Original Article

Hyperbaric oxygen therapy in sudden deafness

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ABSTRACT

HBO (Hyperbaric Oxygen) therapy is used as an adjunct in therapy of wide variety of conditions. The use of HBO therapy in sudden deafness of sensori-neural aetiology is well documented. Between January 98 and July 99, eleven individuals with sudden onset deafness of differing aetiology were referred to the Department of Hyperbaric Medicine at IAM IAF, Bangalore. The treatment schedule in all the four cases was the same i.e. 2.5 ATA x 90 minutes for 5 days in a week. The efficacy of therapy in each case, highlighting the difference in response based on aetiology, is discussed. An attempt is made to identify the factors associated with good response.

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The medical treatment of perceptive deafness (i.e. sudden deafness) and tinnitus has been a disappointment thus far, especially with regard to chronic forms. Neurophysical studies carried out on the cochlea in animals and confirmed by observations in humans prove that degenerative occurrences are largely ascribable to ischaemic damage. The precarious arterial supply of the labyrinth further compounds the problem [1].

Material and methods

Case records of thirteen patients with ENT problems undergoing Hyperbaric Oxygen Therapy between Jan 98 and Jul 99, at IAM were perused. All patients were civilians referred from different hospitals. Of these eleven individuals had sudden onset hearing loss of perceptive type. The details of these patients are given in Tables 1 and 2.

All individuals were subjected to the same format of evaluation, which comprised of :

Initial re-evaluation by the ENT specialist at IAM included a baseline audiometry record. All were subjected to HBO runs at 2.5 ATA for 90 minutes. After 5 exposures repeat audiometry was performed and improvement if any was documented. Improvement in audiometry of >20 dB were given additional 5-7 exposures.

Results

All the cases of acute sensorineural hearing loss did manifest improvement with HBOT (Hyperbaric Oxygen Therapy)

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Table 1

No.	Pt	D	Clinical features			No runs	Post HBOT Symptoms		
			Tinnitus	Vertigo	Nystgmus		Tinnitus	Vertigo	Nystagmus
1	KD	Severe SNHL (L)	+			08	–	–	–
2	SKP	Severe SNHL (R)	+++	++	+*	10	–		
3	VJ	Severe SNHL (R)	+++		–	10	–		
4	M	Severe SNHL (L)	+	–	–	10	–		
5	AAS	Total SNHL (L)	++	–		14	–	–	–
6	VR	Moderate SNHL (L)	++	–		10	–	–	–
7	KSS	Severe SNHL (L)	++	–	–	06	–	–	–
8	SS	Chr SNHL (bilat)	–	–	–	06		–	–
9	VR	Severe SNHL (L)	+++	–	–	10		–	–
10	KKM	Moderate SNHL (L)	–	+	–	07	–		–
11	RM	Total SNHL (R)	++	–	–	13	–		–

Improvement in audiometry records was seen in 8 of the 11 patients (72.72%).

Improvement was a likely outcome when hyperbaric oxygen therapy was administered within 14 days of onset of symptoms. (Eight of the eleven cases reviewed reported to IAM within 2 weeks of onset symptoms i.e. in 72% cases). HBOT was administered as an adjuvant in all cases.

Discussion

With regard to the rationale of hyperbaric oxygen therapy (HBO), the cochlea is indeed an ideal structure as oxygen can be delivered to the inner ear from plasma by diffusion and for the basal turn of the cochlea, by aerial diffusion through the semi-permeable membrane that encloses the fenestra rotunda.

Table 2

Sl no	Patient name	Age	Sex	D	Duration of deafness prior to commencement of HBO	HBO run profile	Inv. before HBO		Post HBO improvement in PTA
							1	2	
1	KD	41	M	Severe SNHL (L)	7 Days	A	+		++
2	SKP	46	M	Severe SNHL (R)	10 days	B	+	–	++
3	VJ	32	M	Severe SNHL (R)	12 days	B	+	–	+
4	M	38	M	Severe SNHL (L)	14 days	B	+	+	+++
5	AAS	53	M	Total SNHL (R)	7 days	B	+	–	++
6	VR	48	M	Moderate SNHL (L)	4 days	B	+		++
7	KSS	24	M	Severe SNHL (L)	7 days	A	+		++
8	SS	20	F	Chr SNHL (bilat)	5-6 years	A	+		
9	VR	33	M	Severe SNHL (L)	3 days	B	+	+	+++
10	KKM	53	M	Moderate SNHL (L)	21days	B	+	–	+
11	RM	46	M	Total SNHL (R)	12 days	B	+	+	++

Average age : 39.45 years SD = 11.15

Legend : A - 2.5 ATA for 90 minutes for 5 - 7 exposures, B - 2.5 ATA x 90 minutes for 7-14 exposures.
1. Audiometry, 2. MRI/CT Scan
+denotes improvement of 10 dB in audiometry
++denotes improvement of 20 dB in audiometry
+++denotes improvement of > 30 dB in audiometry
*Induced by caloric test (cold water)

Table 3

Investigations prior to HBO

Sl No.	Name	Pre-HBO work-up of patients			
		PTA	Imp A	TYMPANOMETRY	ABR
1	KD	+	–	–	–
2	SKP	+	–	–	–
3	VJ	+	–	–	–
4	M	+	–	+	–
5	AAS	+	–	+	–
6	VR	+		–	–
7	KSS	+		+	
8	SS	+	–	–	
9	VR	+	-	+	–
10	KKM	+	–	–	–
11	RM	+		+	+

To function, the auditory system requires a large quantity of oxygen; even more than the brain [1]. Hyperbaric oxygen therapy aims at taking advantage of the effects of the oxygen physically dissolved in the plasma [2]. The inner ear is ideally suited for HBO due to the presence of the eustachian tube which communicates with the nasopharynx. Thereby, it is the middle ear that becomes saturated with oxygen in a hyperbaric environment.

