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**Original article**

# Association of dyslipidemia to asymptomatic sensorineural hearing loss in Patients with systemic lupus erythematosus

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**Abstract:**

**Background:** Sensorineural hearing loss can be the first manifestation in systemic lupus erythematosus patients. It has been described to occur more frequently in females than in males**.** **Aim of the work:** To detect asymptomatic sensorineural hearing loss (ASNHL) in patients with Systemic Lupus Erythematosus (SLE)and to find its relation to lipid profile and other disease parameters. **Patients and Methods:** a case control study conducted on fifty SLE patients (3 males and 47 females)and twenty matched healthy controls (2males and 18 females). SLE disease activity index (SLEDAI) and systemic lupus international collaborative clinics damage index (SLICC DI) scores were done. All patients were subjected to clinical and laboratory investigations. Lipid profile was done for patients and controls. Otoacoustic emission test, Audiometry, were done for patients and controls. **Results:** The mean age of the patients was (28.46 ± 8.8) and the disease duration was(7.12 ±6.33) years. Triacylglycerol(TAG), Cholesterol, Low density lipoprotein

(LDL) cholesterol were significantly higher in SLE patients compared to controls (p= 0.034, 0.005, 0.002respectively). ASNHL was observed in 10 SLE patients (20%). TAG, Cholesterol, LDL cholesterol were significantly higher in patients with SNHL compared to patients without SNHL (p=0.022, 0.038, 0.020 respectively). Oral ulcers, serositis were significantly more prevalent in patients with SNHL compared topatients without SNHL (p=0.036,0.034 respectively). Disease activity (SLEADI) and damage index scores( SLICC DI) were significantly higher in patients with SNHL compared topatients without SNHL (p=0.028, 0.009 respectively).**Conclusion:** Asymptomatic SNHL was found in minority of SLE patients with an association with high levels of TAG, cholesterol, LDL cholesterol suggesting atherosclerosis as a mechanism.

## 1. Introduction:

SLE is a chronic inflammatory autoimmune disease defined by the production of a wide spectrum of autoantibodies and immune complex deposition, which results in numerous organ damage. The influx of proinflammatory cytokines mediates the inflammatory response **[1].**

Sensorineural hearing loss (SNHL) is considered a type of hearing loss caused by a cochlear lesion or abnormalneural conductionin the eighth cranial nerve or the central auditory processing areas of the brain**[2].**

Hearingand vestibular function can be affected by autoimmune diseases as a primary immune-mediated disease or as a systemic autoimmune disease manifestations**[3].**It has been reported thatSensorineural hearing occurred in symptomatic and asymptomatic SLE patients in recent decades, with an incidence ranging from 8% to 57.5 percent **[4].**Sensorineural hearing loss, on the other hand, has been found to occur regardless age of the disease onset**[5].**LDL cholesterol is one of the five major groups of lipoproteins which transport all fat molecules around the body in the extracellular water**[6].**lipoprotein particles in the circulation vary in size, density, and lipid and apolipoprotein composition, can be classified into numerous groups based on physical and

chemical parameters. The major source of atherosclerotic lipid storage is low-density lipoprotein (LDL), whereas high-density lipoprotein (HDL) is not atherogenic and has an inverse relationship with atherosclerotic CVD risk **[7].**Considering traditional risk factors and chronic inflammation, atherosclerosis occurs earlier and more aggressively in SLE patients.**[8].**Dyslipoproteinemia has been related to atherosclerotic cardiovascular complications in SLE patients **[9],** as seen by high levels of LDL cholesterol, very low-density lipoprotein cholesterol, and triglycerides with low levels of high density lipoprotein HDL cholesterol **[10].** This occurs as a result of autoantibodies and cytokines produced in SLE patients, which which modulate the activity of lipoprotein lipase (LPL), a key enzyme in lipid metabolismapoA-I synthesis [11].paraoxonase (PON), and lecithin cholesterol acyltransferase (LCAT) activity all decrease during inflammation, resulting in impaired HDL function; HDL from SLE patients is more proinflammatory than HDL from HCs **[12].** Anti-Ro positivity has beenrelated to the high incidence of low HDL in lupus patients, which may increase the risk of cardiovascular diseases **[13].**

The objectives of this study was to detect asymptomatic sensorineural hearing loss (ASNHL) in SLE patients and find its relation to lipid profile and other clinical parameters.

