

## Pulsatile Tinnitus as a Presenting Symptom for Idiopathic Intracranial Hypertension: Diagnosis and Management

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**Abstract: Objectives:** This pilot selective study aimed to explore relationship between intracranial hypertension (IIH) and development of pulsatile tinnitus (PT) and the impact of its treatment on severity of PT. **Patients & Methods:** All patients presenting to ENT clinic with PT were evaluated and only patients fulfilling Modified Dandy's Criteria for the diagnosis of IIH were enrolled in the study. Patients underwent full otorhinolaryngological examination and tinnitus was graded using the Tinnitus Handicap Inventory (THI). Severity of headache was assessed using the 11-point Numeric Rating Scale (NRS) and detailed ophthalmic examination was performed. All patients underwent lumbar puncture (LP) and opening pressure was measured, then patients were maintained on acetazolamide 1–2 g/day. If patients did not show improvement or if the condition recurred after initial improvement, patients underwent insertion of lumbo-peritoneal shunt (LPS) for permanent drainage. **Results:** Six PT patients had IIH; 5 females and one male with mean age of  $31.5 \pm 6.2$  years and mean BMI of  $35.1 \pm 3.5$  kg/m<sup>2</sup>. All patients had PT that completely disappeared on jugular compression with mean THI score of  $22 \pm 13.3$ . At time of discharge, all patients showed improved headache, tinnitus completely disappeared in five patients, while the 6<sup>th</sup> patient had THI grade 1 with a mean total THI score of  $8 \pm 6.9$  with significant difference versus at admission score. Throughout mean follow-up period of  $15.5 \pm 3.9$  months; one patient showed recurrence of headache and tinnitus and underwent LPS insertion for permanent CSF drainage and patient reported complete relief of her symptoms and tinnitus disappeared completely. **Conclusion:** There is close association between IIH and PT. IIH-directed therapy provided nearly complete relief of tinnitus with resolution of other manifestations. Presence of PT must arouse suspicion for being secondary to increased ICP and could be used as prognostic sign for assessing treatment efficacy and follow-up for the possibility of recurrence.

[Mohamed F. Shindy. **Pulsatile Tinnitus as a Presenting Symptom for Idiopathic Intracranial Hypertension: Diagnosis and Management.** *J Am Sci* 2015;11(6):201-208]. (ISSN: 1545-1003). <http://www.jofamericanscience.org>. 23

**Keywords:** Pulsatile tinnitus, idiopathic intracranial hypertension, Lumbar puncture, Lumbo-peritoneal shunt

### 1. Introduction

Pulsatile tinnitus (PT) is an uncommon otologic symptom, which often presents a diagnostic and management dilemma to the otolaryngologist. This symptom always deserves a thorough evaluation to avoid disastrous consequences from potentially life-threatening associated pathology. However, in most pulsatile tinnitus patients a treatable underlying etiology can be identified (**Harvey et al., 2014**).

Pulsatile tinnitus can be classified by its site of generation as arterial, arteriovenous, or venous. Typical arterial causes are arteriosclerosis, dissection, and fibromuscular dysplasia. Common causes at the arteriovenous junction include arteriovenous fistulae and highly vascularized skull base tumors. Common venous causes are intracranial hypertension and, as predisposing factors, anomalies and normal variants of the basal veins and sinuses. In a series of patients, pulsatile tinnitus was most often due to highly vascularized tumors of the temporal bone (16%), followed by venous normal variants and anomalies (14%) and vascular stenoses (9%). Dural arteriovenous fistulae, inflammatory hyperemia, and

intracranial hypertension (IIH) were tied for fourth place; 8% each, (**Hofmann et al., 2013**).

Pseudotumor cerebri (PTC) is characterized by raised intracranial pressure (ICP) without an identifiable mass, evidence of hydrocephalus, or abnormal cerebrospinal fluid (CSF) content. In the past, most cases of PTC appeared to have no identifiable etiology, and thus, they were classified as "idiopathic intracranial hypertension". Recently, however, a subset of patients with presumed IIH has been found to have evidence of cerebral dural sinus stenoses, particularly involving one or both transverse sinuses (**Radvany et al., 2013**).

