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Original Article

Prognostic Value of Fibrinogen/HDL Ratio in Idiopathic Sudden Sensorineural Hearing Loss

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OBJECTIVE: Vascular causes are most frequently implicated in idiopathic sudden sensorineural hearing loss (ISSHL), and the process has been reported to be associated with fibrinogens and plasma lipoproteins. The purpose of this study was to determine the relationship between serum fibrinogen and high-density lipoprotein (HDL) levels, and the ratios of fibrinogen/HDL and monocyte/HDL, and ISSHL.

METHODS: The present retrospective study included 116 patients with ISSHL. Treatment outcomes was categorised four groups according to Siegel's criteria as follows: complete recovery (Group1), partial recovery (Group 2), slight recovery (Group 3), or no improvement (Group 4). Fibrinogen, HDL and monocyte levels of patients were assessed through routine blood analyses, and fibrinogen/HDL and monocyte/HDL ratios were calculated for each patient.

RESULTS: Fibrinogen values were significantly lower in Group 1 than in Groups 3 and 4, whereas the values were also significantly lower in Group 2 than in Group 3. HDL values in Group 1 were significantly higher than in the other groups. Fibrinogen/HDL values were significantly lower in Group 1 than in Groups 3 and 4. However, there was no significant difference among the groups with regard to monocyte/HDL ratios.

CONCLUSION: To our knowledge, this is the first study investigating the fibrinogen/HDL ratio in ISSHL in the literature. Although fibrinogen/HDL ratio might be a useful prognostic indicator for hearing recovery in patients with ISSHL, further studies with a larger patient population is required to confirm its clinical practicability and reliability.

KEYWORDS: Sudden hearing loss, fibrinogen, high-density lipoprotein, monocytes, prognosis

INTRODUCTION

Sudden sensorineural hearing loss (SSHL) is an otological emergency and has been defined as acute-onset sensorineural hearing loss (SHL) with a hearing threshold of ≥30 dB for 3 consecutive test frequencies occurring within a 72-hour period [1]. The incidence of SSHL varies and has been reported to be 5-20 per 100,000 [2]. The etiology of SSHL has not yet been clearly identified. Most cases are considered to be idiopathic, and vascular theory is one of the most widely accepted causes [3]. Some researchers have indicated that the frequency of idiopathic sudden sensorineural hearing loss (ISSHL) increases in hypercoagulability cases, including sickle-cell anemia and macroglobulinemia; in diseases with increased blood viscosity, such as polycythemia vera and fat embolism; in atherosclerosis and mitral valve diseases; and in systemic vascular diseases such as diabetes mellitus (DM), Buerger's disease, and collagen tissue disease [3,4]. High fibrinogen and high-density lipoprotein (HDL) levels were correlated with the development of ISSHL [5].

In this study, we aimed to evaluate the relationship of serum fibrinogen and HDL levels and the ratios of fibrinogen/HDL and monocyte/HDL with ISSHL to determine their potential prognostic value in ISSHL.

MATERIALS AND METHODS

Patient Selection

This retrospective study included 116 patients who presented to our clinic between January 2014 and May 2016 and were treated for ISSHL diagnosis. SSHL was defined as SHL with a hearing threshold of ≥30 dB for 3 consecutive test frequencies occurring within a 72-hour period. The inclusion criteria were as follows: age, 18-65 years; <7 days between the occurrence of hearing loss and the commencement of treatment; and no prior treatment for ISSHL before enrolment in this study.

This study was presented at the 14th Turkish Rinology, 6th National Otology-Neurotology, 2nd National Head and Neck Congress, 28 April-1 May 2018, Antalya, Turkey

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All patients received 1 mg/kg/d systemic methylprednisolone (Mustafa Nevzat Ilac Sanayi A.S., Istanbul, Turkey) along with a gastroprotective medicine lansoprazole (Nobel Ilac Sanayi A.S., Istanbul, Turkey), 1×30 mg of the oral proton pump inhibitor. The steroid dosage was decreased by 20 mg once in 3 days. Intratympanic dexamethasone (ITS) (Deva Holding A.S., Istanbul, Turkey) and/or hyperbaric oxygen (HBO) therapy were administered as a salvage therapy for patients who responded poorly to the systemic steroid treatment. ITS treatment was applied as 5 doses every other day. After inserting a ventilation tube into the tympanic membrane, approximately 0.5-0.7 cc dexamethasone was injected transtympanically into the cavum tympani. The patients then rested in the supine position for 30 min in a manner that ensured otological positioning by rendering the head 45° to the healthy ear. The patients were instructed to swallow or talk as little as possible during this resting time. The HBO treatment was administered for 10 days as a single session per day, which lasted for 120 min at 2.5 ATA (1 ATA=760 mmHg). The patients who benefited from the treatment during audiometry follow-ups were additionally given HBO treatment as a single session per day for 10 days.

