performance in children and adolescents with cerebral palsy. Eur J Paediatr Neurol. 2010;14:496-502.
3. Hilderley AJ, Fehlings D, Lee GW, Wright FV. Comparison of a robotic-assisted gait training program with a program of functional gait training for children with cerebral palsy: design and methods of a two group randomized controlled cross-over trial. Springer Plus. 2016;5:1886.
4. Vrecar I, Majdic N, Jemec I, Damjan H. Changes in passive range of motion of joints of the lower limbs in children with cerebral palsy after an intense training program on the Lokomat. Rehabilitacija. 2013;12:38-45.
5. Fowler EG, Staudt LA, Greenberg MB, Oppenheim WL. Selective Control Assessment of the Lower Extremity (SCALE): Development, validation, and interrater reliability of a clinical tool for patients with cerebral palsy. Dev Med Child Neurol. 2009;51:607-14.
6. Janda V, Pavlu D. Goniometrie. Brno: Institut pro dalsi vzdelavani pracovniku ve zdravotnictvi; 1993.
7. Alotaibi M, Long T, Kennedy E, Bavishi S. The efficacy of GMFM-88 and GMFM-66 to detect changes in gross motor function in children with cerebral palsy (CP): A literature review. Disabil Rehabil. 2014;36:617-27.
8. Thompson P, Beath T, Bell J. Test-retest reliability of the 10-metre fast walk test and 6-minute walk test in ambulatory school-aged children with cerebral palsy. Dev Med Child Neurol. 2008;50:370-6.
9. Columbo G, Joerg M, Schreier R, Dietz V. Treadmill training of paraplegic patients using a robotic orthosis. J Rehabil Res Dev. 2000;37:693-700.
10. Cohen J. Statistical Power Analysis for the Behavioral Sciences, 2nd edition, New York, 1988.

Pediatric Papilledema at a Tertiary Care Ophthalmological Center

Pediatric papilledema is usually asymptomatic and is diagnosed on routine screening. We conducted a retrospective study to evaluate pediatric papilledema with respect to presentation, etiology and treatment at the neuroophthalmology clinic of a tertiary care eye institute.19 of the 24 children studied had Idiopathic intracranial hypertension.This study stresses upon the interdisciplinary approach for prompt diagnosis and treatment of papilledema.

Keywords: *Diagnosis, Idiopathic intracranial hypertension, Management, Referral.*

Papilledema is defined as optic disc edema secondary to high intracranial pressure, the etiology for which may be known or unknown (idiopathic) [1]. Idiopathic intracranial hypertension (IIH) is typically defined by exclusion using modified Dandy criteria [2]. IIH typically affects obese women of childbearing age, but it may be seen in patients of any age or weight [3]. Obesity and weight gain appear to be risk factors during adolescence but not in pre pubertal age group [4]. Pediatric IIH is diagnosed in many asymptomatic children during a routine encounter [5].

Pediatric central nervous system tumors are the second most common childhood malignancies, and hence is a major etiology of pediatric papilledema. The purpose of this study was to evaluate papilledema in the pediatric age group at the neuro-ophthalmology clinic of a tertiary eye care center.

A review of hospital records of papilledema patients in the pediatric age group (<15 years) was done for the period January, 2016 – December, 2018. Patients with pseudo papilledema and those on previous treatment were excluded from the study. We reviewed the case records of all the patients and extracted information on age and symptoms at presentation, best corrected visual acuity, pupillary response, extraocular movements, diplopia, fundus biomicroscopy and optic disc findings at presentation. Body mass index was calculated for all the patients. Pre pubertal age was considered to be less than 11 years and pubertal between 11 and 15 years. Best corrected visual acuity was measured using Snellen optotypes and visual field was tested using Bjerrums kinetic perimetry. Neuro-imaging of brain (magnetic resonance imaging (MRI) or MR venogram) findings and serological evaluation including complete hemogram, thyroid function tests were recorded. Results of lumbar puncture and cerebrospinal fluid analysis were available for only one patient, due to lack of consent in others.

Twenty-four patients met the inclusion criteria and the mean age was 11.3 years, youngest was a 2-year-old child. Girls were more frequently affected (13, 54.1%). The commonest presenting symptom was headache **(***n*=12**)**, followed by double vision (*n*=7), and defective vision (*n*=6). Few patients presented with sudden onset of ocular deviation (*n*=2), pain on eye movement (*n*=2), radiating neck pain (*n*=2) and frequent blinking (*n*=1). Best corrected visual acuity remained 20/20 in 18 of our patients in both eyes, while 6 (25%) patients presented with visual morbidity. Of those, three had IIH and others were due to secondary causes. Pupillary examination and color vision remained normal in all our patients except in one diagnosed with craniopharyngioma. Sixth nerve palsy was seen in 12.5% (*n*=3) of patients,and 87.5% (*n*=21) patients had enlarged blind spot on visual field assessment. Overall, 23 (96%) patients had bilateral disc edema and one had unilateral disc edema on fundus examination. The most common etiology in our population was found to be IIH in 79% (*n*=19), intracranial tumors in 12.5%, and the rest falling under infective etiology and obstructive hydrocephalus (***Table*I**).

