen syndrome were not included in the study.

All the patients were evaluated by cranial magnetic resonance imaging (MRI) and the radiological findings were defined as follows: empty sella, flattening of the posterior aspect of the globe (FPS), and distention of the perioptic subarachnoid space with or without a tortuous optic nerve. Partial empty sella was defined as the presence of CSF in > 50% of the pituitary fossa based on Mallery et al. [6]. MRI was performed using a 1.5-Tesla scanner (Philips Ingenia, Philips Healthcare, the Netherlands). Brain MRI was acquired using axial T2 fat suppression, axial fluid attenuation inversion recovery (T2 fluid-attenuated inversion recovery), axial and sagittal T1, and coronal T2 without contrast at a plane thickness of 5 mm. MR venography was performed by using a 2D time-of-flight method with an inferior saturation band to eliminate signal intensity from arterial structures. Maximum intensity projections were constructed from the source images and were reviewed along with the source data.

The symptoms and signs were questioned by considering the modified Dandy criteria revised by Friedman et al. [4] in 2014. Demographical findings, major symptoms, clinical features, and opening CSF pressure were recorded for each patient. Opening pressure was recorded in mm CSF.

Patients who were not diagnosed with definite or probable PTC were followed up for PTC complications for 1 year.

NCSS (Number Cruncher Statistical System) 2007 (Kaysville, UT, USA) software was used for the statistical analyses. Descriptive statistical methods (mean, standard deviation, median, frequency, percentage, minimum, and maximum) were used for evaluating the study data. The normality of the distribution of quantitative data was tested with Shapiro–Wilk test and graphical analyses. Student's *t* test was used for comparing the normally distributed quantitative variables between the two groups. Pearson chi-square test, Fisher's exact test, and Fisher–Freeman–Halton test were used to compare the qualitative data. Logistic regression analysis was used to examine the risk factors affecting the CSF pressure level. Statistical significance was assessed at a minimum level of p < 0.05.

Results

This study was carried out with 82 patients who presented to the pediatric neurology clinic of a tertiary hospital between 2018 and 2020 with a preliminary diagnosis of PTC. Their ages ranged from 5 to 18 years, with a mean age of 11.43 ± 3.35 years.

There was no statistically significant correlation between presence of headache and timing of headache and PTC (p > 0.05).

No statistically significant correlation was found between the headache complaint being severe enough to wake the patient up during sleep and the PTC (p > 0.05).

A statistically significant relationship was found between the increase in headache complaints when leaning forward and the PTC (p = 0.014; p < 0.05), and in the group whose complaints increased when leaning forward, the rate of high CSF pressure was higher than in the group whose complaints did not increase when leaning forward. Increased complaints when leaning forward increased the risk of PTC by 4765 times (OR: 4765; %95CI: 1270–17,882).

There was no statistically significant relationship between the duration of the headache complaint lasting > 2 h and the diagnosis of PTC (p > 0.05). Although it was not significant, the duration of the complaint lasting > 2 h increased the risk of PTC by 1.813 times (OR: 1813; %95CI: 642–5113).

A statistically significant relationship was found between the headache complaint being severe enough to keep the patient from playing and the PTC diagnosis (p = 0.019; p < 0.05), and the rate of high CSF pressure level in the group that experienced complaints sufficient to keep them from playing was higher than in the group whose complaints did not keep them from playing. The complaint being severe enough to keep the patient from playing increased the risk of PTC by 2951 times (OR: 2951; %95CI: 886–9826).

There was no statistically significant relationship between vomiting, diplopia, and toxic appearance and PTC (p > 0.05).

Table 1 Evaluation of the patients with preliminary diagnosis of PTC according to descriptive characteristics