We excluded patients who did not comply with the definition of ISSHL (retrocochlear pathology, ototoxicity, perilymph fistula, and acoustic trauma); had a prior history of ISSHL; underwent prior surgery of the affected ear; had any type of ear pathology (tympanic membrane perforation or chronic otitis media); had a history of autoimmune disease; experienced sudden hearing loss owing to ototoxic medication use; or had a history of DM, hyperlipidemia, renal failure, or cardiovascular disease. This study was approved by the Health Science University, Kayseri Training and Research Hospital (52332816/7/23.8.2017) and conforms to the principles of the Declaration of Helsinki.

Audiological Evaluation

All patients were diagnosed after a complete ear-nose-throat examination followed by pure-tone audiometry. Pure-tone thresholds of 250-8,000 Hz and speech discrimination scores were evaluated. The pure-tone average was calculated as the average hearing threshold at frequencies of 500; 1,000; 2,000; and 4,000 Hz. Audiological examinations were performed in soundproof cabins using a brand audiometry device (AC 40; Interacoustics, Denmark) by the same audiologist group. The patients were categorized into 4 groups according to the Siegel's criteria (Table 1) based on their hearing thresholds at the beginning and at the end of the third month as follows: complete recovery (Group 1), partial recovery (Group 2), slight recovery (Group 3), or no improvement (Group 4).

MAIN POINTS

- The fibrinogen/HDL ratios were significantly lower in complete recovery group and there was a significant correlation between the fibrinogen/HDL ratio and hearing recovery.
- The fibrinogen/HDL ratio might be a useful prognostic indicator for hearing recovery in patients with ISSHL,
- There was no significant correlation between the monocyte/HDL ratio and hearing recovery.

Table 1. The study groups according to the Siegel's criteria

Type	Hearing recovery
I. Complete recovery (Group 1)	Final hearing better than 25 dB
II. Partial recovery (Group 2)	More than 15 dB gain, final hearing 25–45 dB
III. Slight recovery (Group 3)	More than 15 dB gain, final hearing poorer than 45 dB
IV. No improvement (Group 4)	Less than 15 dB gain

Biochemical and Hematological Evaluation

Before the treatment began, all patients were evaluated for complete blood count, chemistry panel, fibrinogen test, and HDL level determination. The fibrinogen/HDL and monocyte/HDL ratios were also calculated for each patient.

Statistical Analysis

Data were analyzed using the Statistical Package for the Social Sciences (SPSS), version 22; (IBM Corp.; Armonk, NY, USA). The normality of data distribution was analyzed using the Kolmogorov-Smirnov test. Data were expressed as mean±standard deviation. The chi-square test was used to compare categorical variables, whereas the quantitative data of the groups were compared by the one-way analysis of variance followed by the Tukey's honestly significant difference as the post hoc test. The Pearson correlation coefficient was determined to establish the correlation between the fibrinogen and HDL levels and the fibrinogen/HDL ratio, with the study groups categorized according to the Siegel's criteria. A p value of <0.05 was considered statistically significant for all comparisons.

RESULTS

Overall, 116 patients and 122 ears were included in this study. The bilateral incidence rate was 5.1%. Of the affected 122 ears, 64 were right (52.4%) and 58 were left (47.6%) ears. The difference in laterality was not statistically significant (p=0.97). There were 30, 23, 18, and 45 patients in Groups 1, 2, 3, and 4, respectively. There was no significant difference among the groups in terms of age or gender. The comparisons of demographic data and values of laboratory parameters of the groups are shown in Table 2.