Idiopathic intracranial hypertension is a disease with a predilection for young obese women. IIH is a syndrome characterized by elevated ICP, headache, transient visual obscuration and pulsatile tinnitus. However, patient maintains an alert and oriented mental state with no localizing neurologic findings apart from the false-localizing 6<sup>th</sup> cranial nerve palsy (**Wakerley et al., 2015**).

Neurodiagnostic studies are otherwise normal except for increased ICP. Neuroimaging showed signs

of ICP including empty sella syndrome, lateral sinus collapse, flattened globes and fully unfolded optic nerve sheaths, but there is no evidence of deformity or obstruction of the ventricular system or secondary cause of IIH can be found. However, abnormalities of the pituitary gland and optic nerve sheath were reliable diagnostic signs for IIH (**Hoffmann et al., 2013**).

The most dangerous sequale of IIH is the development of papilledema which is usually present and can lead to optic atrophy with progressive permanent visual loss. The earliest sign of visual loss is constriction of peripheral visual field, usually starting with the inferior nasal quadrant (**Soiberman & Kesler, 2013**).

The current prospective pilot study aimed to explore the relationship between IIH and development of PT and the impact of its treatment on the severity of tinnitus.

## 2. Patients & Methods

The current study was conducted at Departments of Otorhinolaryngology and Neurosurgery, Miami Hospital, Abu Dabi, UEA since Jan 2011 till April 2014 so as to allow a minimum follow-up period of 6 months for the last case enrolled in the study. All patients presenting to ENT outpatient clinic with tinnitus of any grade of severity were enrolled in the preliminary evaluation for the presence of IIH. Selection of patients with IIH was relied on Modified Dandy's Criteria for the diagnosis of IIH (**Dandy, 1937; Friedman & Jacobson, 2002**) including awake and alert patient presenting by signs and symptoms of increased ICP including headache, nausea, vomiting, transient obscuration of vision, papilledema without localizing or focal signs on neurological examination and absence of deformity, obstruction, and displacement of the ventricular system as confirmed by radiological workup. CSF opening pressure in the lateral decubitus position of  $>200$  mmH<sub>2</sub>O in non-obese patients and  $>250$  mm H<sub>2</sub>O in obese patients with CSF free of cytological or chemical abnormalities is confirmatory for IIH.

All enrolled patients underwent detailed history taking including demographic data for age, gender, body weight, height and calculation of body mass index (BMI) defined as weight in kilograms divided by the square of the height in meters (**Bray, 1992**). Patients were graded according to the international classification of BMI into: underweight (BMI $<18.5$  kg/m<sup>2</sup>); normal weight range (BMI=18.5-24.99 kg/m<sup>2</sup>); overweight (BMI=25-29.99 kg/m<sup>2</sup>); Obese (BMI $>30$  kg/m<sup>2</sup>) (**WHO 1995, 2004**).

Patients underwent determination of IIH-related data including date of presentation, presenting symptoms including headache, nausea, vomiting,

neurological symptoms, and ocular symptoms like transient visual loss, loss of central vision, visual field defects, and double vision and medications received and response to it. Also, history included inquire about the presence and duration of any systemic disease, as hypertension, diabetes mellitus or relevant drug history as systemic corticosteroids, oral contraceptive pills, and vitamins.

Physical examination included evaluation of the severity of headache using the 11-point Numeric Rating Scale (NRS) for assessment of pain intensity with numbers from 0 to 10 where 0 indicates no pain and 10 indicates worst pain imaginable (**Collins et al., 1997**). A detailed ophthalmic examination was performed, including best corrected snellen visual acuity, refraction, ocular motility examination, pupil evaluation, color vision, anterior segment examination, intraocular pressure measurement, optic disc examination and degree of papilledema. Visual field testing by either automated Humphrey static or manual Goldmann perimetry. Then, all patients underwent complete neurological for cranial nerve deficits or associate

All patients underwent a full tinnitus history with emphasis around the time of onset of tinnitus and onset of tinnitus annoyance, history of exacerbation of tinnitus by a change in medication, exposure to loud noise or increase in stress, and laterality of tinnitus. Medical and drugs history was taken. Jugular veins compression test; including cessation of PT on jugular vein compression suggests a vascular origin of tinnitus.