| | | The patients with preliminary diagnosis of PTC | | р |
|--|------------------|--|---------------------|---------------------|
| | | Normal $(n=21)$ | PTC (<i>n</i> =61) | |
| Age (years) | Min–max (median) | 6–17 (11) | 6-18 (12) | ^a 0.345 |
| | $Mean \pm SD$ | 11.43 ± 3.35 | 11.77 ± 3.31 | |
| CSF pressure | Min–max (median) | 14.0-20.0 (17.0) | 21-114 (32.0) | |
| | $Mean \pm SD$ | 17.04 ± 1.65 | 36.7 ± 16.05 | |
| Headache | No | 8 (34.8) | 15 (65.2) | ^c 0.235 |
| | Yes | 13 (22) | 46 (78) | |
| | 1 week | 6 (42.9) | 8 (57.1) | ^d 0.220 |
| Headache duration($n = 66$) | 1 month | 4 (14.8) | 23 (85.2) | |
| | 1 year | 2 (16.7) | 10 (83.3) | |
| | >1 year | 1 (16.7) | 5 (83.3) | |
| Headache: wakes the patient up | No | 18 (29) | 44 (71) | ^c 0.221 |
| | Yes | 3 (15) | 17 (85) | |
| Headache: increases when leaning forward | No | 18 (34.6) | 34 (65.4) | ^c 0.014* |
| | Yes | 3 (10) | 27 (90) | |
| Headache: lasts more than 2 h | No | 14 (30.4) | 32 (69.6) | ^c 0.258 |
| | Yes | 7 (19.4) | 29 (80.6) | |
| Headache: keeps the patient from playing | No | 18 (34.0) | 35 (66.0) | ^c 0.019* |
| | Yes | 3 (10.3) | 26 (89.7) | |
| Vomiting | No | 17 (26.6) | 47 (73.4) | ^b 1.000 |
| | Yes | 4 (22.2) | 14 (77.8) | |
| Double vision | No | 20 (27.4) | 53 (72.6) | ^b 0.435 |
| | Yes | 1 (11.1) | 8 (88.9) | |
| Toxic appearance | No | 20 (27.4) | 53 (72.6) | ^b 0.435 |
| | Yes | 1 (11.1) | 8 (88.9) | |

**p* < 0.05

^aStudent's *t* test

^bFisher's exact test

^cPearson chi-square test

^dFisher-Freeman-Halton test^dFisher-Freeman-Halton test

There was no statistically significant correlation between papilledema and PTC (p > 0.05).

A statistically significant relationship was found between papilledema grade and CSF pressure level (p = 0.038; p < 0.05), and the rate of high CSF pressure in the groups with Grades 2–3 and Grade 4 papilledema was higher than that in the group with Grade 1 papilledema. The risk of high CSF pressure increased by 4821 times in patients with Grade 2+3+4 papilledema when compared with patients having Grade 1 papilledema (OR: 4821; %95CI: 983–23,638).

When the reasons for PTC signs on MRI were examined, they included partial empty sella, optic nerve sheath expansion, and optic nerve tortuosity, which did not differ significantly according to CSF pressure levels (p > 0.05). However, the CSF pressure was found to be high in all patients with FPS, which was significant (p = 0.004; p < 0.01). A statistically significant relationship was found between CN 6 paralysis and CSF pressure level (p = 0.048; p < 0.05), and the rate of high CSF pressure in the group with CN 6 paralysis was higher than that in the group without CN 6 paralysis.

The symptom characteristics and clinical features of the patients are shown in Table 1 and Table 2 respectively.

Discussion

Friedman revised the Dandy criteria in 2014 and published an article that guided in the diagnosis of PTC. PTC markers include headache, transient visual obstructions, pulsatile tinnitus, visual loss, and diplopia as well as papilledema [4]. As reported in a study, all of these symptoms may not simultaneously occur in every patient diagnosed with PTC. Table 2Evaluation of thepatients with preliminarydiagnosis of PTC according toclinical features

| | | The patients with preliminary diagno- sis of PTC | | р |
|---|---------------|---|---------------------|---------------------|
| | | Normal $(n=21)$ | PTC (<i>n</i> =61) | |
| Papilledema | No | 4 (21.1) | 15 (78.9) | ^c 0.205 |
| | Yes | 17 (27) | 46 (73) | |
| Papilledema grade (n=63) | Grade 1 | 15 (34.9) | 28 (65.1) | ^d 0.038* |
| | Grade $2+3+4$ | 2 (125) | 18 (90.0) | |
| CN 6 paralysis | No | 21 (28.8) | 51 (70.8) | ^c 0.048* |
| | Yes | 0 (0) | 10 (100) | |
| PTC signs on MRI | | | | |
| 1. Partial epty sella | | 4 (20) | 16 (80) | ^b 0.433 |
| 2. Optic nerve sheath expansio | n | 7 (29.2) | 17 (70.8) | ^b 0.642 |
| 3. Optic nerve tortuosity | | 2 (25) | 6 (75) | ^b 1.000 |
| 1+2+3 (all three) | | 1 (20) | 4 (80) | ^c 1.000 |
| Flattening of the posterior aspect of the globe (FPS) | | 0 (0) | 14 (100) | ^b 0.004* |