IIH in children and adolescents is relatively uncommon and may be associated with puberty and resulting hormonal changes

[6]. In pre-pubertal children, IIH appears to be even less frequent;we found three girls and three boys each in the pre-pubertal age. Children with IIH are reported to have an equal sex distribution [7], though we found a male female ratio of 1:2. Affected adolescents of IIH tend to be overweight, but obesity and weight gain do not appear to be risk factors [8]. In our series one girl was obese, two were overweight; one of whom was in pubertal age. Acute headache and double vision were the common symptoms on initial presentation and none of our patients were picked upon routine examination. We had three patients with sixth nerve palsy as false localizing sign, who presented with sudden squinting.

Visual loss has been reported to occur in children with IIH. Pediatric IIH is just as threatening to vision as the adult form [6], in our study we encountered visual morbidity in three of our patients. Enlarged blind spot, which has been reported to occur in virtually all eyes with papilledema,was found in our patients also. Accurate visual field testing in children is sometimes difficult to perform, and hence difficult to rely on as the only accurate test. We suggest performing a kinetic perimetry in young and uncooperative children. Symmetric papilledema was recorded in eighteen children and one boy had unilateral papilledema.In this series, all our patients were referred to neurophysician and medically managed with oral acetozolamide and responded well to treatment. None of our patients needed Optic nerve sheath decompression.

Brain tumors with the greatest direct threat to the visual pathways are tumors that involve the optic pathway, parasellar tumors, and cerebral hemispheric tumors [8].We had one patient with pilocytic astrocytoma, the commonest cerebral hemispheric lesion which causes vision loss due to secondary optic atrophy following papilledema. Craniopharyngioma, the most common supratentorial tumor of childhood exhibits a bimodal age distribution. In our series, it was diagnosed in a 15-year-old boy with chronic visual deficit in one eye with papilledema [9].Though tuberculosis is common in India, tuberculous brain abscess is rare [10].Our patient with multiple tubercular cerebral abscess and midline shift had papilledema as the primary manifestation and was treated with anti-tuberculous therapy and recovered completely.

In summary, IIH is a common cause of papilledema in Indian children, and they are mostly symptomatic during presentation and respond well to medical management. Prompt diagnosis and proper management can prevent needless blindness resulting from secondary optic atrophy and also play a significant role in saving the life of children. This study emphasizes that ophthalmologists play a key role in monitoring for visual morbidity following papilledema and also stresses upon the interdisciplinary approach for prompt diagnosis and treatment of papilledema.

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REFERENCES

1. Lee AG, Wall M. Papilledema: are we any nearer to a consensus on pathogenesis and treatment? Curr Neurol Neurosci Rep. 2012;12:334-9.
2. Friedman DI, Jacobson DM. Diagnostic criteria for idiopathicintracranial hypertension. Neurology. 2002;59: 1492-95.
3. Dhungana S, Sharrack B, Woodroofe N. Idiopathic intracranial hypertension. ActaNeurol Scand. 2010;121: 71-82.
4. Aylward SC, Reem RE. Pediatric intracranial hypertension. Pediatr Neurol. 2017;66:32-43.
5. Bassan H, Berkner L, Stolovitch C. Asymptomatic idiopathic intracranial hypertension in children. Acta Neurol Scand. 2008;118:251-5.
6. Lessell S. Pediatric pseudotumorcerebri (idiopathic intracranial hypertension).SurvOphthalmol. 1992;37:155-66.
7. Babikian P, Corbett J, Bell W. Idiopathic intracranial hypertension inchildren: The Iowa experience. J Child Neurol. 1994;9:144-9.
8. Edmond JC. Pediatric brain tumors: The neuro-ophthalmic impact. Int OphthalmolClin. 2012;52:95-106.
9. Merchant TE, Pollack IF, Loeffler JS. Brain tumors across the age spectrum: Biology,therapy, and late effects. SeminRadiatOncol. 2010;20:58-66.
10. Andronikou S, Greyling PJ. Devastating yet treatable complication of tuberculous meningitis: The resistant TB abscess. ChildsNervSyst.2009;25:1105-06.

Noonan Syndrome in Thai Children

This study describes clinical features of Noonan syndrome and gene mutations, including *PTPN11, SOS1*, and *BRAF* in the Thai population.Widely spaced eyes were the most common finding from the digital facial analysis technology used in this study.

Keywords: *Facial analysis technology, Gene mutation, PTPN11*.

Noonan syndrome is a genetic disorder with an estimated prevalence of 1 in 1,000 to 2,500 live births [1]. The typical facial features include ptosis, widely spaced eyes, down slanted palpebral fissures, and low set ears [2]. Early and accurate diagnosis of NS is essential as each patient needs an individual treatment regimen, and has distinct recurrent risk and prognosis [3]. Due to limited resources for genetic testing for the disorder, facial analysis technology may be useful to identify new cases. The digital facial analysis technology has previously been used to identify individuals with Noonan syndrome from 20