Patients underwent otoscopy for assessment of drum and middle ear status, tympanometry to test middle ear function and pure tone audiometry to evaluate hearing affection. Assessment of the effects of the tinnitus was graded using the Tinnitus Handicap Inventory (THI) as shown in table 1 (**Newman, 1999**).

Radiological workup included brain MRI for exclusion of intracranial pathology and MR angiography for exclusion of abnormalities of brain sinuses and CT examination of head for exclusion of ear, drum, and sinuses lesions

All patients underwent lumbar puncture, under complete aseptic condition, with the patient in the lateral decubitus position with outstretched legs and as relaxed as possible, or in the seating position. Following injection of 3-cm subcutaneous lidocaine, a spinal needle was inserted into a lower lumbar (e.g., L4/L5) intervertebral space. The opening pressure was measured using a manometer held at the level of the left atrium, prior to the removal of CSF. Generally, an opening pressure  $\leq 200$  mmH<sub>2</sub>O is considered normal, a pressure value of 201-249 mmH<sub>2</sub>O is inconclusive and a pressure  $>250$  mmH<sub>2</sub>O indicated high ICP (**Brodsky & Vaphiades, 1998**). CSF sample was

obtained for cytological and bacteriological examination and then an appropriate volume of CSF was drained. Patients were maintained on medical treatment including acetazolamide 1–2 g/day but its dosage was reduced in patients who were not able to tolerate its side effects including nausea, vomiting, sedation, and metabolic acidosis.

Patients were followed-up for their headache scores, presence and severity of tinnitus, Snellen's visual acuity, grading of papilledema and field of

vision once weekly for one month and monthly for 3 months and then every six months. If patients did not show any improvement in Snellen's visual acuity for a minimum of two lines or visual field defects with persisting disc edema for 6–8 weeks after lumbar puncture and medical treatment, or if the condition recurred after initial improvement, patients were prepared for insertion of lumbo-peritoneal shunt (LPS) for permanent drainage.

**Table (1): Grading the severity of tinnitus (Newman, 1999)**

Grade	Description	Significance	THI=
Slight (Grade 1)	<ol style="list-style-type: none"> <li>1. Tinnitus only heard in quiet environment</li> <li>2. Very easily masked.</li> <li>3. No interference with sleep or daily activities.</li> </ol>	Grade 1 covers most people experiencing but are not troubled by tinnitus.	0-16
Mild (Grade 2)	<ol style="list-style-type: none"> <li>1. Easily masked by environmental sound</li> <li>2. Easily forgotten with activities.</li> <li>3. Occasionally interfere with sleep but with not daily activities.</li> </ol>	The majority of people suffering tinnitus fall into grades 2 and 3.	18-36
Moderate (Grade 3)	<ol style="list-style-type: none"> <li>1. Presence of background/environmental noise</li> <li>2. Daily activities may still be performed.</li> <li>3. Less noticeable when concentrating.</li> <li>4. Not infrequently interferes with sleep and quiet activities.</li> </ol>		38-56
Severe (Grade 4)	<ol style="list-style-type: none"> <li>1. Almost always heard, rarely if ever masked.</li> <li>2. Leads to disturbed sleep pattern</li> <li>3. Can interfere with ability to carry out normal daily activities.</li> <li>4. Quiet activities adversely affected.</li> <li>5. Hearing loss is likely to be present.</li> </ol>	Uncommon.	56-100
Catastrophic (Grade 5)	<ol style="list-style-type: none"> <li>1. All tinnitus symptoms at level of severe or worse.</li> <li>2. Hearing loss likely to be present.</li> <li>3. Associated psychological pathology is likely to be found</li> </ol>	Extremely rare.	