## 2. Patients and Methods:

A case control study, conducted on 50 SLE patients (47 females and 3 males), selected from Rheumatology &Rehabilitation Department, Beni-Suef university hospital. SLE patients werediagnosed according to Systemic Lupus International Collaborating Clinics (SLICC) classification criteria **[14]**and 20 controls matched in age and sex (18 females and 2 males**).**

**Inclusion criteria:**

- Age of SLE patients was ranged from 16 to 59 years old
- Both males and females were included

**Exclusion criteria**: Patients with diabetes mellitus and other rheumatological diseases.

SLE patients were subjected to full clinical examination and laboratory investigations. Patients and controls were subjected to measurement of serum concentrations of total cholesterol, Triacylglycerol(TAG), high and low density lipoprotein(HDL and LDL)cholesterol andvery low density lipoprotein(VLDL) cholesterol using automated enzymatic analysis(Beckman coulter, AU480, USA)after over-night fasting (from 8 to 12 hours) except for water. SLE disease activity was evaluated by the systemic lupus erythematosus disease activity index

(SLEDAI) **[15].**The SLE disease severity was assesed by the Systemic Lupus International Collaborative clinics/America Collage of Rheumatology (SLICC/ACR) damage index**[16].**

**Audiological Evaluation:**

**All patients and controls were subjected to audiological evaluation:(a)** Tonal audiometry, the frequency range 0.25- 8 kHz,Intera-coustics in a sound treated room with TDH 39 earphones. Speech audiometry including speech reception threshold (SRT) using, Arabic spondee words **[17]** and word discrimination score (WDS) using, Arabic phonetically balanced (PB) words **[18]**.**(b)**Otoacoustic emission testing **[19]**.

**Statistical analysis:** The collected data were coded then entered and analyzed using the SPSS version 22 (Statistical package for social science). Descriptive statistics was done for categorical variables by frequency and percentage, and for numerical variables in the form of mean and standard deviation (mean ± SD).

**Ethical Considerations**: The study was approved by the ethical committee of the Faculty ofMedicine, Beni-Suef University**.**Informed written consent was obtained from all participants before sharing in the study, after explaining the objectives of the work**.**

## 3. Results:

SLE patients distributed as47 (94%) females and 3 (6%) males and, their mean age was (28.46 ± 8.8) years (17-57years), and disease duration (7.12 ±6.33) years(1-25years).The other group was 20 healthy controls distributed as18 (90%) females and 2 (10%) males and, their main age was (31.65 ± 9.8) years(19-51years); with no statistically significant difference among cases and controls regarding sex and age.The characteristics of the SLE patients are illustrated in**(Table 1)**.

TAG, Cholesterol, Low density lipoprotein (LDL) cholesterol were significantly higher in SLE patients compared to controls (p= 0.034, 0.005, 0.002respectively)**(Table 2)**.

All the studied healthy control participants had normal Hearing profile. Asymptomatic sensorineural hearing loss (ASNHL) was observed in 10 SLE patients (20%). Five (10%) of the 10 patients had unilateral SNHL while the others (10%) had bilateral SNHL. Mild SNHL was the most common severity in both ears,with 8 patients (16%) having mild SNHL, one patient (2%) having severe SNHL in the right ear, and three patients (6%) having mild SNHL, one patient (2%) having moderate SNHL, and two patients (4%) having severe SNHL in the left ear

Regarding the emission test; 12 patients (24%) required referral while 38 patients (76%)

passed the test in the right ear, 19 patients (38%) required referral while 31 patients (62%) passed the test in the left ear **(Table3)**.Regarding to different frequencies; medium(1000-2000HZ) and low frequencies (250-500HZ) as shown in **(figure 1).**

Oral ulcers, serositis were significantly more prevalent in patients with SNHL compared to patients without SNHL (p=0.036, 0.034 respectively). Disease activity (SLEADI) and damage index(SLICC DI) scores were significantly higher in patients with SNHL compared to patients without SNHL (p=0.028, 0.009 respectively) (**Table 4).**

**Regarding SLE patients:** TA Gwas significantly higher in those with SNHL compared to those without SNHL (189.30 ±125.18 vs. 117.86 ±73.07respectively; p= 0.022). Cholesterol was significantly higher in those with SNHL compared to those without SNHL (216.30 ±46.07 vs. 177.23±60.14respectively; p= 0.038). LDL cholesterol was significantly higher in those with SNHL as compared to those without SNHL. (132.10 ±23.35 vs. 106.43 ±46.74respectively; p=0.020) (**Table 5).**

the numbers of patients with hearing losses were slightly higher among high frequencies(4000-8000HZ) as compared to

