

The Significance of Routine Laboratory Parameters in Patients with Sudden Sensorineural Hearing Loss

Hasan Yasan · Mustafa Tüz · Murat Yarıktas · Giray Aynali · Önder Tomruk · Ömer Akkuş

Received: 26 November 2010 / Accepted: 3 January 2012 / Published online: 15 January 2012
© Association of Otolaryngologists of India 2012

Abstract There are several factors (viral infections, metabolic and ototoxic disorders etc.) accused for the development of sudden sensorineural hearing loss. Some prognostic factors (late onset of treatment etc.) had been evaluated in the literature. There is no sufficient data on the effect of routine laboratory parameters on the development and/or prognosis of sudden sensorineural hearing loss. The aim of this study is to investigate the effects of routine blood chemistry and hematological parameters on the development and prognosis of disease in patients with idiopathic sudden sensorineural hearing loss. One hundred and forty-seven patients with the diagnosis of idiopathic sudden sensorineural hearing loss followed up during the periods of 2000–2010 years were included in this study.

One hundred and three septoplasty patients with no otologic complaints were enrolled as control group. Following the clinical and demographic evaluations, patients with idiopathic sudden sensorineural hearing loss and control groups, and patients treated successfully and patients with poor outcome were compared with each other. Data were analyzed by T test. All hematological and biochemical parameters were compared. Hemoglobin, hematocrit, white blood cell count, total and direct bilirubin, fasting blood glucose level and aspartate aminotransferase were significantly different between idiopathic sudden sensorineural hearing loss and control groups. There was no significantly different parameter between patients treated successfully and patients with poor outcome. Hemoglobin, hematocrit, white blood cell count, total and direct bilirubin, fasting blood glucose level and AST all can be risk factors for SHL, or they can be the result of undetermined pathology, because these parameters have no effect on the prognosis. Other routine parameters seem to have no effect on the development and/or prognosis of idiopathic sudden sensorineural hearing loss.

H. Yasan (✉) · M. Tüz · M. Yarıktas · G. Aynali
Ear, Nose, Throat, Head and Neck Surgery Department, Suleman Demirel University, Moderevler Mah. 142.cad., Çevreyolu, Azim sitesi, A Blok D:11, 32200 Isparta, Turkey
e-mail: dryasan@hotmail.com

M. Tüz
e-mail: mutuz@hotmail.com

M. Yarıktas
e-mail: yariktas@med.sdu.edu.tr

G. Aynali
e-mail: giraynali@yahoo.com

Ö. Tomruk
Emergency Department, Suleman Demirel University, Isparta, Turkey
e-mail: ondertomruk@hotmail.com

Ö. Akkuş
Gaziantep State Hospital Ear, Nose, Throat, Head and Neck Surgery Department, Suleman Demirel University, Gaziantep, Turkey
e-mail: dromerakkus@yahoo.com

Keywords Sudden hearing loss · Risk factor · Prognosis · Audiology

Introduction

The definition of sudden sensorineural hearing loss (SSHL) is acute deterioration of hearing during the last 3 days, of over 30 dB on the three consecutive bone conduction frequencies. There are several etiologic theories proposed for the development of SSHL such as infection, vascular, autoimmune and the rupture theories. The prognosis is also affected by several factors [1, 2]. The aim of this study is to

investigate the possible effects of routine blood chemistry and hematological parameters on the development and prognosis of disease in patients with SSHL.