THI: Tinnitus Handicap Inventory

### Statistical analysis

Obtained data were presented as mean±SD, ranges, numbers and ratios. Results were analyzed using paired t-test for within group variability and Wilcoxon; ranked test for unrelated data (Z-test) and Chi-square test ( $X^2$  test) paired t-test for variability between groups. Statistical analysis was conducted using the SPSS (Version 15, 2006) for Windows statistical package. *P* value <0.05 was considered statistically significant.

### 3. Results

Six patients had tinnitus in association with other manifestations of IIH; 5 females and one male with mean age of 31.5±6.2; range: 28-42 years, but 3 women were younger than 30 years and two females were in range of 30-40 years and the male was aged 42 years. Three females were very obese with BMI>35 kg/m<sup>2</sup>, two females were obese with BMI in range of 30-35 kg/m<sup>2</sup> and the male was overweight with BMI of 29.1 kg/m<sup>2</sup> with a mean BMI of 35.1±3.5; range: 29.1-38.6 kg/m<sup>2</sup>: details patients' data are shown in table 2.

**Table (2): Demographic data of patients had tinnitus in association with IHH**

Data				Frequency	Mean ( $\pm$ SD)	
Age (years)	Strata	<30		3 (50%)	27 $\pm$ 1.7	
		30-40		2 (33.3%)	33 $\pm$ 2.8	
		>40		1 (16.7%)	42	
	Total		6 (100%)	31.5 $\pm$ 6.2		
Gender	Males			1 (16.7%)		
	Females			5 (83.3%)		
BMI data	Weight			6 (100%)	102.5 $\pm$ 11.8	
	Height			6 (100%)	170.7 $\pm$ 1.9	
	BMI	Strata	Overweight (25-30)		1 (16.7%)	29.1
			Obese (>30-35)		2 (33.3%)	34.3 $\pm$ 0.9
			Very obese (>35-40)		3 (50%)	37.7 $\pm$ 0.8
	Total			6 (100%)	35.1 $\pm$ 3.5	

BMI: Body mass index

All patients presented with symptoms suggestive of increased ICP. Mean NRS score of headache at time of presentation was 7.8 $\pm$ 0.8; range: 7-9. Three patients had nausea and vomiting, 4 patients had transient

visual obscuration and reduced visual acuity and 5 patients had bilateral papilledema; 4 of grade 0 and one of grade 2 and one had unilateral papilledema of grade one (Table 3).

**Table (3): IHH-related clinical data**

Data				Findings
Headache				6 (100%)
NRS scoring of headache severity			7	2 (33.3%)
			8	3 (50%)
			9	1 (16.7%)
			Total	7.8 $\pm$ 0.8
Transient visual obscuration				4 (66.7%)
Reduced visual acuity				4 (66.7%)
Snellen's visual acuity			6/6	1 (16.7%)
			6/9-6/18	2 (33.3%)
			6/24-6/60	2 (33.3%)
			HM	1 (16.7%)
Nausea & vomiting				3 (50%)
Diplopia				1 (16.7%)
Disc edema score	Grading	0		4 (66.6%)
		1		1 (16.7%)
		2		1 (16.7%)
	Laterality	Bilateral		5 (83.3%)
		Unilateral		1 (16.7%)

Data are presented as numbers & mean $\pm$ SD; percentages are in parenthesis; NRS: Numeric Rating Scale; HM: Hand movement perception

All of the six patients had vascular tinnitus that completely disappeared on jugular compression. Three patients had mild, 2 had moderate and one had severe tinnitus with a mean score of 22 $\pm$ 13.3; range: 8-44. Mean duration of symptom was 5.5 $\pm$ 1; range: 4-7 months and followed development of headache by a

mean duration of 30 $\pm$ 11.4; range: 15-45 days (Table 4). Four patients received medical treatment for otitis media but treatment was ineffective and one patient had previous myringotomy and insertion of Gromets' tube for pressure equalization but the effect was negligible.