**Table 1:** Characteristics of the systemic lupus erythematosus patients**:**

| **Parameter**<br>**mean ±SD or n(%)** | **SLE cases**<br>**(n=50)** |
|---|---|
| **Gender;**<br>**Male**<br>**Female**<br>**Age (years)** | <br>3(6.0)<br>47(94.0)<br>28.46 ± 8.8 |
| **Disease duration (years)** | 7.12±6.33 (1-25) |
| ***Clinical Symptoms:*** | |
| **Malar rash** | 49 (98.0) |
| **Discoid rash** | 5 (10.0) |
| **Photosensitivity** | 46 (97.9) |
| **Oral ulcer** | 37 (74.0) |
| **Arthritis** | 42 (84.0) |
| **Renal disorders** | 23 (46.0) |
| **Hematologic** | 16 (32.0) |
| **Serositis** | 25 (50.0) |
| **Alopecia** | 26(50.0) |
| **Myositis** | 1 (2.0) |
| **Fever** | 9(18.0) |
| **Neurologic** | 9 (18.0) |
| **Vasculitis** | 2(4.0) |
| ***Laboratory investigations*:** | |
| **ESR (mm/hr)** | 60.6600±33.32500 |
| **CRP (mg/L)** | 15.5664± 14.62573 |
| **Hemoglobin (gm/dl)** | 11.1140 ± 1.76057 |
| **TLC /L** | 10.9280 ± 23.39226 |
| **Platelets /µl** | 255.3200±108.30841 |
| **C3(mg/dl)** | 99.00 ± 40.84 |
| **C4 (mg/dl)** | 19.16 ± 12.79 |
| **Urea (U/L)** | 27.0400 ± 17.45193 |
| **ALT (mmol/L)** | 19.4800 ± 8.29861 |
| **Positive ANA** | 50 (100.0) |
| **Positive anti-dsDNA** | 8 (16.00) |
| **Positive lupus anti-coagulant** | 23 (46.00) |
| ***Medications*:** | |
| **Corticosteroids** | 50 (100.0) |
| **Azathioprine** | 26 (52.0) |
| **Hydroxycloroquine** | 44 (88.0) |
| **Scoring systems:** | |
| **SLEDAI**<br>**SLICC DI** | 16.96 ±7.5<br>0.54 ±0.65 |

SLE: systemic lupus erythematosus, *ESR: Erythrocyte Sedimentation Rate, CRP: C - reactive protein, HG: Hemoglobin Concentration, TLC: Total Leukocyte Count, PLT: Platelets, ALT: Alanine Aminotransferase, C3-C4: Complement. ANA: Antinuclear Antibody, ds-DNA: Anti-double strand DNA. SLEDAI: Systemic Lupus Erythematosus Disease Activity Index, SLICC DI: The Systemic Lupus Erythematosus International Collaborating Clinics Damage Index.*

**Table 2: Comparison of lipid profile between SLE patients and healthy controls:**

| | | Minimum | Maximum | Mean ± SD | p-value |
|---|---|---|---|---|---|
| **TAG (mg/dl)** | SLE Cases | 29.00 | 526.00 | 132.16 ± 89.2 | **0.034*** |
| | Healthy Control | 58.00 | 148.00 | 88.10 ± 23.8 | |
| **Cholesterol (mg/dl)** | SLE Cases | 75.00 | 370.00 | 185.04 ± 59.3 | **0.005*** |
| | Healthy Control | 122.00 | 172.00 | 146.05 ± 14.8 | |
| **LDL (mg/dl)** | SLE Cases | 54.00 | 272.00 | 111.56 ± 44.2 | **0.002*** |
| | Healthy Control | 54.00 | 98.00 | 78.45 ± 11.7 | |
| **HDL (mg/dl)** | SLE Cases | 21.00 | 85.00 | 45.46 ± 13.1 | 0.163 |
| | Healthy Control | 33.00 | 62.00 | 50.00 ± 9.3 | |

*LDL: Low-density lipoprotein, HDL: High-density lipoprotein, TAG: Triglycerides *p-value ≤0.05 is considered statistically significant***.**