Patients and Methods

The patient groups for this study were selected from 52 prospective and 95 retrospective patients with SSHL (study group). Twenty-six prospective and 77 retrospective subjects with isolated nasal septal deviation were included as control group. This study was carried out between the years of 2000 and 2010. The cases of control group were selected among age and sex matched subjects with only nasal septal deviation (otherwise healthy). Pure tone audiograms were taken just before and each day of medical treatment for a period of 14 days. Pure tone audiographic measurements were made by a device of Amplaid 350. Routine blood chemistry and whole blood count values were taken just before the medical treatment. Pure tone averages (averages of 0.5, 1 and 2 kHz) of hearing level before the treatments were compared with those of 14th day of the treatment. The elapsed days between the awareness of hearing loss and starting of treatment was defined as duration of treatment initiation. All the SSHL cases were administered standard

treatment protocol including acyclovir, corticosteroids, rheomacrodex, ginkgo glicozide, moclobamide, vitamin complex, and proton pump inhibitors. Patients with hearing gain less than 10 dB, or those who has not improvement were defined as treatment failure group. Treatment success is defined as 10 dB or more hearing gain. This evaluation was made as comparing the pure tone averages of first and 14th days of treatment. Hearing gain between 10 and 20 dB PTA was defined as limited improvement. Hearing gain more than 20 dB PTA was defined as significant improvement. Exclusion criteria were diabetes mellitus, hepatitis, hematologic diseases, ototoxic drug usage, previous otologic diseases or intervention. The comparisons were made between control (septoplasty patients with nasal septal deviation without ear problems) and study groups (SSHL). SSHL group patients were also compared according to the limited and significant improvements. Data were statistically analyzed by using T test. *P* values lesser than 0.05 were defined as statistically significant.

Results

Demographic criteria of 147 patients with SSHL were as follows; 79 (53.7%) male, 68 (46.3%) female, age range

Table 1 The comparisons of patients with SSHL and control subjects

Parameters	SSHL (N: 147)	Control group (N: 103)	<i>P</i>
Age range (year)	15–55	17–51	–
Mean age (year)	30.81 ± 11.08	30.11 ± 10.27	–
Gender (M/F)	79/68	55/48	–
Duration of treatment onset (day)	1–20 (7.63 ± 5.21)	–	–
MCV (femtoliter)	81.72 ± 8.02	83.1 ± 5.07	.582
Hemoglobin (g/dl)	14.21 ± 1.25	15.78 ± 1.32	.001*
Haematocrit (%)	40.61 ± 4.11	45.82 ± 3.25	.011*
RDW (%)	13.87 ± 1.46	13.01 ± 0.89	.097
Platelet (10 ³ /μl)	256.39 ± 58.19	251.27 ± 56.07	.580
WBC (10 ³ /μl)	8.51 ± 2.32	7.98 ± 2.12	.032*
Total Bilirubin (mg/dl)	0.60 ± 0.35	0.86 ± 0.39	.023*
Direct Bilirubin (mg/dl)	0.20 ± 0.15	0.34 ± 0.17	.001*
Indirect Bilirubin (mg/dl)	0.40 ± 0.21	0.49 ± 0.34	.291
Fasting blood glucose (mg/dl)	112.65 ± 53.20	90.08 ± 7.98	.030*
BUN (mg/dl)	19.01 ± 15.26	19.65 ± 12.81	.921
Creatinin (mg/dl)	1.03 ± 1.24	0.95 ± 0.31	.524
Total lipid (mg/dl)	173.74 ± 37.15	150.61 ± 40.94	.212
Triglyceride (mg/dl)	173.96 ± 120.17	124.19 ± 86.55	.308
HDL (mg/dl)	43.62 ± 12.85	41.22 ± 9.08	.815
LDL (mg/dl)	96.39 ± 35.80	84.25 ± 33.21	.427
VLDL (mg/dl)	36.61 ± 26.87	26.80 ± 18.74	.329
AST (u/l)	26.45 ± 8.60	22.84 ± 6.46	.041*
ALT (u/l)	24.85 ± 12.76	24.12 ± 14.91	.756

MCV mean corpuscular volume, RDW red cell distribution width, WBC white blood cell count, BUN blood urea nitrogen, HDL: high density lipoprotein, LDL: low density lipoprotein, VLDL: very low density lipoprotein, ALT alanine aminotransferase, AST aspartate aminotransferase