**Table (4): Tinnitus-related clinical data**

Data			Findings
Duration of tinnitus complaint	<6 months	Number	3 (50%)
		Duration	4.7±0.6
	>6 months	Number	3 (50%)
		Duration	6.3±0.5
	Total		
Time lag between development of headache and appearance of tinnitus	≤30 days	Number	4 (66.7%)
		Duration	23.8±7.5
	>30 days	Number	2 (33.3%)
		Duration	42.5±3.5
	Total		
Tinnitus grade	1		3 (50%)
	2		2 (33.3%)
	3		1 (16.7%)
Total THI score			22±13.3 (8-44)

Data are presented as numbers & mean±SD; percentages & ranges are in parenthesis; THI: Tinnitus Handicap Inventory

One of the six patients refused the trial of single lumbar puncture and medical treatment and asked for application of LPS during the preliminary lumbar puncture, while the other 5 patients underwent preliminary lumbar puncture and received medical treatment. Mean CSF opening pressure was 313.3±29.8; range: 280-365 mmH<sub>2</sub>O. Two patients were admitted to surgical ICU for one day for

maintenance on oxygen supply and adjustment of their respiration, but none required mechanical ventilation. Four patients were discharged on the 2<sup>nd</sup> day and two patients were discharged on 3<sup>rd</sup> day after lumbar puncture for a mean hospital stay of 2.3±0.5; 2-3 days. Details of operative and immediate postoperative period were shown in table 6.

**Table (6): Management data**

Data				Findings
CSF opening pressure (mmH <sub>2</sub> O)	Strata	<300	Frequency	2 (33.3%)
			Mean	287.5±10.6
	≥300	Frequency	4 (66.7%)	
		Mean	326.3±27.8	
Total				313.3±29.8
Line of management	LP and medical treatment			5 (86.7%)
	LPS insertion			1 (16.7%)
Immediate postoperative stay at	Surgical ICU			2 (33.3%)
	Surgical ward			4 (66.7%)
Hospital stay	2 <sup>nd</sup> PO day			4 (66.7%)
	3 <sup>rd</sup> PO day			2 (33.3%)
	Mean duration (days)			2.3±0.5 (2-3)

Data are presented as numbers & mean±SD; percentages & ranges are in parenthesis; CSF: Cerebrospinal fluid; LP: Lumbar puncture; LPS: Lumboperitoneal shunt; ICU: Intensive care unit; PO: Postoperative

At time of discharge, all patients showed improved headache with a mean at discharge NRS score of 2.8±0.4; range: 2-3 and significant difference versus at admission NRS score. Tinnitus completely

disappeared in five patients, while the 6<sup>th</sup> patient had THI grade 1 with a mean total THI score of 8±6.9; range: 2-20 with significant difference versus at admission THI score (Figs. 1 & 2).