**Table 3: Hearing Profile among studied SLE patients; (N= 50):**

| | | | N (%) |
|---|---|---|---|
| Laterality | | NAD | 40 (80.0) |
| | | Unilateral | 5 (10.0) |
| | | Bilateral | 5 (10.0) |
| Right Ear | Hearing Profile | Normal | 41 (82.0) |
| | | Mild SNHL | 8 (16.0) |
| | | Moderate SNHL | 0 (0.00) |
| | | Severe SNHL | 1 (2.0) |
| | Emission test | Pass | 38 (76.0) |
| | | Refer | 12 (24.0) |
| Left Ear | Hearing Profile | Normal | 44 (88.0) |
| | | Mild SNHL | 3 (6.0) |
| | | Moderate SNHL | 1 (2.0) |
| | | Severe SNHL | 2 (4.0) |
| | Emission test | Pass | 31 (62.0) |
| | | Refer | 19 (38.0) |

*NAD= no abnormalities detected,*

*SNHL:Sensoryneural hearing loss.*

**Table 4: Relation of asymptomatic sensory neural hearing loss to characteristics of SLE patients:**

| | **No SNHL N= 40(%) or mean±SD** | **SNHL N= 10(%)or mean±SD** | **p-value** |
|---|---|---|---|
| **Gender;** | | | |
| Male | 3 (7.5) | 0 (0.00) | 0.372 |
| Female | 37 (92.5) | 10 (100.0) | |
| **Age; (years)** | | | |
| mean ±SD | 27.68 ±8.5 | 32.5 ±9.6 | 0.124 |
| **Disease Duration; (years)** | | | |
| mean ±SD | 6.95 ±6.31 | 7.80 ±6.71 | 0.708 |
| *Clinical Symptoms:* | | | |
| **Malar rash** | 39 (97.5) | 10 (100.0) | 0.614 |
| **Discoid Rash** | 5 (12.5) | 0 (0.00) | 0.239 |
| **Photosensitivity** | 30 (75.0) | 8 (80.0) | 0.741 |
| **Oral Ulcer** | 27 (67.5) | 10 (100.0) | 0.036* |
| **Arthritis** | 35 (87.5) | 7 (70.0) | 0.243 |
| **Renal Disorders** | 17 (42.5) | 6 (60.0) | 0.480 |
| **Hematologic** | 13 (32.5) | 3 (30.0) | 0.880 |
| **Serositis** | 17 (42.5) | 8 (80.0) | 0.034* |
| **Alopecia** | 21 (52.5) | 5 (50.0) | 0.887 |
| **Myositis** | 1 (2.5) | 0 (0.0) | 0.614 |
| **Fever** | 6 (15.0) | 3 (30.0) | 0.269 |
| **Neurologic** | 6 (15.0) | 3 (30.0) | 0.269 |
| **Vasculitis** | 1 (2.5) | 1 (10.0) | 0.279 |
| *Laboratory investigations* : | | | |
| **ESR; (mm/hr)** | 60.60 ±34.88 | 60.90 ±27.83 | 0.980 |
| **CRP (mg/L)** | 14.05 ±13.97 | 21.63 ±16.36 | 0.144 |
| **Hg; (gm/dl)** | 10.97 ±1.81 | 11.69 ±1.51 | 0.251 |
| **TLC; /L** | 6.36 ±2.64 | 6.30 ±1.4 | 0.948 |
| **PLT; /µl** | 264.03 ±111.96 | 243.80 ±69.75 | 0.590 |
| **ALT; (mmol/L)** | 20.28 ±8.09 | 16.30 ±8.77 | 0.178 |
| **UREA; (U/L)** | 27.14 ±18.58 | 26.60 ±12.59 | 0.931 |

| | | | |
|---|---|---|---|
| **ds-DNA** | 17 (42.5) | 6 (60.0) | 0.480 |
| **Lupus Anti-coagulant** | 6 (15.0) | 2 (20.0) | 0.700 |
| **C3 (mg/dl)** | 98.65 ±43.66 | 100.41 ±28.64 | 0.904 |
| **C4 (mg/dl)** | 19.38 ±13.68 | 18.32 ±8.91 | 0.818 |
| Scoring systems: | | | |
| **SLEDAI** | 15.40 ±6.18 | 23.20 ±9.30 | 0.028* |
| **SLICC DI** | 0.38 ±0.49 | 1.20 ±0.79 | 0.009* |