p < 0.05; **p < 0.01

^bFisher's exact test

^cPearson chi-square test

^dFisher–Freeman–Halton test

In fact, as reported in a publication by Favoni et al. [8], cases diagnosed with PTC without findings of papilledema are present in the literature [7, 9].

Pulsatile tinnitus and intermittent vision loss, which were mentioned by Friedman, were not included in the markers for our patients because it is difficult for children to properly describe these symptoms. In a study conducted by Wall et al. [10] in 2015, it was revealed that headache was the most common symptom among the 165 cases diagnosed with PTC. Headache in PTC may be daily or constant but may also be entirely episodic, and the pain may be diffuse or focal. Headache in pediatric PTC is more likely to involve the neck and shoulders, which is perhaps related to the sensitivity toward distention of the spinal root dural sheaths with increased pressure. Importantly, the "classic" high-pressure headache triad of (i) daily headache; (ii) worsening with Valsalva; and (iii) diffuse non-pulsating pain was found to be present in only 36.6% of the children with PTCS [10, 11]. In our patients, headache was present in 75% of the cases with PTC and in 25% of the cases without PTC. In the present study, the characteristics of the headache in pediatric PTC patients were discussed in detail. Headache that lasted for > 2 h, kept the child from playing, and was aggravated by leaning forward was found to be significantly increased in the group with PTC when compared with the other group in this study. We believe that CSF pressure should be checked with LP in every child with such headaches even if papilledema is not detected.

Bassan et al. [12] compared the symptoms of 51 patients diagnosed with PTC in their study. It was found that 27% of the patients had no symptoms, while their CSF pressure was

found to be > 200 mm CSF [12]. Papilledema was detected in all of these patients, but this publication did not provide information about papilledema grade. In a study conducted by Distelmaier et al. [14] in 2006, the need to perform LP in every patient with papilledema even if they do not have complaints was emphasized. But this study also did not provide information about papilledema grade [13–15]. In our study, the detection of Grade 1 papilledema was not found to be statistically significant. However, the presence of Grade ≥ 2 papilledema and CN6 paralysis was found to be an independent risk factor for detecting PTC.

Pseudo papilledema is an entity characterized by varying degrees of erasure and swelling in the optic disc margins in childhood and is often confused with true papilledema [16]. Congenital disc anomalies, hyperopic small discs, and bilateral optic disc drusen (ODD) are often confused with true papilledema (1). ODD are calcified deposits located in the optic nerve head, and its incidence in the population varies between 0.2 and 3% [17]. While large superficial ODDs can be seen easily in fundus examination, deep ODDs located under the optic disc are difficult to detect [18]. Particularly, while the deeply embedded drusen causes optic disc swelling, it does not lead to any abnormalities in the vessels emanating from the disc or microvascular changes such as bleeding and exudate [19]. Therefore, it is important to make the differential diagnosis with true papilledema in the early stage [20-23].

Kovarik et al. [24] showed that among 34 children referred for evaluation of suspected papilledema, 26 actually had pseudo papilledema. Only two of these 34 children

were ultimately diagnosed with papilledema. Similarly, Liu et al. [25] showed that among 52 children referred with a diagnosis of PTC and/or papilledema, 26 actually had pseudo papilledema. Among the 26 children with pseudo papilledema, MRI was performed in 19, CT in 9, and LP in 14 of them, and 12 were treated with medications before referral. These studies emphasize the necessity of an accurate evaluation of the optic disc to avoid overdiagnosis of PTC leading to inappropriate diagnostic testing and treatment [24, 25].