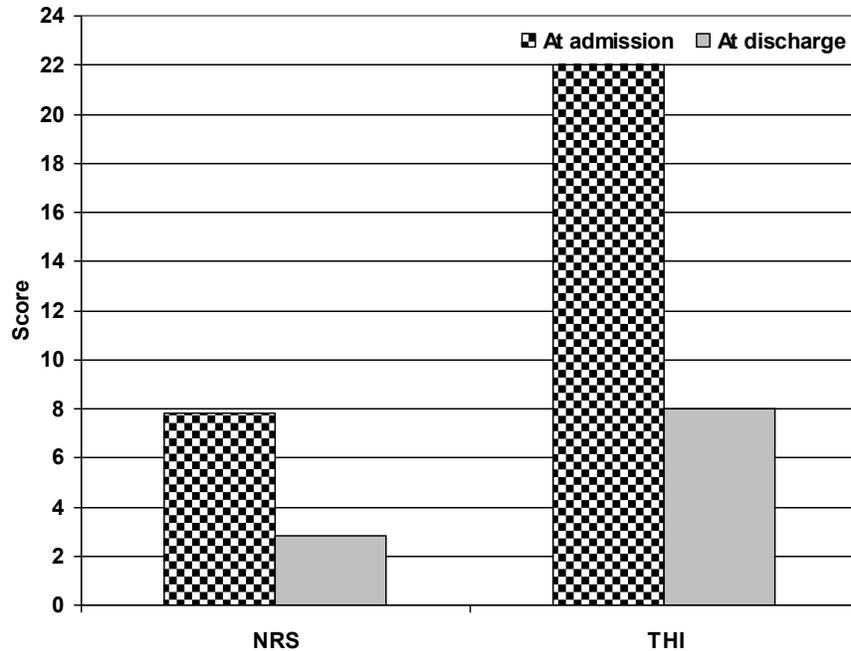


Fig. (1): Mean at admission and discharge NRS and THI scores of studied patients

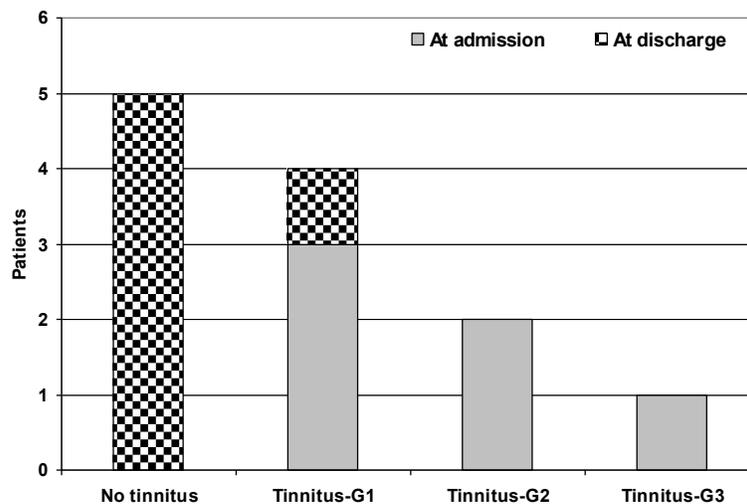


Fig. (2): At admission and discharge tinnitus grading of studied patients

Throughout mean follow-up period of  $15.5 \pm 3.9$ ; range: 9-21 months; one patient showed recurrence of headache and tinnitus and underwent insertion of LPS for permanent CSF drainage and patient reported complete relief of her symptoms and tinnitus disappeared completely. As regards ophthalmic manifestations, 3 patients showed improved visual acuity by two lines, 2 patients showed improvement by one line, while the 6<sup>th</sup> showed no improvement of visual acuity, but all showed resolution of papilledema.

#### 4. Discussion

The current study aimed to selection of patients with IIH, a diagnostic entity that relies on exclusion of other causes of increased intracranial pressure (ICP) despite of the presence of full spectrum of its clinical manifestations, among those presenting by pulsatile tinnitus (PT) so as to explore the relationship between IIH and development of PT and the impact of its treatment on the severity of tinnitus.

Throughout the study period, 6 patients with IIH-related PT were collected, a finding indicating a

possible causal-effect relationship between IIH and PT. in line with this finding, **Skau et al. (2011)** who in their series of IIH found the initial symptoms were headache (94%), visual blurring (82%) and pulsatile tinnitus (65%). Also, **Wall et al. (2014)** in their large prospective cohorts of untreated patients with IIH reported pulse synchronous tinnitus in 52% of patients enrolled in the Idiopathic Intracranial Hypertension Treatment Trial.

Interestingly, majority of studied patients were obese with BMI crossing up to 42 kg/m<sup>2</sup>, a finding indicating an etiological relationship between obesity and development of IIH. This observation supported that previously reported in literature wherein **Pollak et al. (2013)** retrospectively reviewed medical records of patients with IIH and found 82% of patients were overweight at the time of diagnosis and number of recurrences inversely correlated with weight loss and seemed to be influenced by the obstetrical history. **Contreras-Martin & Bueno-Perdomo, (2015)** reported that IIH is more common in young women with higher body weight and is also associated with the use of contraceptive drugs. Also, **Naarden et al. (2015)** found patients with IIH are mainly overweight young women who present with raised ICP evidenced by headache, nausea, vomiting and vision disturbances. **Almarzouqi et al. (2015)** reported that the incidence of IIH in several Middle East countries has been estimated at 2.02-2.2/100,000 in the general population, obesity is a major risk factor for IIH which is associated with an increased risk of severe vision loss.