The fibrinogen levels were significantly lower in Group 1 than in Groups 3 and 4, whereas there was no significant difference between Group 1 and Group 2. In addition, the fibrinogen levels were significantly lower in Group 2 than in Group 3 (Table 3). There were no further significant differences in terms of fibrinogen levels among the groups. The HDL levels in Group 1 were significantly higher than those in all the other groups. However, the difference among the other groups was not statistically significant (Table 4). The fibrinogen/HDL ratios were significantly lower in Group 1 than in Groups 3 and 4, whereas the difference among the other groups was not statistically significant (Table 5). There was no significant difference among the groups regarding their monocyte/HDL ratios.

According to the Pearson correlation coefficient, there was a significant correlation between the fibrinogen and fibrinogen/HDL levels and the study groups (p=0.003 and r=0.273, p=0.003 and r=0.272, respectively), whereas there was no correlation for the HDL levels.

Table 2. Comparison of demographic data and laboratory parameters of the groups

	Group 1 (n=30)	Group 2 (n=23)	Group 3 (n=18)	Group 4 (n=45)	p
Age (y)	39.4	47	46.3	46	0.124
Gender (M/F)	21/9	13/10	12/6	22/23	0.102
Fibrinogen (mg/dL)	280.6±65.2	297.7±90.2	388.6±84.2	341.8±122.4	0.001
HDL (mg/dL)	51.8±9.3	44.1±8.6	47±11.2	48±9.4	0.034
Monocyte (10^3/μL)	0.46±0.23	0.45±0.15	0.51±0.14	0.42±0.29	0.176
Fibrinogen/HDL	5.53±1.37	7.04±2.73	8.62±2.47	7.55±3.63	0.002
Monocyte/HDL	0.0091±0.0052	0.0108±0.0045	0.0113±0.0038	0.0095±0.0072	0.089

HDL: high-density lipoproteins; F: female; M: male; n: number of patients

Table 3. P values derived from the statistical comparison of fibrinogen levels among the groups

	Group 1 (n=30)	Group 2 (n=23)	Group 3 (n=18)	Group 4 (n=45)
Group 1	NA	0.922	0.002	0.046
Group 2	0.922	NA	0.021	0.304
Group 3	0.002	0.021	NA	0.325
Group 4	0.046	0.304	0.325	NA

Analysis of variance/Tukey's honestly significant difference.
NA: not applicable; n: number of patients

Table 4. P values derived from the statistical comparison of high-density lipoprotein levels among the groups

	Group 1 (n=30)	Group 2 (n=23)	Group 3 (n=18)	Group 4 (n=45)
Group 1	NA	0.023	0.326	0.322
Group 2	0.023	NA	0.784	0.406
Group 3	0.326	0.784	NA	0.982
Group 4	0.322	0.406	0.982	NA

Analysis of variance/Tukey's honestly significant difference.
NA: not applicable; n: number of patients

Table 5. P values derived from the statistical comparison of fibrinogen/high-density lipoprotein ratios among the groups

	Group 1 (n=30)	Group 2 (n=23)	Group 3 (n=18)	Group 4 (n=45)
Group 1	NA	0.228	0.002	0.017
Group 2	0.228	NA	0.298	0.896
Group 3	0.002	0.298	NA	0.538
Group 4	0.017	0.896	0.538	NA

Analysis of variance/Tukey's honestly significant difference.
NA: not applicable; n: number of patients

DISCUSSION

Idiopathic sudden sensorineural hearing loss is one of the rare pathologies that can be treated as a part of sensorineural-type hearing losses. Although its etiopathogenesis is not clearly known, it has a relatively good prognosis. The spontaneous recovery rate of ISSHL, independent of treatment, is 32%–65% [6]. Therefore, the efficiency of treatment protocols and the evaluation of prognostic factors have become more demanding. Although several causes, such as infectious, immunological, and inflammatory factors, are associated with the etiopathogenesis of ISSHL, one of the most emphasized theories is the vascular theory. The cochlea is supplied with blood from 2 terminal arteries with very small diameters; therefore, the cochlea is susceptible to vascular trauma because it has no collateral support. According to the vascular theory, hearing loss can be caused by a sudden vascular hemorrhage, an occlusion originating from emboli, vasospasm, or blood viscosity [7-10]. Some studies also purport that ischemic risk factors, such as smoking, hypertension, and hyperlipidemia, are also risk factors for ISSHL, and 35%–40% of patients with ISSHL have hyperlipidemia [4,11]. Many studies have reported that increased cholesterol and triglyceride levels are more prevalent among patients with ISSHL [12]. Some of the experiments performed using animal models have also revealed edema in the cochlear structures of animals [13].

