Effectiveness of Hyperbaric Oxygen Therapy and Systemic Steroids in Idiopathic Sudden Sensorineural Hearing Loss

Systematic Review for the Partial Fulfillment of the Master Degree in Otorhinolaryngology

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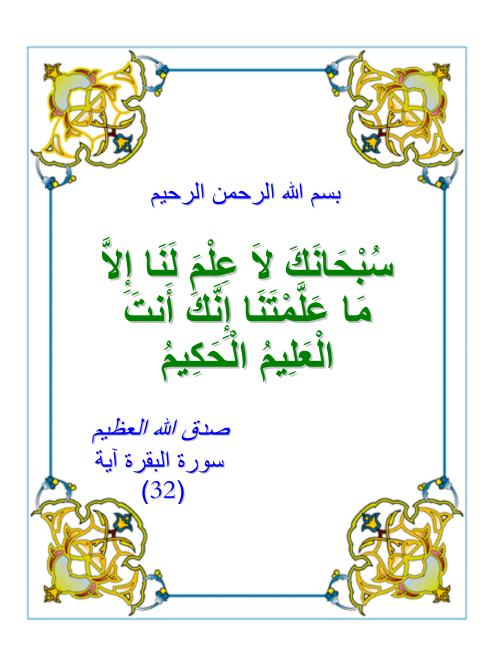
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First and above all, all thanks to **God** the merciful, the compassionate without his help, I could not finish this work.

Words stand short to express my respect and thanks to Professor Dr. Sabry Magdy professor of otorhinolaryngology Ain-Shams University, for his great support and continuous encouragement which have been the main factors to complete this work.

I would like to express my appreciation for Dr. Mohamed Amir, Assistant Professor of otorhinolaryngology Ain-Shams University, for his precious remarks and back up.

I wish to introduce my deep thanks to Dr. Mohamed Shehata, Fellow of Otorhinolaryngology - Ain Shams University for his help, guidance and accurate supervision.

I would like to introduce my great appreciation to all my Professors, Staff and Colleagues.

I am delighted to express my deep gratitude and cardinal thanks to all members of my family, specially My Parents, for their love, care and everlasting support. My overwhelming appreciation, love and respect to the soul of my dad whom I wished to share us these moments.

Special thanks to My Wife, for her great support and care.

All my love for my cute angel Merola Mamdoh

Mamdoh Wasfy

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List of Abbreviations

ATA	Atmosphere absolute
ATP	Adenosine Tri-Phosphate
СВС	Complete Blood Picture
CMV	Cytomegalovirus
CSF	Cerebro-Spinal Fluid
CT	Computed Tomography
DNA	Deoxyribonucleic Acid
НВО	Hyperbaric oxygen
HIV	Human Immunodeficiency Virus
HL	Hearing Loss
ISSNHL	Idiopathic Sudden Sensorineural Hearing Loss
IL1	Interleukin 1
IV	Intravenous
K	Potassium
kHz	Kilo-Hertz
meq	Milliequivalant
mg	Milligram
ml	Milliliter
mV	Millivolt
MRI	Magnetic Resonance Imaging
Na	Sodium
NFkB	Nuclear Factor Kappa B

PG	prostaglandin
PT	patient
PTA	Pure Tone Average
SHL	Sudden Hearing Loss
SDS	Speech Discrimination Score
SNHL	sensorineural hearing loss
SSNHL	Sudden sensorineural hearing loss
TNF	Tumor Necrosis Factor
TTT	treatment

Introduction

Sudden sensorineural hearing loss (SSNHL), also known as acute cochlear labyrinthitis, has long been an enigma for otologists. It is a unilateral process in 98% of patients, with complete spontaneous recovery rates ranging from 32% to 65% cited in the literature (*Horn et al.*, 2005).

SSNHL is an otologic emergency that afflicts approximately 5 to 20 per 100,000 patients annually, although this rate is thought to vary depending on the virulence of annual viruses (*Muzzi et al.*, 2009).

True prevalence of SSNHL may actually be greater than reported rates because patients recovering spontaneously within few hours or days and may never come to medical attention (*Horn et al.*, 2005).

Diagnosis is usually based on patient's history, laboratory, audiologic, imaging tests, and a high suspicion on the part of the otologist. The audiologic definition of SSNHL is hearing loss greater than 30 dB in more than three contiguous frequencies over 3 days or less (*Bennett et al.*, 2005).