Mean cerebro-spinal fluid (CSF) opening pressure of studied patients was 313.3±29.8; range: 280-365 mmH<sub>2</sub>O, which coincided with that reported by **Ambika et al. (2010)** who in their series of 50 IIH patients, CSF opening pressure was 250-350 mmH<sub>2</sub>O in 39 patients and was >350 mmH<sub>2</sub>O in 11 patients. Also, **El-Saadany et al. (2012)** reported that among 22 patients who underwent lumbo-peritoneal shunt (LPS) placement for IIH, 16 patients had severe and fulminant opening CSF pressures with values of more than 400 mmH<sub>2</sub>O.

All studied patients responded to preliminary lumbar puncture (LP) and maintenance on medical treatment and showed significant improvement of their headache scoring and tinnitus grading, with improvement of visual acuity, field of vision and resolution of papilledema. Unfortunately, one patient showed recurrence of headache and tinnitus and underwent insertion of LPS for permanent CSF drainage and patient reported complete relief of her symptoms and tinnitus disappeared completely.

These findings indicated the possibility of ICP control using medical treatment after pressure normalization by lumbar puncture and go in hand with

**Ambika et al. (2010)** who after initial lumbar puncture started medical treatment for all patients and 70% of patients responded, while 30% patients had to undergo LPS. **Skau et al. (2011)** found that in patients with IIH and underwent repeated LP weight-loss is the main predictor of a favorable outcome with respect to CSF pressure as in patients with weight-loss >3.5% of BMI, ICP decreased significantly, while in patients with weight-loss <3.5% of BMI, changes in ICP were insignificant.

These results indicated a causal-effect relationship between increased ICP and development of PT that found to be relieved by ICP control. In support of this opinion, **Kastanioudakis et al. (2013)** presented a 19-year-old morbid obese female had bilateral PT, headache, neck pain, slight imbalance, and visual disturbances with bilateral papilledema and decreased visual acuity. Light digital pressure over ipsilateral internal jugular vein resulted in immediate cessation of PT and hearing improvement. Neuro-radiological studies were normal, but lumbar puncture revealed high ICP. Patient went on a diet and received oral acetazolamide, after two weeks, patient's vision, headache, neck pain, PT and hearing loss initially became stable and afterwards improved gradually to complete resolution of PT and headache after one month of treatment and dieting. **Yri & Jensen, (2014)** documented that aggravation of headache by coughing or straining, relief after CSF withdrawal, retrobulbar pain and PT may suggest intracranial hypertension.

### Conclusion

The obtained results showed close association between IIH and pulsatile tinnitus. IIH-directed therapy, either medical, single drainage or permanent drainage provided nearly complete relief of tinnitus with resolution of other manifestations. Thus, presence of PT must arouse suspicious for being secondary to increased ICP and could be used as prognostic sign for assessing efficacy of treatment and for follow-up for the possibility of recurrence.

### Acknowledgment

The author wishes to acknowledge staff members of Neurosurgery and Neuroimaging Departments, Al-Ahleia Hospital and New Medical Center (NMC), Abu Dabi, UEA for their invaluable assistance for fulfilling this study.

### References

1. Almarzouqi SJ, Morgan ML, Lee AG: Idiopathic intracranial hypertension in the Middle East: A growing concern. *Saudi J Ophthalmol.* 2015; 29(1):26-31.
2. Ambika S, Arjundas D, Noronha V, Anshuman: Clinical profile, evaluation, management and