Fibrinogen is a positive acute-phase protein, and its secretion increases during inflammatory processes. In previous studies, fibrinogen has been associated with cardiovascular disease and several types of cancer such as kidney and lung carcinoma [14]. Fibrinogen has a strong correlation with plasma viscosity [15]. In particular, an increased inclination for aggregation has adverse effects on blood-flow velocity in association with fibrinogen levels [16]. Therefore, decreased blood-flow velocity in veins that is occluded owing to the increased plasma viscosity accelerates the development process and increases thrombotic inclination [17]. An elevated plasma fibrinogen level elevates the inclination for atherosclerosis owing to both increased plasma viscosity and the thrombotic triggering effect of fibrin as a result of degradation. Many studies have shown that this process leads to ISSHL when it occurs in the inner ear [18,19]. A study by Park et al. [20] has also reported that increased fibrinogen levels are associated with poor prognosis. Another study has reported that increased fibrinogen levels measured during the first week of disease were inversely proportional to hearing improvement [21]. In this study, the fibrinogen levels were significantly lower in Group 1 than in Groups 3 and 4. The fibrinogen levels were significantly lower in Group 2 than in Group 3.

Oxidative stress is a key risk factor for atherosclerosis, and such an effect can be caused by the formation of pro-inflammatory and pro-atherogenic oxidized low-density lipoprotein (LDL) in the artery intima [22]. These peroxidation products are important, because they

show a pro-inflammatory effect and cause endothelium dysfunction. HDL exerts its antioxidant effect by decreasing LDL oxidation [23]. This effect is ensured by many diverse enzymes and structural proteins. Elevated LDL and total cholesterol (TC) levels along with decreased HDL levels and an increased TC/HDL ratio are associated with vascular damage and thrombosis [24]. Increased TC, triglyceride, and non-HDL levels are also compatible with a level of hearing loss and poor prognosis [25]. In a systematic review and meta-analysis, Chang et al. [26] found no evidence of serum lipids being associated with SSHL but they could not definitively rule out such an association. In this study, the HDL levels in Group 1 were significantly higher than those in the other groups, but the difference among the other groups was not statistically significant.

Ma et al. [27] found that concomitantly increased fibrinogen and decreased HDL levels were associated with increased recurrent cerebral thrombosis, although increased fibrinogen and decreased HDL levels were not significant when compared separately. Kowalski et al. [28] reported that the concomitance of increased D-dimer, a fibrinogen degradation product, and decreased HDL level was a risk factor for acute pulmonary emboli formation. The fibrinogen and HDL levels were previously evaluated separately in several cardiological and oncological studies, and these levels were associated with inflammatory and atherothrombotic processes. Similarly, these levels were also evaluated in patients with ISSHL, and both the levels were found to have an effect on the etiopathogenesis and were evaluated as prognostic. In this study, we also concluded that both fibrinogen and HDL levels may have a prognostic value in ISSHL. However, our study was the first to evaluate the concomitance of both parameters in ISSHL using the fibrinogen/HDL ratio. In this study, the fibrinogen/HDL ratio was significantly lower in Group 1 than in Groups 3 and 4. In addition, there was a significant correlation between the fibrinogen/HDL ratio and hearing recovery according to the Siegel's criteria. Therefore, we suggest that fibrinogen/HDL ratio may be used as a prognostic indicator in patients with ISSHL.

Previous studies, particularly cardiovascular and cerebrovascular investigations, have demonstrated that the monocyte/HDL ratio is a prominent indicator for atherosclerosis [29, 30]. In a single study on ISSHL, Koçak et al. [31] evaluated the monocyte/HDL ratios in 45 patients with ISSHL and compared these levels with those of 47 healthy volunteers. They did not identify any significant difference between the study and control groups and therefore concluded that the monocyte/HDL ratio did not have any predictive value. Similarly, we did not find any significant difference among the groups in terms of their monocyte/HDL ratios.