* Statistically significant

and mean age were 15–55 and 30.81 ± 11.08 years, respectively. Control group subjects were composed of 103 septoplasty patients of 55 (53.4%) male and 48 (46.6%) female. The age range and mean age were 17–51 and 30.11 ± 10.27 years, respectively (Table 1). The mean PTA of all SSHL patients just before treatment was 58.45 ± 20.48 (35–80) dB. The mean PTA of all SSHL patients at the 14th day of treatment was 32.71 ± 15.38 (5–62) dB. Seventy-seven out of total 147 SSHL cases (52.4%) have had the right ear involvement, and 70 cases (47.6%) have had left ear involvement. Nine patients with bilateral SSHL were not included into the study according to the exclusion criteria (Six have had ototoxic drug usage, three has had uncontrolled diabetes mellitus for 5–10 years duration). One hundred and two (69.4%) out of 147 patients with SSHL had improvement by our standard treatment protocol. Forty-five (30.6%) patients did not have enough improvement in their hearing levels. Seventy-seven (65.7%) out of 102 improved patients were significantly improved SSHL cases. Thirty-five (34.3%) patients had only limited improvement. There were statistically significant difference between the cases of SSHL and those of control subjects with respect to the hemoglobin ($P = .001$), hematocrite ($P = .011$), white blood cell

($P = .032$), total bilirubin ($P = .021$), direct bilirubin ($P = .001$), fasting blood glucose ($P = .030$) and aspartate aminotransferase ($P = .041$) (Table 1). The other laboratory parameters had not a significant difference between study and control groups affecting prognosis of SSHL. None of the routine laboratory parameters had a statistically significant difference between the patients treated successfully and patients with poor outcome (Table 2). The only significant parameter between improved and unimproved patients was the duration from disease onset to the starting of treatment ($P = .002$).

Discussion

SSHL is characterized by sudden-onset hearing loss that can become evident within hours or days [3]. Numerous conditions produce SSHL, including infectious disease, trauma, circulatory disturbance, ototoxicity, and neoplasms, among others. However, the precise mechanism of disease remains to be elucidated. The disturbance of cochlear perfusion seems to have a prominent effect on the development of SSHL. Since the etiology was not determined in most of the cases, the pathology is mostly known

Table 2 Comparisons of SSHL patients with and without improvement following standard treatment protocol

Parameter	Improved patients (N: 102)	Patients without improvement (N: 45)	P
Age range (year)	15–54	15–55	–
Mean age (year)	28.77 ± 9.58	29.26 ± 9.62	.633
Gender (M/F)	55/47	24/21	–
Treatment onset (day)	1–8 (3.96 ± 3.62)	1–20 (10.24 ± 8.12)	.002*
MCV (femtoliter)	83.15 ± 8.33	86.12 ± 7.11	.232
Hemoglobin (g/dl)	13.94 ± 1.60	13.48 ± 1.25	.455
Hematocrit (%)	41.25 ± 4.23	39.84 ± 4.17	.288
RDW (%)	13.60 ± 1.68	13.19 ± 1.10	.452
WBC ($10^3/\mu\text{l}$)	8.21 ± 2.07	8.92 ± 2.39	.507
Platelet ($10^3/\mu\text{l}$)	252.39 ± 55.17	269.13 ± 64.92	.397
Total bilirubin (mg/dl)	0.59 ± 0.28	0.45 ± 0.13	.143
Direct bilirubin (mg/dl)	0.20 ± 0.13	0.19 ± 0.10	.841
Indirect bilirubin (mg/dl)	0.40 ± 0.26	0.33 ± 0.13	.097
Fasting blood glucose (mg/dl)	110.02 ± 50.25	114.24 ± 58.40	.856
BUN (mg/dl)	18.98 ± 8.12	16.75 ± 8.28	.485
Creatinin (mg/dl)	0.91 ± 0.37	1.25 ± 0.65	.424
Total lipid (mg/dl)	170.12 ± 43.23	182.67 ± 33.14	.482
Triglyceride (mg/dl)	175.44 ± 80.59	188.13 ± 60.44	.652
HDL (mg/dl)	41.52 ± 13.25	44.74 ± 12.81	.787
LDL (mg/dl)	91.43 ± 24.81	110.62 ± 37.25	.275
VLDL (mg/dl)	38.71 ± 14.70	35.61 ± 14.23	.814
AST (u/l)	26.13 ± 8.12	24.81 ± 10.41	.421
ALT (u/l)	24.29 ± 10.50	26.27 ± 15.08	.757