Theoretical causes of SSNHL described in the literature include: viral infection, autoimmune syndromes, membrane rupture, Ménière's disease, neoplasm, metabolic, and vascular insults. As early as 1976, Fisch et al. measured perilymphatic

oxygen tension by way of an oxygen electrode inserted in the perilymphatic space and found it to be decreased in some patients with SSNHL. In 1988, Kubo et al. proposed decreased cochlear blood flow and subsequent capillary edema as a possible mechanism of cochlear hypoxia (*Horn et al., 2005*).

Idiopathic sudden sensorineural hearing loss is a diagnosis of exclusion and should be entertained only after a thorough search for known causes (*Haberkamp et al.*, 1999).

Treatment of idiopathic sudden sensorineural hearing loss (ISSHL) is still a problem for the otologist. When a cause of sudden sensorineural hearing loss is clear, the treatment is specific. When a cause cannot be found, treatment of ISSHL is much more controversial (*Topuz et al.*, 2004).

Positive prognostic factors for recovery have included a short interval from onset to initial medical visit, age range 15 to 40 years, absence of vertigo, midfrequency/upsloping audiometric pattern, and early steroid administration (*Horn et al.*, 2005).

Working from a hypothesis of predominantly ischaemic origin, oxygen has a therapeutic advantage in that it easily diffuses across the blood-brain barrier to reach target tissues, and high concentrations are well tolerated. Hyperbaric oxygen therapy has been favored as a treatment for sudden SNHL by many otolaryngologists, and has resulted sometimes in dramatically improved hearing (*Muzzi et al.*, 2009).

Presently, the most commonly used treatment of ISSHL is the systemic administration of steroid hormone, either alone or combined with other treatments. In ISSHL, steroids are thought to increase vascular circulation and to reduce the immune response and presumed inflammation in the cochlea (*Minoda et al.*, 2000).

AIM OF THE WORK

ur study is a systematic review to evaluate the effectiveness of hyperbaric oxygen therapy and systemic steroids in idiopathic sudden sensorineural hearing loss.

ETIOLOGY, PATHOLOGY AND PATHOPHYSIOLOGY OF ISSNHL

Possible Causes of ISSNHL

In the vast majority of patients with ISSNHL, no specific cause can be identified; therefore, the disease is called "Idiopathic" as an exclusion diagnosis (*Fetterman et al.*, 1996).

Theoretical causes of SSNHL described in the literature include viral infection, autoimmune syndromes, membrane rupture, Ménière's disease, neoplasm, metabolic, and vascular insults (*Horn et al.*, 2005).

Some direct and indirect findings support the viral theory of ISSNHL: 1-Temporal association of ISSNHL with active viral upper respiratory illness. 2-Serologic evidence of active viral infection. 3-Histopathologic examination of post mortum human temporal bones. 4-Animal experiments demonstrating virus penetration of inner ear (*Mattox et al.*, 1977).

On the other hand some circumstantial data to support the vascular theory of ISSNHL: 1-Sudden onset. 2-Case reports of sudden deafness with known systemic vascular disease. 3-Histopathologic demonstration of cochlear changes caused by vascular occlusion (*Rauch*, 2004). Certain prothrombotic risk factors and genes have been associated with ISSNHL (*Haynes et al.*, 2007).

Studies have shown that there are alteration in the blood and red cell filterability. And an association between ISSNHL and slow blood flow in the vertebrobasilar system, it has been reported that reduction in cochlear blood flow results in cochlear hypoxia due to edema of the capillary cells (*Banerjee and Parnes*, 2005).

ISSNHL is classified with some of these inner ear disorders as immune mediated. **McCabe**, 1979 first introduced the concept of autoimmune inner ear disease. There may be other immune mediated but not autoimmune causes of ISSNHL (*Freedman et al, 1996*). Also it was found that there is association of ISSNHL with known inner ear diseases as Wegner's disease, Cogan's syndrome and temporal arteritis (*Haynes et al., 2007*).

Cochlear membrane rupture is a cause of sudden hearing loss (SHL), either spontaneous in cases of ISSNHL or occur in a patient with a history of temporal bone trauma and stapedectomy that damage the internal membranes of the cochlea and results in hearing loss. The concept of spontaneous cochlear membrane rupture is different, so there seems to be little objective evidence to support the idea that a substantial percentage of ISSNHL is caused by spontaneous cochlear membrane rupture (*Haynes et al.*, 2007).

Other diseases that can result in sudden hearing loss must be excluded to diagnose ISSNHL as: acoustic neuroma, Meniere's disease, syphilis, diabetes mellitus, multiple sclerosis, mumps, polyarthritis nodosa, Cogan's syndrome and migraines (Fetterman et al., 1996).