Our study had some limitations. First, this was a retrospective study, and it also had a small sample size. In addition, the initial hearing loss degrees of the groups were not homogeneous. Second, although there was no statistically significant difference in terms of age and gender between the groups, the mean age of Group 1 was lesser than the average ages of Groups 2–4. Age is especially important, because a vascular etiology is likely to be more common in patients with ISSHL who are older.

Third, the treatment protocols among the groups were heterogeneous owing to the administration of salvage therapy to non-responsive patients. Analyses of these parameters in further prospective studies that ensure intergroup homogenization may offer a better evaluation of the reliability and efficiency of these results.

CONCLUSION

To the best of our knowledge, this is the first study to investigate the fibrinogen/HDL ratio in patients with ISSHL. Although the fibrinogen/HDL ratio might be a useful prognostic indicator for hearing recovery in patients with ISSHL, further studies with a larger patient population are required to confirm its clinical practicability and reliability.

Ethics Committee Approval: This study was approved by the Health Science University, Kayseri Training and Research Hospital and conforms to the principles of the Declaration of Helsinki. (52332816/7/23.8.2017)

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REFERENCES

1. Byl FM. Seventy-six cases of presumed sudden hearing loss occurring in 1973: prognosis and incidence. Laryngoscope 1977; 87: 817-25. [Crossref]
2. Dallan I, Bruschini L, Nacci A, Bruschini P, Traino C, Rognini F, et al. Transtympanic steroids as a salvage therapy in sudden hearing loss: preliminary results. ORL J Otorhinolaryngol Relat Spec 2006; 68: 247-52. [Crossref]
3. Penido NO, Cruz OL, Zanoni A, Inoue DP. Classification and hearing evolution of patients with sudden sensorineural hearing loss. Braz J Med Biol Res 2009; 42: 712-6. [Crossref]
4. Aimoni C, Bianchini C, Borin M, Ciorba A, Fellin R, Martini A, et al. Diabetes, cardiovascular risk factors and idiopathic sudden sensorineural hearing loss: a case-control study. Audiol Neurotol 2010; 15: 111-5. [Crossref]
5. Marcucci R, Alessandrello Liotta A, Cellai AP, Rogolino A, Berloco P, Leprini E, et al. Cardiovascular and thrombophilic risk factors for idiopathic sudden sensorineural hearing loss. J Thromb Haemost 2005;3: 929-34. [Crossref]
6. Mattox DE, Simmons FB. Natural history of sudden sensorineural hearing loss. Ann Otol Rhinol Laryngol 1977; 86: 463-80. [Crossref]
7. Colclasure JB, Graham SS. Intracranial aneurysm occurring as sensorineural hearing loss. Otolaryngol Head Neck Surg 1981; 89: 283-7. [Crossref]
8. Jaffe BF. Sudden deafness: a local manifestation of systemic disorders: fat emboli, hypercoagulation and infections. Laryngoscope 1970; 80: 788-801. [Crossref]
9. Mattox DE., Lyles CA. Idiopathic sudden sensorineural hearing loss. Am J Otol 1989; 10: 242-7.
10. Capaccio P, Ottaviani F, Cuccarini V, Bottero A, Schindler A, Cesana BM et al. Genetic and acquired prothrombotic risk factors and sudden hearing loss. Laryngoscope 2007; 117: 547-51. [Crossref]
11. Lin HC, Wang CH, Chou YC, Shih CP, Chu YH, Lee JC, et al. The correlation between lipoprotein ratios and hearing outcome in idiopathic sudden