*ESR: Erythrocyte Sedimentation Rate, CRP: C - reactive protein, HG: Hemoglobin Concentration, TLC: Total Leukocyte Count, PLT: Platelets, ALT: Alanine Aminotransferase, C3-C4: Complement. ANA: Antinuclear Antibody, ds-DNA: Anti-double strand DNA. SLEDAI: Systemic Lupus Erythematosus Disease Activity Index, SLICC: The Systemic Lupus Erythematosus International Collaborating Clinics Damage Index. p-value ≤0.05 is considered statistically significant.*

**Table 5: Association of asymptomatic sensory neural hearing loss to lipid profile of SLE patients:**

| | **No SNHL N= 40** | **SNHL N= 10** | **p-value** |
|---|---|---|---|
| **TAG (mg/dl)** | 117.86 ±73.07 | 189.30 ±125.18 | **0.022*** |
| **Cholesterol (mg/dl)** | 177.23 ±60.14 | 216.30 ±46.07 | **0.038*** |
| **LDL (mg/dl)** | 106.43 ±46.74 | 132.10 ±23.35 | **0.020*** |
| **HDL (mg/dl)** | 44.94 ±13.71 | 47.31 ±10.64 | 0.613 |

**p-value ≤0.05 is considered statistically significant*

*LDL: Low-density lipoprotein, HDL: High-density lipoprotein, TAG: Triacylglycerol.*

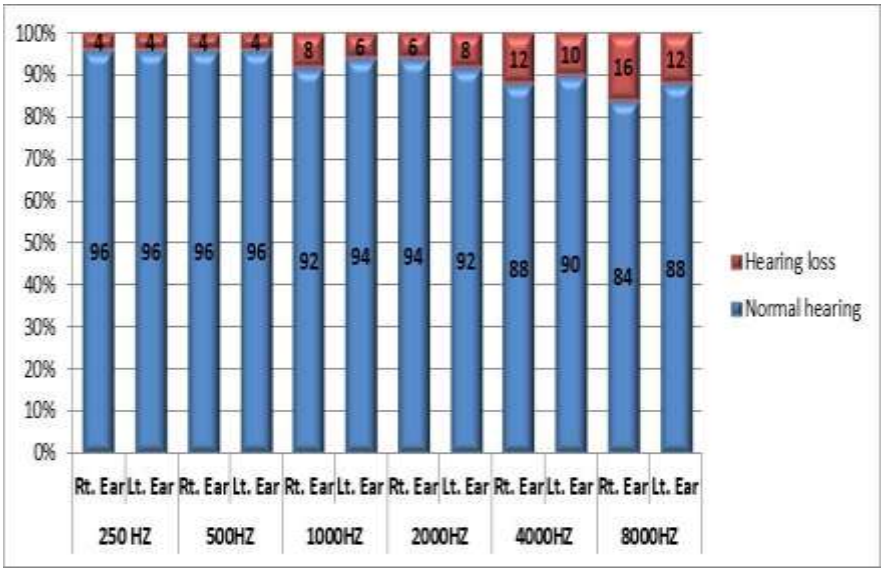


**Figure (1): PTA thresholds in right and left ears among studied SLE patients.**

## 4. Discussion:

Sudden sensorineural hearing loss (SSNHL) is defined as a sudden loss of hearing, usually unilateral, of more than 30 dB in 3 contiguous frequencies of the tonal audiometry. SSNHL estimates an incidence ranging from 5 to 20 per 100.000 people per year. In approximately 75% of cases, a cause cannot be identified. However, it could be a clinical manifestation of Systemic lupus erythematosus (SLE) and Antiphospholipid Syndrome (APS). **[20].** It has been described to occur more frequently in females than in males**[21].**

An early atherosclerosis in small blood vessels probably contributes to hearing loss in SLE**[22].** Regarding SLE patients in this study,SLE patients were distributed as47 (94%) females and 3 (6%) males and. Healthy controls were distributed as18 (90%) females and 2 (10%) males with no statistically significant differences in sex and age, between cases and controls.

Our findings were in agreement with **Roverano's et al[23],**their study included(31femalepatients, 25female healthy participants).

SLE is almost 9 times more common among females than males over the course of life, in the reproductive age period**[24].**After 3 years of the onset of the SLE,the occurrence of dyslipidemia had increasedto 60% or even higher **[25].**High cholesterol levels was estimated in the Systemic Lupus International Collaborating clinics' cohort of 918 SLE patients **[25].**

TAG, Cholesterol, (LDL) cholesterol werehigherin SLE patients compared to controls in this study with a statistically significant diffrence.