MRI findings of PTC include (i) empty sella, (ii) flattening of the posterior aspect of the globe (FPS), (iii) distention of the peri-optic subarachnoid space with or without a tortuous optic nerve, and (iv) transverse venous sinus stenosis [26]. In studies by Görkem et al. [27] and Kohli et al. [28], these four parameters were found to be significant for PTC, but FPS and optic nerve tortuosity were found to be associated with PTC to a greater extent among these four more parameters [27–29]. In the present study, presence of any of the MRI findings was not found to be associated with PTC except FPS. In our patients with asymptomatic Grade 1 papilledema with normal CSF pressure, empty sella and distention of the peri-optic subarachnoid space in the optic nerve sheath were observed. On the other hand, FPS was found to be significantly higher in patients with PTC.

The advantage of our study when compared with other studies is that the control group was composed of patients with a preliminary diagnosis of PTC. The limitation of our study when compared with other studies is that parametric data were not used in the measurement of the optic nerve sheath, and transverse sinus stenosis (TSS) was not checked. Parametric data were used owing to a lack of consensus on this issue. TSS was not checked because MR venography was not performed in every patient.

Conclusion

In the pediatric patients with the preliminary diagnosis of PTC, the headache that increases when leaning forward lasts longer than 2 h; the headache that keeps the child from playing was found to increase the risk for PTC significantly in our study.

Our study demonstrates that having no complaints and Grade 1 papilledema with or without MRI findings except FPS that has been detected incidentally were not found statistically significant between PTC and non-PTC groups. However, the presence of Grade ≥ 2 papilledema and CN6 paralysis was found to be an independent risk factor for PTC.

Acknowledgements We gratefully thank EMIRE BOR for the statistical analysis used in this study.

Data availability The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

Declarations

Conflict of interest The authors declare no competing interests.

References

- Bandyopadhyay S (2001) Pseudotumor cerebri. Arch Neurol 1699–70. https://doi.org/10.1001/archneur.58.10.1699
- Toscano S, Lo Fermo S, Reggio E, Chisari CG, Patti F, Zappia M (2020) An update on idiopathic intracranial hypertension in adults: a look at pathophysiology, diagnostic approach and management. J Neurol 27. https://doi.org/10.1007/s00415-020-09943-9
- Phillips PH, Sheldon CA (2017) Pediatric pseudotumor cerebri syndrome. J Neuroophthalmol 1:33–40. https://doi.org/10.1097/ WNO.00000000000548
- Friedman DI (2014) The pseudotumor cerebri syndrome. Neurol Clin 32(2):363–96. https://doi.org/10.1016/j.ncl.2014.01.001
- Gaier ED, Heidary G (2019) Pediatric idiopathic intracranial hypertension. Semin Neurol 39(6):704–10. https://doi.org/10. 1055/s-0039-1698743
- 6. Mallery RM, Rehmani OF, Woo JH et al (2019) Utility of magnetic resonance imaging features for improving the diagnosis of idiopathic intracranial hypertension without papilledema. Journal of neuro-ophthalmology : the official journal of the North American Neuro-Ophthalmology Society
- Balbi GGM, Matas SL, Len CA, Fraga MM, Sousa IO, Terreri MT (2018) Pseudotumorcerebri in childhood and adolescence: data from a specialized service. Arq Neuropsiquiatr 76(11):751– 55. https://doi.org/10.1590/0004-282X20180131
- Favoni V, Pierangeli G, Toni F, Cirillo L, La Morgia C, Abu-Rumeileh S et al (2018) Idiopathic intracranial hypertensionwithout papilledema (IIHWOP) in chronic refractory headache. Front Neurol 26;9:503. https://doi.org/10.3389/fneur.2018.00503
- Evans RW (1998) Complications of lumbar puncture. Neurol Clin 16(1):83–105 https://doi.org/10.1016/s0733-8619(05)70368-6
- Wall M, Falardeau J, Fletcher WA, Granadier RJ, Lam BL, Longmuir RA et al (2015) NORDIC Idiopathic Intracranial HypertensionStudy Group. Risk factors for poor visual outcome in patients with idiopathicintracranial hypertension. Neurology 1;85(9):799–805. https://doi.org/10.1212/WNL. 0000000000001896
- Burkett JG, Ailani J (2018) An up to date review of pseudotumor cerebri syndrome. Curr Neurol Neurosci Rep 2;18(6):33. https:// doi.org/10.1007/s11910-018-0839-1
- Bassan H, Berkner L, Stolovitch C, Kesler A (2008) Asymptomatic idiopathicintracranial hypertension in children. Acta Neurol Scand 118(4):251–5. https://doi.org/10.1111/j.1600-0404.2008.01007x
- Aylward SC, Reem RE (2017) Pediatric intracranial hypertension. Pediatr Neurol 66:32–43. https://doi.org/10.1016/j.pediatrneurol. 2016.08.010
- Distelmaier F, Sengler U, Messing-Juenger M, Assmann B, Mayatepek E, Rosenbaum T (2006) Pseudotumor cerebri as an important differential diagnosis ofpapilledema in children. Brain Dev 28(3):190–5. https://doi.org/10.1016/j.braindev.2005.07.003
- Boyter, Barmherzig R, Szperka CL (2019) Pseudotumor cerebri syndrome in children. Curr PainHeadache Rep 10;23(8):58. https://doi.org/10.1007/s11916-019-0795-8