Blood supply to the inner ear is by the internal auditory artery which is a branch of the basilar artery, but is more frequently a branch of the inferior cerebellar artery.

Impairment of hearing follows :

a) Interference with the conduction of sound to the inner ear by anything that hampers the passage of sound waves down the external meatus.

b) Impaired efficiency of the middle ear mechanism.

c) As a consequence of failure of the organ of Corti or its central nervous connections.

In otological practice, any patient with impairment of hearing may be described as being deaf. Deafness may also be classified as slight, moderate, severe or total.

Vascular Deafness is perceptive in type, and is attributed to vascular insufficiency. An anatomical imbalance, with spasms of the internal auditory artery or its branches of distribution, causes impaired functioning of the sensory epithelium. If the vestibular branches of these arteries are involved, vertigo will appear. Ischaemia may result from a spasm of atheromas of the internal auditory artery or its terminal branches. Insufficiency of the cochlear vessels results in anoxia of the ganglion cells, reduced circulation in the stria vascularis and reduced secretion of endolymph, which is also qualitatively abnormal.

There follows an accumulation metabolites and an increase of osmotic pressure within the scala media this causes osmotic transference of fluid from perilymphatic and vascular compartments. The result is deafness.

Clinically, ischaemia of the cochlea occurs as perceptive deafness with other inner ear symptoms. The onset of deafness is gradual and very often intermittent or fluctuating. Fluctuating deafness is commonly found to affect low tones and may mistaken for a middle ear deafness due to intermittent Eustachian obstruction. Deafness may or may not be associated with tinnitus and vertigo.

In order to administer HBO effectively in patients with ear disorders, it is essential to evaluate the status of their hearing by means of the following tests.

a) Pure-Tone Audiometry

b) Impedance Audiometry

c) Tympanometry

d) Auditory Brain Response (ABR) is a free field recording. The ABR latency period consists of five to seven peaks measured within the first milli second.

Tinnitus can be defined as a subjective experience of hearing sounds in the ear that has no basis of reality in the environment. Tinnitus must be distinguished from auditory hallucination.

Tinnitus is a simple sound described in terms of monotone noises such as "ringing, raining" etc whereas hallucination is a complex sensation such as the hearing of "voices, music" etc. Tinnitus is a symptom of a disturbance of the auditory mechanism of the sense organ or its central connection. Tinnitus may be intermittent or continuous and may vary in intensity. The following are possible factors for tinnitus :

a) In the external ear, a foreign body and otitis externa may produce the symptoms.

b) In middle ear, manifestations of media are a common cause.

c) In the inner ear, new bone fenestration in the area of the fistula antefenestrum of the bony or otic capsule in otosclerosis causes a persistent tinnitus. Accoustic trauma from gunfire, Meniere's disease, auditory nerve migraine, vestibular neuritis, and eight nerve tumour may all be associated with tinnitus.

Oxygen and the Auditory Function :

The inner ear vascular system, consisting of the stria vascularis, receives blood flow from the basilar artery and from the inferior cerebellar artery. Sympathetic fibres directly innervate arteries, arterioles and some veins of the inner auditory canal and of the modiolus. The blood supply is affected by the injection of pressure agents which determine an increment of the flow. Hypothermia does bring about a clear reduction of haematic flow [4]. High-intensity stimulation causes a reduction in the vascular calibre, with a reduction in the vessel capacity. Noise of a shot or high frequency and high intensity, leads to decreased functioning that can be permanent. Hyperventilation hypocapnia reduces the direct flow. On the contrary, hypercapnia and hypoxaemia lead to a relative increment of the flow due to a mechanism of direct (high CO_2) or indirect (reduced oxygen partial pressure) vasoplegia.

All these data suggest that intravascular pressure, evaluated as arteriolar oxygen and partial pressure of carbon dioxide, influences the vascular mechanism of the inner ear arterioles, and shows that the cochlear flow is regulated by physiological stress response mechanism.

The efficacy of HBO in hearing loss is the correction of sudden deafness. The hypothesis is that the potassium and sodium pump takes care of the ionic balance and the electrophysiological function of the inner ear is blocked by hypoxaemia; and timely compensation for this hypoxaemia (e.g. noise induced) may correct the functional damage.

Conclusion :

HBO therapy has a strong physiologic rationale for use in sudden deafness. All the eleven cases referred to the Institute showed improvement. HBOT was used as an adjuvant in all cases. Therapeutic evaluation of HBOT can be substantiated only by double blind trials.

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