- visual outcome of idiopathic intracranial hypertension in a neuro-ophthalmology clinic of a tertiary referral ophthalmic center in India. *Ann Indian Acad Neurol.* 2010; 13(1):37-41.
3. Bray GA: Pathophysiology of obesity. *Am J Clin Nutr.*, 1992; 55: 488S-94S.
  4. Brodsky MC, Vaphiades M: Magnetic resonance imaging in pseudotumor cerebri. *Ophthalmology*, 1998, 105(5): 1686-93.
  5. Collins S, Moore R, McQuay H: The visual analogue pain intensity scale: What is moderate pain in millimetres. *Pain* 1997, 72:95-7.
  6. Contreras-Martin Y, Bueno-Perdomo JH: Idiopathic intracranial hypertension: Descriptive analysis in our setting. *Neurologia.* 2015; 30(2):106-110.
  7. Dandy WE: Intracranial pressure without brain tumor: diagnosis and treatment. *Annals of Surgery*, 1937; 106: 492–513.
  8. El-Saadany WF, Farhoud A, Zidan I: Lumboperitoneal shunt for idiopathic intracranial hypertension: patients' selection and outcome. *Neurosurg Rev.* 2012; 35(2):239-444.
  9. Friedman DI, Jacobson DM. Diagnostic criteria for idiopathic intracranial hypertension. *Neurology* 2002; 59(10):1492-5.
  10. Harvey RS, Hertzano R, Kelman SE, Eisenman DJ: Pulse-synchronous tinnitus and sigmoid sinus wall anomalies: descriptive epidemiology and the idiopathic intracranial hypertension patient population. *Otol Neurotol.* 2014; 35(1):7-15.
  11. Hoffmann J, Huppertz HJ, Schmidt C, Kunte H, Harms L, Klingebiel R, Wiener E: Morphometric and volumetric MRI changes in idiopathic intracranial hypertension. *Cephalalgia.* 2013; 33(13):1075-84.
  12. Hofmann E, Behr R, Neumann-Haefelin T, Schwager K: Pulsatile tinnitus: imaging and differential diagnosis. *Dtsch Arztebl Int.* 2013; 110(26):451-8.
  13. Kastanioudakis I, Konitsiotis S, Asproudis I, Ziavra N: Venous pulsatile tinnitus due to pseudotumor cerebri syndrome in a young morbid obese female. *Hippokratia.* 2013 Oct;17(4):383.
  14. Naarden MT, Schuitemaker A, Braakman HM, van Doormaal TP, Porro GL, Straver JS: Idiopathic intracranial hypertension and obesity]. *Ned Tijdschr Geneeskd.* 2015; 159: A7980.
  15. Newman CW. Development of the Tinnitus Handicap Inventory. *Arch Otolaryngol Head Neck Surg.* 1999; 122: 143–48.
  16. Pollak L, Zohar E, Glovinsky Y, Huna-Baron R: Reevaluation of presentation and course of idiopathic intracranial hypertension--a large cohort comprehensive study. *Acta Neurol Scand.* 2013; 127(6):406-12.
  17. Radvany MG, Solomon D, Nijjar S, Subramanian PS, Miller NR, Rigamonti D, Blitz A, Gailloud P, Moghekar A: Visual and neurological outcomes following endovascular stenting for pseudotumor cerebri associated with transverse sinus stenosis. *J Neuroophthalmol.* 2013; 33(2):117-22.
  18. Skau M, Sander B, Milea D, Jensen R: Disease activity in idiopathic intracranial hypertension: a 3-month follow-up study. *J Neurol.* 2011; 258(2):277-83.
  19. Soiberman U, Kesler A: Idiopathic intracranial hypertension--what's new in 2012? *Harefuah.* 2013; 152(2):115-8, 121.
  20. Wakerley BR, Tan MH, Ting EY: Idiopathic intracranial hypertension. *Cephalalgia.* 2015; 35(3):248-61.
  21. Wall M, Kupersmith MJ, Kieburz KD, Corbett JJ, Feldon SE, Friedman DI, Katz DM, Keltner JL, Schron EB, McDermott MP; NORDIC Idiopathic Intracranial Hypertension Study Group: The idiopathic intracranial hypertension treatment trial: clinical profile at baseline. *JAMA Neurol.* 2014; 71(6):693-701.
  22. WHO. Physical status: the use and interpretation of anthropometry. Report of a WHO Expert Committee. WHO Technical Report Series 854. Geneva: World Health Organization, 1995.
  23. WHO expert consultation. Appropriate body-mass index for Asian population and its implications for policy and intervention strategies. *The Lancet*, 2004; 157-63.
  24. Yri HM, Jensen RH: Idiopathic intracranial hypertension: Clinical nosography and field-testing of the ICHD diagnostic criteria. A case-control study. *Cephalalgia.* 2014. pii: 0333102414550109.

6/13/2015