- Fard MA, Sahraiyan A, Jalili J, Hejazi M, Suwan Y, Ritch R, Subramanian PS (2019) Optical coherence tomography angiography in papilledema compared with pseudopapilledema. Invest Ophthalmol Vis Sci 2;60(1):168–175. https://doi.org/10.1167/ iovs.18-25453
- Pineles SL, Arnold AC (2012) Fluorescein angiographic identification of optic discdrusen with and without optic disc edema. J Neuroophthalmol 32(1):17–22. https://doi.org/10.1097/WNO. 0b013e31823010b8
- Hamann S, Malmqvist L, Costello F (2018) Optic disc drusen: understanding an oldproblem from a new perspective. Acta Ophthalmol 96(7):673–684. https://doi.org/10.1111/aos.13748
- Chang MY, Pineles SL (2016) Optic disk drusen in children. Surv Ophthalmol 61(6):745–58. https://doi.org/10.1016/j.survophthal. 2016.03.007
- Rotruck J (2018) A review of optic disc drusen in children. Int Ophthalmol Clin 58(4):67–82. https://doi.org/10.1097/IIO. 00000000000236
- 21. Palmer E, Gale J, Crowston JG, Wells AP (2018) Optic nerve head drusen: an update. Neuroophthalmology 42(6):367–84. https://doi.org/10.1097/IIO.0000000000236
- 22. Freund P, Margolin E (2020) Pseudopapilledema 2020: StatPearls[Internet]. Treasure Island (FL): StatPearls Publishing
- Martinez MR, Ophir A (2011) Optical coherence tomography as an adjunctive tool fordiagnosing papilledema in young patients. J Pediatr Ophthalmol Strabismus 48(3):174–81. https://doi.org/10. 3928/01913913-20100719-05

- Kovarik JJ, Doshi PN, Collinge JE, Plager DA (2015) Outcome of pediatric patientsreferred for papilledema. J AAPOS 19(4):344–8. https://doi.org/10.1016/j.jaapos.2015.05.007
- Liu L, Yu MD, Shields CL (2019) Papilledema or pseudopapilledema? Indian J Ophthalmol 67(4):449. https://doi.org/10. 4103/ijo.IJO_2013_18
- Delen F, Peker E, Onay M, Altay ÇM, Tekeli O, Togay Işıkay C (2018) The significance and reliability of imaging findings in pseudotumor cerebri. Neuroophthalmology 43(2):81–90. https:// doi.org/10.1080/01658107.2018.1493514
- 27. Görkem SB, Doğanay S, Canpolat M, Koc G, Dogan MS, Per H et al (2015) MR imaging findings in children with pseudotumor cerebri and comparison with healthy controls. Childs Nerv Syst 31(3):373–80. https://doi.org/10.1007/s00381-014-2579-0
- Kohli AA, Vossough A, Mallery RM, Woo JH, Sheldon CA, Paley GL et al (2019) Magnetic resonance imaging findings in pediatric pseudotumor cerebri syndrome. Pediatr Neurol 99:31–39. https:// doi.org/10.1016/j.pediatrneurol.2019.04.010
- 29. Giridharan N, Patel SK, Ojugbeli A, Nouri A, Shirani P, Grossman AW et al (2018) Understanding the complex pathophysiology ofidiopathic intracranial hypertension and the evolving role of venous sinusstenting: a comprehensive review of the literature. Neurosurg Focus 45(1):10. https:// doi.org/10.3171/2018.4.Focus18100

Publisher's Note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.