In both the right and left ears, the hearing profiles of the SLE patients and controls were examined using (Emission test, Audiometry, and Tympanogram). All of the healthy control subjects in this study had a normal hearing profile.

All of our SLE patients and controls had a type (A) tympanogram, which meansthere is no abnormalities in the middle ear.

In our study, Asymptomatic SNHL was found in ten SLE patients (20 percent). Five of the ten patients (10%) had unilateral SNHL, while the other 10% had bilateral SNHL. In patients with unilateral SNHL, four patients (8%) had hearing loss in the right ear and one patient (2%) had hearing loss in the left ear. Mild SNHL was the most common severity in both ears. In our study, the number of individuals with hearing loss was slightly higher in the high frequencies compared to the middle and low frequencies.

Otoacoustic emission testing (OAES); We classified OAES results as pass or refer in our study; pass for those with normal outer hair cells, and refer for those with abnormal outer

hair cells who are predicted to have hearing loss in the future due to OAEs' predictive ability.

In agreement with **Ferrari et al[22]**, studying 89 female SLE patients, asymptomatic sensorineural hearing loss was found in 14 of the SLE patients, The mild severity was the mild SNHL. The majority of their patients had high-frequency hearing loss. In a study of 89 symptomatic SLE patients, **Batuecas-Caletroet al[26]**, discovered 17 patients (19%) with hearing loss, mostly in high Frequencies. Regarding the clinical manifestations, SNHL had a statistically significantassociationwithOral ulcers and serositisin the present study.

We disagree with **Mokbel et al[27]**, enrolling 20 female SLE patients and showed that lower hearing levels was found inlupus nephritis.. Our findings contrasted with those of **Gomides et al[28],** who enrolled 45 female SLE patients and 45 healthy women (15.6 percent of patients had SNHL) in a study that found no significant differences in all clinical symptoms between non-SNHL and SNHL patients.

In the current study,there was a statistically significant differences in disease activity (SLEADI) and (SLICC DI) scores in patients withSNHL and those without SNHL.

Our study was on the contrary with **Abbasi et al, [4]** who studied 45 SLE patients (11, 1% of patients complained from hearing loss), no significant relationshipwas found between hearing loss and disease activity (SLEDAI) and damage (SLICC DI) scores.

SNHL had a statistically significant relation with TAG, Cholesterol, and LDL cholesterol in the present study**.**

The association between hypercholesterolemia and hearing loss was revealed in a study by **Andreeva et al [29],** (They studied 4,407adults [59.6 percent] women).

In contrast, **Mokbelet al[27]**, found a non-significantly statistical association between SNHL and lipid profile.

The proposed mechanism of dyslipidemia causing SNHL included lipidosis, microvascular disease, metabolic hypoxia, embolism, hereditary hyperviscosity and atherosclerosis of cochlear blood vessels. An increase in plasma level of LDL cholesterol and total cholesterol (TC) are major risk factor for the development of atherosclerosis**[30].**

Normal LDL does not trigger foam cell production when incubated with cultured macrophages or smooth-muscle cells. When LDL undergoes lipid peroxidation, however, it becomes a ligand for scavenger receptors found on endothelial cells and macrophages. The development of cholesterol-laden foam cells is caused by the uptake of oxidised lipoproteins by these receptors in macrophages.

In addition to foam cell formation, oxidized LDL acts in the vessel wall to stimulate the secretion of cytokines and growth factors by endothelial cells, smooth muscle cells, and monocyte-derived macrophages. The consequence is recruitment of more monocytes to the lesion and proliferation of smooth-muscle cells, which synthesize and secrete increased amounts of extracellular matrix, such as collagen thereby causing narrowing of labyrinthine vessels. This will cause reduction in oxygen (hypoxia) and nutrient supply to the inner ear with resultant SNHL **[31]**.

In present study SNHL among studied SLE patients was not related to patient's gender, age and disease duration so we recommended larger sample size and on large geographical scale for better results.

## 5. Conclusion and recommendations:

Asymptomatic SNHL was found in minority of SLE patients with an association withhigh levels of TAG, cholesterol, LDL cholesterol suggesting atherosclerosis as a mechanism. Furthermore, SLE patients need regular audiometry studies and lipid profile analysis for earlier and better treatment.

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