sensorineural hearing loss patients. Clin Otolaryngol 2015; 40: 355-62. [Crossref]
12. Kim CK, Schmalfuss CM, Schofield RS, Sheps DS. Pharmacological treatment of patients with peripheral arterial disease. Drugs 2003; 63: 637-47. [Crossref]
13. Satar B, Ozkaptan Y, Surucu HS, Oztürk H. Ultrastructural effects of hypercholesterolemia on the cochlea. Otol Neurotol 2001; 22: 786-9. [Crossref]
14. Meade TW, Mellows S, Brozovic M, Miller GJ, Chakrabarti RR, North WR, et al. Haemostatic function and ischemic heart disease: The Northwick park heart study. Lancet 1986; 6: 533-7. [Crossref]
15. Letcher RL, Chien S, Pickering TG, Sealey JE, Laragh JH. Direct relationship between blood pressure and blood viscosity in normal and hypertensive subjects: role of fibrinojen and concentration. Am J Med 1981; 70: 1195-202. [Crossref]
16. Schmid-Schoenbein H. Microrheology of erythrocytes, blood viscosity, and the distribution of blood flow in the microcirculation. Int Rev Physiol 1976; 9: 1-62.
17. Somer T, Meiselman HJ. Disorders of blood viscosity. Ann Med 1993; 25: 31-9. [Crossref]
18. Masuda M, Kanzaki S, Minami S, Kikuchi J, Kanzaki J, Sato H, et al. Correlations of inflammatory biomarkers with the onset and prognosis of idiopathic sudden sensorineural hearing loss. Otol Neurotol 2012; 33: 1142-50. [Crossref]
19. Suckfüll M, Wimmer C, Reichel O, Mees K, Schorn K. Hyperfibrinogenemia as a risk factor for sudden hearing loss. Otol Neurotol 2002; 23: 309-11. [Crossref]
20. Park YA, Kong TH, Seo YJ. A sustained increase of plasma fibrinogen in sudden sensorineural hearing loss predicts worse outcome independently. Am J Otolaryngol 2017; 38: 484-7. [Crossref]
21. Kanzaki S. Sakagami M, Hosoi H, Murakami S, Ogawa K. High fibrinogen in peripheral blood correlates with poorer hearing recovery in idiopathic sudden sensorineural hearing loss. PLoS One 2014; 9: e104680. [Crossref]
22. Stocker R, Keaney JF. Role of oxidative modifications in atherosclerosis. Physiol Rev 2004; 84: 1381-478. [Crossref]
23. Navab M, Ananthramaiah GM, Reddy ST, Van Lenten BJ, Ansell BJ, Fonarow GC, et al. The oxidation hypothesis of atherogenesis: the role of oxidized phospholipids and HDL. J Lipid Res 2004; 45: 993-1007. [Crossref]
24. Yilmaz MB, Guray U, Guray Y, Cihan G, Caldir V, Cay S, et al. Lipid profile of patients with aortic stenosis might be predictive of rate of progression. Am Heart J 2004; 147: 915-8. [Crossref]
25. Chen C, Wang M, Fan Z, Zhang D, Lyu Y, Wang H, et al. Correlations between the pathogenesis and prognosis of sudden sensorineural hearing loss and blood lipid. Zhonghua Er Bi Yan Hou Tou Jing Wai Ke Za Zhi 2015; 50: 793-8.
26. Chang IJ, Kang CJ, Yueh CY, Fang KH, Yeh RM, Tsai YT. The relationship between serum lipids and sudden sensorineural hearing loss: a systematic review and meta-analysis. PLoS One 2015; 13: e0121025. [Crossref]
27. Ma X, Ji XM., Fu P, Ding YC, Xue Q, Huang Y. Coexistence of High Fibrinogen and Low High-density Lipoprotein Cholesterol Levels Predicts Recurrent Cerebral Venous Thrombosis. Chin Med J (Engl) 2015; 128: 1732-7. [Crossref]
28. Kowalski J, Jędrzejczyk JT, Barylski M, Ciećwierz J, Sienkiewicz M, Kowalczyk E. Value of D-dimer and HDL cholesterol concentrations in predicting the occurrence of acute pulmonary embolism. Pol Merkur Lekarski 2016; 40: 283-7.
29. Rogacev KS, Zawada AM, Emrich I, Seiler S, Böhm M, Fliser D, et al. Lower Apo A-I and lower HDL-C levels are associated with higher intermediate CD14?? CD16? monocyte counts that predict cardiovascular events in chronic kidney disease. Arterioscler Thromb Vasc Biol 2014; 34: 2120-7. [Crossref]
30. Nadeem R, Singh M, Nida M, Waheed I, Khan A, Ahmed S, et al. Effect of obstructive sleep apnea hypopnea syndrome on lipid profile: a meta-regression analysis. J Clin Sleep Med 2014; 10: 475-89. [Crossref]
31. Koçak HE, Acipayam H, Elbistanlı MS, Yiğider AP, Alakhras W, Kıral MN, et al. Is the Monocyte/HDL Ratio a Prognostic Marker of Idiopathic Sudden Hearing Loss? Otolaryngol Pol 2016; 70: 26-30. [Crossref]