MCV mean corpuscular volume, RDW red cell distribution width, WBC white blood cell count, BUN blood urea nitrogen, HDL high density lipoprotein, LDL low density lipoprotein, VLDL very low density lipoprotein, ALT alanine aminotransferase, AST aspartate aminotransferase

* Statistically significant

as idiopathic SSHL. Gender, regional differences, seasons, and smoking have no effect on the development of disease [1, 4, 5].

However, we have found 53.7% preponderance of male patient. Hemoconcentration is another accused parameter. This is not the case in our study, and this controversy indicate that, hemoconcentration is a result of disorder underlying SSHL instead of etiologic factor. Similarly white blood cell count is high as a result of underlying disorder, not an etiologic factor of SSHL. Because it is statistically significantly higher in SSHL group than control subject, and there is no difference between SSHL patients with or without improvement. The pathology leading to the high level of white blood cell seems to be the cause of SSHL as well. This may be result of some upper respiratory tract infections that is mostly present in the history of patients. This may be explained by viral infection as one of the causes of disease. Vascular disorder is well-known etiopathogenetic factor in the development of SSHL [1]. Although high levels of serum cholesterol, triglyceride and uric acid are known to be vascular risk factors, our study had not a correlation between these risk factors and SSHL development and/or prognosis. The time elapsing from the onset of hearing loss to the initiation of treatment is known to be an important prognostic factor: the sooner the treatment is initiated, the better the outcome. In our study, patients with delayed treatment had poorer outcome. The other factors for poor prognosis are as follows: higher levels of hearing loss at admission, labyrinthine responsiveness disorders, older age and co-morbid systemic disorders [1, 6].

Conclusions

Hemoglobin, hematocrit, white blood cell count, total and direct bilirubin, fasting blood glucose level and AST all can be risk factors for SHL, or they can be the result of undetermined pathologies, because these parameters have no effect on the prognosis. Unknown underlying causes of idiopathic SSHL may be the result of different values of some laboratory parameters between study and control groups. The other routine laboratory parameters seem to have no effect on the development and prognosis of idiopathic sudden sensorineural hearing loss.

References

1. Narozny W, Kuczkowski J, Kot J, Stankiewicz C, Sicko Z, Mikaszewski B (2006) Prognostic factors in sudden sensorineural hearing loss: our experience and a review of the literature. *Ann Otol Rhinol Laryngol* 115(7):553–558
2. Suckfüll M (2009) Perspectives on the pathophysiology and treatment of sudden idiopathic sensorineural hearing loss. *Dtsch Arztebl Int* 106(41):669–675
3. Görür K, Tuncer U, Eskandari G, Ozcan C, Unal M, Ozsahinoglu C (2005) The role of factor V Leiden and prothrombin G20210A mutations in sudden sensorineural hearing loss. *Otol Neurotol* 26(4):599–601
4. Byl FM (1984) Sudden hearing loss: eighth year's experience and suggested prognostic table. *Laryngoscope* 94:647–661
5. Slattery WH, Fisher LM, Iqbal Z, Friedman RA, Liu N (2005) Intratympanic steroid injection for the treatment of idiopathic sudden hearing loss. *Otolaryngol Head Neck Surg* 133:251–259
6. Suckfull M, Mess K (1998) Hemoconcentration as a possible pathogenic factor of sudden hearing loss. *Eur Arch Otolaryngol* 255:281–284